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EPA-450/5-85-001

Inorganic Arsenic NESHAPS: Response to Public Comments on Health, Risk Assessment, and Risk Management

Strategies and Air Standards Division

U.S. ENVIRONMENTAL PROTECTION AGENCY
Office of Air and Radiation
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

April 1985

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1.0 Introduction

The Administrator has decided to regulate certain low-arsenic copper smelters and glass manufacturing plants and not to regulate primary and secondary lead smelters, primary zinc smelters, zinc oxide plants, cotton gins and arsenic chemical plants. The EPA is publishing this document in support of those decisions by providing detailed consideration and response to comments received during the proposal/public comment process. In particular, this document provides detailed responses to comments which are related to the following general topics:

1. The listing of inorganic arsenic as a hazardous air pollutant under section 112;
2. The health effects associated with arsenic exposure;
3. The risk management approach used as a basis for the proposal and
4. The risk assessment methodology.

Also, the Agency has produced companion documents that contain other background information and detailed responses to comments for the specific source categories. The reader is referred to the following list for complementary information:

1.	Low-Arsenic Copper Smelters	EPA-450/3-83-010b
2.	Glass Manufacturing Plants	EPA-450/3-83-011b
3.	Primary and Secondary Lead Smelters, Cotton Gins, Primary Zinc Smelters, Zinc Oxide Plants, Arsenic Chemical Plant	EPA-450/5-85-002

In addition to the above documents, the reader is also referred to the Agency's health effects document, "Health Assessment for Inorganic Arsenic," EPA-450/3-83-021F, from which many of the Agency's responses for comments in Chapters 2 and 3 were drawn.

LIST OF COMMENTERS ON PROPOSED
COPPER SMELTER ARSENIC STANDARDS

Docket Item Number	Commenter and Affiliation[a]
IV-D-1; IV-D-95; IV-D-677, IV-F-10	Susan and Robert Adams
IV-D-2; IV-D-37; IV-D-90	Ms. Teresa Doyle
IV-D-3	Mr. Hugh Kimball
IV-D-4	Ms. Susan Anderson
IV-D-5; IV-D-93; IV-D-530, IV-D-673	Ms. Sheri Reder
IV-D-6	Mr. Eugene Fujimoto
IV-D-7	Ms. Marilyn Muller
IV-D-8	Mr. Craig D. Hilborn
IV-D-9; IV-F-9	Mr. John T. Konecki
IV-D-10	Chris Connery and Mary Scott
IV-D-11; IV-D-127, IV-D-677	Dr. Robert E. Sullivan
IV-D-12	Mr. Thomas M. Skarshaug *et al.*
IV-D-13	Ms. Virginia Nichols
IV-D-14	Mr. Philip H. Abelson
IV-D-15	Ms. Nathallie Fitzgerald
IV-D-16	Mr. James J. Mason
IV-D-17	Mr. T.C. White ASARCO, Inc.
IV-D-18; IV-D-19; IV-D-59; IV-D-64; IV-D-222; IV-D-445; IV-D-602; IV-D-603; IV-D-620, IV-D-621; IV-D-649; IV-D-691; IV-D-702; IV-D-703; IV-D-714; IV-D-716; IV-D-787; IV-D-792; IV-D-793; IV-F-2[b]	Mr. L. W. Lindquist ASARCO, Inc.

Docket Item Number	Commenter and Affiliation[a]
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IV-D-21	Mr. Terry Sullivan
IV-D-22	Mr. Hans Zeisel The University of Chicago Law School
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IV-D-24; IV-D-136	Mr. Harvey S. Poll Puget Sound Air Pollution Control Agency
IV-D-25; OAQPS-79-8/IV-D-3	George and Adriana Hess
IV-D-26	Mr. Steve Burcombe
IV-D-27	Mr. Arnold Cogan Cogan & Associates
IV-D-28	Mr. Frank M. Parker, III Southwest Occupational Health Services, Inc.
IV-D-29	Mr. John J. Sheehan United Steelworkers of America
IV-D-30; IV-D-283; IV-D-383	Mr. Edward S. Watts
IV-D-31	Mrs. Delores Keating
IV-D-32; IV-D-677	Ms. Sharon Rue
IV-D-33	Ms. Joy Nelsen
IV-D-34	"A Concerned Citizen"
IV-D-35; IV-D-593; IV-F-9	Mr. Ralph K. Garrison
IV-D-36	Ms. Barbara Jensen
IV-D-38; IV-F-10	B. J. Kanagy
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Docket Item Number	Commenter and Affiliation[a]
IV-D-40	Ms. Patricia Ives
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IV-D-42	Janis and Gregory McElroy
IV-D-43; IV-D-114; IV-D-438	Fred and Sue Campbell
IV-D-44	David and Ann Beckwith Boberg
IV-D-45	Ms. Susan Konecki
IV-D-46	Vernon and Christine Trevellyan
IV-D-47	Erica and Michael Meade
IV-D-48	Mr. Richard L. Swenson
IV-D-49; IV-D-375	Ms. Elaine Taylor
IV-D-50	Mr. Paul J. Braune
IV-D-51	Ms. Hymen Diamond
IV-D-52	Ms. Nancy Sosnove
IV-D-53	Ms. Terry Patton
IV-D-54	Ms. Patricia Bauer
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IV-D-56	Mr. David Burcombe
IV-D-57	Mr. Michael Higgins
IV-D-58; IV-D-253; IV-D-621; IV-D-683	Mr. Glenn L. Boggs
IV-D-60	Mr. Toby Holmes
IV-D-61	Ms. Laurie E. Martin
IV-D-62	Mr. and Mrs. Donald R. Jopp
IV-D-63; IV-D-435; IV-D-721; IV-F-11	Ms. Irene Blackford

Docket Item Number	Commenter and Affiliation[a]
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IV-D-67	Mr. and Mrs. Leo A. Yuckert
IV-D-68	Ms. Mildred Schiffor
IV-D-69	Mr. R.W. Neuser
IV-D-70	Mr. Douglas P. Coleman Coland, Inc.
IV-D-71	Mr. and Mrs. Al Booze
IV-D-72	Ms. Olivia Watt
IV-D-73; IV-D-105; IV-D-302; IV-D-575	Mr. Robert Krimmel
IV-D-74	Nancy Morgan and Michael Barnes
IV-D-75	Mr. Noel Daley
IV-D-76; IV-D-117; IV-D-443; IV-D-757; IV-F-11	Mr. Frank W. Jackson Vashon-Maury Island Community Council
IV-D-77	Ms. Tammi L. Contris
IV-D-78	Ms. Frances Wotton
IV-D-79	Mr. and Mrs. Fuller
IV-D-80; IV-D-677	Ms. Caroline Hunter Davis
IV-D-81; IV-D-121	Mr. Robert Lipp
IV-D-82	Mary and Stephen Daniel
IV-D-83	Mr. Stanley C. Smith
IV-D-84; IV-D-677	Norene, Vince, and Patricia Gallo
IV-D-85	Mr. Timothy Walsh Greenpeace Northwest

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IV-D-94	Ms. Cheryl Owings
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IV-D-97	Mr. G. R. Finden
IV-D-98	Ms. Laura H. Vaughn
IV-D-99	Ms. Gertrude Quinn
IV-D-100	Ms. Mona Brady
IV-D-101	Ms. Rose Owens
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IV-D-116; IV-D-433	Rev. Merry Kogut

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IV-D-119; IV-D-446; IV-D-648; IV-D-710, 710a, 710b; IV-D-745; IV-D-749; IV-D-759; IV-F-2[b]; OAQPS-79-8/IV-D-33, 33a, 33b	Mr. David D. Doniger Natural Resources Defense Council, Inc.
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IV-D-129	Ms. Dana Griffin
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IV-D-132	Mrs. G.R. Byrski
IV-D-133; IV-D-485; IV-D-621	Mr. Russell I. Lewis
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IV-D-135	Mrs. John E. Erickson
IV-D-137	Mr. Gene Alberts Pacific Sun Ltd.
IV-D-138	George and Norma Newcomb
IV-D-139	Ms. Sue Hanson

Docket Item Number	Commenter and Affiliation[a]
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IV-D-141; OAQPS-79-8/IV-D-7	Mr. David C. Roberts
IV-D-142	Mr. Del Langbauer
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IV-D-144; IV-D-719	Mr. Noel McLane
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IV-D-147	Ms. Johanna H. Mason
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IV-D-155	Mr. Peter K. Schoening Chemical Proof Corporation
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IV-D-175	Mr. and Mrs. George Kahl
IV-D-176	Mr. Harold T. Rock
IV-D-177	Mr. David Walkup
IV-D-178	Mr. Raymond Garner
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IV-D-181	Mr. Richard Balles

Docket Item Number	Commenter and Affiliation[a]
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Docket Item Number	Commenter and Affiliation[a]
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IV-D-212	Mr. Harold E. Jorgenson
IV-D-213	Mr. John Bentson Vale
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IV-D-226	Mr. Daniel S. Dean
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IV-D-229	Mr. Harry D. Maxwell

Docket Item Number	Commenter and Affiliation[a]
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IV-D-234	Mr. Henry Cox
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IV-D-237	Mr. Joseph M. Stadtler
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IV-D-241; OAQPS-79-8/IV-D-12	Ms. Alice Spears
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IV-D-243	Mr. Charles E. Allen
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IV-D-246	Mr. Raymond R. Webster
IV-D-247	Mr. F. Willard White
IV-D-248	Thomas and Rosemary Arnold
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IV-D-251	Ms. Eleanor Schaffer

Docket Item Number	Commenter and Affiliation[a]
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IV-D-272	Ms. Mae Brown
IV-D-273	Mr. Lowell Jorgenson
IV-D-274	Mr. Paul DiMaio
IV-D-275	Frank and Delores Keating

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IV-D-277	Mr. Jack Stutler
IV-D-278	Erwin and Patricia Myers
IV-D-279	K.S. Hammond
IV-D-280	Mrs. F.M. Larson
IV-D-281	Mr. Frank Dlanc
IV-D-282	Florence Irvin and John Jurovich
IV-D-284	Mr. William Dearborn
IV-D-285	Mr. Leon Cunningham
IV-D-286	Mr. Richard Lowery Electric Motor Service Co.
IV-D-287	Mr. Fred Young E. A. Wilcox Co.
IV-D-288	Mr. Kenneth Sprong Harbison-Walker Refractories
IV-D-289	Mr. C.M. Bevis Bevis & Assoc., Inc.
IV-D-290	Mr. Laurence Evoy Pierce County Medical
IV-D-291	Mr. George Leonhard Mount Rainier Council Boy Scouts of America
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IV-D-293	Mr. Joseph Prinse
IV-D-294	Mr. Lee Fedderly
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IV-D-298	Mr. Robert Laughlin
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IV-D-306	Mrs. Robert Schanzenbach
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IV-D-310	Mr. Don Zemek
IV-D-311	Mr. Justice Ashwell
IV-D-312	Harold and Anne Ransom
IV-D-313	Dr. and Mrs. Robert Knapp
IV-D-314	Dr. Richard G. Schoen
IV-D-315	Herbert and Charlotte Weston
IV-D-317	Mr. John Susanj
IV-D-318; IV-D-621	Mr. Coy Brown
IV-D-319; IV-D-621	Mr. Bill Weston
IV-D-320	Mr. and Mrs. John Reed

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IV-D-321	Ms. Ruth Brown
IV-D-322	Mr. George Austin Austin Mac, Inc.
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IV-D-326	Mr. Floyd Martin
IV-D-327	Mrs. Elizabeth Pedersen
IV-D-328	Ms. Laure Nichols
IV-D-330	Mr. John Dyer
IV-D-331	Mr. Kenneth Taylor
IV-D-332	Mr. and Mrs. Fredrick Young
IV-D-333	Mrs. Robert Guddes
IV-D-334	Charles and Thelma Modie
IV-D-335	Ms. Mary L. Mullin
IV-D-336	Mr. John Daly
IV-D-338	Mr. Arlander Bell
IV-D-339	Mr. Walter Kunschak
IV-D-340	Mr. Donald Angle
IV-D-341	Pete and June Zaferin
IV-D-342	Mr. Allan Weydahl Nalco Chemical Co.
IV-D-343	Ms. Greta Dotson
IV-D-344 IV-D-345	Mr. Charles Shaw Mr. Frank Puz
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Docket Item Number	Commenter and Affiliation[a]
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IV-D-351	Mr. Robert Ellener
IV-D-353	Ms. Ella Phillips
IV-D-354	Mrs. Marjorie McMenamin
IV-D-355	Patrick and Nora Duggan
IV-D-356	Ms. Mary McCormack
IV-D-357	Mr. and Mrs. Ervin Lee
IV-D-358	Mr. J.M. Will Tam Engineering Corp.
IV-D-359	Mr. S. Evan Davies S. Evan Davies & Associates
IV-D-360	Ms. Betty J. Roberts
IV-D-361	Mr. and Mrs. Garland Cox
IV-D-362	Ms. Janet Jacobson
IV-D-363	Ms. Frances Coats
IV-D-364	Ms. Ellen Herigstad
IV-D-365	Mr. Fred Wise
IV-D-366	D.M. Manning
IV-D-367	Mr. and Mrs. W. Rieck
IV-D-368	Ms. Olga Williams
IV-D-369	Mr. Bill Merrill
IV-D-370	Mr. and Mrs. Ray Lunger

Docket Item Number	Commenter and Affiliation[a]
IV-D-371	Mr. William Hanar
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IV-D-374	Mr. Louis Burkey
IV-D-376	Mr. Gerald Copp Public Utility District #1 of Chelan County
IV-D-377	Ms. Eva Malovich
IV-D-378	S. Behrman
IV-D-379	Mr. Raymond Wall
IV-D-380	Mr. Jim Wilhelmi, Jr. The Stationers, Inc.
IV-D-381	Mr. George Jowell
IV-D-382; IV-D-621	Mr. Floyd Williams
IV-D-384	Mr. Michael Fabb
IV-D-385	Mr. Ken Reaj
IV-D-386	Mr. William Cammarano, Jr. Cammarano Bros, Inc.
IV-D-387	Mr. M.J. Burgess
IV-D-388	Mr. D.S. Skeie Industrial Mineral Products, Inc.
IV-D-389	Mr. P. McDougal
IV-D-390[c]	Mr. Victor Selvig
IV-D-391	G.D. Shipley
IV-D-392	Robert and Jan Van de Mark
IV-D-393	G.S. and Bernice Tallman
IV-D-394	Ms. Mildred Wall

Docket Item Number	Commenter and Affiliation[a]
IV-D-395	Mr. James Jacobsen
IV-D-396	M.C. Teats
IV-D-397	Mrs. June Gilson
IV-D-398	H.C. Bauman
IV-D-399	Mr. Ronald Roman
IV-D-400	Virginia and John Weaver
IV-D-401	Mr. Manfred Bell
IV-D-402	Mr. Edwin Briggs
IV-D-403	Mr. David Griffiths Cornell, Weinstein & Griffiths
IV-D-404	Ms. Kathryn Keller
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IV-D-407	Mrs. Edna Carlson
IV-D-408	S. Mladervich
IV-D-409	Mr. Glenn Roberts
IV-D-410	Mr. Frank D. Pupo Sam's Tire Service
IV-D-411	Ms. Carol Van Ginhoven
IV-D-412	Mr. Lloyd Skinner
IV-D-413; IV-D-677	Ms. Helen Gabel
IV-D-414; IV-D-677	Mr. Phillip Notermann
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IV-D-417; IV-F-9	Mr. James Garrison

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IV-D-423	Robert and Elnora Turver
IV-D-424	Mrs. Cheryl Curtis
IV-D-425	Mr. and Mrs. Harold Feley
IV-D-426	Walt and Kathy Hansen
IV-D-427	Rev. John Keliner Old St. Peter's Church
IV-D-428	Mr. Robert Burns
IV-D-430	Oleta Kerns
IV-D-431	Mr. Jon Fayst
IV-D-432	Mr. John Ellingson
IV-D-434	M. J. Bunnell
IV-D-436	Mr. G. Patrick Healy
IV-D-437	Ms. Joan Peterson
IV-D-439; IV-D-662; IV-D-676	Margie and Jeff Goulden
IV-D-440	Devitt and Debby Barnett
IV-D-441; IV-D-664; IV-D-676	Dr. John Van Ginhoven
IV-D-442	Mrs. Ray Hund
IV-D-443	Jeanne Snell and Frank Jackson Vashon-Maury Island Community Council

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IV-D-444	Mr. David A. Frew
IV-D-447; IV-D-786	Mr. Stephen Cant State of Washington Dept. of Labor & Industries
IV-D-448	Ms. Anita Fries Ohio State Clearinghouse
IV-D-449; IV-D-620; IV-D-621; IV-F-2[b]	Mr. Donald Robbins ASARCO, Inc.
IV-D-450	Mr. Ron Johnson
IV-D-451	Mr. Marion Brannon
IV-D-452	Ms. Cora Tolstrup
IV-D-454	Mr. Wayne Vanderflute
IV-D-455	Mr. F. Steven Doman
IV-D-456	Mr. Mark Peterson
IV-D-457	Mr. Robert Daniel
IV-D-458	Pat Frostad Motors & Controls Corp.
IV-D-459	Mr. Robert Lawson
IV-D-460	Mr. William Scott
IV-D-461	Mr. Bailey Nieder Tacoma Steel Supply
IV-D-462	Mr. Hugh Wild
IV-D-463	Ms. Elaine Thomas-Sherman
IV-D-464	Mr. Sidney Peyton
IV-D-465	Mr. Paul Foslien
IV-D-466	Mr. Sam Smyth
IV-D-467	Mr. Bill Cope

Docket Item Number	Commenter and Affiliation[a]
IV-D-468	Mr. Albert Behar Pierce County Medical
IV-D-469	Ms. Sheila McCanta
IV-D-470	Mr. Edgar E. King
IV-D-471	Ms. Mary Chouinard
IV-D-472	Rose and Floyd Murphy
IV-D-473	Mr. Russell Johnson
IV-D-474	Ms. Helen Carnahan
IV-D-475	Ms. Lucille Olsen
IV-D-476	Beatrice and George Peterson
IV-D-477	Mr. and Mrs. Carroll Thompson
IV-D-478	Ms. Norma Rozmen
IV-D-479	Ms. Marian Ganz
IV-D-480	Mr. John Gaul
IV-D-481	Ms. Molly LeMay
IV-D-482	Mr. Joseph Petranovich
IV-D-483	Mr. Rohn Burgess
IV-D-484	Mr. Jack McGuirk
IV-D-486	Mr. John Watson
IV-D-487	Mrs. Georgann Gallagher
IV-D-488	Ms. Alvinia Hagen
IV-D-489	Mr. C. Mark Smith Tacoma-Pierce County Economic Development Board
IV-D-490	Mrs. Virginia Loomis
IV-D-491	Delmer Pitts

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IV-D-492	Mr. Robert Heaton
IV-D-493	Dr. Michael J. Jarvis
IV-D-494	Mr. Kenneth J. Haagen
IV-D-495	Mr. E.P. Stiles Pierce County Medical Bureau, Inc.
IV-D-496	Beverly and Lawrence Sawtelle
IV-D-497	Mr. and Mrs. K.W. Mueller
IV-D-498	Ms. Frances Johnson InterAcc Co.
IV-D-499	P. Fischer
IV-D-500	Ms. Betty M. Susan
IV-D-501	Mr. and Mrs. Duane Puyear
IV-D-502	Ms. Marie Bean
IV-D-503	Mr. Thomas G. Stoebe
IV-D-504	Mr. Malcolm N. Thompson United Steelworkers of America Local 25
IV-D-505	Ms. Doris Adams Smelterman's Federal Credit Union
IV-D-506	Mr. John Fink
IV-D-507	Mr. Wayne Harkness
IV-D-508	Herb and Shirley Godfrey
IV-D-509	Mr. and Mrs. A.R. Glenn
IV-D-510	Mr. Donald S. Leinum
IV-D-511	Mr. Paul A. Schulz
IV-D-512	Gary and Nancy Ackman

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IV-D-513	Mr. Bailey Nieder Columbia Energy Co., Inc.
IV-D-514	Mr. E.T. McGrath
IV-D-515	Ms. Beverly M. Migliore Brown University Department of Geological Sciences
IV-D-516	Mr. Fred H. Smith Cochrane Northwest, Inc.
IV-D-517	Ms. Margaret J. Rowan
IV-D-519	Mr. Robert R. Treanton Fick Foundry Co.
IV-D-520; IV-F-9	Ms. Rayna Holtz
IV-D-521	James and Jerry Brandfas
IV-D-522	Mr. Jerry Michael Carlson
IV-D-523	Mr. Wayne S. Moen
IV-D-524; IV-D-554; IV-D-660	Mr. Richard L. Franklin
IV-D-525	Mrs. E. Gerie Fortier
IV-D-526	Ms. Cheryl Kirkwold
IV-D-527	Mr. James D. Gray
IV-D-528	Mr. and Mrs. Al Wegleitner
IV-D-529	Ms. Carol A. Krona
IV-D-531	John and Doris Achman
IV-D-532	Mr. Robert D. Hall
IV-D-533	Mr. and Mrs. W.H. Buzzell
IV-D-534	Ms. Ruth M. Johnson
IV-D-535	Mr. Howard O. Huggard
IV-D-536	Mr. Kenneth Mensching and Family

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IV-D-537	Mr. Robert D. Budd
IV-D-538	Mr. Gregory B. Curwen Gierke, Curwen, Metzler & Bobrick
IV-D-539	Mr. and Mrs. Richard Perkins
IV-D-540	Mr. R.M. Kennard *et al.*
IV-D-541	Mr. T. Russell Mager
IV-D-542	Ronald and JoAnn Roberts
IV-D-543	Mr. and Mrs. Austin E. Atwood
IV-D-544	Ms. Ruby M. Martin
IV-D-545; IV-D-621	Mr. Clyde H. Hupp Pierce County Central Labor Council AFL-CIO
IV-D-546	Mr. Mike D. Perkins Don H. Perkins, Inc.
IV-D-547	Mrs. Leonard Berglund
IV-D-548	Mr. Marion W. Samuelson
IV-D-549; OAQPS-79-8/IV-D-15	Mr. Kenny Scott
IV-D-550	Mr. W.E. Lightfoot Coffman Engineers, Inc.
IV-D-551	Mr. Robert Reinhart
IV-D-552	Mr. Robert F. Griffith
IV-D-553	Mr. W.A. Palmer
IV-D-555	Mr. and Mrs. Clifford Lakin
IV-D-556	Ms. Stephanie Colony
IV-D-557	Mr. Don H. Hinkley
IV-D-558	Mrs. Allan Lindstrom
IV-D-559	Mr. Bob L. Marshall

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IV-D-560	Mr. Kim de Rubertis
IV-D-561	Mr. A.B. Berg Industrial Mineral Products, Inc.
IV-D-562	Mr. David A. Pitts
IV-D-563	Mr. Paul E. Miller
IV-D-564	Mr. Duane A. Lindoff
IV-D-565	Mr. Richard Fundly
IV-D-566	Mr. Robert M. Helsell Wright Schuchart, Inc.
IV-D-567	Mr. R. Eccles
IV-D-568	Mr. Stephen F. Politeo Lilyblad Petroleum, Inc.
IV-D-569	Mr. Stan Sable
IV-D-570	Ms. Mary Susanj
IV-D-571	Ms. Katherine Spiratos Brown University
IV-D-572	Ms. Gretchen C. Gerish
IV-D-573; OAQPS-79-8/IV-D-17	Ms. Mary E. Cosaboom
IV-D-574	Ms. Ellen McComb Smith
IV-D-576; IV-D-699	Mr. Alf G. Anderson
IV-D-578	Adm. James S. Russell
IV-D-579; IV-F-9	Ms. Laurie Lehman
IV-D-580	Ms. Jennifer Paine
IV-D-581	Dr. Colleen R. Carey St. Luke's Medical Bldg.
IV-D-583	Toshio and Suzanne Akamatsu St. Joseph Hospital

Docket Item Number	Commenter and Affiliation[a]
IV-D-585	Mr. Frank B. Terrill
IV-D-586	Ms. Lidona Shelley
IV-D-587	Mr. Brent Hartinger
IV-D-588	Ms. Constance Northey
IV-D-590	Mr. Michael J. Curley
IV-D-591	Ms. Susan M. Hodge
IV-D-592	Ms. Miriam Bishop
IV-D-594	Mr. John Candy
IV-D-595	Mr. Daniel M. Nelson Princeton University Department of Religion
IV-D-596	Mr. Dwight Holcombe
IV-D-597	Mr. Bruce Hoeft
IV-D-598	Mr. Lloyd D. Morrell
IV-D-599	Mr. Elliott McLean
IV-D-600	Ms. Betsy Allen
IV-D-601	Mr. Robert A. Erickson
IV-D-604; IV-D-609	Mr. Gerald S. Pade Friends of the Earth, Northwest Office
IV-D-605	Mr. and Mrs. A. Derby
IV-D-606; IV-D-689	Chris Combs
IV-D-607	Mr. Floyd Oles
IV-D-608; OAQPS-79-8/IV-D-18	Mr. Michael Gregory Sierra Club, Grand Canyon Chapter
IV-D-610	Paul and Sally Borgen

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IV-D-611	Mr. Ake Nygren Boliden Metall AB Sweden
IV-D-612	Mr. Lloyd Dodd L-M-D Electro-Silver Resource
IV-D-613	Ms. Virginia Mitchell
IV-D-614	Mr. James Tracht Pennwalt Corporation
IV-D-615	Mr. Marvin Williams Washington State Labor Council AFL-CIO
IV-D-616	Mr. Arne Bjornberg Mr. Rolf Svedberg Boliden Metall Ab Sweden
IV-D-617; OAQPS-79-8/IV-D-19	Mr. David F. Zoll Chemical Manufacturers Assoc.
IV-D-618	Mr. Christopher DeMuth Office of Management & Budget
IV-D-619	Mr. James H. Boyd Newmont Mining Corporation
IV-D-620	Mr. R. J. Moore, F. C. Schafrick, and J. C. Martin Shear & Gardner (for ASARCO) Dr. Ian T.T. Higgins (for ASARCO)
IV-D-620; IV-F-2[b]	Mr. M. O. Varner, C. K. Guptill, C. R. Counts, and D. E. Holt ASARCO, Inc.
IV-D-621	ASARCO, Inc. *See footnote at end of this section
IV-D-623	Mr. William Mitchell
IV-D-624	Mr. William Woolf

Docket Item Number	Commenter and Affiliation[a]
IV-D-625; OAQPS-79-8/IV-D-20	Mr. J.F. McKenzie Pacific Gas & Electric
IV-D-626; OAQPS-79-8/IV-D-21	Mr. Richard Kamp Smelter Crisis Education Project
IV-D-627	Mr. Thomas C. White
IV-D-628	Mr. E.E. Ives Stearns-Roger Engineering Corp.
IV-D-629	Mr. Brian Baird
IV-D-630	Mr. John Thomas
IV-D-631	Mr. Harmon Rulifson
IV-D-632	Mr. Robert Matthews
IV-D-633	Mr. Dennis Crumbley
IV-D-634	Mr. A.V.J. Prather and K.E. Blase Prather, Seeger, Doolittle & Farmer, Dr. S.H. Lamm (for Kennecott)
IV-D-634; IV-F-2[b]	Mr. R.A. Malone, Dr. L.S. Salmon, Dr. H.A. Lewis (for Kennecott)
IV-D-635	Mr. and Mrs. LeRoy Annis
IV-D-636	Ms. Evelyn Hildebrand
IV-D-637	Ms. Lucy Fitch
IV-D-638	Ms. Julie Reimer
IV-D-639	Mr. Larry Jones
IV-D-640	Mr. Floyd Hoffman, R.E. Johnson, and W.N. Miller Phelps Dodge Corporation
IV-D-640; IV-F-2[b]	Dr. S.H. Lamm, Mr. T.L. Cogut (for Phelps Dodge)
IV-D-640; IV-F-6[b]	Mr. F.P. Mendola Phelps Dodge Corporation

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IV-D-640; IV-D-704; OAQPS-79-8/IV-D-22; OAQPS-79-8/IV-D-32	A. Coy and S. Christiansen Evans, Kitchel & Jenckes (for Phelps Dodge)
IV-D-641; OAQPS-79-8/IV-D-23	Mr. Steven Kuhrtz New Jersey Dept. of Environmental Protection
IV-D-642; IV-D-750	Ms. Yvonne Thomas
IV-D-643	Ms. Jeanette Wakeman
IV-D-644	Ms. Katherine German
IV-D-645; IV-D-763; IV-D-770	Dr. Thomas Douglas Allied Medical Examiners
IV-D-646; IV-D-708 and 708a; IV-D-712; IV-D-767	Mr. Michael Wright United Steelworkers of America
IV-D-647	Mr. Victor Gawley
IV-D-650	Mr. William Evan Wharton School of Finance University of Pennsylvania
IV-D-651; IV-D-653	Mr. James Nolan Puget Sound Air Pollution Control Agency
IV-D-652	Washington State Department of Social & Health Services
IV-D-654	Mr. Doug Sutherland Tacoma-Pierce County Board of Health
IV-D-655	Mrs. P.A. Aarrestad
IV-D-656	Mr. Joseph Shopin
IV-D-657	Mr. Warner Matson
IV-D-659	Mr. Dwight Kipp
IV-D-661	Mr. Douglas Branson
IV-D-663	David and Marti Lambert

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IV-D-664	Dr. John Van Ginhoven
IV-D-665	Mrs. Harold Hartinger
IV-D-669	Mr. Bradley Nakagawa, et al.
IV-D-671	Mr. Warren Wotten
IV-D-672	Ms. Annabelle Reed
IV-D-674	James and Debra Mains
IV-D-675	JonLee Joseph
IV-D-676; IV-D-677; IV-D-777	Sen. Slade Gorton U.S. Senate
IV-D-678	Ms. Susan Macrae Sierra Club
IV-D-679	Mr. Bernard Clouse
IV-D-680; IV-D-681	Mr. Leonard Roberts Office of Budget and Management Ohio State Clearinghouse
IV-D-682; IV-D-773	Mr. Floyd Frost, Ph.D. Washington Department of Social and Health Services
IV-D-684; IV-D-754; IV-D-780	Ms. Darcy L. Wright
IV-D-685	Mr. Jon Nuxoll
IV-D-686	Mrs. T.L. Radke
IV-D-688	Ms. Mary Clark Lee
IV-D-690	Mr. Jack Callinsky
IV-D-692; IV-D-787; IV-D-792; IV-D-793	Mr. Gerald McGrath
IV-D-693; IV-D-764; IV-D-791	Mr. Arthur Dammkoehler Puget Sound Air Pollution Control Agency

Docket Item Number	Commenter and Affiliation[a]
IV-D-694	Donald and Shirley Ferris
IV-D-696	Ms. Gail Nordstrom
IV-D-697	Mr. Everett Lasher
IV-D-698; IV-D-731; IV-D-766; OAQPS-79-8/IV-D-26; OAQPS-79-8 /IV-D-31; OAQPS-79-8/IV-D-34	Mr. Robert Abrams Ms. Mary Lyndon New York State Department of Law
IV-D-700	Sven and Arvi Halstensen Star Electric
IV-D-701	Mr. Jon Hinck Greenpeace, U.S.A.
IV-D-704a; OAQPS-79-8/IV-D-28	Dr. Steven Lamm Consultants in Epidemiology & Occupational Health, Inc.
IV-D-705	Iskra Johnson
IV-D-706	Mr. John Roberts Engineering Plus, Inc.
IV-D-707	Ms. Margaret Wolf
IV-D-709	Mr. Larry Weakly
IV-D-711	Mr. Kurt Blase Prather, Seeger, Doolittle & Farmer
IV-D-713	Mr. Francis Hull
IV-D-715	Mr. Phil Nelson Washington State Department of Ecology
IV-D-717; IV-D-722	Mr. James Harris
IV-D-718	Ms. Eileen Goldgeier Brown University
IV-D-720	Ms. Lizabeth Brenneman
IV-D-724	Mr. William Rodgers, Jr. University of Washington School of Law

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IV-D-725	Mr. Hugh Mitchell
IV-D-726	Mr. Peter Andrews
IV-D-727	Mr. John Calnan
IV-D-728	Mr. Paul Karkainen
IV-D-729	Mr. Timothy Larson University of Washington Department of Civil Engineering
IV-D-730	Ms. Debbie Huntting
IV-D-732	Mr. Peter Murray Vashon Business Assoc.
IV-D-733	Mr. Dan Schueler
IV-D-734	Joseph and Karen Bartle
IV-D-735	Mr. Frank Hagel
IV-D-736	Mr. Robert Evans Purified Air Systems
IV-D-737	Washington Fair Share
IV-D-738; IV-D-751; IV-F-9	Ms. Jeanne Snell Vashon-Maury Island Community Council
IV-D-739	Mr. Douglas Easterling University of Wisconsin Department of Psychology
IV-D-740	Mr. Bruce Mann University of Puget Sound Department of Economics
IV-D-741	Dr. Jesse Tapp Seattle-King County Department of Public Health
IV-D-742	Mrs. Anna Marie Champlain
IV-D-743	Mr. Brian Kameus

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IV-D-744	Ms. Lin Noah
IV-D-746	Kelly Wheat
IV-D-747; OAQPS-79-8/IV-D-24	Dr. Thomas Godar American Lung Association
IV-D-748	Ms. Karen Langbauer
IV-D-752	Mr. Daniel Carlson
IV-D-753	Ms. Kathleen R. Harkins and Mr. Vernon W. Harkins
IV-D-755; IV-D-758; OAQPS-79-8/IV-D-25	Dr. W. Dale Overfield Neurology and Neurosurgery Associates of Tacoma, Inc., P.S.
IV-D-756	Ms. Penny Perka
IV-D-760; IV-D-774	Mr. Nils Lucander
IV-D-761	Ms. Mary-Win O'Brien United Steelworkers of America
IV-D-762	Mr. Richard Dale Smith Port of Tacoma
IV-D-765	Mr. G.D. Schurtz Kennecott
IV-D-768	Ms. Marjorie L. Williams and Ms. Fern Stephan
IV-D-769	Mr. Lance Neitzel
IV-D-771	Mr. Jeffrey Morris and Ms. Cheryl Platt
IV-D-772; OAQPS-79-8/IV-D-16	Dr. Philip J. Landrigan Centers for Disease Control NIOSH Robert A. Taft Laboratories
IV-D-775	Mr. Norman D. Dicks Member of Congress

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IV-D-776	Mr. Rod Chandler Member of Congress
IV-D-778	Mr. John McCain Member of Congress
IV-D-779	Ms. Katherine M. Hayes
IV-D-782	Mr. Ross Schlueter
IV-D-783	Mr. Gary A. Preston
IV-D-784	Mr. Dave Bateman
IV-D-785	Mr. Richard W. Rice Phelps Dodge Corporation
IV-D-788	Mr. R.A. Malone Kennecott
IV-D-789	Mr. M.O. Varner ASARCO, Inc.
IV-D-790	Mr. Richard W. Rice Phelps Dodge Corporation
IV-D-795; OAQPS-79-8/IV-D-9	Ms. Eve R. Simon
IV-D-810	Ms. Denise Fort State of New Mexico Environmental Improvement Division
IV-D-811	Mr. F.C. Schafrick Shea & Gardner (for ASARCO)
IV-D-812	Mr. K.E. Blase Prather, Seeger, Doolittle & Farmer (for Kennecott)
IV-D-813	Mr. S.J. Christiansen Evans, Kitchel & Jenckes (for Phelps Dodge)
IV-D-814	Mr. Gordon Venable State of New Mexico Environmental Improvement Division

Docket Item Number	Commenter and Affiliation[a]
IV-F-1**	Public Hearing transcript Thomas Jefferson Auditorium Department of Agriculture Washington, D.C. November 8, 1983
IV-F-2[b]	Mr. Blake Early Sierra Club
IV-F-3, -4, -5**	Public Hearing transcripts Bicentennial Pavillion Tacoma, Washington November 2-4, 1983
IV-F-6[b]	Mr. Rolf Svedberg Boliden Metall AB Sweden
OAQPS-79-8/IV-D-1	Mr. Thomas J. Koralewski Libbey-Owens-Ford Company
OAQPS-79-8/IV-D-4	Mrs. Robert D. Hartwig
OAQPS-79-8/IV-D-5	Mr. H. E. Dean Plains Cotton Growers, Inc.
OAQPS-79-8/IV-D-8	Mr. Earl W. Sears National Cotton Council of America
OAQPS-79-8/IV-D-27	Mr. J. T. Barr Air Products and Chemicals, Inc.
OAQPS-79-8/IV-D-29	Dr. Samuel Milham, Jr. Washington State Department of Social and Health Services
OAQPS-79-8/IV-D-30	Dr. Ian Higgins University of Michigan School of Public Health
OAQPS-79-8/IV-D-35	Comments Cross Referenced To Other Dockets
OAQPS-79-8/IV-D-36	Hunton & Williams for UARG

Docket Item Number	Commenter and Affiliation[a]
OAQPS-79-8/IV-D-37	Wapora, Inc., "Carcinogens from Municipal Incinerators"

[a]If no affiliation is indicated, commenter is a private citizen.

[b]These docket items contain the written testimonies submitted by commenters at the public hearings, which are essentially identical to their oral presentations.

[c]Not a comment on this standard.

*The IV-D-621 code indicates comments submitted by ASARCO. Numbers 1-16 following 621 indicate sections of ASARCO comments. Naumbers following 1-16 and immediately preceeded by a decimal point indicate subsections, e.g., IV-D-621-15.1 represents comments found in subsection 1 within section 15 of ASARCO's comments.

**In the main text, a one, two, or three digit number following a decimal point indicates the position of the commenter within the order of the speakers at the hearing on that particular day, e.g., IV-F-1.13 represents the thirteenth speaker at the public hearing on November 8, 1983 in Washington, D.C.

2. HEALTH

2.1 HEALTH EFFECTS ASSOCIATED WITH EXPOSURE

2.1.1 Carcinogenicity of Arsenic Emissions

Comment:

A number of comments submitted in response to the listing of arsenic agreed with EPA's conclusion that arsenic should be regarded as a human carcinogen (IV-D-11, IV-D-66, IV-D-158, IV-D-150, IV-D-144, IV-D-590, IV-D-164, IV-D-292, IV-D-420, IV-D-427, IV-D-441, IV-D-152, IV-D-592, IV-D-301, IV-D-388, IV-D-588, IV-D-8, IV-D-411, IV-D-314, IV-D-604, IV-F-3.30*, IV-F-3.31, IV-F-3.37, IV-F-3.38, IV-F-3.43, IV-F-3.46, IV-F-3.60, IV-F-3.103, IV-F-3.72, IV-F-4.4, IV-F-4.6, IV-F-4.9, IV-F-4.25, IV-F-4.28, IV-F-4.31, IV-F-4.50, IV-F-4.66, IV-F-5.15, IV-D-709, IV-D-717, IV-D-718, IV-D-756, IV-D-768, IV-D-722, IV-D-726, IV-D-16 IV-D-772, IV-D-705, IV-D-710, IV-D-742, IV-D-746, IV-D-427, IV-D-515, IV-D-530, IV-D-541, IV-D-622, IV-D-630, IV-D-644, IV-D-673, IV-D-676). One writer (IV-D-66) indicated that he knows arsenic is a carcinogen and that according to ASARCO's Michael Varner, high levels of arsenic caused cancer in pre-WWII workers. A second writer (IV-D-411) spoke of her experiences as a biologist and cancer victim. She is fully convinced that arsenic, in any amount, causes cancer. A third writer (IV-D-164) referred to a Tacoma area Veterinarian's statement that there is "an unusually high incidence of cancer among hogs".

Comment:

Testimony was also offered on the carcinogenicity of inorganic arsenic. One commenter (IV-F-3.52) expressed concern for the effects of arsenic on children growing up with as high as ten times the normal amount of a known carcinogen in their bodies. The Washington State League of Women Voters (IV-F-4.11) in their testimony, referred to arsenic as a hazardous air pollutant and known carcinogen.

* See footnotes in Chapter 1 for description of the decimal system used to identify public comment.

Comment:

Some commenters discussed the types of cancer caused by arsenic, specifically, lung and skin cancer (IV-D-115, IV-D-32, IV-D-106, IV-D-412, IV-F-3.4, IV-F-3.41, IV-F-3.42, IV-F-4.9, IV-F-4.66, IV-F-4.71, IV-D-429, IV-D-137, IV-D-111, IV-D-621-16.12, IV-D-611, IV-D-622, IV-D-670, IV-D-676). Some individuals felt that arsenic caused lung cancer (IV-D-4, IV-F-3.6, IV-F-3.7, IV-F-3.5, IV-F-4.4, IV-F-4.68 IV-D-141, IV-D-146, IV-F-4.71). According to one correspondent (IV-D-4), lung cancer is two times as common near arsenic emitting smelters. Another correspondent (IV-D-141) indicated that EPA cites the cancer risks of arsenic, but cannot determine how much it takes to cause lung cancer. Another correspondent (IV-D-146) stated the odds of getting lung cancer may be slightly above average for smelter workers.

Comment:

One individual (IV-F-3.73) testified that arsenic in the air would increase the risk of lung cancer. A second commenter (IV-F-4.62) stated his belief that arsenic caused an increased level of lung cancer in smelter workers. A third individual, speaking as a member of a smelter union, expressed the opinion that exposure to inorganic arsenic posed health risks. "We know what arsenic has done to too many of our union brothers and sisters in the Tacoma Smelter and other copper smelters. It was the deaths of our members which provided the conclusive evidence that arsenic causes lung cancer" (IV-F-4.4).

Response:

The EPA agrees with the commenters. The present status of inorganic arsenic as a human and experimental animal carcinogen has been closely investigated by agencies such as the National Institutes of Occupational Safety and Health, scientific organizations such as the National Academy of Science and the International Agency for Research on Cancer (IARC), and in a number of individual assessments.

The EPA has estimated the relative carcinogenic potencies of a number of chemical substances and has ranked arsenic within the first quartile of 52 suspect carcinogens among such other suspect human carcinogens as DDT,

PCBs, Aldrin, and B[a]P. In addition, the IARC characterizes arsenic as carcinogenic to humans.[1]

Epidemiological studies of copper smelter workers in the U.S., Sweden, and Japan have strongly suggested an increased risk of respiratory cancer resulting from exposure to airborne inorganic arsenic. The EPA's Health Assessment Document has reviewed 12 such studies, of which 11 have shown a positive association between exposure to arsenic and lung cancer.[2] The range of the statistical mortality rates from lung cancer in smelter workers above the expected lung cancer mortality rates in the non-exposed population indicates a 3-fold to 12-fold increase in the risk of lung cancer as a result of airborne arsenic exposure.

Proportionate mortality studies of arsenical pesticide workers have also shown an increased risk of lung cancer mortality in a range of 3 to 16 times that expected. A study of German vintners using arsenical pesticides found a significant increase in lung cancer mortality above the expected rate.

With respect to nonoccupationally exposed groups, arsenic contaminated drinking water studies and studies of patients using arsenical medicinals have demonstrated a skin cancer prevalence rate in these exposure groups. A study of Taiwan residents consuming high levels of arsenic in drinking water showed a 10-fold increase in the risk of skin cancer. Precancerous hyperpigmentation and hyperkeratosis were evident in many other arsenic contaminated drinking water studies. Keratonic lesions, hyperpigmentation, and epitheliomas were found to be prevalent in individuals using arsenical medicinals.

Comment:

Several commenters expressed concern about the correlation between lung cancer and the ASARCO Tacoma smelter emisisons. Five individuals noted that Tacoma ranked below the national average and/or as fifth among the state's 10 largest cities for lung cancer (IV-D-264, IV-D-255, IV-D-256, IV-D-330, IV-D-402, IV-F-4.38).

Response:

While EPA agrees that studies of the lung cancer rates in the vicinity of Tacoma generally do not show elevated lung cancer rates, the Agency does not agree that these findings are sufficient to discount the potential cancer risks from exposure to ambient arsenic. The power of any epidemiological study in the detection of risk is limited. Particularly for more common forms of cancer such as lung cancer, a large increase in observed cancers would be necessary to distinguish the effect of a specific carcinogen from a relaitvely high background incidence. The Tacoma data may indicate that the problem is not "epidemic" in nature, and in this regard are not inconsistent with EPA's risk estimates.

Comment:

A few comments (IV-D-593, IV-F-3.72, IV-D-571, IV-D-622, IV-D-630, IV-D-676) referred only to skin cancer. One writer (IV-D-593) commented that EPA's own scientists had acknowledged a relationship between arsenic exposure and skin cancer but that the Agency ignored this effect because it is curable. An individual (IV-F-3.72) thought there's a possibility of skin cancer transmitted to human being through hand-mouth dust contact. However, two commenters felt that arsenic does not cause skin cancer (IV-D-621-14.4, IV-D-345). Two correspondents discussed skin cancer in relation to the smelter emissions. One correspondent (IV-D-391) wrote that although his family had lived near the ASARCO Tacoma smelter for a total of over 800 person-years, not one had ever had skin cancer. Another correspondent (IV-D-597) stated that two family members had developed skin cancer, fortunately treatable, at various times in their lives (IV-D-597).

Response:

The EPA reviewed case-control studies of populations or individuals exposed to arsenic-contaminated drinking water, and arsenical medicinals.[4] Most of the studies demonstrated a positive association between exposure, either by ingestion or dermal absorption, and the manifestation of skin cancer. A study of 40,000 persons in Taiwan exposed to arsenic in the drinking water

found a significant prevalence of skin cancer over that of 7,500 other Taiwanese who drank water free of arsenic. A study of a village in Chile dependent on arsenic contaminated drinking water showed precancerous skin formations in many of the inhabitants. Similar etiological studies in the U. S. where relatively high arsenic contamination existed in the drinking water have not demonstrated a prevalence of skin cancer. Case reports of patients that were treated with arsenical medicinals showed histopathic manifestations including hyperpigmentation, keratotic lesions and epitheliomas. While the bulk of the evidence does correlate skin cancer with exposure to arsenic contaminated drinking water and arsenical medicinals, the results of the case reports were not quantitative to the extent that risk from exposure could be modeled. Furthermore, in reviewing the primary route of individual exposure to inorganic arsenic, EPA determined that the primary exposure pathway affecting the most numbers of people is the airborn dispersion of inorganic arsenic from the emissions of certain industrial processes. Therefore, in estimating risk only epidemiological data that involved this exposure pathway were assessed.

Comment:

One commenter (IV-F-5.21) referred to the difference in individual susceptibility to cancer from arsenic exposure. According to this commenter:

"A lot of people think that because they can get by without having cancer from arsenic that everybody is the same and in the same boat but I think some people can get by without this cancer...or the smelter fumes and not get cancer but other ones will".

Response:

Individual susceptibility to carcinogenesis is a consideration in the overall assessment of risk. The U. S. population represents a very diverse, genetically heterogeneous group that is exposed to a variety of toxic agents. The National Academy of Sciences has stated that:

"Genetic variability to carcinogenesis is well-documented, and it is

also known that individuals who are deficient in immunological competence (for genetic or environmental reasons) are particularly susceptible to some forms of cancer. It seems, therefore, that even if we were to postulate an average threshold for a particular cancer induced by a particular agent, we would in practice need a series of thresholds for different individuals. It would be extremely difficult, in practice, to establish a single threshold."[5]

The EPA agrees with NAS' observations on this subject and, EPA, for this reason, tends to give less weight to view that a threshold for airborne arsenic exposure exists below which no possibility of cancer can arise. Considering the potential for variation in susceptibility, it is unlikely that practical thresholds could be determined with any degree of certainty. In addition, the inhalation exposure data base involves healthy male workers and therefore does not provide adequate information for EPA to assess risk to subpopulations with potentially higher susceptability.

Comment:

Examples of community members (mostly employed by and/or living in the vicinity of the ASARCO Tacoma smelter who had developed cancer (IV-D-79, IV-D-133, IV-D-139, IV-D-428, IV-F-4.52, IV-F-5.18) were provided. Examples were also given of those, in similar circumstances, who hadn't developed cancer (IV-D-30, IV-D-133, IV-D-139, IV-D-277, IV-D-326, IV-D172, IV-D-181, IV-D-208, IV-D-210, IV-D-265, IV-D-229, IV-D-383, IV-D-457, IV-D-345, IV-D-306, IV-D-333, IV-D-356, IV-D-359, IV-D-282, IV-D-324, IV-F-4.5, IV-F-4.21, IV-F-4.52, IV-D31, IV-D-58, IV-D-134, 481,8343, IV-D-504, IV-D-193, IV-D-179, IV-D-601, IV-D-485, IV-D362, IV-D-372, IV-D-270, IV-F-4.44, IV-F-4.49, IV-F-4.58).

Comment:

Several commenters disagreed with the potential positive correlation between arsenic and lung cancer (IV-F-4.60, IV-D-304, IV-D-316, IV-D-338, IV-D257, IV-D-274, IV-D-303, IV-D-312, IV-D-502, IV-D-185, IV-D-196, IV-D-160, IV-D-167, IV-D-168, IV-D-311, IV-D-242, IV-D-494, IV-D-232, IV-D-621-14.12, IV-D-621-16.11, IV-D-343). One writer (IV-D-494) stated that arsenic emitted by the smelter has been in the air for over sixty years and research has shown a below average rate for lung cancer in the Tacoma area. Good scientific work would not ignore past real experience and make life and death predictions

based on short term measurements and estimates. Another writer (IV-D-232) asserted that no link has been established between arsenic and lung cancer. An individual (IV-F-4.60) testified that arsenic air pollution has not been shown to produce high lung cancer rates in the study populations.

Comment:

Many of the commenters claimed that there was no connection between the smelter emissions and lung cancer (IV-D-212, IV-D-213, IV-D-215, IV-D-354, IV-D-367, IV-D-355, IV-D-349, IV-D-382, IV-D-340, IV-F-3.2, IV-D-621-12.6, IV-D-621-12.11, IV-D-621-12.17, IV-D-621-12.22, IV-D-621-13, IV-D-621-14.4, IV-D-621-14.7, IV-D-621-15.9, IV-D-621-16.2, IV-D-695, IV-D-697, IV-D-718, IV-D-621-12.8, IV-D-621-15.9, IV-D-621-16.11, IV-D-350, IV-D-518, IV-D-523, IV-D-550, IV-D-555, IV-D-568, IV-D-612, IV-D-607, IV-D-522, IV-D-561, IV-D-621-5, IV-D-625). Dr. Samuel Milham (IV-F-3.2) investigated lung cancer mortality by census tract in the Tacoma area and found no difference in lung cancer mortality in census tracts closest to the smelter when workers were removed (although smelter emissions were not mentioned in the description of this study).

Comment:

Other writers felt that ASARCO emissions do not cause cancer (IV-D-335, IV-D-487, IV-D-381, IV-D-472, IV-D-154, IV-D-166, IV-D-233, IV-D-226, IV-D-250, IV-D-489, IV-D-251, IV-D-377, IV-D-68, IV-D-621-12.3, IV-D-621-12.15, IV-D-62112.11, IV-D-621-12.23, IV-D-621-14.3, IV-D-621-6, IV-D-343, IV-D-534, IV-D621-14.9). According to one writer (IV-D-489), epidemiological studies have not proven a direct relationship between cancer and the levels of emissions currently found in the areas around primary copper smelters.

Comment:

An individual (IV-F-3.15) commented that attributing a certain number of deaths per year from cancer due to smelter emissions was "sheer nonsense" and that while body chemistry can trigger cancerous conditions in certain individuals, determining the degree of tolerance and the amount of chemical intake the average individual could withstand could be pure guesswork.

Comment:

Some people disagreed with an association between arsenic and cancer on other grounds (IV-D-249, IV-D-187, IV-D-407, IV-D-192, IV-D-149, IV-D-358, IV-D-275, IV-F-3.18, IV-D-45, IV-F-3.52, IV-F-3.59, IV-F-5.8, IV-D-16 (IV-D-772), IV-D-621-15.6). A chronic disease epidemiologist (IV-D-45) maintained that the relationship of dose and cancer risk which supports the NESHAP may be nothing more than a relationship between age and risk of cancer. A correspondent (IV-D-149) indicated that many experiments in animals have shown trivalent inorganic arsenicals incapable of causing cancer. Five individuals (IV-D-621-120, IV-D-621-15.6, IV-D-621-12.8, IV-D-621-16.12, IV-D-621-12.15), commented that arsenic is actually a nutrient and/or has a protective effect on the human system.

Comment:

Testimony regarding the lack of a positive relationship between arsenic and cancer was also provided. One individual (IV-F-3.11) noted that while certain epidemiological studies based on industrial exposures seem to implicate arsenic as a carcinogen, most community based studies have not provided confirmation and that there is uniform support from the animal literature denying the carcinogenicity of arsenic. Nancy Frost (IV-F-4.72), in support of her father, Douglas V. Frost, Ph.D., a nutrition biochemist, submitted a newspaper article that he wrote in which he described arsenic as an essential nutrient and not a pollutant. Dr. Frost concluded that "no arsenical had been found to cause cancer experimentally in animals and the presumed link between arsenic and cancer in human's was an unproven and untestable association. Ms. Frost also submitted a paper published by her father entitled "What Do Losses in Selenium and Arsenic Bioavailability Signify for Health?" in which Dr. Frost asserted that, "there are many more likely causes for the cancers in humans for which arsenic is blamed".

Community Health Studies:

Kennecott (IV-D-634) submitted testimony by two epidemiologists stating that review of the community epidemiology studies referenced in EPA's draft

health assessment and other such studies shows no support for EPA's statement that excess mortality or morbidity exists for populations living near arsenic emitting sources. One commenter believed that in total, these studies represent a sufficient population group to have shown such effects if they existed. The commenters reviewed 10 lung cancer mortality studies. They claimed there were only three with any positive findings, and they questioned these findings. These 10 studies included the following:

1. Blot and Fraumeni, 1975. Lung cancer mortality was shown to be significantly higher among males and females in 36 U.S. counties with copper, lead, and zinc smelters and refineries than in the rest of U.S. counties. The increase, corrected for demographic variables, was 17 percent for males and 15 percent for females over the years 1950-1969.

2. Lyon, et al. 1977. Using a population based cancer registry, addresses at diagnosis of lung cancer cases are compared to malignant lymphoma controls to assess the possible carcinogenic effect of the Salt Lake City copper smelter. The distribution of distances from the Smelter of lung cancer cases and lymphoma controls was similar.

3. Rom, et al. 1982. Using the same methodology as Lyon, lung cancer cases around the El Paso, Texas, smelter were shown to have the same distance distribution from the smelter as breast and prostate cancer controls.

4. Greaves, et al. 1981. Greaves, using the same methods as Lyon and Rom, studied the distances of residences at diagnosis or death of lung cancer cases and controls (prostate, colon and breast cancers) from ten copper smelters and one lead-zinc smelter. The distance distribution of lung cancer was not significantly different from the distribution of the control cancers in any of the areas studied.

5. Pershagen, et al. 1977. Mortality in the region around the Ronnskar Smelter in northern Sweden was studied. The population residing within 15 km of the smelter was compared to the population residing 200 km away. The lung cancer mortality in the exposed population (<15 km) was significantly higher in men than in the comparison population, but not significantly different in comparison to national rates. When the occupationally exposed

cases are removed, the lung cancer Standard Mortality Rate (SMR) was reduced and was no longer statistically significantly different than the comparison population.

6. Matinoski, et al. 1976. Cancer mortality reported on death certificates was studied in census tracts in Baltimore around a chemical plant producing calcium and lead arsenate, arsenic acid, cupric acetoarsenite (Paris green), and sodium arsenite. An increase in lung cancer was seen in the census tract containing the plant in the years 1966-1974 in males only. No increase was seen in an earlier time period (1958-1962). Residents of the city who died elsewhere were not ascertained. In the census tract where the plant was located, the average soil arsenic level was 63 ppm. Removing plant workers from the high lung cancer census tract did not eliminate the high male lung cancer mortality rate.

7. Polissar, et al. 1979. Lung cancer mortality by census tract was examined around the Tacoma, Washington, copper smelter. Two surrogates for arsenic exposure were used: (1) distance of the census tract to the smelter, and (2) concentration of sulfur dioxide over background for each census tract. There was no excess risk of lung cancer for persons living near the smelter.

8. Hartley, et al. 1982. Lung cancer mortality in the 35 census tracts in Tacoma, Washington, was examined for the 21 years 1950-1970, using the death certificate address for assignment to census tract. Lung cancer mortality was no higher in the census tracts near the smelter than in those farther away.

9. Milham, et al. 1982. Class rosters of children enrolled at the Ruston elementary school (100 yards from the Tacoma, Washington, smelter) were examined. A cohort of 283 children who were enrolled for three or more years during the years 1900-1919 was developed. Surviving cohort members were contacted and death records were obtained for decedent members. Using life table comparisons, mortality of men in this cohort was shown to be favorable (more survivors to 1980 than expected). It also did not appear that lung cancer was increased in the male cohort (1 lung cancer death among 20 for whom death certificates were obtained). Forty percent of the men in this cohort were employed at the smelter at some time.

10. Newman, et al. 1976. Although this was primarily a study of lung cancer cell type in two Montana copper mining and smelting counties, it demonstrated an increase in lung cancer incidence in both men and women in the towns of Butte and Anaconda, but the same increase was not seen in the counties as a whole.

Also, commenters identified several morbidity studies and they are summarized below.

Community Morbidity Studies

1. Milham and Strong, 1974. In the population around the Tacoma smelter, children were shown to have increased levels of arsenic in hair and urine. Urinary arsenic decreased with distance from the smelter. Mean urinary arsenic for children living within .5 miles of the smelter was .30 ppm (normal .014). Vacuum cleaner dust and attic dust contained over 1000 ppm of arsenic.

2. Morse, et al. 1979. Children exposed to arsenic in air and drinking water in Ajo, Arizona, near a copper mine and smelter were studied. Hair and urinary arsenic were elevated in children and decreased with distance from the smelter. No clinical or hematologic abnormalities attributable to arsenic were found.

3. Baker, et al. 1977. In 19 U.S. towns with primary nonferrous smelters, one to five year old children were studied for arsenic, lead and cadmium absorption. Urine arsenic was elevated near 10 of 11 copper smelters.

4. Milham 1977. Hearing, hematologic status and school attendance of children living in Ruston, Washington (near the Tacoma Smelter), were the same as children living further away from the smelter. The Ruston children have increased levels of urinary and hair arsenic.

5. Nordstrom, et al. 1978. Frequencies of congenital malformations were studied in offspring of female employees of the Ronnskar Smelter and in the populations living near the smelter. In the offspring of the employees, the frequency of multiple malformations was increased. However, there was no increase in total frequency of malformations or in type of malformations in the population around the smelter.

6. Nordstrom, et al. 1978. Frequency of spontaneous abortion and birthweight distributions in female smelter employees and women who lived near the smelter were examined. Women working at the smelter had an increased frequency of spontaneous abortion and low birthweight infants. Women living near the smelter showed no increase in spontaneous abortions, but had a tendency to have infants slightly lighter than women who lived at a distance from the smelter.

The commenter said the Matanoski census track study, showing an increase in lung cancer near a plant in Baltimore, MD, producing arsenical compounds, is probably an aberration. The study found increased lung cancer rates only in males and only between the years 1966-1974. Increases were not seen in the years 1958-1962 despite the fact that the smelter had been operating since about 1900. Furthermore, the commenters claimed that studies of communities with higher potential arsenic exposure showed no increase in lung cancer in persons residing near higher arsenic emitting sources. Three studies they cited to show this were the Pershagen et al., 1977 study of the region around the Ronnskar smelter in northern Sweden, and studies by Polissar, et al., 1979 and Hartly et al., 1982 of census tracts around the ASARCO-Tacoma copper smelter. The studies of Tacoma showed no increase in lung cancer. The Pershagen study showed no significant increase once occupationally-exposed men were removed. The commenters said that emissions of arsenic are lower from the Baltimore plant than from the smelters, and that maximal soil arsenic levels near the Baltimore plant are only 10 percent of those in the Tacoma area. Thus, they said that any increases in lung cancer mortality should also have been seen at these plants, and that the Matanoski study results are an aberration.

The commenters noted that a study by Blot and Fraumeni showed increased lung cancer in males and females for the years 1950-1969 in 36 counties with lead, copper, zinc smelters and refineries when compared to all U.S. counties. The commenters said that weak points of the study include the fact that smelting counties were not separated from refining counties and no data on arsenic exposure are available. They also stated that since lung cancer mortality varies by a factor of 2 from state to state, it is more valid to compare smelting counties with other counties in the same state.

They claimed that if lung cancer rates are compared with rates for all other counties in the same state during the same time period, the lung cancer excess disappears.

The commenters said the Newman et al., 1976 study showed excess lung cancer rates in two cities near copper mining and smelting facilities, but not in the county as a whole. One epidemiologist proposed that since residential information was obtained from hospital and tumor registry files, elevated rates in the towns may have been caused by migration from rural areas to the towns after retirement.

The commenters' review of six community morbidity studies showed only one finding with possible potential significance. The Nordstrom et al., 1978 study found that women living near a Swedish smelter delivered infants weighing slightly less on average than those at distances from the smelter. No increase in spontaneous abortions or congenital malformations was observed in this group. The commenter did not know if any significant health problems could be associated with low birthweight , and could not tell if the findings were an aberration or might be possible to duplicate in future studies.

The commenters concluded that no significant increases in mortality or morbidity had been shown in areas around high and low arsenic emitting sources. One commenter said he believed that, in total, the studies represented a significant population group exposed for a number of years, and that if there were discernable increases in lung cancer or other morbidity, these studies should have shown them. He did not attempt to calculate the total population represented by the studies, and his conclusion that the studies should have been able to detect any lung cancer increased was based on his judgment and the judgment of other epidemiologists he had contacted.

Comment:

On behalf of Phelps Dodge (IV-D-704a), CEOH submitted a translation of a 1981 report by the Swedish National Health Board expert committee. The report reviewed the Nordstrom et al., studies relating arsenic exposure to women living near a Swedish smelter to low birthweight of their babies. The report cited several factors which may affect birthweight:

- age and birth year of the mother
- Parity (number of children born previously) and earlier pregnancies
- smoking habits
- health status
- social factors (social group membership, occupation, etc.)

The committee reported that these factors have not been adequately considered and may account for the variation in birthweights between groups. The committee further reported that population groups chosen in the studies were not homogeneous in important respects. There was no reporting of an age factor or of a factor of previously experienced deliveries in this study. The committee believed these factors to substantially influence birth rates and felt them necessary to account for in such a study. Social factors also were not controlled in the studies. Furthermore, there appeared to be confusion in the concepts of pregnancy order and parity in the reporting of the studies, making results difficult to interpret and unreliable. The committee said that the deficiencies cited in the study call into question the authors' statistical analysis showing differences among the groups.

The committee described Nordstrom's exposure data as vague. Exposure is described in terms of residence location (areas A through D and parishes in the Skellefteae area) or employment in a department at the smelter. Information on environment lacks detail, and there is nothing in the papers reviewed to indicate that exposures in areas A through D differ from each other. A recent article does indicate increased urinary arsenic concentrations in women living in the Skellefteae area and lower concentrations in women living further from the smelter. For occupationally exposed employees, the committee found department not to be a fine enough classification to determine exposure.

Due to the problems discussed above, the committee could not conclude from the studies that birthweights are lower for women living near the smelter.

Nordstrom's study also reported increased frequency of chromosomal abnormalities in smelter workers; however, the committee noted that the

control population is not described with respect to selection, size, and age distribution. They believed new analyses would be necessary before this finding could be supported.

The studies found that children of mothers working at the smelter have increased malformation frequencies. However, the committee presented data showing that the frequency of diagnosing and reporting malformations varies greatly between different hospitals and clinics. This makes comparison between time periods (as Nordstrom's studies have done) suspect. Furthermore, the committee deemed the population groups studied small and the numbers of malformations small. Such small numbers the committee called unreliable.

The committee also noted deficiencies in the original analyses of spontaneous abortion, and noted that reanalyses of areas around the smelter have found no significant differences in frequency of spontaneous abortion.

Other commenters (IV-D-640, IV-D-621-16.10, IV-F-1.16) referred to the same study. They characterized the study as the only study that alleges health effects from community exposure. The commenters reported that the study claims decreases in birthweight and increases in multiple malformation frequency among offspring of residents near the Ronnskar smelter in Sweden. The commenters pointed out that this birthweight study had found no difference for births of parity 1 or parity 2 and that Nordstrom's analysis had given no consideration to known factors affecting birthweight such as smoking history, maternal age and increased parity, social class, and gestational age. The commenters felt there were too many difficulties with the study to accept its results. They noted that the Swedish National Health Board expert committee report of 1981 concluded that study design and execution problems prevented these findings from being accepted at face value.

Response:

The EPA has reviewed community mortality and morbidity studies of areas in the vicinity of smelters emitting arsenic, and arsenic pesticide manufacturing plants.[6] For a number of reasons, these studies are confounding or inconclusive in demonstrating either a positive or negative association of lung cancer to community exposure to inorganic arsenic (see pages 7-50-52 in

the Health Assessment Document). For the most part, these studies did not observe the length of time people lived near smelters, nor did they account for migration patterns. In addition, arsenic exposure levels correlated with distance from the emitting source were not determined. Lung cancer morbidity was derived by inspecting death certificates and comparing rates of lung cancer morbidity in the community with national, county, or States rates. Such procedures may undercount lung cancer SMRs within the community because individuals with lung cancer may move away to receive treatment, or patients diagnosed as having lung cancer may have died of other causes. Generally, community studies do lack the statistical power to detect the increased lung cancer risk to the exposed public and EPA does not expect such studies to produce positive findings.

In the series of Nordstrom et al. studies, it has been repeatedly shown that these studies are flawed for a number of reasons. The Health Assessment Document for Inorganic Arsenic also cautions: "These studies (Nordstrom's) were not designated specifically to study effects of arsenic but rather to study the effects, in general, of the smelter work pollutants on neighboring (proximate) populations, the diverse agents involved preclude making conclusive statements about the specific effects of arsenic." In addition, unbeknownst to the EPA at the time of the HAD publication, the Swedish National Health Board Expert Committee published a report in 1981 that questioned almost every finding in the Nordstrom studies. It is therefore highly questionable whether the Nordstrom studies are suitable for making determinations regarding the potential human reproductive effects caused by arsenic exposure.

Thus, in view of the fact that the community studies did not produce a clear understanding of risk associated with arsenic exposure near a smelter, EPA resorted to the best etiological data base in characterizing inorganic arsenic as a human carcinogen: smelter worker exposure studies. While various animal studies have not demonstrated arsenic carcinogenicity despite using different chemical forms, routes of exposure, and different experimental species, various human epidemiological investigations have showed a consistent

association between airborne exposure to inorganic arsenic, and respiratory cancer in humans. The occupational studies compared the cancer risk (adjusted for age, sex, and other variables) among groups or cohorts exposed to various concentrations of inorganic arsenic with control groups not so exposed. Furthermore, the methodology followed the exposed groups prospectively over time to determine latency, and doseresponse relationships. Epidemiological studies of smelter workers exposed to inorganic arsenic have demonstrated an increased risk of lung cancer mortality 3 to 12 times the expected mortality rate of nonexposed population groups.

2.1.2 Toxicity of Arsenic Emissions/Smelter Emissions

Comment:

Some individuals (many living near the ASARCO-Tacoma smelter) commented on the toxicity of arsenic (IV-D-301, IV-D-108, IV-D-439, IV-D-116, IV-D-76, IV-D-709, IV-D-769, IV-D-720, IV-D-721, IV-D-734, IV-D-756, IV-D-698, IV-D-705, IV-D-16 (IV-D-772), IV-E-621-14.3, IV-D-730, IV-D-779, IV-D-736, IV-D-16 (IV-D-702), IV-D-524, IV-D-554, IV-D-576, IV-D-630, IV-D-674, IV-D-675, IV-D-676, IV-D-662, IV-D-677, IV-D-694, IV-D-520, IV-D-433, IV-D-438). One writer (IV-D-108) stated that the "garlic-smell" of the arsenic in the smelter plume was indicative of its toxic character. A second writer (IV-D-439) called arsenic a known poison and questioned the health danger of this compound at low concentrations. A third writer (IV-D-116) asked "why ASARCO (has) the right to daily poison (him) with its filthy arsenic emissions." A fourth writer (IV-D-76) maintained that a dose of arsenic as small as half the weight of a pin can be fatal to small children; a daily dose as small as 3.5 milligrams is likewise fatal to infants. However, one commenter (IV-D-621-16.2) noted that the body can detoxify/excrete arsenic.

Comment:

Testimony was also given on the toxicity of arsenic (IV-F-3.6, IV-F-3.11, IV-F-3.38), calling arsenic "a very toxic substance"... and that "if you put a teaspoon of it on your Wheaties, it will kill you right now...but not from lung cancer (IV-F-3.73). Another individual (IV-F-3.11) noted that concerns have been voiced in various U.S. communities and that the basis

for these concerns includes a well founded appreciation for arsenic as a lethal agent of historical as well as modern significance.

Comment:

Cases of physical ailments resulting from arsenic exposure were submitted by community members of the ASARCO smelter (IV-D-571, IV-D-622, IV-D-755, IV-D-758). According to one writer (IV-D-158), arsenic exposure in high doses causes increased incidences of chromosomal aberrations and neurological problems. Another writer (IV-D-593) referred to a statement made by Dr. Karle Mottet of the University of Washington that arsenic may cause cardiovascular problems. Another writer (IV-D-273), employed by ASARCO for years, felt that the only lasting damage that he sustained from the arsenic itself was a perforated septum.

Comment:

Commenters also discussed physical ailments (IV-F-4.6, IV-F-3.38, IV-F-3.34, IV-F-4.45). One person (IV-F-3.38) asked about other "less dramatic" health effects resulting from arsenic exposure including angina and high blood pressure. Another commenter (IV-F-3.34) noted swelling and certain described edema, especially of the lower limbs, face and ankles, and a garlic odor to the breath and body sweat. This may be associated with nausea, vomiting or diarrhea. There can be depression of the bone marrow.

Comment:

Another commenter (IV-F-3.11) claimed there is also no reason to conclude, based on either theoretical or any practical considerations, that ambient arsenic concentrations from smelters cause or contribute to any other disease processes.

One individual (IV-F-3.57) stated that ASARCO's emissions cause chromosomal aberrations and a variety of neurological problems. Another person (IV-F-3.53) maintained that community residents have unexplained breathing difficulties, gastrointestinal problems and mysterious allergies which are attributable to the smelter emissions.

Comment:

Some comments were submitted which claimed that the fumes from ASARCO were damaging to the public's health (IV-D-94, IV-D-430, IV-D-599, IV-D-597, IV-F-3.20, IV-F-3.29) and caused burning of the lungs (IV-D-89), eyes, nose, and throat (IV-D-115, IV-D-428). One writer (IV-D-89) indicated that smokestack emissions caused illness, discomfort, and severe stomach upset. Another writer (IV-D-597) commented that his wife grew up a quarter mile from the smelter smokestacks and neither she nor her brother have a sense of smell (IV-D-597). One commenter (IV-F-4.19) stated that when sailing near the smelter the smoke occasionally comes straight down onto the water and causes sore throats and general discomfort.

Comment:

A few correspondents discussed the potential adverse health effects associated with the inhalation of smelter emissions in general. Two correspondents (IV-D-113, IV-D-15) asked about the health implications of the inhalation of toxic smelter emissions. Another correspondent (VI-D-32) commented that on windless days, she cannot jog without getting a chemical taste in her mouth. Another individual (IV-F-3.10) stated that from a health standpoint primary interest is in the particles inhaled into the lower lung, approximately in the range of 0.5 microns to 5 microns. Another correspondent (IV-D-360) submitted: "I jog every day and it causes no problem."

Comment:

Four correspondents noted that arsenic-related health effects other than lung cancer have not been addressed by EPA (IV-D-32, IV-D-85, IV-D-314, IV-C-168).

Response:

The EPA agrees with the commenters that arsenic is toxic to humans. As discussed in EPA's health asessment document, the acute and chronic toxicity

of arsenic is dependent on the chemical form. Inorganic trivalent arsenic is more acutely toxic than inorganic pentavalent arsenic. The complex organic arsenic form is regarded as nontoxic. Acute effects seen after oral exposure include gastroenteritis, diarrhea, and cardiovascular effects.[7] These effects can cause death. The precise lethal dose of inorganic arsenic is unknown, however, the lethal dose of arsenic trioxide is estimated to range from 70 to 180 milligrams.

Neurotoxic effects in humans have been observed following ingestion of inorganic arsenic. These effects have varied with length and type of exposure, as well as the pathway of exposure. Neuropathies have been associated with chronic and acute exposures to high levels of inorganic arsenic, and have included: peripheral nervous system effects characterized by numbing or tingling in the hands and feet; neuralgia; peripheral neuritis, muscular weakness, and memory loss. However, specific dose-response relationships have not been established, especially to chronic low level airborne arsenic exposure.

Cardiovascular effects of inorganic arsenic exposure have been observed. A study in Taiwan indicated an occurrence of peripheral vasculopathy in a population exposed to high levels of inorganic arsenic in the drinking water, characterized by poor circulation resulting in gangrene of the feet, legs or fingers. In epidemiological studies of smelter workers, peripheral vascular disease has generally not been observed, although a few smelter studies have found a significant increase in cardiovascular mortality.[8] Studies of one copper smelter by Lee and Fraumeni (1969) and Lee-Feldstein (1983) found a significant increase in cardiovascular mortality in the workers (SMR=118 and SMR=129, respectively). No relationship to duration of arsenic exposure was found. Higgins, et al. (1982), reported on the same smelter workers, and found that cardiovascular mortality increased with increasing ceiling arsenic exposure among smokers at 500-4999 $\mu g/m^3$ (SMR=165). No effect was seen among nonsmokers. However, Lubine, et al. (1981) did not find an excess of cardiovascular disease in their cohort study of the same smelter workers (SMR=108). The confounding and conflicting

findings of smelter exposure studies suggests that further research is needed in this area.

Respiratory effects other than cancer have been observed in smelter workers exposed to high airborne arsenic levels. Pulmonary insufficiency and tracheobronchitis have been observed in smelter workers in the roaster and furnace areas. Septal perforations and rhinopharyngolaryngitis has also been seen in copper smelter workers. However, the limited information (dose-response data) did not permit the Agency to perform a risk assessment for these particular health effects.

Comment:

Several general comments were submitted to the effect that arsenic is an established health hazard (IV-D-421, IV-D-412, IV-D-419, IV-D-32, IV-D-401, IV-D-115, IV-D-81, IV-D-112, IV-D-106, C-140, IV-D-92, IV-D-292, IV-D-62 IV-F-3.30, IV-F-3.43, IV-F-4.34). One writer (IV-D-81) expressed concern about the effect of ASARCO arsenic emissions on the health of the citizens in the community and particularly, on the health of his children. A second writer (IV-D-412) simply maintained that arsenic causes pain and death. A resident (IV-F-3.43) testified: "We are saying that we don't think arsenic is safe. It's not sufficient for ASARCO to roll out a bunch of Eastern scientists to come in here and tell us, 'Hey, folks, don't worry. We've read the data; we've studied the issue.'"

Response:

Section 112 requires that EPA set standards that provide a "ample margin of safety." Where a health effects threshold can be determined, this requirement can be met by establishing the standard at a level that insures that the exposure threshold is highly unlikely to be exceeded. Where identifiable thresholds do not exist or are indeterminate, as with carcinogens including inorganic arsenic, any level of control selected short of an absolute ban on emissions, may pose a finite carcinogenic risk.

The EPA believes that the final inorganic arsenic standards which permits some level of residual risk provides an ample margin of safety to protect public health.

Comment:

Correspondents (IV-D-76, IV-D-51, IV-D-32) discussed the potential adverse health effects associated with the inhalation of arsenic. One correspondent (IV-D-76) said that the risks from inhalation of (arsenic) in the air and dust are also substantial and that the primary concern is for infants, children, and pregnant women. Another correspondent (IV-D-51) questioned to what extent the inhalation of arsenic is lethal. One correspondent (IV-D-32) expressed concern about the possibility of children absorbing arsenic by breathing playground dust.

Comment:

Many correspondents submitted comments referring to studies which have shown high levels of arsenic in the blood, hair and/or urine of children living near the copper smelters (IV-D-112, IV-D-76, IV-D-9, IV-D-11, IV-D-21, IV-D-106, IV-D-107, IV-D-166, IV-D-422, IV-D-426, IV-D-417, IV-D-164, IV-D-90, IV-D-33, IV-D-66 IV-D-404, IV-D-375). One writer (IV-D-166) claimed claimed that (his) six year old son (had) the highest content of arsenic found in the urine of all the children tested in the Olympia-Tacoma-Vashon Island area. A second writer (IV-D-164) mentioned that urinary arsenic levels twice normal were found in Island children and referred to an article in a local paper in which children aged 0-5 months showed the highest arsenic levels.

Comment:

Testimony regarding arsenic tissue levels in children was also provided (IV-F-3.2, IV-F-3.4, IV-F-3.15, IV-F-3.41, IV-F-3.53, IV-F-3.57, IV-F-3.60, IV-F-3.74, IV-F-4.15, IV-F-4.49, IV-F-4.60, IV-F-4.62, IV-F-4.68, IV-F-5.8,

IV-F-5.18, IV-F-4.71). Samuel Milham Jr., M.D., M.P.H. (IV-F-3.2) discussed a study he had conducted in which he determined that children who lived near ASARCO had higher urinary and hair arsenic levels than those who lived farther away. He attributed these high levels to the inhalation route of exposure. Graphs on urinary and hair arsenic levels in children were submitted by Dr. Milham (IV-F-3.81 -- IV-F-3.95).

Comment:

The authors of a published report entitled Monitoring and Reducing Toxic Intake of Children Near the Tacoma Smelter and in South Park, Seattle (IV-F-4.73) discussed in their testimonies the main results of this study. the Tacoma Smelter and concluded that children with pica, or who eat dirt and other materials they shouldn't, may have a significant arsenic intake. For example, some children who live near the smelter have three times the normal amount of arsenic in their urine. Also, some hair samples contained 20 times the usual amount of arsenic. Hair analysis can give a doctor an idea of the long-term ingestion of arsenic". Mr. John Roberts (IV-F-4.10), coauthor of this study, testified that both ingestion and breathing are important routes of entry of arsenic, into children.

Comment:

Comments were also made that no illness resulted from an increase in arsenic tissue levels. Dr. Milham (IV-F-3.2) indicated that hearing, chromosomal analyses, growth and development, and blood levels were all normal and that no anemia was found in the population studied (in contrast to other morbidity studies of arsenic human tissue contamination). Another individual (IV-F-3.6) testified that although arsenic levels in physiologic samples from children are elevated close to the Tacoma smelter, no increase in illness or deaths has been demonstrated.

Comment:

Some individuals (IV-D-604, IV-F-4.15, IV-D-593, IV-F-3.55, IV-D-719, IV-D-726, IV-D-768, IV-D-741, IV-D-621-14.10, IV-D-579, IV-D-738, IV-D-670) expressed concern about the cumulative effects of arsenic exposure. A person (IV-F-3.55) stated that to accurately gauge our exposure to arsenic,

EPA must include the historical and continuing accumulation of arsenic. However, two commenters indicated that arsenic does not accumulate (IV-D-621-15.6, IV-D-621-16.12).

Response:

Individuals residing in the vicinity of sources of airborne arsenic exposure, especially high arsenic copper smelters, may be at risk for increased intake because of the concomitant exposure to arsenic in the air, and arsenic deposited from the air onto soil and dust. Children may be more susceptible than adults. A Japanese study of arsenic poisoning of young children that had consumed arsenic-contaminated infant milk formula showed a number of indications of central nervous system involvement.[9] Follow-up studies showed significant cases of abnormal brain patterns, masked cognitive deficiencies, severe hearing loss and behavioral problems.

Unfortunately, no specific dose-response curves were developed either in the child poisoning studies or in the female smelter worker studies relating arsenic exposure to the manifestation of an effect. In the latter study no certainty was expressed that indeed airborne arsenic exposure caused the observed spontaneous abortion rate. Although indicative of a positive response to arsenic exposure, no extrapolation of an estimate of risk can be done with the data base. With respect to risk to children absorbing arsenic by inhaling playground dust, no inferences of risk can be made from the arsenic ingestion and poisoning studies of Japanese children. In addition, the mechanisms of inorganic arsenic deposition onto soil surfaces from smelter emissions and consequent adsorption onto soil and dust surfaces are not well understood. Given the extent of knowledge concerning deposition, transport and surface clearance of inorganic arsenic as it passes from the air media to soil and dust, EPA cannot accurately assess the cumulative effects of arsenic exposure nor was EPA able to assess the relative risk of these noncarcinogenic health responses. However, EPA in its decision making process is aware of the possible risk to sensitive individuals, and does consider this in conjunction with results of quantitative risk modeling.

Comment:

A few correspondents (IV-D-60,IV-D-76, IV-D-114) discussed the potential adverse health effects associated with the ingestion of arsenic through sources other than vegetables grown in local gardens such as ingestion of arsenic in seafood (IV-D-718, IV-D-739, IV-D-515, IV-D-530).

Comment:

One correspondent maintained that several populations ingest more arsenic in their drinking water than those who live near smelters will be exposed to, and that lifetime studies of those persons show no ill effects (this statement was documented with the following report: EPA 600/1-81-064).

Response:

As discussed in the previous response, the final risk assessment addresses only the inhalation of arsenic emitted by the smelter. The form of arsenic contained in seafood and its toxicological properties are different from the inorganic forms of arsenic regulated under the proposed air emissions standard. While shellfish and other marine foods have the highest arsenic level of any food category, the arsenic in marine species is stored in complex organoarsenical forms. Based on recent reports these forms are assimilated by man and rapidly excreted intact. They are not metabolized like the inorganic forms being regulated. Toxicologically, the organic forms of arsenic contained in seafood are relatively inert.

Comment:

A few people questioned the level at which arsenic presents a health hazard (IV-D-164, IV-D-267, IV-F-4.28, IV-F-5.15, IV-D-622, IV-D-756).

Comment:

Concern was also expressed about the health hazards of smelter emissions (IV-D-595, IV-D-110, IV-D-105, IV-D-83, IV-D-404, IV-D-375, IV-D-329, IV-D-592, IV-F-3.58, IV-F-3.70, IV-F-4.10, IV-F-4.28, IV-F-4.52, IV-F-5.3). One writer

(IV-D-110), with no specific reference to arsenic, described a disease involving a breakdown of the immune system which she attributed to the daily bombardment of toxic chemicals; she referred to an article which appeared in the L.A. Times concerning the ASARCO Tacoma smelter emissions. A second writer (IV-D-105) indicated that smelter workers will have an obvious health hazard. The populace within several miles will have a less obvious but real health loss. A third writer (IV-D-83) has been concerned about the smelter pollution and how it might affect the health of his family for years.

Response:

Clinical pathology reports of arsenic exposure have reported on the role of inorganic arsenic as an immunosuppressant in humans. This is evident in the use of arsenical medicinals in the treatment of steroid-responding disorders, and as a lymphocytostatic agent. Reports of chronic consumption of high arsenic contaminated drinking water supports the immunosuppressant role of arsenic. Chilean children exposed to the water displayed histories of chronic cough and bronchitis.[10] Other arsenic exposure studies have observed the occurrence of herpes simplex, and chronic pulmonary infections and this is evidence of arsenic as an immunosuppressant. Therefore, it is possible, although not yet clearly defined, that long-term exposure to airborne arsenic in the vicinity of copper smelters may contribute to disease patterns within the community. Further research in this area is needed to describe a possible association.

2.1.3 Teratogenicity/Reproductive Effects

A number of commenters expressed concern regarding the potential adverse effects of ASARCO emissions on the fetus (IV-D-4, IV-D-593, IV-D-604, IV-F-3.37, IV-F-3.41, IV-D-158, IV-F-3.42, IV-F-3.53, IV-F-3.57, IV-F-4.6, IV-F-4.11, IV-F-4.68, IV-F-5.7, IV-F-5.8). One correspondent (IV-D-593) referred to a statement made by Dr. Karle Mottet of the University of Washington at a meeting in Tacoma in which he indicated that arsenic may cause birth defects. Another correspondent (IV-D-604) questioned whether EPA was concerned about

potential birth defects. One writer (IV-D-158) stated that it is apparent that arsenic exposure cause increased incidence of birth defects and miscarriages. One individual (IV-F-5.7) stated that the more immediate risk of the involuntarily terminated pregnancy or even a retarded child is more threatening and of greater concern than the possibility of getting cancer in the more distant future. A student (IV-F-4.68) from the University of Berkely claimed that fetuses, newborns, and children are particularly vulnerable to the effects of arsenic toxicity. She submitted a paper that she wrote entitled ASARCO Arsenic Toxicity and the Public Health (IV-F-4.71).

Response:

Teratogenic effects of arsenic compounds have been observed in animal studies using a variety of species.[11] One study observed malformations in hamster fetuses following intravenous injection of sodium arsenate into the pregnant female on the eighth day of gestation. Exencephaly, encephaloceles, and skeletal defects were observed. Another hamster study of similar design observed embryos with a delay in neural fold elevation and neural tube closure with arsenate exposure. In experiments with mice, increased fetal resorption, decreased fetal weights, cleft lip, fork ribs and fused vertebrae were apparent following a single injection of sodium arsenate (intraperitoneally). Other animal studies have exhibited clear indications of fetal teratogenicity. Animal studies on the effects arsenic may have on postnatal growth and development have not observed any effect.

Swedish studies (Nordstrom and co-workers) of female smelter workers, and of females residing in the vicinity of smelters, have suggested an increase in the rate of spontaneous abortions resulting from exposure to smelter pollutants.[12] Female smelter workers showed a prevalence of spontaneous abortions which was 17 percent above what was expected. Women who worked directly on smelter processes showed a spontaneous abortion rate 28 percent higher than other female smelter workers. Women residing in the vicinity of the smelter displayed a spontaneous abortion rate 7 to 11 percent above the expected rate, with the highest rate in the area closest

to the smelter. However, many female smelter workers were reported to reside in this area.

However, Nordstrom et al. studies are flawed for a number of reasons. The Health Assessment Document cautions: "These studies (Nordstrom's) were not designed specifically to study effects of arsenic but rather to study the effects, in general, of the smelter work. While data from these studies suggest a low-level effect of smelter pollutants on neighboring (proximate) populations, the diverse agents involved preclude making conclusive statements about the specific effects of arsenic." In addition, unbeknownst to EPA at the time of the HAD publication, the Swedish National Health Board Expert Committee published a report in 1981 that questioned almost every finding in the Nordstrom studies. In the Administrator's judgment, the Nordstrom studies are not suitable for making determinations regarding the potential human reproductive effects caused by arsenic exposure. Therefore, it is not possible to relate arsenic exposure to the reproductive effects observed. The risk assessment methodology employed by EPA focused on the risk of respiratory cancer. However, EPA, in its overall evaluation of adverse health effects, will qualitatively regard other indications of arsenic exposure as well. Two health studies are being undertaken by the Washington State Department of Social and Health Services to assess the potential impact of smelter pollutants, especially arsenic. The study parameters will include incidences of reduced birth weight, and teratogenic effects (oral cleft) in areas affected by smelter emissions. These observations will be compared to areas remote from smelter emissions to determine the effects from ASARCO Tacoma smelter pollutants (see page 2-42).

2.1.4 Systemic Effects of Arsenic Emissions/Smelter Emissions

Comment:

One writer (IV-D-41) questioned why kidney damage, a "main effect of arsenic" exposure, was not being considered. Another writer (IV-D-404) claimed that both arsenic and cadmium are known to cause kidney failure in humans. One individual (IV-F-3.37) stated that arsenic accumulates in and is excreted from the kidneys.

Comment:

Four correspondents (IV-D-94, IV-D-430, IV-D-417, IV-D-592) addressed the impact of ASARCO emissions on asthmatics. One correspondent (IV-D-417) asserted that the dark cloud from ASARCO Tacoma has caused asthma sufferers increased agony.

Comment:

Many people also testified that they had respiratory problems associated with the ASARCO smelter emissions (IV-F-3.20, IV-F-3.24, IV-F-3.57, IV-F-4.10, IV-F-4.36, IV-F-4.52, IV-F-5.4, IV-F-5.10, IV-F-5.11, IV-F-5.16). One person (IV-F-5.10) testified that some people in the area (of the smelter) have respiratory and sinus problems. Another person (IV-F-5.4) stated that there are days when the odor in the air is so bad that they develop an asthmatic condition and cannot breathe. Another person (IV-F-5.11), asserted that as a youngster he lived in one of five closest houses to the ASARCO Tacoma smokestack and he recalled playing with extensive pain in his lungs.

Comment:

In contrast, six correspondents maintained that, although they had lived near and/or worked at ASARCO Tacoma for many years, they had never had any respiratory problems (IV-D-265, IV-D-233, IV-D-362, IV-D-391, IV-D-465, IV-D-215). One correspondent (IV-D-391) commented that his family had lived within one and one-half miles of the smelter for a combined total of over 800 years and not experienced any serious respiratory disorders.

Response:

Systemic effects other than cancer, resulting from chronic exposure to airborne inorganic arsenic have been noted in epidemiological studies. One study of smelter workers handling refined arsenic displayed nasal septum perforation and rhinopharyn-golaryngitis. Workers in roaster, furnace and convertor smelter processes showed tracheobronchitis and pulmonary insufficiency. Hepatic effects have been observed in arsenic ingestion studies

involving chronic exposure to high arsenic contaminated water or arsenical medicinals. These effects are cirrhosis and hypertension. Other observed chronic systemic effects are reversible anemia, and reduced hemoglobin production. Chronic renal effects related to arsenic ingestion or inhalation are not well characterized. Chilean children exposed to arsenic in drinking water showed a chronic cough and bronchitic history.

Chronic systemic effects other than cancer of either high level or low level inhalation exposure to airborne inorganic arsenic from copper smelters are not well understood or defined. Dose specific responses in the aforementioned studies were not reported. Therefore, a determination of increased risk of noncancerous systemic effects within the community affected by smelter pollutants cannot be evaluated at this time.

Comment:

Comments were submitted regarding tissue levels of arsenic in other community members (IV-F-3.67, IV-D-428, IV-D-418, IV-D-428, IV-D-604, IV-F-3.21). One writer (IV-D-428) maintained that he and his wife had blood tests which showed lead and arsenic contamination. Another writer stated that emissions from the smelter had poisoned the blood of three generations in the town of Ruston, Washington. One individual (IV-F-3.21) testified that his urine tested positive for an arsenic contamination level of 20 micrograms per liter. Arsenic tissue levels in animals was discussed. One correspondent (IV-D-164) indicated that a local butcher had noted that the livers of slaughtered animals were unusually spotted. One commenter (IV-F-3.37) referred to a television documentary, "Green Grow the Profits," in which arsenic was initially reported in the livers of poultry and later found in the white meat as well.

Response:

Urinary arsenic levels have been shown to increase when arsenic is inhaled. Arsenic may also be excreted via hair. The studies cited above provide additional evidence for EPA's assertion that the population is

being exposed through inhalation to arsenic emitted by the smelter. The urinary arsenic studies also indicate that exposure is highest near the plant. This is in agreement with both modeling and ambient monitoring results.

However, EPA does not routinely use tissue levels as a measure of public exposure to smelter emissions or lung cancer risks. The primary reason is that arsenic concentrations in tissue reflect many factors in addition to the inhalation of arsenic emitted by the smelter. Diet, in particular the consumption of seafood, can occur and result in increases or decreases in tissue levels. Individual metabolism can also cause variations in the amount of arsenic excreted. Individuals living in the same area from which tissue samples are taken on the same day may show a range of arsenic levels. Therefore, arsenic levels in tissue may not be good estimators for exposure to air emissions from ASARCO since other sources of exposure can contribute to arsenic concentrations uncovered in the tissues.

2.1.5 Dermal Effects of Arsenic Emissions/Smelter Emissions

Comment:

Some correspondents (IV-138, IV-D-247, IV-D-613, IV-D-622, IV-D-16 (IV-D-772)) discussed the dermal effects which they believed were associated with ASARCO Tacoma emissions. One writer attributed his wife's itching, welting rash to the handling of objects left outside which had accumulated dust emitted from the ASARCO smokestacks. Another writer (IV-D-217) who worked at ASARCO Tacoma for 36 years claimed that he suffered from skin irritation.

Comment:

Dermal effects connected with arsenic and/or employment at copper smelters were also discussed at the hearings. One individual (IV-F-4.27) who worked in some of the "worst areas" of the smelter stated that there were some skin irritations, but these were taken care of with no ill effects (IV-F-4.27). Another person (IV-F-3.18) commented that arsenic is a skin

irritant if not handled properly but that it would kill bacteria, and germs. Another individual (IV-F-4.60) testified that arsenic in significant or toxic levels, would produce skin pigmentation of certain areas of the skin, the neck, eyelids, nipples and arm pits and that the skin may be thickened in these areas.

Response:

Although there are no known cases of skin disorders resulting from arsenic inhalation in man, chronic oral exposure to arsenic induces a sequence of changes in skin epithelium, proceeding from hyperpigmentation to hyperkeratosis, characterized as keratin proliferation of a verrucose nature and leading, in some cases, to late onset skin cancers.[13] The U.S. EPA is presently examining this information, along with information from other studies, in order to determine whether quantitative does-response relationships, similar to those seen for skin cancer, can be established for these precancerous skin lesions. However, health effects other than lung cancer which could result from chronic low-level exposure to arsenic have not been sufficiently documented for EPA to quantitatively estimate or model. This kind of health risk is considered by EPA in a qualitative manner during the decision-making process.

2.1.6 Potential for Health Effects from the Ingestion of Arsenic Contaminated Vegetables

Comment:

According to one resident, the King (Seattle) and Pierce (Tacoma) County Health Departments distributed booklets which warned against the consumption of certain vegetables grown in local gardens because of cadmium, arsenic, and other heavy metals in the soil which had accumulated from smelter effluent (IV-D-11). Many of the comments attested to this warning from the County Health Departments (IV-D-9, IV-D-21, IV-D-32, IV-D-49, IV-D-76, IV-D-404, IV-D-375, IV-D-164, IV-D-292, IV-D-434, IV-D-428, IV-F-3.51, IV-F-3.53, IV-F-3.60, IV-F-4.15). Other individuals, with no specific reference to this warning, indicated that they would no longer grow and/or consume local vegetables because of soil contamination (IV-D-21, IV-D,#3, IV-D-605, IV-D-38, IV-D-47, IV-D-71, IV-D-91, IV-D-92, IV-D-100, IV-D-591, IV-D-158, IV-D-439, IV-F-3.37, IV-F-3.53, IV-F-4.52, IV-D-82, IV-D-104). One

correspondent (IV-D-74) stated that lives have already been altered by it (the ASARCO smelter), and that gardening plans and dietary routines had to be changed.

Comment:

Testimony was also given on changes that had to be made with respect to the consumption of local fruits and vegetables because of the ASARCO Tacoma smoke stack emissions. One individual (IV-F-5.4) testified that her fruit has to be peeled because it has a bad taste and tackiness that can't be washed off. Another person (IV-F-3.72) admitted giving up spinach, kale, chard, potatoes, carrots, and other vegetables because of ASARCO (IV-F-3.72).

Comment:

Many residents reported that the foliage in their gardens and yards were burnt or damaged in some fashion by smelter emissons (IV-D-23, IV-D-89, IV-D-94, IV-D-138, IV-D-417, IV-D-418, IV-D-425, IV-D-427, IV-F-3.24, IV-F-3.29, IV-F-3.38, IV-F-4.41, IV-F-5.13, IV-F-5.22).

Comment:

Comments were submitted in which concern was expressed about high arsenic levels in the vegetation and/or soil (IV-D-34, IV-D-90, IV-D-116, IV-D-138, IV-D-419, IV-D-364, IV-F-3.20, IV-F-3.21, IV-F-3.55, IV-F-3.57, IV-F-3.74, IV-F-4.10, IV-F-4.15, IV-F-5.8, IV-F-5.22, IV-D-718, IV-D-739, IV-D-515, IV-D-530, IV-D-576, IV-D-666, IV-D-676, IV-D-694, IV-D-584, IV-D-605).

Comment:

In contrast, numerous commenters claimed that their vegetables suffered no ill effects from the ASARCO Tacoma smelter emissions (IV-D-130, IV-D-139, IV-D-135, IV-D-475, IV-D-210, IV-D-372, IV-D-306, IV-D-365, IV-D-273, IV-D-599, IV-D-348, IV-D-479, IV-D-341, IV-D-253, IV-D-201, IV-D-218, IV-D-242, IV-D-249, IV-D-451, IV-D-270, IV-D-159, IV-D-176, IV-D-166, IV-D-282, IV-D-277, IV-D-352, IV-D-345, IV-D-327, IV-D-298, IV-D-487, IV-D-354, IV-D-355, IV-D-490, IV-D-477, IV-D-394, IV-D-379, IV-D-385, IV-D-473, IV-D-482, IV-D-601, IV-D-476, IV-D-265, IV-D-266, IV-D-377, IV-D-397, IV-D-472, IV-F-3.23, IV-F-3.49, IV-F-4.3, IV-F-4.5, IV-F-4.37, IV-F-4.49, IV-F-4.69, IV-F-5.5, IV-D-625-13, IV-D-725,

IV-D-784, IV-D-769, IV-D-699, IV-D-700, IV-D-705, IV-D-533, IV-D-539, IV-D-613, IV-D-623, IV-D-624). One writer (IV-D-135) observed that the vegetation in the "Point Defiance Park" next to the smelter was unaffected. Another writer (IV-D-451) maintained that mixtures of arsenic and flour or cereal spread on plants as a pesticide had been consumed by humans and livestock with no ill effects. Another writer (IV-D-345) indicated that family members had been eating from their gardens since 1909 and felt that no harm had come to any of them.

Comment:

One individual (IV-F-3.13) stated that it's quite obvious that any harmful effects would depend on the amount of a given vegetable eaten. Previous calculations have shown that it would be impossible for anyone to consume toxic amounts of any vegetable grown in the area.

Response:

The deposition of airborne inorganic arsenic emissions from inorganic arsenic sources onto the soil surface is of concern to EPA. The EPA is cooperating with various state agencies in a comprehensive study of smelter emissions from the ASARCO smelter to determine the routes of exposure responsible for the elevated urinary arsenic levels found in children residing near the smelter. Because the Clean Air Act limits the scope of exposure assessment to hazardous substances in the air, this study is directed under the authority of Superfund (Comprehensive Environmental Response, Compensation, and Liability Act, CERCLA). The multimedia approach will include exposure assessments of inhalation of arsenic in the air or in resuspended dust; ingestion of arsenic from vegetables, drinking water, and ingestion of soil by children. These exposure media will be sampled concurrently with urine, and statistical methods will be applied to determine which exposures have caused the elevated urinary arsenic levels and what remedial actions may be needed to reduce these exposures. Assessment will also be made of the potential health problems associated with lead and cadmium emissions from the smelter (see page 2-42).

2.1.7 Effects of Arsenic Emissions/Smelter Emissions on Other Biological Systems

Comment:

According to the submitted comments, bees and bottom fish are the types of wildlife most affected by the ASARCO Tacoma smelter emissions (IV-D-422, IV-D-418, IV-D-106, IV-D-107, IV-D-115, IV-D-404, IV-F-3.21, IV-F-3.57). One correspondent (IV-D-412) wrote that a Seattle daily newspaper reported scientific research linking arsenic emissions to widespread failure of beehives north of the plant and that bottom fish have been shown to have high levels of arsenic. Four references were made to Dr. J. Bromenshank's study on arsenic levels in bees ranging from "12 ppm at the South end of the Island (a world's record) to 2 ppm at the North end of the Island" (IV-D-9, IV-D-76, IV-F-3.53, IV-F-3.60). Dr. Bromenshank (IV-F-3.17) discussed his study at the hearings and stated that the (arsenic) levels are certainly high enough to equal or exceed those reported to be hazardous or lethal to honeybees and that arsenic typically acts as a stomach poison in insects. Dr. Bromenshank submitted data sheets from his study (IV-F-3.100--IV-F-3.103). Other concerns about the effects of arsenic on the bee population ranged from fatalities of bees due to arsenic contamination (IV-D-38, IV-D-90, IV-D-783, IV-D-705) to the residents' inability to consume local honey (IV-D-47).

Comment:

Comments were submitted regarding the effects of the ASARCO-Tacoma smokestack emissions on other animals (IV-D-581, IV-D-20, IV-D-434, IV-D-76, IV-D-599, IV-D-784, IV-D-137, IV-D-429). One correspondent (IV-D-20) questioned the effect of arsenic and other heavy metal emissions on the entire animal life chain. Another correspondent (IV-D-76) questioned the risks posed by the consumption of fish and shellfish from the area. A correspondent (IV-D-599) questioned the correlation between arsenic exposure and birth defects in fish and wildlife.

Comment:

In contrast, two correspondents (IV-D-218, IV-D-160) felt that the ASARCO-Tacoma emissions are not harmful to the wildlife. One correspondent maintained that there is no shortage of slugs, snails, grasshoppers, tent caterpillars, or gypsy moths around the smelter.

Response:

Section 112 of the Clean Air Act specifically requires the EPA Administrator to establish standards for hazardous airborne pollutants which provide an ample margin of safety to protect the public health. Therefore, consideration of ecological damage to aquatic organisms and other biota would be secondary to evaluation of direct human health effects under section 112 of the Clean Air Act. However, general ecological effects are being investigated under Superfund and other statutes to determine impacts of arsenic emissions from the smelter. Briefly summarized, these efforts include: (1) An assessment of the effects on aquatic life of contaminated discharges into Commencement Bay from the ASARCO smelter and other industries, and of sediments and water that are known to be contaminated; (2) A determination of whether or not additional studies are warranted under Superfund to investigate adverse effects of smelter emissions on other plant and animal life. These studies may include samples of tissue levels of arsenic in livestock (see page 2-42).

2.1.8 ASARCO-Tacoma Smelter Emissions/Arsenic Not a Health Hazard

Several people opposed the proposed standard and/or shutdown of the ASARCO-Tacoma smelter, claiming that the smelter was not a health hazard (IV-D-62116.12, IV-D-621-14.9, IV-D-621-5, IV-D-621-6, IV-D-509, IV-V-568, IV-D-14, IV-D-621-14.17, IV-D-621-14.7, IV-D-621-6.1, IV-D-621-14.2, IV-D-525, IV-D-536, IV-D-621-14.14, IV-D-621-15.2, IV-D-6621-15.6, IV-D-621-15.9, IV-F-3.15, IV-D-547, IV-D-760, IV-D-695, IV-D-323, IV-D-337, IV-D-343). Comments were received from those who had lived near the smelter, who were employed by the smelter, and who both lived by and worked for the smelter. Comments were also submitted by those who, although they made no mention of living near or working for ASARCO-Tacoma, felt that the plant didn't pose a health hazard.

Comment:

Many comments were submitted by individuals who lived in the vicinity of ASARCO (IV-D-262, IV-D-222, IV-D-146, IV-D-184, IV-D-242, IV-D-605 IV-D-367, IV-D-354, IV-D-298, IV-D-352, IV-D-377, IV-D-327, IV-D-369, IV-D-66, IV-D-479, IV-D-341, IV-D-472, IV-D-469, IV-D-470, IV-D-508, IV-D-471, IV-D-176, IV-D-394, IV-D-655, IV-D-360, IV-D-372, IV-D-391, IV-D-211, IV-D-318, IV-F-3.22 IV-F-3.25, IV-F-3.26, IV-F-3.27, IV-F-3.35, IV-F-3.39, IV-F-3.45, IV-F-3.50, IV-F-3.52, IV-F-4.3, IV-F-4.7, IV-F-4.12, IV-F-4.30, IV-F-4.33, IV-F-4.44, IV-F-4.53, IV-D-280, IV-D-315, IV-D-393, IV-D-517, IV-D-525, IV-D-532, IV-D-533, IV-D-534, IV-D-539, IV-D-134, IV-D-544, IV-D-547, IV-D-548, IV-D-552, IV-D-615, IV-D-623, IV-D-624, IV-D-633, IV-D-636, IV-D-659). One writer (IV-D-66) stated that having lived in Tacoma 55 out of 66 years, he found no evidence of ill health from arsenic emissions including the health of his mother, age 103, who had lived in the vicinity longer. Another writer (IV-D-176) indicated that he had lived within a few miles of the smelter since 1937 and had suffered no ill effects. Another writer (IV-D-360) asserted that her family had lived in the five mile radius of the smelter for almost thirty years, that she raised four children in that area, and that her children were healthy adults beginning to have children of their own.

Comment:

Testimony was also given by those who lived near ASARCO-Tacoma. One person (IV-F-4.5) testified that he had five neighbors who lived in the area of the smelter for over 60 and 70 years and hadn't complained of any ill effects (IV-F-4.5). Another individual (IV-F-4.32) lived within 7 blocks of the smelter for the last 57 years and had not suffered any health effects from the smelter nor had his children who had attended a school 3 blocks south of the plant. One woman (IV-F-4.49) who also lived 7 blocks from the smelter felt that the stress from making a living was more of a health hazard than the arsenic from the plant. She also stated that studies conducted in the U.S. and Sweden indicate no increased illness or mortality associated with community exposure to smelters emissions and no increased rate of lung cancer has been observed among persons exposed to ASARCO-Tacoma emissions.

Comment:

Numerous commenters discussed the lack of a health hazard associated with their past and/or present employment with ASARCO (IV-D-182, IV-D-197, IV-D-208, IV-D-212, IV-D-218, IV-D-220, IV-D-221, IV-D-166, IV-D-225, IV-D-226, IV-D-229, IV-D-159, IV-D-268, IV-D-223, IV-D-387, IV-D-504, IV-D-486, IV-D-490, IV-D-656, IV-D-506, IV-D-385, IV-D-311, IV-D-289, IV-D-293, IV-D-299, IV-F-3.59, IV-F-4.8, IV-F-4.40, IV-F-4.42, IV-F-4.58, IV-D-285, IV-D-350, IV-D-512,IV-D-518, IV-D-522, IV-D-532, IV -D-347, IV-D-544, IV-D-547, IV-D-558, IV-D-562, IV-D-613, IV-D-623, IV-D-636, IV-D-647, IV-D-563). One correspondent (IV-D-221) submitted that he has been working for the Tacoma smelter for 24 and a half years, that he carried arsenic every day for 18 years without a mask, and that he is now 75 years old and in the best of health. Another writer (IV-D-486) commented that during fourteen years of employment at the Tacoma smelter, he had suffered no ill effects.

In his testimony one retiree (IV-F-4.13) indicated that he had worked at the plant for 32 years at various jobs and never felt sick throughout his employment. Another person (IV-F-4.45) employed by ASARCO for 14 years claimed that "in one week I breathed, inhaled and ingested more arsenic powder than local residents would in 50 years and I can say that there have been no ill effects to me". Another person (IV-F-5.6) testified that during his approximately thirty years of employment with ASARCO, he and his coworkers were exposed to arsenic dust for "hours on end" and that they are all in fairly good health.

Comment:

Thirty individuals based their opinions of the adverse health effects caused by the smelter emissions on their experiences while both living near and working at the plant (IV-D-199, IV-D-249, IV-D-236, IV-D-233, IV-D-202, IV-D-165, IV-D-397, IV-D-482, IV-D-473, IV-D-492, IV-D-453, IV-D-379, IV-D-348, IV-D-407, IV-D-306, IV-D-364, IV-D-335, IV-D-287, IV-D-196, IV-D-297, IV-D-334, IV-F-3.18, IV-F-3.47, IV-F-4.17, IV-F-4.29, IV-F-4.36, IV-F-4.37, IV-F-4.64, IV-F-5.17, IV-F-5.19, IV-F-5.5). One correspondent (IV-D-407) wrote: "My home has been within 1/2 mile of the smelter for 65

years. My husband has worked there for 44 years. My son worked there for 6 months. My father worked there also. None of these men including myself have had any ill effects from the smelter". Another correspondent (IV-D-199) maintained that he had lived and worked in the Tacoma area for 31 years and he felt that the ASARCO-Tacoma smelter emissions had no ill effects on anyone's health in that period of time.

Comment:

Several people generally felt that the ASARCO smelter posed no health hazards (IV-D-304, IV-D-266, IV-D-250, IV-D-253, IV-D-160, IV-D-326, IV-D-460, IV-D-272, IV-D-505, IV-D-279, IV-D-291, IV-D-320, IV-D-474, IV-D-371, IV-D-399, IV-D-373, IV-D-321, IV-D-331, IV-D-339, IV-D-370, IV-D-276, IV-D-72, IV-D-204, IV-D-227, IV-D-269, IV-D-406, IV-D-450). One correspondent (IV-D-276) submitted that to date, no definite health problems have been proven that will and do exist on current emissions from the plant. Another correspondent (IV-D-460) said: "Since the EPA cannot show that the emissions from ASARCO-Tacoma are harmful to this community, it would seem prudent to me for you to drop your case".

Comment:

Testimony was given by one individual (IV-F-3.28) in which he referred to arsenic's medicinal uses: "In medicine it is used in treatment of anemia to build up red corpuscles of the blood and hemoglobin content. It has a tonic effect on the general nervous system and it is also considered by many authorities to have antiperiodic action, as in malaria. It is known to be effective in various chronic skin diseases. It is used in the treatment of certain forms of dyspepsia, Hodgkin's disease, neuralgia, rheumatoid arthritis, chorea, asthma, hay fever, psoriasis, pemphigus, occasionally in chronic eczema, tuberculosis, diabetes, leprosy, and syphilis".

Response:

A clear absence of adverse health effects, especially lung cancer, has not been demonstrated by various community health studies. Although subject to several shortcomings, several national community studies have indicated

an increase risk of lung cancer in people residing near smelters.[14] (See the summary of the community studies on page 2-9.) Other studies have not demonstrated an excess of lung cancer mortality in communities surrounding smelters (Rom et al. (1982); Lyon et al. (1977); Frost et al. (1983)). Due to the inherent problems with such studies and the inconsistent findings that they have produced, the community studies have not produced a clear understanding of the nature and magnitude of public risk near arsenic sources.

However, uncertain results and negative observations may not be construed as an absence of risk to the public in view of the strong epidemiological association between inorganic arsenic and lung cancer in smelter workers. The EPA is taking the prudent action of reducing the risk of lung cancer resulting from chronic community exposure to airborne arsenic emissions from smelters. The Regulatory Council (an inter-governmental agency cancer policy work group) has observed:

> "The failure of an epidemiological study to detect an association between the occurrence of cancer and exposure to a specific substance should not be taken to indicate necessarily that the substance is not carcinogenic.
>
> Because it is unacceptable to allow exposure to potential carcinogens to continue until human cancer actually occurs, regulatory agencies should not wait for epidemiological evidence before taking action to limit human exposure to chemicals considered to be carcinogenic."[15]

2.1.9 Multiple Chemical Exposure: Synergistic/Additive Effects

Comment:

Several comments were submitted concerning the synergistic/additive effects of exposure to multiple substances (IV-D-114, IV-D-322, CC, IV-F-3.37, IV-F-3.55, IV-F-4.43, IV-F-4.50, IV-D-416, IV-D-438, IV-D-35, IV-D-6, IV-D-718, IV-D-719, IV-D-710, IV-D-427, IV-D-541, IV-D-670, IV-D-57). One writer stated that due to multiple contaminants from ASARCO-Tacoma smelter,

single substance studies of health effects are inadequate. Another writer (IV-D-114) asked to what degree do (arsenic, cadmium, sulfur dioxide, lead, etc.) interact with each other and with other industrial substances to create additional toxicity? Another writer felt that "by proposing an arsenic standard separate from other pollutants coming from the ASARCO smelter, the problem is divided into many segments. Each of them is less dangerous than the sum" (IV-D-593). "One person (IV-F-3.7) cited the conclusions of a study by Lee and Fraumeni and testified: "We know that the ASARCO smelter emits both the sulfur dioxide and the arsenic trioxide, which could mean that a synergistic effect is already in place in those for us who live downwind from the emissions." Another individual (IV-F-4.31) stated that arsenic probably becomes more toxic when it acts synergistically with other substances so that the total exposure is greater than the sum of the individual levels of pollution.

Response:

The EPA realizes there may exist a concomitant risk associated with exposure to air pollutants from smelters. The Agency believes that consideration of all environmental concerns associated with smelter emissions is a necessary and important element in the risk management process. Consequently EPA considered the impact of the proposed standard on emissions of other pollutants, and the actions being taken under other environmental statutes to address other environmental impacts of the smelter. The emission of cadmium, lead, and antimony, for example, present in particulate matter will also be controlled under the proposed arsenic standard.

The risk associated with SO_2 exposure have been statistically isolated from risks associated with arsenic exposure. The data indicate that SO_2 exposure does not explain the excess lung cancer rates observed. Also, indications of excess lung cancers have been found in occupational settings other than primary copper smelters where concomitant exposure to SO_2 and other trace metals would not occur. The arsenic potency estimates (unit risk estimates) for both types of occupational settings are approximately of the same magnitude. These observations lead EPA to believe that excess lung cancer risks are associated only with arsenic exposure.

Although the Agency is aware that risk associated with exposure other than inorganic arsenic inhalation may occur, the available data are mostly inadequate as a basis for the Agency to produce meaningful additional analyses. However, EPA and other Agencies are conducting or planning to conduct further studies in and around Tacoma, Washington, to enhance the available data base and to provide more insight as to the nature of other routes of exposure and corresponding public risk. Such studies and other activities are summarized below. These studies should provide useful information on such impacts for all the smelters, although EPA realizes that the ASARCO-Tacoma facility was smelting rather unique kinds of feed material.

(1) Superfund Activities

Elevated levels of arsenic have been found in the hair and urine of residents living near the ASARCO-Tacoma smelter. Additionally, concentrations of arsenic are substantially above background in various environmental media, including soil, air, household dust, and vegetation. The Superfund law (Comprehensive Environmental Response, Compensation, and Liability Act, CERCLA) is being used to address this multimedia arsenic contamination. Superfund will also be used to evaluate potential problems from cadmium which has been found in elevated levels in garden soil and vegetables near the smelter.

Unlike most other environmental laws, Superfund can be used to correct problems resulting from past practices and spanning all environmental media. Based in part upon the elevated levels of arsenic in environmental media and in urine samples of residents near the ASARCO smelter, a segment of the Commencement Bay area (part of Commencement Bay and adjacent lands) was designated as a Superfund (National Priority List) site in 1980. This site is known as the Commencement Bay Near-Shore Tideflats Superfund site, and includes parts of Tacoma/Ruston/Vashon Island, the Commencement Bay Tideflats area, and the water adjacent to these areas.

On May 2, 1983, EPA and the Washington Department of Ecology (WDOE) signed a Cooperative Agreement making WDOE the lead agency in investigating this Superfund site (with funds provided by the EPA Superfund program and matching funds from the State) and in ensuring that needed remedial actions are taken. This Cooperative Agreement is divided into two tasks, the Ruston-Vashon Task and the Nearshore-Tideflats Task.

Ruston-Vashon Task-Investigations under this Task are focusing upon the issues specifically related to the ASARCO smelter. An exposure assessment study designed by the University of Washington (with assistance from the Centers for Disease Control, WDOE, the State and local health agencies, EPA and the Puget Sound Air Pollution Control Agency) began in January of 1985. The purpose of this study is to determine the routes of exposure responsible for the elevated urinary arsenic levels found in children living near the smelter. Since these exposure routes may include inhalation of arsenic in air and in resuspended dust, ingestion of arsenic from vegetables and drinking water and ingestion of soil and dust by children, several of these media will be sampled concurrently with urine. Statistical methods will then be used to determine which exposures are responsible for the elevated urinary arsenic levels, providing information on the remedial actions that may be needed to reduce these exposures.

Peripheral neuropathies (damage to nerves in the periphery of the body, such as those in the arms or legs) have been found in persons exposed to high levels of inorganic arsenic. Additionally, laboratory experiments have shown that high levels of arsenic can affect the synthesis of hemoglobin in exposed animals, resulting in higher than normal levels of uroporphyrins in the urine. The investigation conducted by the University of Washington includes urinary porphyrin analyses and peripheral neuropathy testing to provide preliminary data on the effects of arsenic in the smelter community.

Work is also being done as part of the Ruston-Vashon Superfund effort to assess the potential exposures resulting from cadmium emissions from the smelter. Cadmium levels above background have been found in the soil

and vegetation near the ASARCO smelter, prompting the local health agencies to suggest that the growth of certain vegetables (e.g., leafy) be discontinued. Existing data on cadmium levels in garden soil and vegetables are now being reviewed, and additional data will be collected, if necessary, to assess what health problems, if any, may result from the levels of cadmium now in the soil.

Several commenters expressed concern that ASARCO was damaging plant and animal life in the vicinity of the smelter. No studies have been done on the effects of these emissions, except for the pollutant sulfur dioxide (SO_2). The SO_2 plant studies done show sharp contrasts in opinion and reflect conditions existing approximately ten years ago. However, analyses of livestock tissue for levels of arsenic and other metals are being considered under the Ruston/Vashon Superfund Task.

Nearshore-Tideflats Task--Contamination of aquatic life in Commencement Bay and the possible effects of this contamination on consumers of seafood have been investigated in previous studies by NOAA (National Oceanic and Atmospheric Administration), Tacoma-Pierce County Health Department (TPCHD) and EPA. A NOAA report issued in 1980 reported the presence of tumors in fish caught in Commencement Bay and higher than background metals levels in limited areas (e.g., near ASARCO and other industries). As a follow-up to this study, EPA analyzed additional samples of aquatic life from the Bay in 1982. Using these data, the TPCHD concluded that there did not appear to be short-term or long-term health risk from consumption of fish caught in the Bay (except in Hylebos Waterway). TPCHD recommended, however, that more data be developed for contaminants in fish at the Point Defiance dock (near ASARCO) as well as at other areas in the Bay.

These additional data are being collected as a part of the Superfund investigations under the Nearshore-Tideflats Task of the EPA/WDOE Cooperative Agreement. Under this Task, WDOE is analyzing the levels of contaminants in Commencement Bay sediment and aquatic life and is

investigating the sources of pollution that are responsible for these contaminants. The effects of these contaminants on aquatic life and the risk to consumers of eating seafood from the Bay are also being assessed. In response to the initial results of this Superfund study, the TPCHD has modified their previous advisory. They now recommend that individuals not consume bottom fish or crab caught from the Commencement Bay Waterways and limit consumption of fish and crabs caught in other areas of the bay. Upon completion of the Superfund investigations, remedial actions will be designed to control the discharge of contaminants to the Bay or remove existing contaminants of concern (e.g., by removal of sediment).

(2) Non-Superfund Activities

Health Related--Two health related studies being conducted by the Washington State Department of Social and Health Services (DSHS) to assess potential impacts from smelter emissions are nearing completion. In these studies, the incidences of reduced birth weight and oral cleft (a birth defect that is easily detected) were compiled in areas near the smelter and in control areas where exposures to smelter emissions are minimal. The incidences in the two areas will then be compared in an attempt to assess the smelter effects.

Dr. Tom Burbacher of the University of Washington is determining the levels of arsenic in stillbirths and in newborns who have died soon after birth. Samples of placenta from women living close to the smelter may also be analyzed if funding is available. These studies will provide information on the levels of arsenic in these various tissues and of the potential for arsenic to be transferred to the fetus during development.

Lead Emissions - The Clean Air Act directs the Administrator to establish air quality criteria and to propose and promulgate primary and secondary National Ambient Air Quality Standards (NAAQS) for air pollutants emitted from numerous and diverse sources that may reasonably be anticipated to endanger public health or welfare. Primary standards are to be set at a level which, in the judgment of the Administrator is required to protect public health with an adequate margin of safety. Secondary standards must specify a level of air quality which, in the judgment of the Administrator and

based on the air quality criteria, is required to protect public welfare from any known or anticipated adverse effects. In 1978, EPA established the primary and secondary standards for lead at a level of 1.5 micrograms per cubic meter (maximum arithmetic mean) averaged over a calendar quarter. The control programs to meet the NAAQS are embodied in the State implementation plans (SIPs) which are developed by the State and local air agencies. Lead is one of six pollutants for which the Agency has developed a NAAQS.

The SIP that WDOE developed for lead in Washington has recently been approved by EPA. Existing monitoring results suggested that the area around the ASARCO smelter was meeting the NAAQS. However, to verify these monitoring results PSAPCA and Region 10 EPA utilized smelter lead emissions data in a dispersion model to estimate the expected maximum ambient lead concentrations around the smelter. The results of this modeling showed that lead emissions from the smelter would not violate the NAAQS even at full operating capacity if ASARCO installed the controls required to reduce the emissions of other pollutants.

In 1972, Dr. Sam Milham of DSHS studied the levels of lead in blood and of blood enzymes expected to be affected by lead in children living near the ASARCO smelter. These studies showed values within normal limits for these children. However, to ensure that excessive lead exposure is not occurring in children in the Tacoma area as a result of previous emissions of lead from ASARCO or other environmental sources of lead, additional testing may be done in the future by the state or local health agencies.

Water/Solid Waste--Prior to ASARCO's decision to close its copper smelter WDOE was reviewing ASARCO's NPDES permit (National Pollutant Discharge Elimination System Permit) to determine what limits should be included in this permit to control the discharge of arsenic and other potentially hazardous pollutants into Commencement Bay. Final modifications to this permit will be made after copper smelting has stopped and the environmental impacts from remaining activities (e.g. the arsenic plant) can be assessed. WDOE will require ASARCO to determine which sources

of pollution have led to the environmental damage in the Bay off-shore of ASARCO. Pollution resulting from run-off of contaminated water from the smelting facility and from movement of pollutants through groundwater on site are both being investigated and may need to be controlled.

Prior to the closure announcement several actions were also being taken by WDOE and PSAPCA to deal with the environmental problems that may result from the use and disposal of ASARCO slag. The potential for emissions of arsenic into air and water at the smelter during the slag cooling process is no longer an issue because of closure. Since slag will no longer be produced, concerns regarding its use as sand-blasting material have also decreased. However, ASARCO as well as several log sort yards in the area have used ASARCO slag as fill material in the past. Because mobilization of the metals from slag into the Bay area from these fill areas is occurring, more extensive studies are being done and WDOE will be working with ASARCO and the owners of these yards to develop remedial actions (e.g. diversion of storm-water from the yards) that can mitigate this mobilization.

Honey Bees--Preliminary results from research done by Dr. Jerry Bromenshenk in 1983 on honey bees in the Puget Sound area show elevated levels of arsenic and cadmium in bees in the ASARCO smelter area. Analysis of these data suggests that at least for arsenic, and possibly cadmium, the source of this bee contamination may be current or past emissions from the ASARCO smelter. Dr. Bromenshenk's brood survival results as well as reports from beekeepers in the smelter area suggest that honey bee survival may be affected by these contaminants, although these data must be further substantiated. Dr. Bromenshenk collected additional data in Puget Sound during 1984 using EPA research money. The results of his study should be available by the summer of 1985.

2.2 RISK ASSESSMENT

2.2.1 Evidence for the Existence of a Threshold For Arsenic

Several commenters criticized the model used by EPA in the development of the risk assessment. Criticism focused on the use of a linear non-threshold

model. In addition flaws were pointed out in the epidemiology studies used by EPA in the development of the unit risk estimate.

Comment:

Several commenters (IV-D-621-14.15, IV-D-621-14.17, IV-D-621-16.12, IV-3-621-14.7, IV-F-3.3, IV-F-3.6, IV-F-3.9, IV-F-3.11, IV-F-3.15, IV-F-5.11, IV-F-1.3, IV-F-3.39, IV-F-3.52, IV-D-294, IV-D-611) stated that a threshold for arsenic existed below which exposure to arsenic did not pose a risk to human health or that the risk was not substantial.

Comment:

One commenter (IV-F-3.3) said that even though EPA has taken the point of view that there is no acceptable amount based on the continuation of the line from the data that's available back down to zero exposure, that there probably is a threshold.

Comment:

Other commenters (IV-F-1.6, IV-F-3.15, IV-F-5.11) addressed a specific level at which the threshold exists and cited the study by Higgins as evidence. One commenter (IV-D-611) cited findings of no excess lung cancer mortality among smelter workers in a Swedish plant at levels above 200 $\mu g/m^3$ as support for the findings of Higgins Anaconda study. Another commenter (IV-F-1.6) stated that there is no evidence of increased risk to people who have exposures below 500 $\mu g/m^3$ based on studies of smelter workers.

Comment:

Others (IV-D-754, IV-D-708, IV-D-617, IV-D-747, IV-D-427, IV-D-530, IV-D-580, IV-D-673) commented that there is no threshold for arsenic and generally supported the no threshold presumption regarding dose-response relationship for human exposure to arsenic.

Comment:

A commenter (IV-F-3.11) stated that theoretical predictions suggest that if arsenic is a carcinogen, then it acts at some epigenetic site and that it is now widely considered that epigenetic carcinogens probably do have a threshold. No evidence was cited.

Comment:

Another commenter stated that arsenic is not a genotoxic substance in in-vitro tests and it has not been shown to be carcinogenic in animals despite numerous attempts. Therefore, there is no basis in fact for the application of a linear non-threshold model. Other commenters (IV-D-621-14.11, IV-D-16) also remarked that arsenic does not act on DNA.

Response: The Non-Threshold Hypothesis

In evaluating the public health hazards associated with exposure to inorganic arsenic, EPA has maintained that in the absence of sound scientific evidence to the contrary, such substances must be considered to pose some finite risk of cancer at any exposure level above zero. Support for the non-threshold hypothesis for carcinogenic substances is derived from sound scientific judgment. For the most part substantiation of the non-threshold hypothesis can be found in policy set forth by the Occupational Health and Safety Administration (OSHA),[16] the Consumer Product Safety Commission (CPSC), the Food and Drug Administration (FDA), the Food Safety and Quality Service, the President's Regulatory Council,[17] and the National Academy of Science.[18]

Epidemiological data support a strong association between chronic exposure to airborne arsenic and lung cancer in humans. In the absence of clear evidence to the contrary, EPA has assumed that if a carcinogenic response occurs at dose levels or exposure levels in a study, then responses at all lower doses will occur at a rate that can be determined by an appropriate extrapolation model.

Some commenters have challenged this position by asserting that certain studies have demonstrated no carcinogenic effect below a certain level.

The threshold argument contends that there exist doses of carcinogens that are so low that they will not cause cancer when human populations are exposed.

It remains EPA's belief, however, that not enough is known about the true mechanisms of initiating carcinoma in human cells and, at present, such mechanisms can only be postulated. Unlike most clastogenic agents, arsenic does not appear to directly damage DNA. However, arsenic does seem to have a genetic effect through some interference with DNA synthesis. Nordenson et al.[19] and Crossen[20] have observed that arsenic induces chromosomal aberrations and sister chromatid exchange (SCE) only when it is present during DNA replication. In addition, arsenic has been known to be a sulfhydryl reagent, and as such it can exhibit a number of thiol-dependent enzyme systems.[21]

Therefore another possible mechanism of carcinogenesis for arsenic is the inhibition of DNA repair enzymes. Another possible mechanism for the action of arsenic is that it may replace phosphorus within the backbone of DNA. This may be one reason arsenic is clastogenic. At present there is no single, well founded explanation describing how arsenic breaks chromosomes or induces SCE. Given this evidence of interference with DNA synthesis, especially chromosomal abberrations, SCE, and inhibition of DNA repair systems, it is not realistic to presume a level of arsenic in the environment that will have a zero effect on the exposed population. Genetic diversity and individual differences in the body's capability to defend itself against the metabolic intrusion of foreign substances greatly discounts the likelihood of a level of exposure of a carcinogen that will not result in an adverse health effect. The most extensive information on carcinogenesis is with ionizing radiation, and certain comparisons can be made with respect to some experimental evidence in animal bio-assays implicating thresholds in some animal tissues, but for the most part thresholds have not been established for most tissues.

The National Research Council of the National Academy of Sciences has noted:

> "If an effect can be caused by a single hit, a single molecule, or a single unit of exposure, then the effect in question cannot

have a threshold in the dose-response relationship, no matter how unlikely it is that the single hit or event will produce the effect (cancer). Mutations in prokaryotic and eukaryotic cells can be caused by a single cluster of ion pairs which were produced by a single beam of ionizing radiation. We would expect that mutations can be caused by a single molecule or perhaps group of molecules in proximity to the DNA. The necessary conclusion from this result is that the dose-response relationship for radiation and chemical mutagenesis cannot have a threshold and must be linear, at least at low doses."[22]

Occupational exposure studies have demonstrated a strong association between chronic exposure to airborne inorganic arsenic and lung cancer.[23] Over 10000 smelter workers have been retrospectively studied spanning the latency period of carcinogenesis. The results are that 11 of the 12 published epidemiological reports of smelter workers in the U.S., Sweden and Japan have shown a 2-fold to 12-fold increase in lung cancer mortality above the expected rate. The increase in lung cancer mortality is evident even when exposure to other pollutants in the workplace was accounted for, i.e., cigarette smoke, sulfur dioxide.

Commenters have contended that because mutagenesis has not been clearly established, and carcinogenesis has not been clearly demonstrated in animal studies despite varying doses and varying animal species, the assertion of a direct acting mechanism of arsenic is unfounded. These commenters go on to suggest an epigenetic mechanism, or possible promoting effect of inorganic arsenic. They offer such evidence as substantiation for a level of exposure in the community that could be tolerated, and that would not result in cancer. The evidence, however, of smelter worker studies showing a positive carcinogenic association to inorganic arsenic transcends the lack of animal evidence. The Regulatory Council considers properly conducted epidemiologic studies that show a statistically significant association between human exposure to a substance and increased risk to cancer as good presumptive evidence that the substance is carcinogenic.[24] Known carcinogens are those

substances associated with cancer in humans. Because the present state of scientific awareness on the mechanisms of cancer are largely theoretical, and are the subject of ongoing research, it is appropriate and prudent that EPA not accept the argument of the existence of a threshold for human exposure to inorganic arsenic until sound evidence in support of thresholds for chemical carcinogens is presented. The NAS has further elaborated:

> "The human population in the United States - the population we are trying to protect - is a large, diverse, and genetically heterogeneous group exposed to a variety of toxic agents. Genetic variability to carcinogenesis is well-documented, and it is also known that individuals who are deficient in immunological competence (for genetic or environmental reasons) are particularly susceptible to some forms of cancer. It seems, therefore, that even if we were to postulate an average threshold for a particular cancer induced by a particular agent, we would in practice need a series of thresholds for different individuals. It would be difficult, in practice, to establish a single threshold.
>
> We (National Academy of Science) conclude from these arguments that, despite all the complexities of chemical carcinogenesis, thresholds in the dose-response relationships do not appear to exist for direct-acting carcinogens. If they do exist, they are unlikely to be detected and hence, impossible to use. This means that there can be no totally "safe" exposure to a particular carcinogen, nor can the term "margin of safety" have any meaning. Any dose of a carcinogen must be considered to be associated with a risk, even if that risk is vanishingly small; estimates must be made of that risk."[25]

2.2.2 The Linear, Non-Theshold Dose/Response Model

Comments were generally critical of the use by EPA's Carcinogen Assessment Group (CAG) of a linear, non-threshold model to derive an arsenic unit risk factor. These commenters (IV-F-3.12, IV-F-3.15, IV-D-189, IV-D-711, IV-D-568, IV-D-640, IV-D-625, IV-D-621-7.1, IV-D-621-15.2, IV-F-1.6, IV-D-617, IV-D-618, OAQPS 79-8, IV-D-27) viewed the model as extremely conservative and

"deliberately designed to lead to a rough upper limit of risk that could be considerably lower". Another commenter (IV-D-621-7.1) claimed that a zero intercept linear absolute model may be an unsatisfactory representation of the relationship between exposure and disease.

Comment:

Other commenters approved of EPA's method of deriving unit risk estimates using the linear non-threshold model. One commenter (IV-D-708) noted that use of a model which overestimates risk is consistent with public health policy although the unit risk estimate is likely to be upperbound.

Response:

While EPA agrees that the linear, non-threshold model is conservative in nature and would tend to provide a plausible upper bound to the risk range, the Agency does not believe that the assumptions upon which it is based or that the results of its use are unreasonable. The dose response model with linearity at low dose was adopted for low dose extrapolation by EPA because at the time of its introduction,
it had the best, albeit limited, scientific basis of any current mathematical extrapolation model.[26] The EPA described this basis most recently in a Federal Register notice announcing the availability of Water Quality Criteria Documents:[27]

> "There is really no scientific basis for any mathematical extrapolation model which relates carcinogen exposure to cancer risks at the extremely low levels of concentration that must be delt with in evaluating the environmental hazards. For practical reasons, such low levels of risk cannot be measured directly either using animal experiments or epidemiologic studies. We must, therefore, depend on our current understanding of the mechanisms of carcinogenesis for guidance as to which risk model to use. At the present time, the dominant view of the carcinogenic process involves the concept that most agents which cause cancer also cause irreversible damage to DNA. This position is reflected by the fact that a very large proportion of agents which

cause cancer are also mutagenic. There is reason to expect that the quantal type of biological response that is characteristic of mutagenesis is associated with a linear non-threshold dose-response relationship. Indeed, there is substantial evidence from mutagenesis studies with both ionizing radiation and with a wide variety of chemicals that this type of dose-response model is the appropriate one to use. This is particularly true at the lower end of the dose-response curve; at higher doses, there can be upward curvature, probably reflecting the effects of multistage processes on the mutagenic response. The linear non-threshold dose-response relationship is also consistent with the relatively few epidemiological studies of cancer responses to specific agents that contain enough information to make the evaluation possible (e.g., radiation-induced leukemia, breast and thyroid cancer, skin cancer induced by aflatoxin in the diet). There is also some evidence from animal experiments that is consistent with the linear non-threshold hypothesis (e.g., liver tumors induced in mice by 2-acetylaminofluorene in the large scale ED_{01} study at the National Center for Toxicological Research, and initiation stage of the two-stage carcinogenesis model in the rat liver and mouse skin)."

2.3 EPIDEMIOLOGIC STUDIES

2.3.1 Critique of Epidemiologic Studies

Several commenters (about 10) focused on flaws present in the epidemiological studies chosen by EPA for the determination of the unit risk estimate for lung cancer due to airborne exposure to arsenic. The comments generally focused on the studies by Lee-Feldstein (1983), Higgins, (1982), Enterline and Marsh (1982) and Brown and Chu (1983). An overview of the major criticisms is presented separately for each study.

Critisims of the Lee-Feldstein Study

Comments (IV-D-711, IV-D-640, IV-F-3.15, IV-F-1.6) were received which questioned the use of data from the 1983 Lee-Feldstein follow-up of Anaconda smelter workers. One commenter stated that the data show poor fit for any combination of data or models chosen. The EPA was criticized for incorporating

only medium and light exposures from this study in order to fit the linear no threshold model. Comments also claim that Lee-Feldstein did not use an appropriate exposure classification so that exposure groups overlapped resulting in the likelihood that someone with an exposure of 1000 $\mu g/m^3$ could be in the heavy, medium or light exposure group. One commenter (IV-D-708) stated that the lack of fit of the Lee-Fieldstein data is due to the method of characterizing exposure rather than any inherent deviation from linearity.

Response:

The Lee-Feldstein Study (1983) has a number of features which support its use in making quantitative risk estimates of lung cancer from exposure to airborne arsenic.[28] It was a large study involving a relatively large number of respiratory cancer deaths. Eight thousand forty-seven male smelter workers were observed for mortality rates from 1938 through 1977 for a total of 192,476 person years of follow-up observations. Altogether 3550 deaths were observed of which 302 deaths were caused by lung cancer. Expected number of cancer deaths were calculated on an age-adjusted basis using the combined mortality of the white male population of Idaho, Wyoming and Montana. Workers were categorized according to length of employment as well as the level of exposure to airborne arsenic. These two factors were correlated with lung cancer mortality. Exposure to arsenic was estimated from 702 samples collected at 56 sampling locations at the smelter during the years 1943 - 1958. These exposures were categorized as heavy, medium and light, and were average levels of airborne arsenic of 11.27, 0.58, and 0.27 mg/m^3 respectively. Follow-up was conducted of workers who had been exposed for 15 years or more. Analysis of the data by EPA shows that the risk for the high-exposure category with an exposure duration greater than 25 years does not agree with the risks for the other groups.[29] Therefore, EPA decided to use low and medium exposure groups to estimate risk.

Criticisms of Higgins Study

The findings of the Higgins study of Anaconda smelter workers was cited by some commenters as providing evidence for the existence of a threshold for lung cancer. One commenter (IV-F-3.15) pointed to the Higgins data to

criticize EPA's assumption that the same linear relationship of risk to exposure level is found at all levels and that there may not be levels of exposure where the risk increases more rapidly than at other exposure levels. Higgins data demonstrate little or no risk change between the lower two exposure groups and a doubling of risk between the upper two exposure groups. Strength is also given to this study due to the proper classification of exposure categories as opposed to the methodology used by Lee-Feldstein (IV-F-3.15, IV-F-1.6). The EPA's fit of Higgins data is questioned although an adequate fit from both the absolute and relative risk models is demonstrated. The criticism focuses on the point that analysis by ceiling exposure indicates heterogeneity of data and because Higgins used an unequal sampling technique, the heavy group dominates the analysis and thus the unit risk calculated from these data only applies to high or very high exposure groups (IV-D-711, IV-D-640).

With regard to the slight deficit in lung cancer mortality for persons whose "ceiling" arsenic was below 500 $\mu g/m^3$, another commenter (IV-D-708) stated that the data with respect to low ceiling doses do not approach statistical significance. Other criticisms include the fact that Higgins only used 20 percent of the available cohort, problems with estimations of exposure and the hypothesis that lung cancer risk is dependent on the highest 30-day dose rather than cumulative exposure.

Response:

Higgins et al. studied 1800 workers at the Anaconda Smelter.[30] The cohort consisted of workers classified in Lee-Feldstein study as heavily exposed, and a random sample of 20% of employees classified as having received medium and light exposures to arsenic. This cohort was 22% of Anaconda workers. Higgins et al. examined industrial hygiene records during 1943-1965 and calculated average air concentrations of arsenic for 18 smelter departments. For 17 other departments with no available measurements, arsenic air levels were estimated or inferred by analogy to known measurements. Based on duration of employment within each department, workers were assigned a time weighted average (TWA) arsenic category, and a ceiling arsenic category.

TWA values were calculated as a function of length of time a worker spent in a given department, and the average arsenic concentration in that department. Ceiling level was defined as the highest arsenic level a worker was exposed to for a period of 30 days or more. In addition, workers were assigned by cumulative arsenic exposure which was calculated as the product of the average arsenic concentration for each department during 1943 - 1965 times the length of employment in that department; the individual's department exposures were summed over his entire work history. Thus cumulative exposure was an estimation of total dose of arsenic a worker received over a lifetime. Higgins et al. grouped TWA and ceiling exposure data into four exposure categories; low (<100 $\mu g/m^3$), medium (100-499 $\mu g/m^3$), high (500-4999 $\mu g/m^3$), and very high (>5000 $\mu g/m^3$). Cumulative exposure data was categorized as low, medium, high, and very high with values of 500, 500-2000, 2000-1200, and greater than 12000 $\mu g/m^3$ - years, respectively. The study showed that exposure to airborne inorganic arsenic was strongly related to increased risk of respiratory cancer mortality. Under the TWA exposure classification system a gradient response was observed, with SMRs ranging from 138 in the low category to 704 in the very high exposure category. Observed increases in lung cancer mortality were statistically significant except in the low exposure category. Ceiling level exposures showed mortality increases to be significant only in the high or very high categories, but a dose-response was observed. SMRs were 129 and 116 in the low and medium categories, respectively. Increases in lung cancer mortality were observed to be significant for cumulative exposure groups above 2000 $\mu g/m^3$ years with lifetime ceilings above 500 $\mu g/m^3$.

Commenters take the findings of no significant increase in lung cancer mortality at ceiling exposure less than 500 $\mu g/m^3$ as evidence of a threshold for arsenic exposure. This hypothesis would represent a mechanism of carcinogenesis suggesting a tolerable dose of arsenic exposure, or a no-observed-effect-level. The power of Higgins et al. study to detect increased lung cancer risk in low exposure levels considerably weakens this hypothesis. The Occupational Safety and Health Administration (OSHA) recently analyzed the ability of the Higgins et al. study to detect a 1.5 fold increase in risk of lung cancer mortality to workers exposed to 150 $\mu g/m^3$

of arsenic for 15 years.[31] The statistical power of Higgins et al. to detect a 1.5 fold lung cancer risk for ceiling exposure categories of less than 100 $\mu g/m^3$ and 100-500 $\mu g/m^3$ exposure level showed a power estimate of only 37%. The study had less than 37% chance of detecting a true 50% excess cancer risk. OSHA estimated the power of the study to detect increased lung cancer risk in the TWA exposure category of less than 100 $\mu g/m^3$ to be only 31%. OSHA concluded that:

> "Most epidemiologic investigators, when initiating a study, attempt to choose a study cohort of sufficient size to have at least 80% power to detect a true difference in the variable of interest. Therefore, the statistical power of Higgins et al., all of which are less than 40%, are much lower than desirable.... Given the low statistical power of the study by Higgins and colleagues to detect increased respiratory cancer risk among workers in the low and medium exposure categories, and given the dose-response gradients observed in their study, it is appropriate to consider excesses of respiratory cancers as evidence of potential risk, even if such excesses are not statistically significant. Hence, the respiratory cancer SMRs of 138, 129, and 116 in the low TWA exposure category, low ceiling category, and medium ceiling category respectively should not be disregarded."[32]

Therefore, in view of the low statistical power of the Higgins et al. study to detect excess lung cancer mortality in low TWA and ceiling exposure categories, and because the mechanism of thresholds for carcinogenic agents is currently not supported with good scientific evidence, EPA cannot accept the argument that Higgins et al. proves the existence of an exposure to arsenic that will not result in an adverse health effect.

Criticisms of the Enterline and Marsh 1982

Use of the study by Enterline and Marsh was criticized for two basic reasons. First, commenters (IV-F-1.6, IV-D-625.5, IV-F-3.15) noted that neither duration of exposure nor time since first exposure contributed strongly to respiratory cancer excess. The excess also held for workers

with short exposure and with short latent periods as well. In other words, exess relative risk of Tacoma smelter workers based on urinary arsenic levels appeared to be independent of cumulative risk. The EPA was criticized for correlating urine arsenic levels into air arsenic levels resulting in an inadequate fit of the data (IV-F-1.6, IV-D-711, IV-D-640). A second criticism was based on the methodology EPA used in fitting the data. One commenter (IV-D-711) made the claim that when a "y" intercept was allowed, the relative risk model had an excellent fit.

Response:

Enterline and Marsh studied a cohort of 2802 men employed at the ASARCO smelter for a year or more from 1960-1964.[33] Their mortality experience was observed through 1976. During the study period, 104 deaths from lung cancer were recorded. Respiratory cancer mortality was significantly increased compared to U.S. males and Washington State males (SMR = 198.1 and 189.4, respectively).

To investigate dose-response, the data were assembled by dividing the total person years of observation into 5 groups based on cumulative arsenic exposure (0-lag), and based on cumulative arsenic exposure up to 10 years prior to the year of observation (10-year lag). Arsenic exposure was estimated on the basis of representative average urinary arsenic levels for workers in a given smelter work area. The assumption was there exists a good correlation between airborne arsenic concentrations and urinary arsenic levels. Enterline converted urinary levels to estimated airborne levels using a conversion factor of 0.304. Thus, a urinary level of 100 μg/l of arsenic was roughly equivalent to 30.4 $\mu g/m^3$ of arsenic in the air. In response to the specific comment in EPA's use of this conversion data in risk analysis, it must be noted that the derivation of airborne arsenic concentrations from urinary levels was the protocol of the Enterline and Marsh cohort study. The Occupational Safety and Health Administration (OSHA) recently reviewed this protocol in establishing rules governing workplace exposure to inorganic arsenic and found that,"a urinary arsenic level is a biological indicator of arsenic exposure that would reflect protection provided by respiratory use."[34] Furthermore, OSHA stated that,

"Because the men studied by Pinto et al. (Enterline) were asked not to eat seafood, which would be the major source of urinary arsenic in the absence of air exposure, Pinto et al's (Enterline's) assumption of zero urinary arsenic from zero air arsenic exposure appears reasonable. Therefore, OSHA considers Pinto et al's (Enterline's) correlation coefficient to be the best available measure of the relationship between urinary arsenic and airborne arsenic and it has been used by a number of scientists."[35]

Cumulative exposure categories, expressed as micrograms of arsenic per liter years (µg As/l-years) were: <500; 500-1500; 1500-3000; 3000-5000; and > 7000. SMRs for lung cancer ranged from 155 to 246 in these categories. There appeared to be no increase in SMRs with increasing dose. For workers with less than 10 years of exposure, SMRs were highest one to two decades after the date of hire (suggesting a short latency period). Likewise, for workers employed 10-19 years, the SMR was highest 20-29 years after the date of hire. These observations seem to suggest that short exposures have a disproportionally greater effect than long exposures, and that effects of early exposure tend to diminish with time.

However, reanalysis of the data by Enterline and Marsh in which observations were restricted to retired workers over age 65 showed a clearer dose-response gradient. When lung cancer mortality was analyzed by latency from initial exposure and duration of employment, SMRs were significantly in excess during the first 10-19 years after cessation of exposure. When lung cancer mortality was examined by duration of employment and by average exposure, SMRs increased both with increasing duration and increasing average exposure.[36] Enterline and Marsh concluded from this that both duration of exposure and intensity of exposure contributed to respiratory cancer mortality.[37]

The EPA considers the Enterline and Marsh study amenable to quantitative estimation of risk to exposure of airborn arsenic. The study involved the entire cohort of workers at the ASARCO-Tacoma smelter. Individual exposure histories were estimated, and the exposure estimates based on a 10 year lag

probably yield a more realistic dose-response than those that do not utilize a lag. Analysis of absolute risk by group submitted before OSHA hearings found that cumulative exposure data and 10 year lag data produced a strong linear trend of increasing risk with increasing cumulative dose.[38] Thus, the data presented are not inconsistent with the linear non-threshold model.

2.3.2 Negative Studies

Many comments (IV-F-1.1, IV-F-1.3, IV-F-3.2, IV-F-4.60, OAQPS 79-8, IV-D-27, IV-F-4.62, IV-F-4.38, IV-F-4.14, IV-F-3.11, IV-F-3.12, IV-F-3.6, IV-F-1.14, IV-D-773, IV-D-652, IV-F-4.4) were received regarding the absence of health effects, particularly an increase in mortality due to lung cancer, within the Tacoma community. One commenter (IV-D-621-5) provided the full text of epidemiology statistics which demonstrate that there is no actual support that there is increased lung cancer in communities near smelters. Several studies were cited which demonstrated no increased risk of lung cancer in residents residing near copper smelters. These included Polissar et al. (1979), Hartley et al. (1982), Milham 1982, Frost (1983). Another commenter (IV-D-710) cited problems with these studies such as small sample sizes, lack of correction for confounding variables and flawed methodologies as reasons for the inability to detect an increased risk of lung cancer in the community.

Comment:

A number of commenters (IV-F-3.11, IV-D-609, IV-D-708, IV-F-4.43, IV-D-710) questioned the extrapolation from occupational studies to determine risks in the community. Concerns were based on the uncertainty inherent in such extrapolations in the development of unit risk and the possibility that such risks could be higher because of such uncertainties.

Comment:

Criticism focused on the use of occupational studies where exposures were much higher than ambient levels found in the community (IV-D-695,

IV-D-627, IV-F-3.11, IV-F-4.43, IV-D-621-15.7, IV-F-3.12, IV-F-3.57, IV-F-3.6, IV-F-3.15, IV-F-4.60, IV-D-621-15.9) A claim was made that the statistical data base used by EPA is weak and inadequate for determining carcinogenic risk from low level arsenic exposure (IV-D-71, IV-D-640).

Response:

It is not unreasonable to estimate risk of respiratory cancer from chronic airborne arsenic exposure based on observations derived from statistically valid occupational exposure studies. A causal association between exposure to a chemical agent and the manifestation of cancer in humans in the context of prolonged worker exposure to that agent is a valid and sound epidemiologic method of assuming the agent is carcinogenic in humans. Once this has been established, as in the case of inorganic arsenic, then exposure factors, and dose-response gradients documented in occupational studies become a good basis of estimating risk in the general population. A 3-fold to 11-fold increase in risk of respiratory cancer has been observed in over ten epidemiologic studies of smelter workers exposed to airborne arsenic.[39] This strong association relating human exposure to lung cancer has prompted the International Agency for Research and Cancer, the World Health Organization Arsenic Working Group, the Chemical Manufacturers Association, the Occupational Health Safety Administration,[40] and the National Toxicology Program to identify inorganic arsenic as a human carcinogen.[41] Four epidemiologic studies demonstrated a good dose-response relationship and provided a good basis for risk assessment; they were: Brown and Chu (1983); Lee-Feldstein (1983); Higgins et al. (1982) and Enterline and Marsh (1982).

Dose-response curves from these studies were used to estimate unit risk of exposure to $1\mu g/m^3$ of airborne arsenic. The linear non-threshold approach in estimating risk to lung cancer was employed by EPA, because, as the Office of Technology Assessment of the U.S. Congress has pointed

> "Such linear models are conservative in that, if they err, they overestimate the amount of disease to be expected. All government agencies that use extrapolation employ linear models for predicting

cancer incidence. Other models project risks that decrease more rapidly than dose, and they are advanced as alternatives to the linear model. The choice of a model is important because, if an acceptable level of risk were decided on, almost any other model would allow higher exposures than do linear models."[42]

Commenters have raised the issue of the appropriateness of extrapolating from medium and high exposure levels discerned in occupational studies to low level community exposure. Despite methodological differences between smelter studies used by EPA to generate dose-response gradients, the studies found a dose-response relationship in which increasing exposure to airborne arsenic was correlated with increasing lung cancer risk. The World Health Organization recently stated that,

> "The use of the linear non-threshold model is recommended for extrapolation of risks from relatively high dose levels, where cancer responses can be measured, to relatively low doses, which are of concern in environmental protection where such risks are too small to be measured directly either through animal or human epidemiological studies. The linear non-threshold model has been generally accepted amongst regulatory bodies in the USA for chemical carcinogens and for ionizing radiation on an international basis. The linear non-threshold philosophy was accepted by a Task Group on Air Pollution and Cancer in Stockholm in 1977. The scientific justification for use of a linear non-threshold extrapolation model stems from several sources: the similarity between carcinogenesis and mutagenesis as processes which both have DNA as target molecules, the strong evidence of the linearity of dose-response relationships for mutagenesis, the evidence for the linearity of the DNA binding of chemical carcinogens in the liver and skin, the evidence for the linearity in the dose-response relationship in the initiation stage of the mouse 2-stage turmorigenesis model, and the rough consistency with the linearity of the dose-response relationships for several epidemiological studies; for example, aflatoxin

and liver cancer, leukemia and radiation. This rationale for the linear non-threshold dose-response model is strongest for the genotoxic carcinogens."[43]

2.3.3 Criticism of Model-Unit Risk Estimate

Comment:

Commenters (IV-F-3.12, IV-F-3.15, IV-D-711, IV-D-640, IV-D-621-7.1) questioned the fit of the data in the models claiming that in most cases the fit is not adequate. Criticism focused on the Lee-Feldstein data which show no fit when the relative risk model is used and poor fit when the absolute risk model is used. One commenter (IV-D-621-7.1) criticized the use of p-value to assess "goodness of fit" stating that this method is not satisfactory because the value depends on the magnitude of discrepancies between observed and expected and the size of the study. The same commenter stated that the lack of fit as assessed by EPA's approach may arise from misclassification of exposure, incomplete follow-up or misclassification of disease resulting in errors in the data. Based on this approach, the commenter feels EPA should exclude the Lee-Feldstein data.

Comment:

One commenter (IV-F-1.6) said that the Lee-Feldstein data should not be used in the estimation of unit risk because the data do not fit the linear non-threshold model and only fit it inadequately when the heavy exposure group is removed from analysis. The Brown-Chu analysis of the same data should not be used either because it uses out of date data and analyzes only workers employed past age 55.

Comment:

One commenter agreed with EPA's unit risk estimate based on the linear absolute analysis of Higgins in preference to the relative risk analysis but claims tht EPA's value of 4.90×10^{-3} may be an error in calculation and that the value is actually 2.67×10^{-3}. The same commenter claims that the analysis provided by EPA supports at best only 2 estimates of unit risk:

1.25×10^{-3} from Brown and Chu and 4.90×10^{-3} from Higgins resulting in a geometric mean of 2.47×10^{-3} or 1.83×10^{-3} if the correct value using Higgins is used.

Comment:

One person (IV-F-1.6) commented that EPA's most recent health assessment uses the absolute risk, analyzing a method that underaccounts for the age related incidence of lung cancer.

Comment:

One commenter (IV-F-3.45) voiced concern over the unit risk estimates. He questioned why absolute risk shows a dose-response relationship while relative risk does not. He offered the explanation that the groups were exposed many years ago and therefore are older. Thus workers with highest levels of exposure are expected to have an increased incidence of lung cancer merely because of age and this could explain the linear relationship between absolute risk and exposure.

Comment:

One commenter (IV-D-609) stated that use of the linear model, which is based on an extrapolation from occupational studies that have their own uncertainties leads to uncertainties in unit risk. Another commenter (OAQPS 79-8/IV-D-27) felt that the Agency has failed to be clear and explicit in its description of both the unit risk estimate and the exposure estimate methodologies and should have explained that both are designed to overstate the probable actual value.

Response:

The data from the various epidemiological studies used for the purpose of deriving a unit risk estimate were statistically analyzed to assess the appropriate fit of the data with both absolute and relative risk models. In every case a linear model fitted the data better than the corresponding quadratic model. In most cases, the fits of the quadratic model could be rejected at the 0.01 level, with the exception of the two smallest data

sets (Higgins et al. absolute risk, and Ott et al.). In Higgins et al. the fit was very marginal (p=0.017). However, for each data set a linear model provided an adequate fit. In every case, the absolute-risk linear model fitted the data better than the relative risk model. The p-values for the its of the absolute risk models ranged from 0.025 to 0.75.

The unit risk is defined as the lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed to an average arsenic concentration of 1 $\mu g/m^3$ throughout a 70 year lifetime. A computed unit risk for each of the studies was used when the chi-square goodness-of-fit p-value was greater than 0.01. The unit risks derived from linear models ranged from 0.0013 to 0.0136. The unit risk derived from the linear absolute-risk models are considered to be the most reliable, because although derived from 5 different sets of data from 4 independent investigations of smelter workers, involving 2 distinct smelter worker cohorts, these estimates were quite consistent, ranging from 0.0013 to 0.0076. To establish a single unit risk estimate for arsenic, first a geometric mean of the data sets within distinct exposed populations was obtained, and then a final estimate was made based on taking a geometric mean of those values. The final estimate is 4.29×10^{-3}.

Admittedly there are uncertainties in the unit risk process. Estimates were made from epidemiological studies in which exposures to arsenic occurred only after employment age was reached. It was assumed in deriving risk estimates through either the relative or absolute risk models that the increase in age-specific mortality rates of lung cancer was a function only of cumulative exposures. The models did not consider how the exposures accumulated. Thus, even though this assumption results in an adequate description of the data, it may be in error when applied to exposures that began early in life. In addition, risk assessment is always constrained by the fact that it depends on original data as reported and analyzed by the investigator who's primary objectives were to examine the incidence of disease and not to determine quantitative risk.

Comment:

A number of commenters (IV-F-4.4, IV-F-3.11, IV-F-1.6, IV-F-3.57, IV-F-4.60, IV-F-4.62, IV-F-1.3) addressed the use of epidemiological studies in risk assessment and their power to detect an increase in cancer incidence above background levels. One commenter (IV-F-4.4) stated that it is not possible using the scientific methods available today to detect 1 or 2 additional cancers over the background rate of cancer that exists in every community with or without a copper smelter.

Comment:

One individual (IV-D-621-14.3) stated that given the fact that migration hinders epidemiology studies, it is unlikely that it will be possible to detect risks of 1 or 2 percent.

Comment:

One person (IV-F-4.1) said that we don't have the capacity to detect small risks from the smelter at this time. The risk would have to be quite large in order to really detect it in a population the size of Tacoma over a short period of years.

Comment:

One commenter (IV-F-1.6) stated that epidemiologic study techniques are too imprecise to measure small increases in death rates from lung cancer. Because of the large number of people needed to measure a slightly increased cancer rate, it may not be possible to definitely answer the question of risk from lower levels of airborne arsenic.

Comment:

Other commenters (IV-D-741, IV-D-621-14.11, IV-D-621-14.8) felt that additional information is needed about the health effects of arsenic and the carcinogenic mechanism of arsenic.

Response:

The EPA agrees with commenters with regard to the difficulty of detecting an increase cancer incidence within the community. Increased health risk to residents of the Tacoma area cannot be measured directly. While epidemiological studies have revealed an association between occupational exposure to ambient arsenic, such associations may not be measurable in the general public because of the presence of many confounding factors. These include the public's greater diversity and mobility, lack of consolidated medical records, lack of historical exposure data over each individual's lifetime, public exposure to many carcinogens besides arsenic, and the long latency period of cancer. Irrefutable proof that arsenic causes cancer in the community would require at least 95 percent certainty about the scientific facts. Since 95 percent certainty is unobtainable for most conceivable cases of low level exposure to carcinogens due to the size of the population or length of time necessary to follow a smaller population, this requirement would preclude the promulgation of environmental standards. Such an approach would not be in consistent with the language or the spirit of section 112.

In the evaluation of inorganic arsenic emissions under section 112, EPA has followed a policy in which the nature and relative magnitude of health hazards are the primary consideration. Regulatory decisions must be made on the basis of the best information available since perfect data can never be obtained. In this case EPA has evaluated the potential detrimental effects to human health caused by pollutant exposure based on the best scientific information currently available. For arsenic this represents epidemiologic studies of individuals occupationally exposed to levels of arsenic higher than are present in ambient air.

Comment:

The CEOH report submitted by several commenters (IV-D-634, IV-D-704, IV-D711, IV-D-640) thought that the variable "D" used in EPA's equations (presented in the draft Health Assessment Document) was more accurately described as incremental exposure exposure above ambient levels rather than exposure as measured in an environmental setting. The general population is exposed to some background level of arsenic. The same commenters said

that EPA had provided no estimate of uncertainty in the unit risk estimates nor had EPA characterized its degree of conservatism.

Response:

The commenters have raised a valid point and their reasoning on this matter reflects understanding of the Agency's exposure and risk assessment. Since arsenic is a naturally-occuring element in the earth's crust, it is no surprise that EPA has detected some arsenic at almost all arsenic monitoring sites. Therefore, each individual probably inhales some arsenic every year over his entire life. So, strictly speaking, the dose of exposure that is used in the linear nonthreshold model would be that incremental exposure above the national average ambient levels. Since the national average is quite small in relation to the concentrations predicted around many of the sources of concern, this correction is not meaningful (see Chapter 3 of the Health Assessment Document).

Indirectly, EPA had provided some measure of uncertainty in the unit risk by displaying the range of values that were calculated for the human studies with reasonable exposure/risk data. As the Health Assessment Document indicates, the values ranged from 0.0013 to 0.0136 per microgram per cubic meter of air. The unit risk estimate of 0.00429 was a single point "best estimate" for the exposure/risk relationship at occupational levels of exposure. However, the Agency has no way to quantify the uncertainty of applying this same relationship at ambient levels. There are no studies that are sensitive enough to detect the predicted excesses in lung cancer in the community. Based on experience with other pollutant data, the Agency believes that the linear, nonthreshold model produces plausible upperbound estimates of public risk (given that the exposure is accurately known), but how much of an upperbound estimate is not known.

Comment:

CEOH (IV-D-634, IV-D-704, IV-D-711, IV-D-640) provided an alternative analysis for deriving a unit risk estimate. Their estimates of unit risk from the Enterline and Marsh data were 4.49×10^{-3} for zero-lag data and 4.5×10^{-3} for a ten year lag. These estimates represent a reduction of 34 percent to 40 percent over the unit risk calculated by EPA. The commenters used EPA's equation, but with an intercept term (b_0):

$$O_i = E_i + PYR_i\ (b_o + b_1 D_i)$$

where: O_i = number of lung cancer deaths predicted by the model for the ith exposure group,

E_i = number of expected deaths based on U.S. white male mortality rates

PYR_i = person-years of observation in the ith group, taken from Table 5-33 of the June 1983 draft Health Document

b_o, b_1 = constants (the intercept and slope, respectively)

D_i = cumulative exposure to arsenic in Mg/liter-yrs

They claimed an improved fit over EPA's model using this model ($X^2 = 0.57$, $p>0.60$). Next, they performed a similar analysis on the data from the Brown and Chu and the Higgins et. al. data, and in a fashion similar to EPA's analysis, calculated the geometric mean of the individual unit risk estimates. In another report, CEOH derived what they termed worst-case risk estimates by fitting linear absolute and relative risk models with intercepts to the five data sets used by EPA. The commenter's worst case estimate derived in this manner was 2.67×10^{-3}.

Response:

The commenters desired to account for the possibility that smelter workers were at a higher than normal lung cancer risk group, and EPA, by not accounting for this possibility, has overstated the unit risk estimate. If the commenters supposition was true, then one would detect greater than expected lung cancer incidence rates in the very low exposure groups of smelter workers.

The Agency considered this possibility since the Lee-Feldstein and the Enterline and Marsh data appear to support the commenter's hypothesis. However, the Agency did not modify their analysis as suggested. There was not a consistent observation of increased lung cancer in the low exposure groups in all the studies. As a number of other commenters pointed out to the Agency, the Higgins et. al. data indicated a less than expected lung cancer

rate for the low exposure group (not statistically significant) . There was no consistent observation of this increased cancer risk at low exposure from study to study. The EPA had other reasons for not modifying its

analysis. As already discussed in the earlier sections of this chapter, the Agency believes that there are credible scientific theories for adopting the linear nonthreshold model. Upon reviewing its previous analysis with the commenters concept in mind (Figures 7-2 thru 7-9 in the health assessment document), the Agency noted that the absolute linear nonthreshold model mathematically described the data within the confidence limits of each risk value for the low exposure groups. Thus, EPA's linear model is adequately describing the data in this region of exposure.

Finally, EPA believes that the two approaches are producing approximately the same results. The commenter's estimate falls within the range of unit risk estimates that the Agency had calculated from study to study (0.0013 to 0.0136 per microgram per cubic meter of air) and so does not significantly change the Agency's perception of arsenic's carcinogenic potency.

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5 Drinking Water and Health. National Academy of Sciences, National Research Council, Wash., D.C., 1977, pp II-21.

6 Health Assessment Document, pp. 7-50 - 7-52.

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8 Ibid. pp. 5-11.

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10 Health Assessment Document, pp. 5-21.

11 Ibid, pp. 5-10 - 5-17.

12 Norstrom, Op. cit.

13 Health Assessment Document, pp. 9-9, 9-10.

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3. LISTING OF ARSENIC

Comment:

Some commenters (IV-D-641, IV-D-622, IV-F-4.67, IV-D-708a, IV-D-741, IV-D-747) expressed support for EPA's decision to list arsenic as a hazardous air pollutant under section 112 of the Clean Air Act. However, another (IV-F-3.11/IV-D-62115.6), questioned EPA's listing of arsenic as a hazardous air pollutant, saying that this listing was based on determinations that "there is a high probability that inorganic arsenic is carcinogenic to humans" but that evidence to support this hypothesis is not unequivocal.

One commenter (IV-D-710) said that to remove arsenic from the list of hazardous pollutants, EPA would have to show that it "clearly" is not hazardous under section 112 of the Clean Air Act. The commenter said that it cannot be shown that arsenic is safe to breathe at ambient levels. He judged that there is substantial evidence that arsenic is a carcinogen, and that as such it must be regarded as posing a cancer hazard at all dose levels. Thus, the commenter reasoned arsenic must remain on the list. Assertions that the risk is "small" or "acceptable" does not provide a legally supportable basis for removing a substance from the hazardous pollutant list.

Response:

Under section 122 of the Clean Air Act, EPA was specially directed to list arsenic as a hazardous air pollutant if the Administrator determined that emissions "into the ambient air will cause or contribute to air emissions which may reasonably be anticipated to endanger public health." Upon review of the available data, the Administrator listed inorganic arsenic as a hazardous air pollutant under section 112. The Adminstrator's decision to list was based on EPA findings that "there is a high probability that inorganic arsenic is carcinogenic to humans and that there is significant public exposure to inorganic arsenic." Evidence for this is summarized in the Federal Register (44 FR 37886, June 5, 1980, 48 FR 33113, July 20, 1983) and EPA's Health Assessment Document for Arsenic (EPA-600/8-83-021F).

The data and documents supporting the listing are filed under Docket Number OAQPS-79-8 and are available for public inspection and copying at EPA's Central Docket section in Washington, D.C.

The Administrator stated at the time of proposal, and many commenters agreed, that there are uncertainties in the health data base and that a significant public health risk in the general community has not been absolutely proven. But, neither the language of the Act nor prudent public health protection policy requires absolute proof of health risks before the Agency invokes its authority to act under section 112.

When the decision to propose inorganic standards was made, the Administrator was aware, via an updated draft document entitled "Health Assessment Document for Inorganic Arsenic" (EPA-600/8-83-021) of issues and the data subsequently presented by the dissenting commenters and was considered when the Agency proposed the inorganic arsenic standards. On balance, however, this draft document presented a strong case for inorganic arsenic being a human carcinogen. In November, 1983, the Science Advisory Board, an advisory group of nationally prominent scientists from outside EPA, concurred with the report's conclusion that the weight of evidence places inorganic arsenic in a group of pollutants that are characterized as "carcinogenic to humans." This conclusion is based on two general observations. First, associations between cancer and inorganic arsenic exposure have been demonstrated in occupational groups, such as in copper smelters, pesticide manufacturing and agricultural work, and in non-occupational populations using arsenical drugs or consuming arsenic-contaminated drinking water and/or food. Second, the results from several independent human studies have consistently demonstrated the same study findings, high relative risks, and specificity of tumor sites (skin and lungs). The EPA has now published these conclusions in the final health document (EPA-600/8-83-021F).

Others have made similar findings regarding inorganic arsenic's carcinogenicity. Widely-respected scientific groups such as the National Cancer Institute and the National Academy of Sciences have concluded there is substantial evidence that inorganic arsenic is carcinogenic to humans and the International Agency for Research on Cancer (IARC) has stated there is sufficient evidence that inorganic arsenic is carcinogenic to humans.

In addition, the Occupational Safety and Health Administration, also recently reviewed the substantial body of evidence and concluded that inorganic arsenic "is clearly a human carcinogen" (45 FR 19584).

After a substance is listed as a hazardous air pollutant, section 112 of the Act requires the Administrator to subject the listing decision to public review during the proposal of the hazardous emission standards for that pollutant and to continue with the promulgation of standards unless the Administrator finds, on the basis of information presented by commenters, "that such pollutant is clearly not a hazardous air pollutant" (section 112 (b)(1)(B)). Thus, in the July 20, 1983 proposal, the Agency specifically requested comments on the listing decision and the Administrator's findings. After reviewing all the public comments and considering the available human health data, the Administrator has affirmed his judgment that inorganic arsenic is a probable human carcinogen and is appropriately listed as a hazardous air pollutant under section 112.

Comment:

One commenter (IV-D-625) requested that EPA identify specific inorganic arsenic compounds as hazardous rather than just grouping them all in the category of "inorganic arsenic." The commenter felt that a further breakdown was appropriate since not all inorganic arsenic compounds were of toxicological concern and cited the June, 1983 draft health assessment, in which EPA stated that elemental arsenic was of "little toxicological interest." According to the commenter, EPA showed evidence that trivalent and pentavalent oxides have adverse health effects but did not establish whether other inorganic forms are hazardous. The commenter said solubility was not considered in the hazard determination. Therefore, the commenter felt EPA should identify which compounds produce the risks estimated, and which are expected to produce greater or lesser risks.

Response:

The keystone of the inorganic arsenic listing decision is the relatively large human health data base that has successfully linked excess lung cancer and total arsenic exposure arsenic exposure in the workplace. Because of the

known chemical composition of the plant products or by-products, the Agency believes that, although total arsenic was measured for the occupational studies, the particular arsenic compounds involved primary were 1) inorganic pentavalent arsenic in the pesticides manufacturing workplace, and 2) inorganic trivalent arsenic in the smelter workplace. In reviewing this health data base in EPA's health assessment document (OAQPS 79-8, II-A-13, EPA-600/8-83-021F), it is apparent to the Agency that exposure to both forms of inorganic arsenic, i.e., the inorganic trivalent arsenic and the inorganic pentavalent arsenic are linked with increased risk cancer risks and the potencies of each form of inorganic arsenic are approximately the same magnitude. Thus, based on the health effects data, it makes little sense to separate arsenic compounds by valency or by specific compound.

In addition, identifying and quantifying the various arsenic compounds present in an unknown matrix is not a routine analytical matter. The Agency has worked with several analytical researchers, Dr. Edwin Woolson of the U.S. Department of Agriculture and Dr. Kurt Irgolic of Texas A&M University, who have much experience in speciating various forms of arsenic in matrices. The Agency realizes that arsenic speciation techniques are in the developmental stage and not readily adapted to a regulatory program. Thus, the Administrator has determined that separate regulation of several forms of inorganic arsenic is unnecessary and impractical.

Comment:

One commenter (IV-D-617) felt that in the application of section 112 to hazardous air pollutants, more explicit provisions should be made for a decision not to list a pollutant if EPA is unable to determine that a significant health risk exists. The commenter endorsed EPA's conditioning of its decision to list arsenic on an intention to establish standards for some source categories and not for those deemed to pose insignificant risks. The commenter noted that exposure should be considered at the time of listing to determine if a significant section 112 health risk exists and that EPA should not list (or should delist) a pollutant when public exposure to that pollutant does not create a significant section 112 health risk.

Response:

Exposure was considered in the decision to list arsenic as a hazardous pollutant under the Clean Air Act (44 FR 37886, June 5, 1980). The arsenic emissions from primary copper smelters and glass plants were determined to pose significant public exposure. The evidence for significant exposure at the time of listing is contained in the listing docket [Docket No. OAQPS 79-8 II-A-6]. If the Administrator were to determine that there is clearly no significant risk, section 112 regulations would not be promulgated.

4.0 EXPOSURE AND RISK DETERMINATION

This chapter is divided into three sections. The first (section 4.1) contains comments on the exposure and risk determination models for which a fairly detailed technical description of the model is required in response.

The second part of the chapter (section 4.2) contains the response to the comments in section 4.1. The response section contains an overview of the exposure and risk models and explains the assumptions and uncertainties questioned by the commenters.

The third section of the chapter (section 4.3) contains additional comments and responses. Many of the comments in this third section are on health risk management and policy issues. The responses to these comments generally do not require the detailed description of the model (given in the BIDs). But they may require a description of the chemistry and fate of arsenic, or of policy under section 112 of the Clean Air Act, or of court cases which may have a bearing on risk management policy. Because of the different nature of responses to these comments, they are located in a separate section (section 4.3).

4.1 COMMENT SUMMARIES

The comments in section 4.1 are divided into 9 subcategories, which include:

- factors not considered in the exposure/risk estimation,
- degree of conservatism of estimates,
- criticisms of input data and general modeling assumptions,
- reasons for use of the dispersion model versus ambient air monitoring data,
- criticism of the exposure estimation model,
- criticism of the unit risk estimate,
- miscellaneous criticisms of the model,
- numerical estimates of risk and exposure, and
- uses of the model and risk estimates.

Responses to the comments in this section are given in section 4.2.

4.1.1 Factors Not Considered in the Exposure/Risk Estimation

Comment:

Several commenters (IV-D-677, IV-D-575, IV-D-571, IV-F-4.11,IV-D-670, IV-F-9) maintained that EPA did not model or consider health risks other than lung cancer, an omission which results in underestimation of risk. Other potential health effects cited by the commenters include non-fatal cancers, general health, and birth defects.

Two commenters (IV-F-4.11, IV-D-710) thought EPA should consider workplace exposure to arsenic. Since they did not, exposure and risk were underestimated, according to the commenters.

Some commenters (IV-F-4.11, IV-F-3.57, IV-D-710, IV-D-632, IV-D-757) felt that the risks to sensitive subpopulations were not considered, causing the model to underestimate risk. Others (IV-F-3.20, IV-F-3.55, IV-F-4.50, IV-D-754, IV-F-9, IV-F-10) said EPA should consider sensitive groups in the rulemaking. Two (IV-F-4.15, IV-D-757) said sensitive groups may include infants, pregnant women, and people with respiratory problems. Commenter IV-D-757 pointed out that that studies used as a basis for the unit risk reflected healthy male worker exposure risks. Commenter IV-D-604 asked if there is a statistical distribution for susceptibility to cancer which could be incorporated in a risk estimation procedure.

4.1.2 Degree of Conservatism of Estimates

Comment:

Several commenters said EPA's risk estimate was not conservative, since there are many uncertainties and a variety of factors were not taken into account. The commenters judged that risk may, in fact, be underestimated. (These commenters include IV-F-3.42, IV-D-710, IV-D-698, IV-D-608, and IV-D-579.) One commenter (IV-F-3.42) called the model a "middle-of-the-road" approach, neither excessively conservative nor reckless. Commenter IV-D-710 said EPA cannot know if the model over- or under-predicts. Two others (IV-F-3.55, IV-D-731) thought the exposure estimate in particular was an underestimate. One commenter (IV-D-708a) said that there is a high degree of uncertainty in EPA's estimates.

Comment:

Many commenters said EPA's model was excessively conservative or "worst case," and that risk has been overstated. (These include IV-F-3.15, IV-F-3.12, IV-F-1.6, IV-F-3.9, IV-D-198, IV-D-210, IV-D-321, IV-D-330, IV-D-356, IV-D-362, IV-D-484, IV-D-486, IV-D-499, IV-F-4.4, IV-D-120, Air Products, IV-F-1.2, IV-D-617, IV-D-621-7, IV-D-621-16.10, IV-D-621-14.7, IV-D-621-15.9, IV-D-621-15.7, IV-D-621-15.2, and IV-D-621-5, IV-D-708a, OAQPS 79-8/IV-D-27, IV-F-9.) Specific criticisms of the model given by these commenters are listed in sections 4.1.3 through 4.1.7.

Comment:

Some commenters (IV-F-5.6, IV-D-330, OAQPS 79-8, IV-D-27, IV-F-4.4, IV-D-522, IV-D-708a, IV-F-10) said the model was just guesswork or speculation without any real scientific basis. Others (IV-D-210, IV-D-342, IV-D-489, IV-D-504, IV-F-4.17, IV-D-529, IV-D-731) said the risk estimates were questionable or wrong, but did not give specific criticisms. ASARCO (IV-F-3.9, IV-D-621-15.2) did not agree with the model.

4.1.3 Criticism of Model - Input Data and General Assumptions

Comment:

Several commenters said the results of the risk determinations are in error because they are based on poor or inaccurate data. (These commenters include IV-D-167, IV-D-168, IV-D-215, IV-D-222, IV-D-232, IV-D-254, IV-D-267, IV-D-276, IV-D-238, IV-D-316, IV-D-330, IV-D-362, IV-D-499, IV-F-3.45, IV-D-157, IV-D-579, IV-D-621-16.4, IV-D-645, IV-D-538, and IV-D-568.) Some commented specifically on the inaccuracy of the epidemiology data and unit risk estimate. Such comments are addressed in section 2.0.

Several commenters said that, in particular, the results of the exposure modeling portion of the risk determination were in error because they were based on inaccurate data. (These commenters included IV-D-165, IV-D-169, IV-D-232 IV-D-330, IV-F-1.7, IV-F-1.8, IV-F-3.9, IV-F-4.38, IV-F-11.) Commenters (IV-F-3.9, IV-D-591, IV-D-14.16, IV-D-710, IV-D-579, IV-F-5.7, IV-D-741, IV-D-793) specifically mentioned emissions data. Others thought ambient

arsenic concentration data rather than a dispersion model should have been used and some commenters disagreed with the dispersion model results. These comments are included in the section on "Criticisms of the Exposure Model."

One commenter (OAQPS-79-8/IV-D-27) said EPA failed to consider available data or check the model against such data.

Comment:

One commenter (IV-D-238) requested that EPA make a new risk determination based on accurate data.

Comment:

Two commenters (IV-D-330, OAQPS-79-8/IV-D-27) said there is no solid scientific basis for the kind of mathematical modeling EPA has done to estimate health risk.

Comment:

Several commenters (IV-D-164, IV-D-167, IV-D-232, IV-D-330, IV-D-499, IV-D-504) said that use of unjustifiable assumptions and faulty reasoning were major defects in EPA's model. One commenter (IV-F-5.14) said EPA's risk determination model treats assumptions as facts.

Comment:

Another commenter (IV-D-600) heard that EPA's statistical model was based on data from another plant.

4.1.4 Criticism of Model - Use of Dispersion Model Versus Ambient Monitoring Data

Comment:

Several commenters thought monitored ambient concentrations rather than concentrations estimated by a dispersion model should be used in the exposure analysis. They thought this would make results more accurate. (These commenters include IV-F-3.15, IV-F-4.11, IV-F-3.45, IV-D-125, IV-D-20, IV-D-67, IV-D-342, IV-F-4.71, IV-D-621-16.10, IV-D-621-16.4, IV-D-622, IV-D-608, and IV-D-621-10, IV-D-708a, IV-D-703, IV-D-793.)

One commenter (IV-D-609) said EPA did not attempt to measure actual levels of arsenic in the local community even though technology and cost were not prohibitive. Therefore, the dispersion model cannot be validated. Another commenter (IV-F-9) sugested that ambient arsenic data be collected by a disinterested party (not ASARCO).

One commenter (IV-F-3.15/IV-D-621-15.9) said that EPA should estimate risk for persons exposed to average environmental levels of 0.05 to 1.0 $\mu g/m^3$. The commenters pointed out the fact that EPA has not followed this approach but has instead estimated the upper limits of risk still marginally consistent with the data.

4.1.5 Criticism of Model - Exposure Estimation

Comment:

Four commenters (IV-D-698, IV-D-608, IV-D-729, IV-D-749) said using a 20 km radius was unrealistic and could underestimate exposure. Commenter IV-D-698 said the 20 km radius might be conservative for fugitive emissions, but was not adequate for stack emissions. On the other hand, one commenter (IV-F-9) felt that exposure from fugitive emissions may not decrease with distance as rapidly as EPA expects.

Two other commenters (IV-F-3.37, IV-F-3.17) said EPA's exposure estimates should not be confined to the immediate vicinity of the sources, since stack emissions can travel great distances and affect other communities.

Comment:

Several commenters (IV-D-710, IV-D-617, IV-D-618, IV-D-120) said the assumption that a person would be exposed continuously over 70 years was conservative and unrealistic. Another (IV-D-618) said annual individual risk should be used rather than "maximum lifetime risk" since a person would probably not be exposed to the same level for 70 years. Another commenter (IV-D-622) also called for a more realistic appraisal of the length of time an individual lives in one area.

Some commenters (IV-D-617, IV-D-621-16.10, IV-D-621-14.8, IV-F-1.6, IV-F-11) said exposure should be measured under existing conditions rather than estimated. Two commenters (IV-D-621-16.10, IV-D-608) said that public exposure should be measured with the source

operating and with it not operating to determine arsenic exposure due to the smelter.

Comment:

One commenter (IV-D-621-16.10) said that since Enumeration District/ Block Group (ED/BG) data and maps were not given out by EPA, there is no way to judge the accuracy of the exposure assessments.

One commenter (IV-D-621-16.10) said getting a map of the area, counting homes, and applying a factor for the average number of people per home would better estimate population than using national census data.

One commenter (IV-D-621-16.10) said 1980 census data should be used rather than 1970 data.

Comment:

Two commenters (IV-D-297, IV-F-4.17) said they did not question EPA's exposure estimates since EPA has better knowledge of that area than they do.

Comment:

One commenter (OAQPS-79-8/IV-D-27) said the Agency had failed to be clear and explicit in its description of the exposure estimation methodology. He also said the most probable way for exposure estimates to differ is downward.

4.1.6 Criticism of Model - Unit Risk Estimate

NOTE: Detailed comments on the derivation of the unit risk estimate are included in section 2.0; however, some general comments on the effects of the unit risk estimate on model outcome are included here.

Comment:

Several commenters (IV-F-3.15, IV-F-1.2, IV-F-4.4, IV-F-3.42, IV-D-617, IV-D-621-15.9) felt the unit risk estimate was an extremely conservative or upper limit estimate. One commenter (IV-F-3.15) said it was the maximum estimated possible risk per unit of exposure.

One commenter (IV-D-710) said EPA's use of the linear no-threshold model is generally regarded as conservative, but it may not be conservative given the uncertainties in the unit risk estimation procedure. He noted the unit risk estimate has increased by a factor of 1.45 since proposal as new data were considered.

Comment:

Several commenters (IV-F-3.15, IV-F-1.2, IV-F-4.4, IV-F-3.57, IV-D-617, IV-D-621-15.9, IV-D-604) questioned the use of the linear no-threshold model, while another commenter (IV-D-622) said the linear model should be used.

Comment:

Some commenters (IV-F-4.4, IV-D-627, IV-D-604) questioned the validity of using data on workers (exposed to high levels of arsenic) to estimate risk to the general public (exposed to low levels of arsenic).

Comment:

Two commenters (IV-D-297, IV-F-4.17) said they did not question EPA's exposure estimates, but they did disagree with the way the risk estimates were derived from the exposure estimates.

4.1.7 Miscellaneous Criticisms of the Model

Comment:

Several commenters (IV-D-164, IV-D-322, IV-D-339, IV-D-362, IV-D-398, IV-D-427) said that EPA's health risk analysis is based on an unrealistic computer model.

Comment:

Two commenters (IV-F-4.4, IV-D-708a) said EPA's final results are an upper bound, and the risk may, in fact, be zero. Another commenter (IV-D-621-15.9) said based on his judgment and review of the data, zero is the most likely estimate of increased risk. Another (IV-D-621-15.7) felt that the range given about the risk estimate should include 0. The commenter said that statistics from the epidemiology studies may follow a

normal rather than log-normal distribution. This assumption would give a range of -7 to +16. Another commenter (IV-F-9) suggested a statistical analysis of validity of the model and the confidence limits.

One commenter (OAQPS-79-8/IV-D-27) said EPA's explanation of the risk determination process was unclear.

One commenter (IV-D-609) said that, according to the model, most cancers will occur a large distance from the smelter due to the magnitude of the population being exposed.

One commenter (IV-D-525) asked if health impacts consider whether victims smoke, their age, family history of cancer, or other environmental circumstances.

Comment:

Two commenters (IV-D-600, IV-D-525) asked if there had actually been increased cancer deaths in areas near sources. One commenter(IV-D-652) believed lung cancer increases might be detectable in the Tacoma, Washington area by community studies. Data from such studies should be used instead of a risk model. One commenter (IV-D-773) said studies had not shown any increased risk of lung cancer and that the model's predictions are unlikely.

Comment:

One commenter (IV-F-3.57) objected to current risk assessment practices which start with the assumption that a substance is harmless and then try to prove this assumption. He stated it would be more appropriate to start with the assumption of harmfulness and try to disprove it.

4.1.8 Uses of Model and Risk Estimates

Comment:

One commenter (IV-F-3.12) stated that according to EPA, the purpose of its risk assessment is to "give a rough estimate of the potential cancer hazard that can be used to guide regulatory decisions." He felt that this regulatory purpose would be difficult to achieve, considering that the rough

estimate of risk was obtained by EPA in a series of steps, each of which was deliberately and consistently designed to inflate the risk.

Comment:

Other commenters (IV-F-3.4, IV-D-622) said that the risk model (composed of the health and exposure models) does not necessarily mirror real life situations; they are used to monitor change in variables such as emission levels and resulting or ambient air concentrations. The commenters continued by saying that the risk model is used to predict the effects of reduced emissions on the potential reductions in risk to public health.

Comment:

One commenter (IV-D-622) said the dispersion model and health risk model do not necessarily mirror real life situations. He said they are used to model changes in variables such as emission levels and resulting changes in ambient air concentrations, and can be used to predict potential reductions in public health risk.

Comment:

One commenter (IV-D-617) said health risk is too uncertain to be used to estimate actual health effects posed by a source. However, he said that since the unit risk estimate is the same for all sources of arsenic, exposure estimates can be used to rank the relative severity of the sources, without assigning actual risk values.

One commenter (IV-D-617) stated that realistic estimates of risk should be used in evaluating residual risk and that measurement of actual public risk should be used whenever possible. Two other commenters (IV-D-20, IV-D-67) agreed that actual health tests of the public should be used in setting standards.

One commenter (IV-F-3.42) supported EPA's approach to this problem of making a risk assessment that is as quantitative as possible and considers both health and economics. Another (IV-D-741) said risk analysis is a necessary part of rulemaking and is the only available way to set standards

with a margin of safety. Others (IV-D-708a, IV-D-735) supported the use of risk assessment as a factor in decision-making, but results should be used with caution.

Comment:

One commenter (IV-D-621-14.7) noted that risk assessment is used by all regulatory agencies, industries, and environmental groups. The commenter said it is mandated under TSCA and FIFRA. Uses are as follows:

- Target levels of risk are needed to take action.

- Risk estimates can aid in setting agency priorities.

- Risk assessment can help analyze the effects of a proposed action.

- Going through the risk assessment procedure can help establish what facts are known and unknown.

- The results of a risk assessment cannot be compared directly with countable cases in the community.

Comment:

One commenter (IV-D-621-14.9) said average risk will vary depending on the radius from the plant considered and the size of the population used.

Comment:

One commenter (IV-D-621-15.9) said that the health assessment gives a high estimate of risk, but does not indicate the likelihood that this risk actually exists.

Comment:

One commenter (IV-D-641) felt that the range given around the risk factors is inadequate.

4.2 RESPONSE TO COMMENTS ON THE EXPOSURE AND RISK ESTIMATION PROCEDURE

Several commenters were critical of the mathematical models EPA used to estimate human exposure to arsenic and cancer risk in the vicinity of inorganic arsenic sources. This section deals with the preceding comments on the exposure model which was used during proposal of the standards. This model has been previously explained in the Federal Register notice of proposal (48 FR 33112, July 20, 1983) and in Appendix E of the background document for the proposed standards EPA-450/3-83-010a. Supplemental information on the dispersion and human exposure models in can be found in previous EPA studies of the models [Docket No. (A-80-40), II-A-69, II-A-42, and II-A-72].

The Agency was aware of the shortcomings of the proposal analysis. Since the proposal, EPA has completed extensive site-specific air quality modeling analyses and has compared the predicted concentrations to the monitored air quality data collected near several sources. These analyses, the key assumptions and a discussion of the uncertainties are described in detail in the Appendices of the Background Information Documents as listed in the Introduction.

4.2.1 Need for a Model to Estimate Exposure and Risk

The Human Exposure Model (HEM) is used to make quantitative estimates of public exposure, current risk, and risk reductions associated with proposed or final NESHAP. These quantitative estimates are considered by EPA in its decision-making process. Although there are underlying uncertainties in the model, EPA considers this methodology a reasonable approach to the estimation of health risks and the best tool available to EPA for predicting the probable effects of a standard.

It is not feasible to measure exposure to ambient arsenic in the nearby area directly. It would require a large number of monitors to establish concentrations to which all persons living near urban sources are exposed.

Exposure will vary with distance and direction from the plant. Furthermore, there is no way that ambient air quality monitors can predict that future ambient concentrations may be if arsenic emissions are reduced as a result of a promulgated standard. However, atmospheric dispersion models can be used to estimate these directional variations in exposure and to predict exposure under different emissions control scenarios. Also, existing monitored data can be used to check or validate the model predictions.

Increased health risk to nearby residents cannot be readily measured either. Epidemiological studies have revealed an association between occupational exposure to ambient arsenic and lung cancer (EPA-600/8-83-021f), but such associations are not readily measurable in the general public because of the presence of many confounding factors. These include the public's greater diversity and mobility, lack of consolidated medical records, lack of historical exposure data over each individual's lifetime, public exposure to many carcinogens besides arsenic, and the long latency period of cancer. Because of such factors, increases in cancer observed in the public can rarely be assigned to a specific chemical or emissions source.

In addition, the increased risk estimates are a fraction of the average lung cancer rates and make such predictions difficult to detect (see chapter 2). Therefore, in the case of inorganic arsenic, public risk is estimated by using air dispersion models and site-specific population data to estimate exposure. Next, the Agency and then applies the exposure/risk relationship as derived from the occupational studies to estimate public risks. Although plagued with uncertainty, quantitative estimates of risk are desirable for decisionmaking and risk assessment methods used in the arsenic analysis are the best tools currently available to EPA to obtain such estimates.

4.2.2. Uses of the Human Exposure Model

While it is true that risk estimates obtained from the HEM are considered in decision-making, they are not used as precise predictions. There are many uncertainties in the model, so the numbers obtained may

over- or underestimate actual risk, as several commenters correctly pointed out to the Agency (see Appendix C of Background Information Document, EPA-450/3-83-010b)

The model results can be used in the decision-making process for making relative comparisons. For example, modeled risk estimates can be used to compare the relative severity of risks from different sources of arsenic (i.e., different plants or industries). And they can be used to compare the relative risk reductions which could be achieved by two or more emissions control options.

4.2.3 Specific Purposes and Scope of the Model

The HEM estimates public exposure to ambient arsenic under baseline conditions (i.e., no NESHAP) and under the proposed NESHAP and other regulatory alternatives. The model also predicts lung cancer risks associated with these exposures.

Risk is expressed in two ways - the "maximum lifetime risk" and "annual incidence." The maximum lifetime risk is the lifetime risk of developing cancer for the individual or individuals estimated to live in the area of highest ambient arsenic concentrations as determined by the exposure model. The aggregate risk is the summation of the risks to people living around a source. It is expressed as incidence of cancer among the total population after 70 years of exposure. For statistical convenience, the aggregate risk is often divided by 70 and expressed as cancer incidence per year.

Both measures of risk are based on lung cancer incidence. Risks from other potential arsenic-related health affects were not modeled. In the judgement of the Agency epidemiologic studies show a strong dose-response correlation between lung cancer and inhalation of of arsenic by smelter workers; the smelter studies did not show a correlation between arsenic exposure and other health effects such as skin cancer (EPA-600/8-83-021F). Thus, increased lung cancer incidence is the likely effect of public inhalation of arsenic.

Health effects other than cancer which could result from chronic low-level exposure to arsenic have not been well documented. For example, cardiovascular effects have been noted. However, the data are limited and

health effects are not consistently demonstrated to the point where a dose-response relationship can be developed (EPA-600/8-83-021F). For this reason, health risks other than cancer cannot be quantitatively estimated or modeled. These other potential risks are considered by EPA in a qualitative manner during the decision-making procedure; the Administrator, upon reviewing the risk assessment results and the associated uncertainties, recognizes that the lung cancer risk estimates may not represent the entire spectrum of public health effects associated with inorganic arsenic exposure.

The arsenic lung cancer dose/response relationship was derived from generally healthy male smelter worker's records. Thus, the distribution of the public's susceptibility to cancer due to arsenic exposure is unknown, so risk to sensitive subpopulations or individuals could not be considered quantitatively in EPA's model. As stated in the background document for the proposed standards, this is one of the uncertainties in risk assessment. The EPA in its decision-making is aware of the possible risk to sensitive individuals and considers this by realizing that the risk estimates are not true measures of actual risks but may vary considerably from the Agency's estimates.

It should also be noted that risks from exposure to arsenic in other media besides air (e.g., water, food) and risks from chemicals other than arsenic were not modeled. These issues are discussed in the chapter on the piecemeal approach (section 5).

4.2.4 Elements of the Exposure/Risk Model

4.2.4.1 Overview. Risk estimates are calculated in a series of steps and require several types of data. An overview of the procedure is given here. First, emissions data or emissions estimates and meteorological data are entered into a dispersion model which calculates the expected long-term ambient arsenic concentrations at various distances and directions from the plant. Census data are used to estimate the number and location of people living near the plant. Then the modeled concentrations are matched to this population distribution using an exposure model. "Exposure", as determined

by the model, is the number of people multiplied by the ambient concentrations to which they are exposed. (The units are people - $\mu g/m^3$).

Once the exposure is calculated, lifetime aggregate risk is estimated by multiplying the exposure results by a "unit risk estimate". The unit risk factor is defined as the lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed throughout their lifetimes to an average concentration of 1 $\mu g/m^3$ in the air they breath. The unit risk is calculated from dose-response curves which are developed from epidemiology studies.

The resulting lifetime incidence is, therefore, the aggreggate risk expected in the exposed population over 70 years. Annual incidence is calculated by dividing the lifetime incidence by 70. The "maximum lifetime risk", or the lifetime risk of developing cancer for those people exposed to the highest concentration determined by the model, can also be calculated by multiplying the highest concentration to which people are exposed times the unit risk estimate.

4.2.4.2 Details of Each Element of the Model and Associated Uncertainties. The various elements of the exposure and risk estimation model, the various assumptions and uncertainties in the modeling procedure and the modifications which have been made to the model since proposal are discussed in detail in the referenced BIDs, e.g. Appendix C of the Background Information Document-EPA450-3/83-010b.

Uncertainties. The method of matching concentrations and populations within about 3 km of the plant was criticized by commenters because people within about 3 km were assigned by the model to live at and be exposed to concentrations at receptors sites located over water or other unlikely spots. Commenters felt that this was obviously unrealistic. When estimating risks for arsenic NESHAP promugation documentation, EPA checked the location of the most exposed individual on small-scale U.S.G.S. maps to insure that such location was either accurate or realistic. However, when calculating annual incidence, EPA's experience is that corrections in the exposure model to more closely account for unrealistic placement of people tend

to make insignificant changes to the estimate. The corrected locations, rather than being at points over water, will be located somewhere else where the arsenic concentrations may be higher, lower, or about the same. When several hundred to several thousand poeple are exposed, the "corrected" exposures seem to be about the same. It is EPA's judgment that given all the uncertainties in the exposure modeling corrections are not normally warranted for calculating annual incidence.

The exposure model also assumes that people are continuously exposed to the average ambient arsenic concentration at their residence. In reality, people travel within and beyond the local area. They are exposed to different concentrations at their workplaces, schools, shopping centers, etc. It would be extremely difficult to model local travel and exposures, and any result would be uncertain. Even if the Agency were to collect detailed information on the public at large near a source, these data would not necessarily reflect mobility and migration patterns of past or future generations. Therefore, exposure is modeled using the concentration at the population centroid nearest their residence, where it is likely people spend the majority of their time. It is not known if this over- or underestimates actual exposure.

For the exposure model and unit risk factor, it is also assumed people stay at the same location and are exposed to the same concentration for 70 years. Human mobility and variable lifespans make this assumption unrealistic. However, long-term individual mobility cannot be modeled for the same reasons as given for modeling individual daily mobility. Another problem is that sources do not emit at a constant annual level for 70 years. Since many sources have been reducing emissions over the past decade, the use of current figures may underestimate risk from previous exposure. Predicted future emission rates under various control scenarios are also uncertain. If they are too high or low, the lifetime risk may be over-or underestimated. Similarly, if the population grows in the future,

aggregate risk would increase. This is not currently modeled. The intent of the model is to present risk estimates to a hypothetical population under a "snapshot" of emissions and population distribution scenario.

As a result of the combined effect of all the above assumptions and uncertainties, the model may or may not underestimate exposure depending on the actual circumstances. On balance, however, EPA believes that the methodology represents a reasonable approach given the inherent uncertainties of exposure modeling.

In the proposal analysis, the unit risk estimate for arsenic was extrapolated from workplace epidemiology studies using a linear non-threshold model to estimate risks for the general populations exposed to the arsenic levels characteristic of the ambient air. This was a weighted average of values obtained from 3 epidemiologic studies [Docket No. (A-80-40) III-B-1 and (OAQPS-79-8) II-A-7]. A 95% confidence limit around this estimate produced a range of 7.5×10^{-4} to 1.2×10^{-2} for the risk factor.

The proposal risk estimates were given as ranges because of the range ascribed to the unit risk factor. The range reflected only the uncertainty around the unit risk factor resulting from the combination of results from 3 epidemiologic studies to produce one unit risk estimate. The range did not reflect uncertainties in the use of a linear vs. other type of dose-response model or uncertainties in the exposure estimates in the epidemiology studies. It also did not consider uncertainties in the dispersion and public exposure models. Therefore, the range cannot be used as a "statistical confidence interval" around the risk estimates predicted by the modeling procedure as a whole. Actual risk could lie outside of the range. A range was presented to give the reader an idea of the wide margin of uncertainty in the risk assessment.

Since proposal, EPA's unit risk factor for arsenic has changed from 2.95×10^{-3} to 4.29×10^{-3}. The new value is based on 5 epidemiologic studies (EPA-600/8-83-021F). A detailed summary of the derivation of the current unit risk factor is given in each of the BIDs. Specific procedures used to calculate unit risk and comments on the unit risk estimate are addressed

in section 2.0. Uncertainties in the unit risk factor contribute uncertainty to the aggregate risk and maximum lifetime risk as predicted by the HEM.

The unit risk factor, which defines the relationship between exposure and lung cancer risks in the linear, non-threshold model was derived from workplace epidemiological studies. The Agency has assumed that the same dose/response relationsip calculated at the higher exposures characteristic of the workplace holds at the lower public exposure levels. There are no arsenic data available to confirm EPA's assumption. As mentioned in the Federal Register notice, data on other human carcinogens have indicated that the linear, non-threshold model provides a plausible, upper-bound limit on public risk at lower exposure levels if the exposure is accurately quantified. Thus, as a matter of prudent public health protection policy, and based on EPA's understanding of the health effects data, the Agency has selected the linear, non-threshold model to estimate cancer risks.

When using the risk model for decision-making purposes, it is important to recognize the sources of uncertainty in the final output. Some issues raised which the model did not consider quantitatively are:

- effects of exposure on sensitive subpopulations,
- effects of exposure to other carcinogens on a person's probability of contracting cancer when exposed to arsenic,
- workplace exposure and exposures at locations other than the population centroid of the census area where people currently live,
- probability of arsenic-related health effects other than lung cancer.

To the extent possible, the Administrator considers these factors qualitatively in his decision-making process, along with the estimates made using the exposure and risk models. He understands that the lung cancer risks may not present the total health risk picture for the arsenic sources.

4.2.6 Summary

The EPA has estimated public exposure to ambient arsenic and associated risks using the Human Exposure Model. While EPA recognizes that there are uncertainties in the risk estimates, the Agency believes that the methods used represent a reasonable approach and the results reflect the best estimates that the Agency can produce within the available resources. Where possible, EPA has confirmed the predicted concentration profile by obtaining available arsenic data and comparing these data to the predictions. In several cases, EPA conducted site-specific air dispersion analysis, considering on-site or local meteorology and terrain features to improve its risk estimates.

4.3 ADDITIONAL COMMENTS AND RESPONSES ON RISK DETERMINATION AND MANAGEMENT ISSUES

4.3.1 Estimation of Exposure Through Measurement of Urinary Arsenic Concentrations

Comment:

One commenter (IV-D-593) suggested that exposure could be measured by measuring arsenic in the hair and urine of children. Another commenter (IV-F-3.2/IV-D-621-14.2) has studied urinary arsenic concentrations in the vicinity of the ASARCO-Tacoma smelter. His results are as follows:

(1) Urinary arsenic levels show a linear decrease with distance from the stack up to a distance of 2-1/2 to 3 miles. After that point, levels are what he considers normal.

(2) Part of the exposure appears to be due to inhalation because urinary arsenic levels in children near the smelter correlated with wind direction.

(3) These findings indicate that low-level emissions contribute more to exposure than stack emissions. Stack emissions would blow over people near the plant where the highest urinary arsenic concentration is found. No basic trend in urinary arsenic levels over time was observed in spite of the fact that stack emissions had been reduced.

(4) Urinary arsenic levels correlate well with concentrations at ASARCO's monitoring stations.

(5) Vacuum cleaner samples taken at homes where urinary arsenic was sampled show a linear decrease to 2-1/2 miles, and then level off.

(6) Relatively few samples were taken due to the expense involved.

ASARCO (IV-D-621-13) included a description of a urinary arsenic study done by the State government in 1976. The study was designed to investigate arsenic exposure to Vashon Island residents. Samples from 22 residents and 110 school children were collected on a day when winds were blowing toward Vashon Island and exposure levels were estimated to be at a maximum. For the northern end of the Island, the average urinary arsenic concentration found was 0.03 ppm, and for the southern end (which is closer to the smelter) it was 0.02 ppm. About 0.014 ppm was considered normal. Arsenic concentration did not appear to correlate well with age, and the averages by age group ranged from 0.018 to 0.036 ppm. The mean arsenic level for people who had recently eaten seafood was 0.09 ppm. Another sampling was planned, but ASARCO felt these results indicated that Vashon Island residents were not being exposed to detrimental levels of arsenic.

One commenter (IV-F-5.7/IV-D-621-15.9) said it is not clear that total excess arsenic exposure in local children is due to current smelter emissions. Even in the absence of current emissions, children may have

increased arsenic levels. Another commenter (IV-D-630) said children near the smelter have high urinary arsenic levels indicating high exposure. He did not provide any specific data.

Response:

Urinary arsenic levels have been shown to increase when arsenic is inhaled (EPA-600/8-83-021F). Several of the urinary arsenic studies cited above provide additional evidence for EPA's assertion that the population is being exposed through inhalation to arsenic emitted by the smelter. The urinary arsenic studies also indicate that exposure is highest near the plant. This is in agreement with both modeling and ambient monitoring results.

However, EPA has not used urinary arsenic concentration as a measure of public exposure to smelter emissions or lung cancer risks. The primary reason is that urinary arsenic levels reflect many factors in addition to the inhalation of arsenic emitted by the smelter. Diet, in particular the consumption of seafood, can account for increases and decreases in urinary arsenic concentrations. Also, the particular species of arsenic compound will affect the way the body reacts to and the rate in which the blood stream absorbs the contaminant. Individual metabolism and age may also cause variations in the amount of arsenic excreted. As shown in the study ASARCO refers to, individuals living in the same area from which urine samples were taken on the same day showed a range of arsenic levels. Thus, urinary arsenic levels cannot be used to estimate exposure to air emissions from ASARCO only, because other sources of exposure can contribute to arsenic concentrations measured in urine. To get a good "map" of exposure, one would have to measure urinary arsenic levels in many individuals living at many different locations at different times of the year under a variety of wind conditions. Dispersion and exposure modeling is a much more practical approach.

There are no data to support commenters' contention that there is no risk if arsenic levels are measured as "normal." According to modeling and ASARCO and EPA monitoring data, people beyond 2-1/2 km from the

smelter are inhaling measurable levels of arsenic. Under EPA's presumption there is no threshold for arsenic and some risk must be recognized to exist even at low doses (see section 2.0, on health effects). Modeling is the only approach available to EPA to quantify these risks due to low level exposure. These risks are then considered in setting standards.

4.3.2. Miscellaneous Comments on Risk Determination

Comment:

One commenter (IV-D-625) suggested EPA provide separate risk estimates for each arsenic compound or valence identified as hazardous.

Response:

For reasons described in section 3, EPA has decided to regulate inorganic arsenic compounds as a class rather than establishing separate standards for individual compounds. Several problems would arise if EPA tried to do a separate risk analysis for each compound. First, there are monitoring, sampling, and analysis problems which would make it difficult to estimate emissions of each. Second, reactions in the atmosphere may convert trivalent arsenic to the pentavalent form; some of each form may continue to exist in the atmosphere. Thus the chemical compounds emitted may not be the same compounds to which people are exposed. Atmospheric reactions are uncertain and cannot be adequately modeled at this time. This uncertainty in the form of arsenic received by receptors makes exposure analyses for individual compounds extremely difficult. Third, several workplace epidemiologic studies link exposure to both trivalent and pentavalent inorganic arsenic with lung cancer. Analysis of the data indicate that the potency of arsenic in either valence state is approximately the same. These studies provide an adequate basis for risk analysis for inorganic arsenic as a category without further separation of compounds.

Comment:

Two commenters (IV-F-1.6, IV-D-621-16.10) said that the unit risk estimate is based on lung cancer mortality data rather than incidence data,

so "cancer incidence per year" is a misnomer. It should be cancer "mortality."

Response:

This statement is accurate. The term "cancer incidence per year" should be read to mean "incidence of fatal lung cancers per year." However, with lung cancer, there is little difference between incidence and mortality since about 90 percent of those who contract lung cancer die within 5 years.

Comment:

One commenter (IV-D-621-14.8) questioned whether the annual average, geometric mean, or maximum 24-hour concentration would be used in the risk estimation if good ambient data were available.

Response:

Long term (at least annual average) concentrations are the basis for EPA's risk model. Cancer risks are proportional to long-term exposure to arsenic (under EPA's modeling assumptions). Therefore, long-term average ambient air concentrations of inorganic arsenic should be used in the exposure model.

Comment:

One commenter (OAQPS-79-8/IV-D-27) said EPA failed to consider available data or check the model against it. He said a similar flaw led the 5th Circuit Court to set aside the CPSC formaldehyde insulation rule.

Response:

The case cited by the commenter, Gulf South Insulation v. Consumer Products Safety Commission, 701 F.2d 1137 (5th Cir. 1983) deals with the Consumer Product Safety Act. The EPA notes that the legal analysis in this case cannot be directly applied to actions under Section 112 of the Clean Air Act, because Section 112 imposes defferent requirements from the CPS Act. For example, under Section 2058 (f)(3)(A) of the Consumer Product Safety Act, the Commission is expressly prohibited from promulgating a safety rule unless it finds that the product that will be subject to the rule poses an unreasonable risk of injury. Section 2060 (c) provides

that a consumer product safety rule shall not be affirmed "unless the Commission's findings....are supported by substantial evidence on the record taken as a whole. "The court read this as justification for a stricter acrutinization of the Commission's actions than the "arbitrary and capricious" standard would allow. 701 F.2d at 1142.

It is believed EPA's data and risk estimation procedure satisfy the requirements of Section 112 and show that arsenic emitted by copper smelters and glass plants may reasonably be anticipated to result in an increase in lung cancer. Therefore, EPA is proceeding with promulgation of emission standards.

The commenter cites inadequacy of the data base as a reason the formaldehyde rule was set aside and as a possible court challenge to EPA's arsenic NESHAP. The data base CPSC used to perform its formaldehyde insulation risk analysis was deemed inadequate by the court. This was largely because emissions and exposure to formaldehyde were determined by testing of unrepresentative buildings by a variety of test methods, some of which have not been approved. Secondly, in estimating health risk from exposure, CPSC extrapolated from the results of one rat study rather than using human epidemiologic data.

The EPA's arsenic data base is believed to be adequate to support a risk analysis which can meet the "reasonably anticipated" criteria of section 112. The EPA has used test data obtained by approved methods from various sources in generating emissions data. Pollutant dispersion and public exposure have been calculated using reasonable, widely accepted dispersion and exposure models. Since the proposal, EPA used ambient monitoring data collected by the States and the companies and compared this data to the air dispersion results. Health risk has been estimated from 5 human epidemiologic studies which link ambient arsenic exposure to increased lung cancer mortality (EPA-600/8-83-021F). While there are uncertainties in the data bases and modeling procedure, EPA has sufficient evidence that arsenic emissions from primary copper smelters and glass manufacturing plants may reasonably be anticipated to pose a significant public health risk and should be regulated under section 112 of the Clean Air Act.

4.3.3 Uses of Risk Analyses in the Decision-Making Process

Comment:

One commenter (OAQPS-79-8/IV-D-27) cited several court decisions which he said had a bearing on the use of risk assessment by regulatory agencies. He cited a Supreme Court decision made in Industrial Union Department of the AFL-CIO v. American Petroleum Institute, 448 US 607 (1980), saying that a significant risk at expected exposures must be demonstrated and that the proposed remedy must provide a significant reduction in that risk before a rule can be promulgated. In Monsanto v. Kennedy 613 F.2d 947 (D.C.Cir. 1979) he said the courts stated that risks cannot be inferred or assumed but must be found by a reliable scientific process. In Marshall Minerals v. FDA 0661 F.2d 409 (5th Cir. 1981) he said the Court specifically rejected the position that all neoplastic response may be deemed cancer and said that such substances must be evaluated for risk under the intended conditions of use.

Response:

In response to the first comment EPA judges that inorganic arsenic does pose a significant risk, and that these standards significantly reduce that risk.

The Monsanto Co. v. Kennedy case centers on the definition of a food additive and the determination if a substance (acrylonitrile) used in food packaging materials should be considered a food additive under the Federal Food, Drug, and Cosmetic Act. It appeared the FDA Commissioner relied on the general principle of diffusion in establishing that acrylonitrile present in beverage cans in very low quantities would migrate into food in significant amounts. Tests showing migration had been done on cans with higher levels of acrylonitrile. But migration into food from the cans in question was too low to detect. The court ruled that in such a case the Commissioner had the latitude to consider migration insignificant and was not mandated to regulate low levels of the substance in packaging as a food additive. They remanded the decision to the FDA Commissioner for further consideration 613 F.2d. at 955-956 (1979).

In this rulemaking, risk has not just been "assumed" as claimed by the commenter. Evidence is presented in the Health Assessment Document and in the listing decision published in the Federal Register (EPA-600/8-83-021f and 44 FR 37886, June 5, 1980). The calculation of risk for the arsenic sources is presented in Section 4.2. Reliable scientific evidence has been used where available in making this risk determination.

In Marshall Minerals v. FDA, FDA, supra denied a request for a public hearing on a petition for food additive regulation. The issues referred to by the commenter were considered by the court because under section 348 (c)(3)(A) the FDA is to make an evaluation of whether the food additive "under the conditions of use" induces cancer when ingested by man or animal.

The commenter implies EPA has no evidence arsenic causes cancer in humans and has not considered conditions of public exposure. The EPA has evidence that inhalation of arsenic by smelter workers causes increased lung cancer incidence and mortality (EPA-600/8-83-021F). Lung cancer, not just neoplastic responses, is associated with arsenic exposure.

Comment:

One commenter (IV-D-698) said section 112 of the Clean Air Act does not authorize reliance on risk analysis to identify the level to which emissions must be controlled. Another commenter (IV-D-590) saw the use of risk assessment in standards development as inconsistent with the section 112 directive to provide an ample margin of safety. Another commenter (IV-D-710) said risk estimates were too uncertain to be used to justify non-application of available controls, and that available technology should be applied to all sources of emissions of a plant before risk assessment is used in decision-making.

The Office of Management and Budget (OMB), on the other hand, (IV-D-618) believes risk assessment should be used by EPA at all stages of its standards-setting procedure. Currently, OMB said, EPA uses it to estimate residual risk after BAT and to determine if further control is necessary. OMB felt that risk assessment should be used in determining whether BAT need be applied. The commenter stated that the best estimate of likely effects rather than conservative estimates should be used.

Response:

The EPA believes that the quantitative estimation of health risk is a reasonable and necessary part of the decision to regulate sources under section 112 of the Clean Air Act. For carcinogenic pollutants such as arsenic for which a health effect threshold has not been conclusively demonstrated, any level of control short of an absolute ban may pose finite health risks. The EPA believes Congress did not intend that emissions standards of zero must be set for such pollutants. Such an intent could cause wide-scale industrial shut-down and considerable economic disruption. Thus, standards which permit some level of residual risk must be considered to provide the "ample margin of safety" to protect public health specified in section 112 of the Clean Air Act.

Risk assessment models represent the best, and often the only, tool available to EPA to determine the health impacts associated with various control alternatives. Therefore, EPA believes that it is appropriate that risk assessment play a role, along with other criteria, in the section 112 regulatory process. In setting standards for a plant or source category, EPA will examine current controls and each regulatory option including application of various technologies, substituting feedstock materials, and closing the plant. The control efficiency, technical feasibility, cost, and reductions in risk as estimated by the risk assessment will be among the impacts considered for each option. In choosing the control option, the Administrator considers whether the estimated risks remaining after each successively more stringent option are unreasonable. This is a judgemental evaluation of the estimated maximum lifetime risk and cancer incidences per year remaining after each control option, the impacts (including economic impacts) of further reducing those risks, and the benefits of the substance or activity producing the risk.

In all cases where risks and other parameters are estimated, the significant uncertainties associated with these numbers will be weighted carefully in reaching the final decision.

Comment:

Several commenters expressed opinions on which measure of risk should be used by EPA in regulatory development. These follow:

OMB (IV-D-618) felt that aggregate (population) risk is a better measure of public health than individual risk. According to OMB, individual risks should only be considered if they are unusually high. Commenter IV-D-673 believed the size of the exposed population and aggregate risk should be used in determining whether a source should be allowed to continue operation. Commenters IV-F-1.17 and IV-D-401 also favored using cancer incidence rate to set priorities, and controlling situations where many people are exposed before those in which few people are exposed.

One commenter (IV-D-618) said that EPA needs to decide what weighting to give the estimated risk for the most exposed individual in comparison to the estimated aggregate population risk. He stressed that a decision to give more weight to the most exposed individuals would likely result in a more extensive regulatory intervention without commensurate public health gains.

One commenter (IV-D-617) pointed out that no consideration is given to the number of people exposed to the maximum lifetime risk. On the other hand, one commenter (IV-D-641) felt that people living in low population density areas should not be subjected to higher individual risks than those living in high density areas.

Response:

The EPA represents cancer risk in two ways. The first is individual risk or "maximum lifetime risk", and the second is annual incidence, a measure of population or aggregate risk. These are described in section 4.2. Such measures aid EPA in estimating if the emissions standard will protect both the highly exposed individual and the public at large. Therefore, EPA considers both expressions of risk in their regulatory development procedure.

5.0 PIECEMEAL APPROACH

5.1 EXPOSURE TO CHEMICALS OTHER THAN ARSENIC

Comment:

Several commenters (IV-D-61, IV-D-120, IV-D-142, IV-D-164, IV-D-443, IV-D-541, IV-D-592, IV-D-731, IV-F-3.37, IV-F-3.51, IV-F-3.55,IV-F-11) maintained that the environmental and health problems due to smelters (mostly made in reference to ASARCO-Tacoma) are not confined to current emissions of arsenic to the atmosphere. They cited environmental problems in other media and problems caused by other pollutants from historical emissions, as well as current and future emissions. These commenters urged EPA to find a way to look at the problem as a whole. They objected to EPA's piecemeal approach.

Several commenters (IV-D-20, IV-D-61, IV-D-69, IV-D-85, IV-D-87, IV-D-104, IV-D-106, IV-D-111, IV-D-112, IV-D-115, IV-D-137, IV-D-164, IV-D-416, IV-D-417, IV-D-427, IV-D-429, IV-D-541, IV-D-551, IV-D-557, IV-D-597, IV-D-666, IV-D-677, IV-D-698, IV-D-710, IV-D-719, IV-D-731, IV-F-3.31, IV-F-3.37, IV-F-3.73, IV-F-3.103, IV-F-4.11, IV-F-4.31, IV-F-4.43, IV-F-9, IV-F-11) said EPA should take into account public exposure to hazardous chemicals other than arsenic. Many (IV-D-6, IV-D-11, IV-D-13, IV-D-21, IV-D-35, IV-D-36, IV-D-38, IV-D-39, IV-D-41, IV-D-43, IV-D-61, IV-D-71, IV-D-76, IV-D-114, IV-D-144, IV-D-164, IV-D-404, IV-D-438, IV-D-443, IV-D-554, IV-D-558, IV-D-592, IV-D-593, IV-D-644, IV-D-660, IV-D-666, IV-D-670, IV-D-677, IV-D-705, IV-D-710, IV-F-3.17, IV-F-3.31, IV-F-3.37, IV-F-3.40, IV-F-3.55, IV-F-4.6, IV-F-4.11, IV-F-4.12, IV-F-4.19, IV-F-4.50, IV-F-5.1, IV-F-5.9, IV-F-5.13, IV-F-9, IV-F-11) specifically mentioned that risk from cadmium, SO_2, lead, copper and antimony should also be considered. Another (IV-F-4.31) noted that EPA should consider the possible synergistic effects among various pollutants. Some (IV-D-593, IV-F-4.43) said that by dividing the problem up into many segments and considering only arsenic, total risk could be underestimated since small segments appear less dangerous than the total picture. Another (IV-D-20) asked that EPA advise the public of the risks of cigarette smoking coupled with arsenic exposure.

One commenter (IV-F-4.50) said that while EPA may be able by law to consider the effects of multiple chemicals separately, those who live with the problem of multiple exposure cannot. Others (IV-F-3.73, IV-D-754) said that the same system that requires EPA to investigate the smelter ties the hands of the investigators by forcing a piecemeal approach. He continued by stating that the studies are too narrow in scope, taking into account only part of the pollutant source and only some of the health effects.

Three commenters (IV-D-70, IV-D-164, IV-F-4.43) said that regulation would be more efficient if other pollutants were considered. One (IV-F-4.43) suggested tradeoffs between different pollutants could be allowed to reduce total risk to a target number. One commenter (IV-D-164) felt that when the piecemeal approach was used, regulations causing a significant change do not result. Another commenter (IV-D-70) suggested it might be simpler and more efficient for some industries to regulate hazardous air pollutants by category rather than by specific pollutant.

One commenter (IV-D-120) said that section 112 of the Clean Air Act is limited only to airborne emissions, resulting in a dependence on Superfund and other statutes for significant reduction in health risk. He continued by stating that many people have been frustrated about the piecemeal approach of the statutes to addressing pollution. Other commenters (IV-D-571, IV-D-783) stated that under the Clean Air Act, EPA has the authority and responsibility to take into account such factors as "non-air quality health and environmental impact" when formulating regulations (section 119 (2)(C)(3)(b)(3)).

Several commenters (IV-D-427, IV-D-592, IV-D-593, IV-D-698, IV-F-3.55, IV-F-4.31, IV-F-9, IV-F-10, IV-F-11) said that arsenic has accumulated in the environment over the years, and can reenter the air, water, or food chain and expose people. They noted that considering current emissions without regard to past history results in underestimation of exposure and risk. One commenter (IV-D-571) requested that information concerning the additional load of hazardous waste be included in the determination of the level of the standard.

Some commenters (IV-D-116, IV-D-433, IV-D-515, IV-D-520, IV-D-571, IV-D-579, IV-D-591, IV-D-592, IV-D-698, IV-D-710, IV-F-3.42, IV-F-4.10, IV-F-5.7, IV-F-9, IV-F-10, IV-F-11) suggested that EPA consider more complex routes of exposure from arsenic emissions. In addition to ambient arsenic levels, the commenter said that reentrainment of dust and secondary ingestion via food, water, and soil should be considered. Two commenters (IV-F-3.42, IV-F-4.10) contended that these routes are especially important for children. However, another commenter (IV-D-621-15.6) said that arsenic concentrations in soil and water were generally low, and arsenic in the soil generally forms insoluble complexes with amorphous aluminum or iron oxide. He noted that this makes arsenic less hazardous, even if dust is reentrained. Therefore, he concluded that EPA and the public should not be overly concerned about these exposure pathways.

Response:

The EPA's estimates of the health risks posed by arsenic emissions from the primary copper smelters do not include those risks attributable to emissions of other regulated air pollutants or pollutants that are candidates for regulation. For this reason, EPA's approach has been criticized as a partial response to the total health hazard and one which perpetuates an interactive strategy that is less efficient and less desirable to all parties concerned.

Although this rulemaking addresses inorganic arsenic emissions, and is being conducted under section 112, the Administrator believes that it is unreasonable to fail to consider the other pollutants emitted by a source and other potential environmental impacts. Because new control technologies and smelter processes that affect arsenic emissions also affect other pollutants, the Agency believes that consideration of all environmental concerns is a necessary and important element in the risk management process. Consequently, EPA considered the impact of the inorganic arsenic copper smelter standards on emissions of other pollutants and the actions

being taken under other environmental statutes to address other environmental impacts of the smelter. Specific actions considered included actions being taken by various state agencies and EPA to reduce PM and SO_2 emissions from smelters and actions being taken to reduce occupational exposures to arsenic.

Although the primary copper smelter standards are directed toward reducing inorganic arsenic emissions, they regulate particulate matter emissions. Consequently, emissions of other pollutants (e.g., cadmium, lead or antimony) which are also present in particulate matter will also be reduced under the standards. Emissions of gaseous pollutants, such as SO_2, are not limited by today's standards; however, there are other regulations that limit emissions of these pollutants from the primary copper smelters. The Administrator has considered in the development of standards the control actions (and the compliance schedule) that are being taken by the various smelters.

As indicated in a previous section, EPA's assessment of human exposure to arsenic presently being emitted from primary copper smelters includes the use of available monitored air concentrations. At the same time, EPA recognizes that some portion of the arsenic to which nearby residents are exposed may not be subject to prediction by dispersion models that consider only current emissions from the source. There is a growing body of scientific data indicating that historical emissions of arsenic from smelters may, through accumulation in soil and dust, deposition on or incorporation into food, and via re-entrainment into the air, contribute to human exposure.

The EPA has attempted to evaluate and consider such risks explicitly in the regulatory decision process through the use of monitored data, which should include the effects of re-entrained arsenic, to estimate health risks. The smaller the estimated ambient concentrations in comparison to those measured by the air quality monitors, the greater the concern that

sources of arsenic other than current, direct air emissions are contributing to ambient levels. However, as previously mentioned, EPA's predicted arsenic concentrations are about the same or sometimes even higher than the measured values collected near the stet at El Paso, air dispersion model consistently underestimated measured concentrations. Although the available data do not allow the Agency to accurately quantify the impact of other exposure pathways, the Administrator has considered this potential in the regulatory decision.

Other environmental impacts of the smelter are being studied by EPA and other agencies and efforts are underway to assess the several problems identified by public comments. The 1980 Comprehensive Environmental Response Compensation and Liability Act (Superfund) designed for EPA to take actions needed to protect public health from exposure to hazardous substances in all environmental media, is being used to investigate other pollutants, such as cadmium and lead, and to remedy the problems resulting from multimedia exposure to these pollutants in the vicinity of the ASARCO-Tacoma smelter (see Chapter 2). Several investigations funded in part or entirely by the Superfund program are underway or being developed to study the potential health problems resulting from the historical accumulation of arsenic, lead, and cadmium. The EPA believes that this work will aid in the characterization and resolution of the environmental problems associated with the ASARCO-Tacoma smelter's operations as well as those problems associated with the other primary copper smelters.

The Administrator also recognizes that even at the control levels required by these standards that some degree of accumulation of arsenic and heavy metals in the soil may occur. The EPA believes, however, that the present levels of these materials in other environmental media are largely the result of the much higher emissions from the smelter before effective control equipment was installed. Emissions have decreased significantly over the past 20 to 30 years. Although the standards will not eliminate arsenic and heavy metal deposition, EPA believes that the controls will further reduce emissions significantly and will reduce the rate of accumulation in the environment.

6.0 EPA'S STATUTORY OBLIGATION UNDER SECTION 112

This section contains comments and responses concerning EPA's statutory obligation under section 112. These comments have been divided into four major subsections:

6.1 Acceptable Risk/Ample Margin of Safety
6.2 BAT Approach
6.3 Economics/Costs As A Decision-Making Criterion
6.4 Recommended Action in the Face of Uncertainty

6.1 ACCEPTABLE RISK/AMPLE MARGIN OF SAFETY

6.1.1 EPA's Responsibility Under the Clean Air Act

Comment:

Some commenters (IV-D-53, IV-D-106, IV-D-107, IV-D-137, IV-D-144, IV-D-158, IV-D-164, IV-D-411, IV-D-439, IV-D-627, IV-D-718, IV-D-724, IV-D-731, IV-D-747, IV-F-1.18, IV-F-3.31, IV-F-3.40, IV-F-3.57, IV-F-4.3, IV-F-4.6, IV-F-4.11, IV-F-4.15, IV-F-4.24, IV-F-4.50, IV-F-4.55, IV-F-4.59, IV-F-4.66) pointed out to EPA that the Clean Air Act of 1970 and 1977 requires that the public must be protected with an "ample margin of safety" from hazardous pollutants. Some said that EPA should implement the Clean Air Act as passed by Congress (IV-D-66, IV-D-107, IV-D-224, IV-D-411, IV-D-662, IV-F-1.18). One commenter (IV-D-45) said that it should not be up to the discretion of either the public or EPA to waive the law as established by the Clean Air Act to provide an ample margin of safety for toxic emissions.

Some commenters (IV-D-25, IV-D-39, IV-D-53, IV-D-104, IV-D-106, IV-D-107, IV-D-111, IV-D-112, IV-D-115, IV-D-137, IV-D-144, IV-D-158, IV-D-161, IV-D-414, IV-D-420, IV-D-422, IV-D-429, IV-D-437, IV-D-443, IV-D-731, IV-D-747, IV-D-530, IV-D-541, IV-D-580, IV-D-632, IV-D-662, IV-D-663, IV-D-666, IV-D-673, IV-D-677.3, IV-D-677.5, IV-D-677.7, IV-D-677.8, IV-F-1.16, IV-F-1.17, IV-F-3.31, IV-F-3.60, IV-F-3.103, IV-F-4.6, IV-F-4.43, IV-F-4.66, IV-F-9, IV-F-10) said that EPA's

proposed arsenic standards are entirely inadequate because the standards would not provide an "ample margin of safety" from toxic emissions. Others (IV-D-111, IV-D-115) said that the proposal does not comply with EPA's obligation to protect the public. Others (IV-D-698, IV-F-1.18, IV-D-732) said that EPA had failed to provide the protection of public health required by section 112 of the Clean Air Act. Still another commenter (IV-D-660) urged EPA to issue standards requiring a reduction in emissions to levels which permit the public to live not only safely, but pleasantly. The United Steelworkers of America (IV-D-708) said that the standards might not assure an adequate margin of safety to the exposed public.

One commenter (IV-F-1.103), who felt the proposed standards fell short of providing an ample margin of protection, was concerned that in proposing a standard which would allow such residual risks the present EPA administration is attempting to establish a precedent for a weaker risk exposure criterion than has been used as the basis for other environmental protection standards. Another commenter (IV-F-1.18) said that the current level of risk that EPA has proposed under the "margin of safety" mandate, makes one shudder to speculate the level of protection the Agency might provide where the statutory mandate is not as explicit.

Some commenters (IV-D-137, IV-D-259, IV-D-310, IV-D-545/IV-D-621-16.6/IV-F-24) said that the standards set by EPA are adequate to protect the health of the citizens living in the local communities. One commenter (IV-D-545/IV-D-621-16.6/IV-F-24) qualified his position by stating that if the proposed standard is not adopted, further delays in the reduction of arsenic emissions would result. Others (IV-D-567, IV-D-568, IV-D-621-6, IV-D-621-7, IV-D-628, IV-F-3.12, IV-F-3.18) said that the current emission standards provide "an ample margin of safety" to protect public health. Others (IV-D-621-15.1, IV-D-621-15.7, IV-F-3.15) said that in their judgement current exposure levels provide a vast margin of safety with respect to pulmonary carcinogenic risk from airborne arsenic exposure in the environs of the ASARCO smelter.

One commenter (IV-D-621-15.9/IV-F-3.15) said that the proposed regulations will most likely reduce the total amount of arsenic emissions from the smelter but the airborne levels of arsenic may or may not be reduced proportionally. The commenter continued saying that since lung cancer risk at current levels of exposure is not expected, there would not be any reduction in health risk as a result of new regulation, rather only an increase in the margin of safety would be attained.

Response:

Section 112 does require that EPA set standards that provide an "ample margin of safety." Where a health effects threshold can be determined, this requirement can be met by establishing the standard at a level that insures that the exposure threshold is highly unlikely to be exceeded. Where identifiable thresholds do not exist or are indeterminate, as with carcinogens, any level of control selected short of an absolute ban on emissions, may pose a finite carcinogenic risk.

In establishing the appropriate level of control for carcinogens, therefore, the Administrator views the objective as a judgement of the extent to which the estimated risk of cancer must be reduced before the degree of control can be considered amply protective. Two choices are available: either the emission standards must be set at zero to eliminate the risk of cancer altogether, or some residual risk must be permitted. Neither the language nor the legislative history of section 112 reveals any specific Congressional intent on how to apply the phrase "provides an ample margin of safety to protect the public health" to non-threshold pollutants like inorganic arsenic that present cancer risks at any level of exposure (48 FR 33116, July 20, 1983).

In the absence of specific direction from section 112, in recognition of the drastic economic consequences that could follow a requirement to eliminate all risk from carcinogenic emissions (see Zero Risk section), EPA believes that it is not the intent of this section to totally eliminate

all risks. Therefore, EPA believes that the final inorganic arsenic standards which permits some level of residual risk provides that is not unreasonable in light of the impacts associated with requiring further control.

6.1.2 What is an Acceptable Risk Level

Comment:

Some commenters (IV-D-164, IV-F-4.59) defined acceptable risk as that which provides an ample margin of safety. One commenter (IV-D-144) agreed with a newspaper article which stated that "ample" means what any intelligent Congress would have been aiming at all along; it denotes the point where we begin to prefer savings over greater safety. Another commenter (IV-D-25) referred to Webster's dictionary for the meaning of the word "ample." He said that it means "of large size, extent, capacity, volume, or scope" and "more than adequate."

One commenter (IV-F-1.18) stated that the determination of an acceptable risk level is a societal decision and each pollutant must be looked at separately. Another commenter (IV-D-621-14.7) felt that risk management, unlike risk assessment, is not a scientific decision; it depends on politics, economics, technology, and public perception of the extent of the risk. Still another (IV-F-4.11) felt that determination of ample margin of safety is a policy issue that involves weighing properly identified risks and benefits.

Response:

The EPA agrees with the commenters who stated that the determination of an acceptable risk level depends not only upon health considerations but upon economics and technology. However, neither the language nor the legislative history of section 112 reveals any specific Congressional intent on how to apply the phrase "provides an ample margin of safety to protect public health" to nonthreshold pollutants like inorganic arsenic that may present cancer risks at any level of exposure. (See previous response for further details on EPA's risk management approach.)

Comment:

Several opinions were expressed concerning what should constitute an acceptable level of risk. Some commenters (IV-D-618, IV-D-627, IV-F-3.55) stated that it is very difficult to determine an acceptable level of risk because it is not easy to prove where the line between danger and safety lies. However, the Chemical Manufacturers Association (IV-D-617) noted that in the past EPA has identified levels of increased risk that are so negligible that they can be deemed fully consistent with the protection of public health.

One commenter (IV-F-3.3) stated that acceptable risk levels have varied from 0.10 for OSHA standards to zero. He did not think that anyone knows what level of health risk is acceptable. Another (IV-F-4.62) felt that one microgram per cubic meter should provide an ample margin of safety for the community. One commenter (IV-D-657) believed that the small health risk that currently exists is acceptable at this point in time, while others (IV-D-630, IV-D-622, IV-D-722) thought it is not acceptable. One commenter said traces of arsenic in children's urine is unacceptable. Another said that closure of the plant may create greater overall public risk than exists presently.

One commenter (IV-F-3.3) felt that while the acceptable level is being determined, ASARCO-Tacoma should continue to reduce arsenic emissions. In contrast, the Chemical Manufacturers Association (IV-D-617) felt that the question of what level of risk is significant must be answered by EPA before establishing regulations. Similarly, another commenter (IV-D-621-14.7) stated that it is necessary for EPA to make a judgment about what level of risk would be unacceptable, in order for EPA to set priorities. Finally, one commenter (IV-D-621-14.11) felt that effective regulation requires a determination of what levels of residual risk, if any, should be tolerated. He asked whether the level of arsenic is low enough now or can be made low enough that it is judged to be a risk not worth further action.

One commenter (IV-D-609) stated that by not promulgating an ambient air quality standard, EPA has allowed any measured concentration levels in the community to be considered acceptable. In contrast, other commenters

(IV-D-621-16.10, IV-F-1.6) felt that the arsenic proposal appears to contain an unannounced policy definition of acceptable risk for environmental exposure.

Some commenters (IV-D-621-16.10, IV-F-1.6) said that EPA's arsenic proposal appears to be based on a policy decision that "unacceptable risk" exists when the maximum lifetime risk to some exposed population is greater than 10^{-4}. Another commenter (IV-D-621-14.9) pointed out that EPA's normal acceptable risk level is 1 in 100,000. Another commenter (IV-D-718) stated that EPA has drifted away from the previous acceptable lifetime risk levels of 1 in 1,000,000 and 1 in 100,000. She states that EPA's proposed standards will set the level down to 2 in 100, and that this is not fulfilling the responsibilities of EPA.

One commenter (IV-D-241) said that the controversy over the proposed standards centers around the question, "What is acceptable risk?" He continued saying that one cannot determine a risk/benefit ratio until an agreement is reached concerning how many arsenic-related deaths are "acceptable." The Clean Air Act as it now stands, he said, sets the risk factor (for all toxins) at 1 in a million, an unhappy compromise as it is, since it is obvious that there is no safe threshold for toxic emissions. However, according to the commenter, the newly proposed arsenic emissions regulations would condone an estimated national ratio of 9.4 to 150 deaths per 10,000 lifetime exposures to such emissions. Where is the concern for the public in such a ratio, and who benefits, he asked?

The New Jersey Department of Environmental Protection (IV-D-641) suggested that EPA initially concentrate on controlling existing sources to below a risk of 1,000 in one million. LAER could be required for sources which exceed that risk level. BAT would be required for existing sources with risks between 1,000 in a million and 1 in a million. NESHAPS regulation would not be required for sources with risks of less than 1 in a million.

The Natural Resources Defense Council (NRDC) (IV-D-710b) stated that they could accept an Agency policy of technology-forcing which stops at the

point where the residual emissions are predicted to create an additional risk level of one in a million. At that point, NRDC suggested that EPA should move on to the next unregulated hazardous pollutant. In the future, NRDC said, it may be possible to further reduce the risk, but if EPA were working seriously on the backlog of unregulated hazardous pollutants, then for the present the protection of public health would be better served by moving on to the next substance.

Response:

Many of these commenters, in effect, are advocating that EPA establish a target risk level for setting standards under section 112. Under this approach, a fixed numerical risk or expected cancer incidence rate target could be used in determining the degree of control required for carcinogens. Although EPA finds the concept of an established "acceptable" risk level appealing, it suffers from several drawbacks. First, the Agency perceives substantial difficulty in determining such levels. This perception was borne out by the wide range of opinions of what constituted acceptability in the minds of the commenters. Second, although current quantitative risk assessment techniques for chemical carcinogens are useful decision-making tools, considerable uncertainties are associated with the techniques at their current stage of development. Consequently, the Administrator believes that in using quantitative risk assessments, he should generally be free to consider that actual cancer risks may be significantly above or below those predicted by the estimated procedures, and not be bound by a fixed target. Third, a fixed target level fails to provide the flexibiity necessary for an appropriate response. For example, where risks could be reduced beyond the target without significant costs, that should be permitted. Likewise, where attainment of the risk-based goal would eliminate a highly beneficial or necessary activity, the decision-maker should be able to consider less stringent standards. The EPA agrees with those commenters who perceived that specific acceptable risk levels are very

difficult to set and are not reasonable as a basis for regulation. After reflecting on the various points presented, the Administrator supports the concept of reducing public risks to the extent possible considering the uncertainty, technical feasibility, environmental, economic, energy, and other impacts on society and industry.

6.1.3 Zero Risk/Zero Exposure

Comment:

Many commenters (IV-D-8, IV-D-9, IV-D-33, IV-D-69, IV-D-71, IV-D-73, IV-D-88, IV-D-102, IV-D-105, IV-D-116, IV-D-144, IV-D-152, IV-D-161, IV-D-301, IV-D-302, IV-D-329, IV-D-401, IV-D-420, IV-D-424, IV-D-433, IV-D-440, IV-D-575, IV-D-583, IV-D-590, IV-D-596, IV-D-598, IV-D-610, IV-D-644, IV-D-661, IV-D-664, IV-D-676.1, IV-D-676.3, IV-D-676.4, IV-D-721, IV-D-725, IV-D-727, IV-D-730, IV-D-783, IV-D-709, IV-D-734, IV-D-744, IV-D-752, IV-D-753, IV-D-768, IV-D-778, IV-D-781, IV-D-784, IV-F-3.7, IV-F-3.29, IV-F-3.37, IV-F-3.51, IV-F-3.65, IV-F-3.66, IV-F-3.70, IV-F-3.74, IV-F-4.3, IV-F-4.28, IV-F-4.59, IV-F-5.13, IV-F-5.15, IV-F-5.22, (IV-D-1, IV-D-57, IV-D-62, IV-D-67, IV-D-72, IV-D-75, IV-D-98, IV-D-144, IV-D-163, IV-D-301, IV-D-400, IV-D-427, IV-D-524, IV-D-556, IV-D-557, IV-D-582, IV-D-598, IV-D-660, IV-D-677.1, IV-D-686, IV-D-689, IV-D-710, IV-F-3.38, IV-F-3.40, IV-F-3.51, IV-F-4.4, IV-F-3.103, IV-F-9, IV-F-10, IV-F-11) (IV-D-109, IV-D-161, IV-D-292, IV-D-329, IV-D-424, IV-D-587, IV-F-1.17, IV-F-3.65, IV-F-3.66, IV-F-3.70, IV-F-4.28, IV-F-5.13) IV-D-1, IV-D-57, IV-D-62, IV-D-67, IV-D-72, IV-D-75, IV-D-98, thought the emission standard should be set at a zero level for arsenic and other pollutants. The commenters reasoned that a zero level would protect the health and welfare of the community. Some commenters (IV-D-710, IV-F-4.66) felt that, using currently available information, it is not feasible for EPA to determine that any exposure to arsenic greater than zero will provide an ample margin of safety. Another commenter (IV-F-4.28) felt that if EPA permits

any emissions of a known carcinogen, a decision has been made by the Agency to use human subjects in research.

The Natural Resources Defense Council (IV-D-710) elaborated upon this thought by stating that the "ample margin of safety" requirement signifies a firmly held goal that no one should lose his or her life or health on account of toxic air pollution. NRDC continued by saying that the absence of identifiable thresholds does not permit the Agency to deem some rates of death or serious illness "insignificant." If one person living near a plant contracts cancer and dies, there has been a health effect of the most serious and final nature in NRDC's view. The fact that only one person died does not make the effect insignificant according to NRDC. The commenter representing NRDC said expressing the effect in terms of an individual risk only means that the death is acceptable because the cancer strikes randomly from a pool of people and the victim may not even be identified.

The Attorney General's Office of the State of New York (IV-D-698) stated that the courts have recognized that EPA may legitimately require zero emissions of hazardous air pollutants in order to meet section 112's mandate. They cited the case of United States v. Borden, Inc., 572 F. Supp. 684, 688 (D. Mass. 1983).

Another commenter (IV-D-25) said that EPA must establish standards for completely safe operations, free from any possibility of causing illness and/or impaired health through arsenic emissions to comply with the ample margin of safety requirement.

Some commenters (IV-D-621-15.2, IV-F-3.9) stated that there is a built in conflict suggested by an overall EPA policy based upon a zero-risk (no-threshold) level for hazardous pollutants, and the use of a risk assessment and risk management approach to determine what levels of risk are acceptable. Another commenter (IV-D-621-14.11) pointed out that since EPA uses a linear model, there will always be some residual risk unless all sources are cut to zero (i.e., the plant is closed and the contaminated soil is removed). One commenter (IV-F-3.3) interpreted the use of the linear no-threshold model to mean that EPA thinks there is no acceptable exposure level for arsenic.

Some commenters (IV-F-4.6, IV-F-3.103, IV-F-9) stated that it is not now possible to completely eliminate arsenic emissions from certain industrial processes, but EPA should have zero or near zero emission levels as an ultimate goal.

Some commenters (IV-D-621-16.9, IV-F-3.18, IV-F-4.15) stated that zero risk from arsenic can never be achieved. One commenter (IV-F-4.15) noted that even if the ASARCO plant closed, some residual risk would still remain from the build up of arsenic in the soil.

Some commenters (IV-D-146, IV-D-545, IV-D-621-16.6, IV-F-4.24) said that it is neither possible nor desirable to eliminate all risks and that life is filled with risks. Therefore, they did not see a necessity for a standard based on zero risk. In support of this view, one commenter (IV-D-621-16.2/IV-F-3.18) noted that even if manmade pollution is totally controlled, pollution caused by natural disasters (fires, storms, earthquakes, volcanic eruptions, etc.) cannot be controlled.

Several commenters (IV-D-125, IV-D-180, IV-D-621-15.2, IV-D-724, IV-F-4.2, IV-F-3.78) thought that a zero emission level is not possible or needed. Economic infeasibility was cited by some commenters (IV-D-125, IV-F-3.78) as a reason. One commenter (IV-D-154) did not believe one life was worth unlimited cost. Some commenters (IV-F-4.2, IV-F-3.78) stated that as a standard approaches the zero risk level, compliance costs increase rapidly, while the benefits are hard to quantify. Another (IV-D-125) went on to say that disasterous economic consequences would result if zero risk was required. He called the concept economic suicide and alarmism. Another (IV-D-724) said a zero risk level for arsenic and other carcinogens would prove socially catastrophic given the pervasiveness of at least minimal levels of carcinogenic emissions from American industries. In support of this view, some commenters (IV-D-617, IV-D-622, IV-F-3.4) felt that it was not the intent of EPA or Congress to interpret section 112 as requiring a zero emission level for arsenic. They concluded that such a requirement would shut down major segments of American industry. [See section entitled "Economics as a Decision Making Criterion Under

Section 112."] The Chemical Manufacturers Association (CMA) (IV-D-617) continued by saying that the agency's rejection of a zero-risk interpretation of section 112 is amply supported by legislative materials and a variety of administrative and judicial decisions in the health and safety area. Citing the legislative history of the 1977 Clean Air Act Amendments, CMA stated that Congress specifically rejected the suggestion that an ample margin of safety for no-threshold pollutants requires zero emission standards. CMA cited 1977 Legislative History at 1030-31, 2577-79, 2594, House Report, 1978; Comm. Print No. 16, Senate Comm. on Environment and Public Works.

Response:

The EPA and other public health agencies and groups have, as a matter of prudent health policy, taken the position that in the absence of identifiable effect thresholds, carcinogens may pose some risk of cancer at any exposure level above zero. In establishing margins of safety for carcinogens, therefore, the task is to determine how low the risk of the occurrence of cancer in an exposed persons or the projected incidence in an exposed population must be driven before a margin of safety can be considered ample to protect the public health. Only two approaches are available for performing this task: either the emission standards must be set at zero to eliminate the risk of cancer incidence altogether, or some residual risk must be permitted. The Administrator does not believe that section 112 expresses an intent to eliminate totally all risks from emissions of airborne carcinogens. Section 112 standards which permit some residual risk can, in the Administrator's judgment, therefore, provide an ample margin of safety to protect the public health.

This view is based on several additional factors. Foremost among these is the belief that if Congress had intended the drastic results that would flow from a requirement to eliminate all risk from emissions of carcinogens, it would have spoken with much greater clarity.

A requirement that the risk from atmospheric carcinogenic emissions be reduced to zero would produce massive social dislocations, given the pervasiveness of at least minimal levels of carcinogenic emissions in key American industries. Since few such industries could soon operate in compliance with zero-emission standards, closure would be the only legal alternative. Among the important activities affected would be the generation of electricity from either coal-burning or nuclear energy; the manufacturing of steel; the mining, smelting, or refining of virtually any mineral (e.g., copper, iron, lead, zinc, and limestone); the manufacture of synthetic organic chemicals; and the refining, storage, or dispensing of any petroleum product. That Congress had no intention of mandating such results seems self-evident.

The conclusion that Congress did not contemplate closure of the nation's basic industries, or even widespread industry closures, is also supported by the history and language of section 112. First, Congress in 1970 gave the subject of plant closures only brief consideration in connection with section 112. While the legislative history makes clear that the Administrator is empowered to set standards under section 112 that result in plant or industry closures where appropriate, it is by no means clear that Congress intended that result for all non-threshold hazardous pollutants, or even that Congress really focused on the problem. Indeed, the very limited nature of the legislative history itself compels the conclusion that closure of the nation's basic industries, irrespective of the actual levels of risk involved, could not have been contemplated. That conclusion becomes even more inescapable in light of the 1977 Amendments, which added radioactive substances - long regarded as confirmed carcinogens and emitted from a wide variety of sources - to the coverage of the Act, with no mention anywhere of industry closures as the inevitable consequence.

The language of section 112 is also consistent with this arsenic standard. In using the phrase "margin of safety," Congress was borrowing a concept from the field of engineering, where it had previously employed the term. By prescribing the use of a margin of safety for the load factors of

underground mine hoist cables in the 1969 Mine Safety Act, for example, Congress surely did not intend to suggest that the safety factor must guarantee a failure risk of zero. Indeed, no reputable engineer would say that even with a margin of safety an "adequately strong" hoist cable presents a failure risk of absolutely zero.

Nor does the use of the term "safety" necessarily imply a zero-risk concept. Where Congress has intended to require safety from the risk of cancer to be absolute, it has known how to express that intention clearly, as it did in the Delaney Clause of the Food and Drug Act, prohibiting the use of any food additive found to induce cancer in man or animal at any level of exposure. This provision was enacted years before section 112, and the absence of comparable specificity in section 112 suggests that "an ample margin of safety to protect the public health" need not be interpreted as requiring the complete elimination of all risks.

In interpreting the margin of safety concept in section 112 of the Clean Air Act, moreover, there is no reason to believe that Congress intended to make air pollution practically the sole facet of American life from which the government would attempt to eliminate risk entirely.

Not only is there no indication, as noted above, that Congress considered the inevitable consequences of such a decision, but such an interpretation would also be quite incongruous in view of the provisions of numerous other public health statutes enacted during or since 1970. These statutes deal with, among other things, environmental carcinogens to which people are equally or more exposed, and they all permit consideration of factors other than risk in setting standards or taking comparable actions.

In particular, the recent enactment of the Toxic Substances Control Act, which was intended to address the problem of toxic substances comprehensively, supports the view that where Congress has specifically considered the problem of reducing risks posed by environmental exposure to carcinogens, it has not required complete elimination of those risks. Lastly, as several commenters pointed out, closing down the copper smelter and other inorganic arsenic sources will not completely eliminate the risks associated

with exposure. Since arsenic is a naturally occurring element in the earth's crust, airborne arsenic has been detected in the air almost everywhere the Agency has sampled for it. However, the measured levels of arsenic in other areas are generally several orders of magnitude below the levels measured in Tacoma. Thus, the Agency suspects that, even with plant shutdown and soil cleanup, Tacoma residents will always be exposed to some inorganic arsenic.

Taken together, the Administrator believes that these statutes provide strong evidence that the complete elimination of risk from environmental exposure to carcinogens is 1) a virtually impossible assignment, and 2) not the task with which he has been charged by Congress.

6.1.4 Comparative Risk

Comment:

Commenters sought a framework for analysis of risk. Many suggested that comparisons of risk levels to those associated with other societal and environmental factors might be appropriate. Both voluntary and involuntary risks were used as a basis of comparison. One commenter (IV-D-668) stated that the comparison of voluntary vs. involuntary risks is an unfair one. One commenter (IV-D-721) said comparing cancer risk caused by the smelter to background cancer incidence is inappropriate. He likened it to justifying a murder by comparing it with the background incidence of accidental death. Some commenters (IV-F-3.57, IV-F-4.68, IV-F-4.71, IV-F-5.18) stated that it was unfair for the residents of the Tacoma area to be subjected to the same risks as smelter employees, because the employees accepted the risks associated with arsenic exposure when they decided to work for ASARCO. One commenter (IV-D-164/IV-D-666) said that as an adult, she might be willing to tolerate the kind of risk associated with EPA's current proposal for arsenic emissions at ASARCO, but children should not have to accept such risks. Another commenter (IV-F-3.6) stated that while individuals need to take responsibility for personal health practices, such as smoking, society must take responsibility for public health measures which decrease involuntary exposure to know harmful substances.

The Office of Management and Budget (IV-D-618) compared EPA's estimate of annual cancer risks from various source categories to death risk from accidents, homicide, and natural background radiation. They concluded that further risk reductions are warranted when the annual risk to the most exposed individual is greater than other risks routinely encountered in daily life.

Similarly, the Pacific Gas and Electric Company (IV-D-625) argued that significance is a function of relative risk because, as other risks are reduced, previously insignificant risks become significant. They continued by saying that a risk that is relatively high (significant) in one area might be totally insignificant in another area, since current total every day health risks vary considerably in different areas due to differences in: traffic hazards, crime rates, earthquakes, floods, storms, landslides, subsidence hazards, occupational hazards, lifestyle choices, economic well being, and environmental pollution hazards. They point out that although EPA is only authorized to regulate environmental pollution hazards, requirements to reduce environmental hazards could preempt public and private resources that might otherwise have been used to reduce far greater hazards of another nature. Therefore, all sources or risks should be considered when determining significance. On the other hand, one commenter (IV-D-9) felt that other risks had no bearing on the arsenic decision and did not need to be considered.

In general, the selection of comparative risks was dependent upon whether the commenter felt that the risk associated with proposed standard was acceptable or unacceptable.

6.1.4.1 Risk Associated With Proposed Standard is Acceptable. The following comparisons were made to illustrate that the risks associated with the arsenic emissions from ASARCO-Tacoma are much less than those associated with other voluntary and involuntary risks.

Voluntary Risks

- risk associated with cigarette smoking and the use of tobacco products (IV-D-15, IV-D-120, IV-D-130, IV-D-141, IV-D-146, IV-D-187, IV-D-227, IV-D-246, IV-D-265, IV-D-267, IV-D-313, IV-D-322, IV-D-355, IV-D-359, IV-D-361, IV-D-364, IV-D-382, IV-D-453, IV-D-548, IV-D-607, IV-D-613, IV-D-621-12.1, IV-D-621-12.5, IV-D-621-12.10, IV-D-621-12.11, IV-D-621-12.13, IV-D-621-12.22, IV-D-623, IV-D-645, IV-D-647, IV-D-657, IV-F-3.18, IV-F-4.25, IV-F-4.30, IV-F-4.32, IV-F-4.54, IV-F-4.60).

- risk associated with drinking alcohol (IV-D-128, IV-D-246, IV-D-452, IV-D-453, IV-D-621-12.5, IV-D-645, IV-D-647, IV-F-4.8, IV-F-4.25, IV-F-4.30).

- risk associated with drug abuse (IV-D-246, IV-D-645, IV-F-4.8, IV-F-4.25, IV-F-4.49).

- risk associated with driving a car (IV-D-146, IV-D-472, IV-D-613, IV-D-760, IV-F-4.53) and with auto traffic (IV-F-4.49).

- risk associated with the use of lawn fertilizers (IV-D-613).

- risk due to radiation associated with living in a brick or stone building (IV-D-128).

- risk associated with different life styles (IV-D-377, IV-D-647).

- risk associated with eating processed food (IV-D-132).

Involuntary Risks

- health risk associated with mobile source pollution (IV-D-31, IV-D-230, IV-D-247, IV-D-254, IV-D-265, IV-D-313, IV-D-322, IV-D-359, IV-D-382, IV-D-453, IV-D-472, IV-D-621-12.5, IV-D-621-12.12, IV-D-621-12.13, IV-D-621-12.24, IV-D-623, IV-D-636, IV-D-657, IV-F-4.30).

- risk of accidents caused by drunk drivers (IV-D-621-12.24).

- risk associated with air pollution from wood burning heaters (IV-D-364, IV-D-535, IV-D-613, IV-D-657).

- risk associated with CO_2 pollution (IV-F-4.30).

- risk associated with breathing dust (IV-D-322).
- risk associated with emissions from other local industries (IV-D-271, IV-D-272, IV-D-280, IV-D-313).

- risk associated with living in polluted cities (IV-D-525, IV-D-616, IV-F-5.14).

- risk associated with living with a smoker (IV-D-128, IV-F-3.47).
- risk associated with DDT (IV-D-322).

- risk associated with toxic contaminants in medicines (IV-D-453).

- risk associated with contaminants in flour (IV-D-621-12.5, IV-F-5.12).

- risk associated with arsenic residues in seafood (IV-D-537).

- risk associated with toxic residues in vegetables and meat (IV-D-323, IV-D-472).

- risk associated with pesticide residues in vegetables (IV-D-452), and grains (IV-F-4.30).

- risk associated with eating vegetables that contain natural pesticides made by plants to protect themselves from insects, fungus and animals (IV-F-4.44).

- risk associated with artificial food coloring and food preservatives (IV-D-278, IV-D-322, IV-D-406, IV-D-452, IV-D-472).

- risk associated with fats present in such foods as meat and buttermilk; these fats can be broken down in the body to mutagenic substances (IV-F-4.44).

- risk associated with burned and brown foods, including everything from carmelized sugar to toast, that contain mutagenic substances (IV-F-4.44).

- risk associated with living (IV-D-187, IV-D-353, IV-D-472).

- risk associated with heart disease (IV-D-621-12.16).

- risk of being kidnapped (IV-F-4.49).

- risk of being murdered (IV-D-621-12.24).

- risk associated with war (IV-D-384).

- risk of drowning (IV-D-760).

- risk associated with natural disasters: floods (IV-D-621-12.24) and volcanic eruptions (IV-D-621-12.13).

- risk associated with untraviolet radiation from sunlight (IV-D-621-12.13).

- risks that existed in past years when automobile and smelter emissions were higher than they are today (IV-D-695, IV-D-366).

6.1.4.2 Risk Associated With Proposed Standard is Unacceptable. The following comparisons were made by other commenters to illustrate that the risks associated with the arsenic emissions from ASARCO-Tacoma are much more than those associated with other involuntary risks. They also provided guidelines for what the commenters believe is an acceptable level of risk.

Involuntary Risks

- risk of botulism from canned foods (IV-D-71).

- risk associated with nuclear radiation and fallout is at least as great as the risk associated with exposure to heavy metals (IV-D-41).

- cancer risk to the general population (IV-D-120, IV-D-164, IV-D-590, IV-D-666, IV-F-1.17, IV-F-4.71).

- risk levels in other industrial geographic areas (IV-D-114, IV-D-142, IV-D-582).

- risk levels comparable to natural background levels of arsenic in other communities (IV-D-721, IV-D-771, IV-F-3.43, IV-F-3.54, IV-F-4.6, IV-F-3.103). Three commenters (IV-D-734, IV-F-3.43, IV-F-3.54) went on to state that the body or urinary levels of arsenic in persons living around ASARCO-Tacoma should not be greater than are normally found elsewhere in the country. Some commenters (IV-D-593, IV-D-643) specifically mentioned that children's urinary arsenic levels should be within a normal range.

- risk levels in other communities affected by arsenic emissions (IV-D-114, IV-D-438, IV-D-443, IV-F-3.20, IV-F-3.53, IV-F-3.58, IV-F-4.43, IV-F-4.52, IV-F-5.18, IV-F-3.103, IV-F-11). One commenter (IV-F-4.43) said that the risk to Tacoma is ten times the combined total risk to 14 other communities that have copper smelters.

- risk levels associated with hazardous compounds that are regulated by other agencies, such as FDA (IV-D-621-14.9, IV-F-3.43). One commenter (IV-D-621-14.9) suggested that EPA adopt FDA's stringent action level for carcinogens (1 in 1,000,000) because the aggregate risk for arsenic, SO_2, cadmium, etc. could be quite large.

- risk levels associated with other environmental standards (IV-D-541, IV-D-580, IV-F-11). One commenter (IV-F-1.18) stated that arsenic is about as carcinogenic as DDT, EDB, chlordane and heptachlor, all of which have been banned by EPA regulation under FIFRA. He also added that FIFRA does not impose as stringent a requirement for the maintenance of an ample margin of safety as does the Clean Air Act.

Another commenter (IV-F-11) said that limits on such pollutants as benzene, vinyl chloride, and dioxin are more restrictive than the proposed arsenic standard.

- risk levels associated with other EPA regulated hazardous air pollutants (IV-D-710, IV-F-1.18, IV-F-3.43).

- risk levels associated with other EPA regulated air-borne carcinogens (IV-D-142, IV-D-147, IV-D-314). Other commenters mentioned benzene (IV-D-443, IV-D-621-14.9), dioxin (IV-D-443) and vinyl chloride (IV-D-120, IV-D-443, IV-D-621.14.9, IV-F-1.18). Elaborating further, one commenter (IV-F-1.18) stated that EPA regulations for vinyl chloride reduced the lifetime risk of cancer to 1 in a million. He continued by stating that the emission standards for many major vinyl chloride sources were even set at zero. However, he noted that according to the Health Assessment Document for arsenic (p. 5-145), arsenic is three times more carcinogenic than vinyl chloride. Another commenter (IV-D-621-14.9) stated that the unit risk estimates for both vinyl chloride and benzene are on the order of 1:100,000, whereas, the unit risk estimate for arsenic is about 400 times higher.

Response:

Comparing the risks associated with arsenic exposure to risks associated with activities such as cigarette smoking and drinking alcohol is inappropriate, because risks due to arsenic exposure are largely involuntary. That is people who live near the smelter may be unaware of their inorganic arsenic exposure or, because of their circumstances, cannot relocate in some other more acceptable area.

Many commenters mentioned involuntary risks that they perceived to be either less or more than the risks associated with the proposed standard. Comparing the risk levels associated with these involuntary risks (particularly those associated with environmental hazards or contaminated food products) to the risk levels associated with the inorganic arsenic emissions, may not be appropriate because different risk methodologies and different assumptions may have been used to calculate them. (See the discussion of the uncertainties associated with the risk determination model, see Section 4.2.) However, EPA understands the desire of the public to seek a reference for relating to the estimated risk levels associated with inorganic arsenic source categories. The EPA believes that comparing the estimated increased lung cancer risk associated with inorganic arsenic source categories to national lung cancer rates provides a useful perspective (see Table 6-1).

Table 6-1. National Annual Cancer and Lung Cancer Rates - All Ages (1982)[a]

	Annual Deaths[bc] Per 100,000	Percent of[c] Total Deaths
Malignant neoplasms of respiratory and intrathoracic organs	50.2	5.8
Malignant neoplasms, including neoplasms of lymphatic and hematopoietic tissues (cancer-all forms)	188.1	21.9

[a] Source: "Monthly Vital Statistics Report," National Center for Health Statistics, Vol 31, No 13, October 5, 1983.

[b] Based on a ten percent sample of deaths

[c] Rates are not age-adjusted.

6.2 BAT APPROACH

Overview:

At the time of proposal EPA used a series of steps in deciding what the level of Section 112 standards should be. This series of steps included one step in which a determination was made concerning what level of control constitutes best available technology (BAT). As expressed in the preamble to the proposed standard (48 FR 33116), EPA's policy for implementation of Section 112 was as follows:

> 1. Source categories are identified on the basis of estimates of their potential to result in significant risk because risk to public health is the dominant theme of Section 112. A significant risk is considered to be associated with a source category when the weight of the health evidence indicates a strong likelihood that the substance emitted by the source category is a human carcinogen and either individuals or larger population groups are significantly exposed to the substance as emitted from the source category.
>
> 2. All source categories that are estimated to result in significant risks are evaluated and the current level of control ascertained. That control may result voluntarily or from State, local or other Federal

regulations. Whether the level of control meets the definition of BAT (considering cost and other impacts) then is determined. The BAT determination in this case can take into account such factors as the potential for improved control, the economic impacts of improved control on the source category, and the age and remaining useful life of the facilities.

3. The use of risk estimates generally has been confined to areas of broad comparisons, e.g., in selecting source categories to evaluate, and in assessing the incremental change in risk that results from application of various control options. The use of risk estimates in an absolute sense is avoided because of the many uncertainties of the estimates. These uncertainties are compounded as the focus is narrowed. In other words, in evaluating specific sources, as opposed to source categories, the uncertainties associated with the risk estimates increase dramatically.

4. Cost-effectiveness is one of the criteria used in selecting BAT. However, the use of cost-effectiveness in the BAT selection may result in some apparent disparities in risk improvement at some sources. Risk estimates are highly uncertain while technology and cost are generally well understood and provide an objective means of determining reasonableness of control.

5. If in the judgment of the Administrator, if the residual risks after BAT are unreasonable, then the source category must be controlled to a more stringent level. Whether the estimated risks remaining after the application of BAT are unreasonable will be decided in light of a judgmental evaluation of the estimated residual risks (and

their uncertainties), the economic, energy and environmental impacts of further reducing those risks, the readily available benefits of the substance or activity producing the risks and the availability of substitutes and possible health effects resulting from their use.

The public comments indicated that the risk management approach, as described in the July 20, 1983, Federal Register notice of proposal, did not give sufficient consideration to the protection of public health. Evidently, some commenters saw the selection of BAT as the final step in the decision-making process. Also, there seemed to be some level of misunderstanding as to what BAT represented and some confusion between similar terms used in other EPA programs such as "best available control technology" (BACT) found in the Prevention of Significant Deterioration program and "best available technology" (BAT) in the water program. Based on consideration of the public comments (as described on the following pages), the above concerns of possible public misinterpretation, and the recent experiences that the Agency has had with other pollutants, the Administrator has decided to refine the risk management process described in the proposal.

The EPA's refined strategy for risk management under section 112 provides for the comprehensive assessment of candidate source categories to evaluate current control levels and associated health risks as well as options for further control, the health risks reduction obtainable and the associated costs and economic impacts. Based on this assessment, EPA selects a level of control which in the judgment of the Administrator reduces health risks to the greatest extent possible, cognizant of the other impacts of regulation. The EPA believes this approach is both rational and consistent with the requirements of section 112. The major steps in EPA's procedures are outlined below.

1. Source categories are identified on the basis of estimates of their potential to result in significant risk. A significant risk is considered to be associated with a source category when the weight of the

health evidence indicates a strong likelihood that the substance emitted by the source category is a carcinogen and either individuals or larger population groups are significantly exposed to the substance as emitted from the source category.

2. All source categories that are estimated to result in significant risks are evaluated and the current level of control ascertained. The EPA examines the various options available to reduce emissions from these sources, including controls similar to those imposed under Section 111 of the Clean Air Act (New Source Performance Standards), the use of substitute feedstock materials, and closing a plant. Options are examined in terms of control efficiency, technical feasibility, costs, and the reductions in risk that they achieve. If a source category is not already required to apply the selected emissions reduction option, EPA will set the Section 112 standard which reflects the level of control of the selected option. If a category is already controlled (for example, by other EPA standards, other Federal, State, or local requirements, or standard industry practice) to the selected level, and EPA expects that the level of control will be required for these and new sources (EPA will continue to monitor this), a Section 112 standard would be redundant and need not be established. The level of control selected by the Administrator may be different for new and existing sources within a source category because of higher costs associated with retrofitting controls on existing sources. When selecting the control option, the Administrator considers whether the estimated risks remaining after application of each level of control are unreasonable. This is of a judgmental evaluation of the estimated maximum lifetime risk and cancer incidences per year remaining after application of each control option, the impacts, including economic impacts, of further reducing those risks, and the readily available benefits of the substance or activity producing the risks. In all cases where risks and other parameters are estimated, the significant uncertainties associated with those numbers will also be considered in reaching the final decision.

As can be seen when comparing the current risk management approach to the one given in the proposal, the term "BAT" has been removed. This change reflects something more than just a revision in terms: this is a refined approach used selecting the final control option as a basis for the Section 112 regulation. Instead of the previous multi-step process, this approach incorporates an amalgam of elements of the BAT residual risk approach combined with the elements of the two risk-based alternatives set forth in the proposal. With the refined approach there is no separate step to determine the appropriate level of control and then to examine the reasonableness of the residual risks. Rather, these two steps are combined into a single selection process which involves considering simultaneously the possible control options and the technical, economic, public health, and other implications of each option. This refinement, the Administrator believes, is both rational and more consistent with the language of section 112, and, as seen by reading the following comment summaries, it responds to many concerns of the commenters on this proposal.

Comment:

Many commenters (IV-D-61, IV-D-74, IV-D-114, IV-D-142, IV-D-147, IV-D-301, IV-D-346, IV-D-401, IV-D-438, IV-D-443, IV-D-524, IV-D-541, IV-D-557, IV-D-593, IV-D-604, IV-D-608, IV-D-609, IV-D-618, IV-D-660, IV-D-662, IV-D-663, IV-D-677.3, IV-D-747, IV-F-1.1, IV-F-1.18, IV-F-3.31, IV-F-3.58, IV-F-3.60, IV-F-3.103, IV-F-4.15, IV-F-9, IV-F-11) objected to what they saw as EPA's setting standards for ASARCO based on "best available technology" (BAT). Commenters felt that basing a standard on BAT placed primary emphasis on issues other than health, such as affordability, technology and economics. The commenters felt that health concerns were the appropriate primary emphasis.

Other commenters (IV-D-154, IV-D-231, IV-D-237, IV-D-271, IV-D-288, IV-D-399, IV-D-464, IV-D-480, IV-D-519, IV-D-622, IV-F-3.7, IV-F-3.8, IV-F-3.50, IV-F-4.49, IV-F-9, IV-F-11) favored basing a standard on BAT,

calling it a reasonable, logical approach. Some felt it reasonable in the face of uncertainty concerning health risk. Others felt it an equitable approach until other smelters were required to install similar levels of controls. One (IV-D-622) said that anything less than BAT would be unacceptable.

Response:

It is important to keep in mind the fact that the application of BAT was only one step within the risk management approach as described at proposal in deciding what level of control should be applied to a source category. Standards for hazardous air pollutants were not to be based on BAT unless, in the Administrator's judgment, the residual health risk levels after the application of BAT were not unreasonable. Within the context of the risk management approach discussed at proposal, EPA judged that all source categories of a hazardous air pollutant which are estimated to result in significant risk should be at a minimum controlled to a level which reflects BAT. Each such source category would then be controlled to a greater degree if, in the judgment of the Administrator, it was necessary to prevent unreasonable risks.

Thus, the proposed decision-making process begins and ends with the consideration of risks because the Agency views its primary mission under the section 112 as the reduction of public risk.

As a practical matter, there is a certain portion of the Agency's time spent on evaluating factors that are not directly related to reduction of air pollution risks but are important in the overall selection of the appropriate control option. For instance, has the technology been demonstrated at other installations as a means to reduce emissions? If required, can the control device actually be used safely on the process or the stack gases? Will the control technology create a pollution problem in another medium such as the water or land? Is the control technology so expensive that its application will surely shut the plant down? The EPA agrees with those commenters who felt that answers to these kinds of questions must be part of the control option selection process. Such analyses are part of the Agency's refined risk management approach.

The Agency agrees with those commenters who perceived that the protection of public health did not weigh very heavily in the selection of BAT; yet the Agency did not disregard the reduction of public risks. The effectiveness of the control equipment to reduce emissions (and risks) was weighed against the costs to install and operate that control equipment. Also, to the extent possible EPA considered the impacts from the pollution controls on other environmental media such as soil and water. After BAT was selected, the Agency reviewed the level of residual risks (and their uncertainties), determined if they were unreasonable, and considered requiring controls beyond BAT. Thus, the risk management policy, as outlined in the proposal, considered the protection of public health in each of the three steps for selecting the controls to be used as a basis for regulation.

Comment:

Several commenters (IV-D-386, IV-D-399, IV-D-466, IV-F-3.44, IV-F-4.51) noted that basing standards on BAT allowed for continued improvement. As new technology becomes available and economically feasible, they saw it as appropriate to require that technology for control of emissions. Several commenters (IV-D-269, IV-D-271, IV-D-372, IV-D-373, IV-D-386, IV-D-403) said that as long as ASARCO is making and is willing to make noticeable improvements, the plant should be allowed to continue operation. One commenter (IV-D-710) said EPA must revise standards periodically to continue to approach the statutory goal of complete public health protection as rapidly as possible. Another (IV-D-483) viewed EPA's role as one of helping industry work out its environmental problems by giving them technology, giving them easily attainable standards to meet over reasonable periods of time, then setting tougher standards as time and technology move forward.

Comment:

Some commenters (IV-F-3.29, IV-F-3.31, IV-F-3.103, IV-F-3.31, IV-F-9) objected to basing a standard on BAT because they felt it eliminated any incentive on the part of the smelter to develop improved control technology. One commenter (IV-F-1.17) complained that EPA needs to be pushed to require

even today's state-of-the-art controls, let alone any technological innovations. Still other commenters (IV-D-73, IV-D-302, IV-D-575) advocated requiring emissions to be essentially zero. The commenters said this would force ASARCO to design and build totally effective anti-pollution equipment. Another (IV-D-580) said that it seems that EPA is allowing existing technology and its costs to call the shots rather than forcing technology under the Clean Air Act. He called this an undesirable precedent. Another (IV-D-698) rejected standards based on BAT. He advocated technology-forcing standards under section 112 of the Clean Air Act. Another commenter (IV-D-710) said that the law recognizes that a standard may be set at a level which reflects a projection of what can be achieved by sources in the foreseeable future.

Response:

The EPA agrees that continued improvement in arsenic emissions control is a desirable goal. However, the agency must be reasonable. The EPA cannot set a rapidly moving target because it would be difficult if not impossible for industry to comply.

For example, since it may take industry up to several years to design, purchase and install control equipment, the controls could be outdated before they are in operation. Once again, the company would have to begin designing and purchasing the latest controls. The costs to the company would be very high and, most importantly, the hazardous emissions are not being effectively reduced; the company never gains expertise in the operation of the controls. In general, the Agency does not plan to use such an approach. It could prove infeasible for the Agency to implement.

In some circumstances, EPA's risk management approach will be forcing technology. Where source owners (or source category) are required to apply all the controls they can afford and the residual risks remain unacceptable, the owners have only two operations available. Either the source must devise some control option that is more effective than state-of-the-art controls (technology-forcing) or close down their operations which are posing the health risk problems. In this scenario, the owners must take some serious steps to reduce further or eliminate their emissions.

In addition, EPA periodically reviews the final emissions standards. The review considers the availability of improved emission control technologies, process modifications or substitute materials. The Agency then determines if there is a need for a change in the standards. The decision process will focus on the amount of risk reduction that may result from developing a more stringent standard. If the risk reduction is significant, the Agency will carefully consider the possibility of more restrictive standards.

Comment:

Some commenters (IV-F-1.7, IV-F-1.10) objected to basing a standard on BAT because they saw it as penalizing those smelters which had installed controls in the past. They said the cost-effectiveness analysis which underlies BAT determinations makes it appear more costly for those who have made improvements in the past and rewards those who have postponed installing controls. In particular, one commenter (IV-F-1.10) saw it as unfair to ASARCO-Tacoma because ASARCO had previously installed controls.

Another (IV-F-1.1) said that any approach where those who can afford it pay for it, and those that cannot may have a lesser degree of control, creates artificial competitive disadvantages.

Response:

It is hard to understand how cost effectiveness analysis could work against a facility which had installed controls previously if the previously-installed controls are effective ones. Cost effectiveness ratios are expressed as the cost in dollars of adding additional controls divided by the additional emission reductions which could be achieved in megagrams per year. If a facility has installed effective control technology, additional emission reductions achievable by additional control would probably be small. A small emission reduction would cause the cost effectiveness ratio to be a large number and, therefore, less attractive as a control alternative.

If a facility has installed ineffective control technology, additional control technology may offer significant additional emission reductions. A large emission reduction would cause the cost effectiveness ratio to be small. A low cost effectiveness ratio indicates that the control technology under consideration may be reasonable if the cost to the industry is affordable.

Comment:

Some commenters (IV-D-489, IV-D-515, IV-F-3.7) expressed strong support for continued efforts to develop control technology for arsenic emissions.

Comment:

One commenter (IV-D-710) said BAT is not the best technology in a technical sense; rather, it is the best control available considering economic, energy, and environmental impacts. The commenter inferred that the term "BAT" was borrowed from the Clean Water Act in which it establishes the test for toxic water pollutant standards. But, he said that in the Clean Water Act, BAT denotes more stringent standards than BAT as articulated for hazardous air pollutants regulated under the Clean Air Act. He explained that although EPA takes costs and other factors into account when establishing BAT for water pollutants, the best performing facilities provide a floor below which BAT may not slip. The commenter cited a 1982 notice regarding water pollutant effluent limitations: "BAT limitations, in general, represent the best existing performance of technology in the industrial category or subcategory" (47 FR 46435, October 18, 1982). The commenter objected to EPA's failure to recognize such a floor for hazardous air pollutants. He felt that standards set for hazardous air pollutants based on the BAT approach have in the past fallen short of requiring technology even as good as the best already in use.

Response:

The EPA does not agree that the term BAT, as defined within the Clean Water Act and its subsequent regulations, provides a floor below which BAT may not

slip. The Federal Register notice which this commenter cites continues with the following:

> "In arriving at BAT, the Agency considers the age of the equipment and facilities involved, the process employed, the engineering aspects of control technologies, process changes, the cost of achieving such effluent reduction, and non-water quality environmental impacts. The Administrator retains considerable discretion in assigning the weight to be accorded these factors" (47 FR 46435, October 18, 1982).

Thus, in the Clean Water Program, BAT may not necessarily reflect in all cases the best performing control technologies because of case specific differences which for some sources make this level of performance impossible to attain.

Comment:

A commenter (IV-D-710) found BAT as defined and implemented for hazardous air pollutants indistinguishable from the test applicable to New Source Performance Standards under section 111. He said that this implementation was contrary to Congress' intent that EPA set more stringent requirements under Section 112 than under section 111.

The commenter (IV-D-710) provided a recommended alternative to EPA's BAT approach. He argued that at a minimum, Section 112 must be interpreted to mandate standards which require technology at least as good as the best in use now or available in the foreseeable future. The commenter said that the required technology should include all design, operational, and maintenance improvements that can be installed at present or within reasonable lead times. Another commenter (IV-D-572) agreed with the first commenter's (IV-D-710) reasoning, saying that ASARCO must set pollution control levels at the lowest possible levels, not on achievable levels which are claimed to be affordable. Another (IV-D-778) felt EPA had based its standard on "Best Affordable Technology" rather than "Best Available Techhnology", and

he objected to this. Similarly, another commenter (IV-D-721) objected to EPA's policy of allowing a company to install only the available technology it says is affordable. He said this allows the company to resist development or installation of further control technologies. The first commenter (IV-D-710) went on to say that it was only after this stringent minimum has been applied that risk assessments should be used.

The commenter referred to a settlement agreement for litigation over the vinyl chloride standard as evidence that EPA had once embraced his recommended approach. He further urged EPA to return to the approach taken with vinyl chloride. Another (IV-D-731) said EPA's analysis of available technology and selection of BAT is less stringent than existing regulations for other hazardous air pollutants.

Response:

The commenters are arguing for a minimum requirement for all sources of hazardous air pollutants. The minimum requirement, being fostered by the commenters appears to be the best technology in use now or available in the foreseeable future, regardless of cost of current emission levels or current risk estimates. After this minimum level of control has been applied, the commenters would favor examination of residual risk.

This approach is similar to the one discussed at proposal but differs in the way in which the minimum level of control would be chosen as a first step. The EPA's implementation policy at proposal included requiring best available technology considering economic, energy, and environmental impacts. The commenters would apply a more stringent minimum requirement before examining residual risk.

As previously discussed in this section, EPA has refined the approach described at proposal to one in which the Administrator considers all factors and impacts together in making his decision. Based on EPA's experience to date, using the approach suggested by the commenters would have serious economic consequences on certain source categories (and the surrounding communities) such as the primary copper smelters. In addition,

applying the best technically available controls may provide little or no reduction in risks if the existing sources are already applying some control measures or if the application of further control creates a hazard in another medium. The Administrator feels that such information must be considered in the process of selecting the appropriate level of controls and in certain situations, applying the most stringent level of control, regardless of its economic and environmental impacts, does not constitute sound public policy. The above concerns are part of the reason why EPA has refined the decision-making process.

Comment:

When danger from emissions remaining after application of this minimum level of control remains great, a commenter (IV-D-710) stressed that EPA must set more stringent standards. One commenter (IV-D-718) said that if any doubt remains that additional controls might be warranted, it is EPA's legal and moral obligation to go beyond BAT. In some cases the commenter foresaw no alternatives to closing a plant.

Another commenter (IV-D-571) offered some suggestions for going beyond BAT. He favored using technology-forcing criteria or emission taxes to make going beyond BAT possible.

Response:

The Agency agrees with the commenters. As stated earlier in EPA's risk management approach, the Administrator considers the health risk estimated to remain after implementation of a control option. If, in his judgment, the residual risk is unreasonable, he will require a more stringent control option which may include plant closure.

Comment:

One commenter (IV-D-617) felt that alternatives considered in selecting BAT should be limited to technologies that have been demonstrated to be

feasible and effective for the source category under consideration. The commenter was basically supportive of the BAT approach, noting several advantages. He said it allows EPA to determine whether the costs and other impacts of a control requirement are disproportionate to the resulting emission reduction benefits. Another virtue he cited is that it allows recognition that BAT may already exist in certain source categories and that no standards need to be established for those categories. The commenter felt that this approach agreed with his opinion that controls should only be as stringent as needed to eliminate a section 112 risk and that controls should not be required when they are not necessary.

The commenter predicted that when BAT is selected with attention to cost effectiveness and is applied to sources which have been pre-sorted on the basis of population exposure, it is unlikely that residual risks will be unreasonable.

Response:

As discussed in previous responses in this section, EPA has refined its decision-making procedures. However, the selected control option's feasibility and effectiveness, costs, current control levels and risk estimates are still considered in the decision-making process.

Comment:

One commenter (IV-D-710) said that there is no legal basis for the determination of whether the risk remaining after application of BAT is reasonable. He called the judgmental evaluation of risk remaining, the impacts (including economic) of further reducing the risk, and the benefits of the substance producing the risk a cost-benefit analysis. The commenter contended that there is no basis in the Clean Air Act for applying a cost-benefit analysis under section 112.

The commenter said that in practice, the analysis of residual risk is nothing more than a repetition of the analysis of BAT because EPA had never come to a conclusion that a standard should go beyond BAT.

The commenter further argued that risk estimates being as uncertain as they are, in implementing a statute requiring a precautionary, preventative approach, EPA cannot rationally use risk assessments as a basis for not requiring the use of available emission controls on all sources of arsenic emissions.

Response:

As EPA pointed out at proposal, the statute requirements does not accommodate air carcinogens that may pose health risks at any levels of exposure. Therefore, EPA has adopted a pragmatic approach to regulate such pollutants after considering residual risks, costs and other factors. Rejecting the idea of zero risk and massive plant closures, EPA does consider costs and risk reductions achievable in selecting the control option for the standard. The concept presented by the commenter of not examining residual risk and costs and other impacts of reducing risk still further is one which EPA rejects.

The EPA has not had to make a decision to go beyond BAT. The EPA and industry have been able to find solutions which have allowed for continued operation. The option is still there, however. If the Administrator should determine that measures more stringent than BAT (including plant closure) are required to protect the public health, he would act to require those more stringent measures.

Comment:

One commenter (IV-D-618) saw large differences across source categories in the level of costs EPA found reasonable in determining BAT. The commenter said he could find no clear criteria applied in a consistent fashion which would differentiate among the controls considered to be BAT.

Response:

BAT determinations included consideration of feasibility and economic, energy, and environmental impacts. Cost was only one part of the consideration.

Comment:

One commenter (IV-D-641) said that BAT based on economics should be used only as a baseline. Where health risk is significantly higher than for other plants, the commenter felt that LAER should apply.

Response:

The EPA agrees in concept with the commenter. Where health risk is unreasonably high, stringent control measures must be applied. The level of control selected may be even more stringent than what might be considered lowest achievable emission reduction (LAER). The EPA is not using predetermined levels of control in the standard selection process.

Comment:

One commenter (IV-D-698) said that EPA's analysis of available control technology does not meet the Clean Air Act requirements. The commenter said that EPA purports to have established BAT for each source category, yet technology-based standards are only allowable when emissions standards are not feasible. The commenter said EPA had not demonstrated the infeasibility of emissions standards. Another commenter (IV-D-609) said that adoption of BAT as an approach to emission control would require additional Congressional legislation.

Two commenters (IV-D-621-5, IV-D-621-15.1) favored the BAT approach over the approach of setting an ambient air standard. Specifically, one (IV-D-621-15.1) felt that the proper way to deal with control of fugitive arsenic emissions is by means of identifying the source and determining what, if any action can be taken to control or reduce the emissions.

Response:

The commenters appear to be confusing the decision on the level of the standard with the decision on the format of the standard. The Clean Air Act

specifies that a section 112 standard must be expressed as an emission limit unless it is infeasible to do so. In that case, section 112 states that the Administrator may instead promulgate a design, equipment, operational, work practice standard.

Under both the past and the current approach, when selecting a control option EPA considers available technology which could be used to meet the standard, associated costs of that control technology and the level of residual risks.

Comment:

One commenter (IV-D-604) saw the BAT approach as creating a de facto air quality standard. The commenter further reasoned that since the same proposed BAT applies to both low and high arsenic feed copper smelters, EPA has created two margins of safety for the public.

Response:

The commenter appears to have misunderstood the concept of BAT as it was presented at proposal. BAT, because it depended on economic, energy, and environmental factors, could be set at different control levels for different source categories. As mentioned earlier, determining BAT was not the final step of the decision-making process. BAT was the selected control option if the residual risks were not unreasonable in light of the impacts of requiring controls beyond BAT.

The commenter appears to have observed two levels of residual risk, surmising that the Administrator has defined two different numbers for risk levels that are not unreasonable. This concept is incorrect. A range of residual risks could be considered not unreasonable depending on the outcome of the evaluation of the factors used in the decision-making process.

Comment:

One commenter (IV-D-144) stated that EPA's approach to determining acceptable risk requires that EPA estimate the cancer risk remaining for the population after controls are in place. Then EPA determines if the remaining cancer risk is acceptable taking into account the costs and technical feasibility of reducing the risk further. The commenter suggested that the degree of risk be defined first. Then, the economic and social costs of reducing this risk need to be assessed. Finally an acceptable level of risk can be determined.

Response:

All the factors the commenter suggested for inclusion in the rulemaking process are included in the Administrator's considerations. However, the order of consideration may differ. The commenter is suggesting that changing the order of factor consideration will affect the final decision. The refined policy calls for the simultaneous consideration of all relevant factors and so accommodates the commenter's concerns.

Comment:

One commenter (IV-F-4.59) said that an ample margin of safety is related to what is technologically feasible.

One commenter (IV-F-1.18) felt that the Clean Air Act should require the best available technology (BAT), even if BAT drives a company out of business. Congress should then decide if plant closure is an unacceptable tradeoff between risk reduction and the cost of compliance.

Response:

The commenters are expressing opinions about the role that technology, its capabilities and its costs, should play in determining the level of a standard. As defined in the proposal preamble, the requirement of BAT would not drive a source category out of business; however, individual sources within a large source category might be impacted in this manner. However,

both the BAT and the current risk managmement approaches focus on the reasonableness of the residual risks and does not stop at determining technologically feasible control options.

Comment:

One commenter (IV-D-609/IV-F-4.15) stated that it does appear that Congress intended Section 112 to cause shutdown of any industry that either cannot or will not comply with air quality standards protective of public health. He noted that the Senate committee that enacted the Clean Air Act denied that the concept of technical feasibility could be used as the basis for establishing ambient air standards, saying that the public health is more important than the question of whether the early achievement of ambient air quality standards protective of health is technically feasible. The commenter quoted the Senate Committee report (S. Rep. N. 1196):

> "In the Committee discussions, considerable concern was expressed regarding the use of the concept of technical feasibility as the basis of ambient air standards. The Committee determined that (1) the health of the people is more important than the question of whether the early achievement of (ambient air quality) standards protective of public health is technically feasible; and (2) the growth of the pollution load in many areas even with the application of available technology, would still be deleterious to public health.
>
> "Therefore, the Committee determined that existing sources of pollutants either should meet the standard of the law or be closed down . . ."

Response:

The commenter is referring to national ambient air quality standards instead of the hazardous emission standards presented in this package. Unlike the criteria pollutants, the health effects associated with arsenic public exposure levels are not documented and the protection of public

health presents a more difficult determination. However, EPA agrees that public health is the primary concern for section 112 standards as it is when setting ambient air quality standards. Technical feasibility is also of concern but only one factor considered when the Administrator has determined that the public health risks are not unreasonable.

6.3 ECONOMICS AS A DECISION-MAKING CRITERION UNDER SECTION 112

Comment:

Some commenters (IV-D-439, IV-D-541, IV-D-557, IV-D-630, IV-D-710, IV-D-724, IV-D-754, IV-D-778, IV-F-3.7, IV-F-3.31, IV-F-4.6, IV-F-4.15) stated that EPA is required to place the protection of public health and the environment, not costs or the availability of technology, as the primary consideration in developing standards. Other commenters (IV-D-25, IV-D-106, IV-D-112, IV-D-137, IV-D-698, IV-F-3.1) said that the proposed arsenic standard was based on economic feasibility, an action which is against the legal mandate of the Clean Air Act to provide an ample margin of safety.

One commenter (IV-D-224) said the foundation of EPA and the Clean Air Act is to protect people's health and the environment, not to attack their health and well-being for the sake of the financial well-being of the copper industry. Another commenter (IV-D-641) said that EPA should not avoid its responsibility to protect health through case-by-case acceptance of high risks by locality to avoid closure of a major local industry. According to the commenter, such a policy could result in the location of inadequately controlled facilities in economically depressed areas.

Some commenters (IV-D-710, IV-F-4.6) stated that Congress had no intention of authorizing EPA to perform cost-benefit analyses when setting hazardous air pollutant standards. Another (IV-D-747) thought cost-benefit analysis was one of several factors which might be considered, but should never be the basis of standards for hazardous pollutants. He felt in this case EPA has overemphasized costs to industry and underemphasized health

costs and benefits to individuals and society. One commenter (IV-D-609/ IV-F-4.15) stated that it does appear that Congress intended section 112 to cause shutdown of any industry that either cannot or will not comply with air quality standards protective of public health.

Some commenters (IV-F-4.59, IV-D-718, IV-D-724, IV-D-731) felt that EPA's proposed regulation implies that economic risks can be considered under section 112 of the Clean Air Act although section 112 itself does not allow this. One commenter (IV-F-4.59) also said that it was not the intent of Congress to include economic analyses in the decision-making process. He continued by pointing out that the regulatory process starts with BAT, and if that is not stringent enough, more controls must be added until an ample margin of safety is reached without regard to economic factors.

Similarly, the Attorney General's Office of the State of New York (IV-D-698) stated that section 112 has been violated by establishing BAT based on costs. They continued by saying that EPA must either set emission limits or establish technology-forcing performance standards. They contended that EPA has not demonstrated that emission standards are not feasible as required in the Clean Air Act.

Both the Natural Resources Defense Council (IV-D-710) and the Attorney General's Office of the State of New York (IV-D-698) stated that the New Source Performance Standards were clearly intended by Congress to apply to less dangerous pollutants. They continued by saying that Congress explicitly provided authority in section 111 to consider costs. However, the commenters said, the Act does not mention that economics should be factored into section 112. Congress intended, and the law requires, EPA to set more stringent, more protective standards for hazardous air pollutants regulated under section 112. New York State (IV-D-698) went on to say that EPA has simply abandoned section 112's requirements and followed the easier path laid by section 111.

One commenter (IV-F-3.7) pointed out that while section 317 of the Clean Air Act mandates that an economic impact assessment be made, it also

states that this information does not affect or alter the final decision in setting standards.

Another commenter (IV-F-4.71) noted that the Toxic Substances Control Act states that, while economic impacts of EPA decisions must be considered, they must not prevent implementation of the strictest standards necessary to protect public health.

One commenter (IV-D-466) asked EPA to use sound scientific basis with plenty of weight on economic effects in regulating arsenic emissions. Others (IV-F-3.4, IV-D-728) said that economic data for the local area are important and must be considered in setting standards or policies.

The Chemical Manufacturers Association (IV-D-617) stated that the margin of safety concept embodied in the Clean Air Act Amendments was intended by Congress as a means of providing a "reasonable degree of protection" for public health, not as an instrument for eliminating environmental health risks entirely. Citing legislative history, this commenter continued by stating that Congress was well aware that equating the term "margin of safety" with absence of risk would be "an illusion" that "ignores all economic and social consequences." (1977 Legislative History at 2578, House Report).

Response:

Section 112 of the Clean Air Act is a potentially powerful tool which does not provide explicitly, either in language or legislative history, for the weighing of the benefits of control against the control costs. At face value, section 112 could be construed to require regulation even when the costs clearly exceed any measurable benefit. A total disregard for economics would result in a zero risk philosophy. However, this philosophy has been dismissed by EPA as being impractical (see Section 6.1.3 on Zero Risk). In view of this, EPA has sought to construct an approach to the implementation of section 112 which will not necessitate the establishment of regulations which would impose costs unreasonably disproportionate to the benefits obtainable.

This approach considers current control levels and associated health risks as well as options for further control, the health risk reductions obtainable and the associated costs and economic impacts. Based on this assessment, EPA selects a level of control which in the judgment of the Administrator reduces health risks to the greatest extent possible, cognizant of the significance of the residual risks and the societal impacts of the regulations. The EPA believes this approach is both rational and consistent with the requirements of section 112.

6.4 RECOMMENDED ACTION IN FACE OF UNCERTAINTY

Comment:

Many commenters (IV-D-162, IV-D-170, IV-D-177, IV-D-179, IV-D-181, IV-D-185, IV-D-193, IV-D-196, IV-D-212, IV-D-221, IV-D-229, IV-D-230, IV-D-250, IV-D-281, IV-D-298, IV-D-299, IV-D-312, IV-D-326, IV-D-333, IV-D-316, IV-D-339, IV-D-349, IV-D-367, IV-D-370, IV-D-371, IV-D-372, IV-D-373, IV-D-382, IV-D-383, IV-D-456, IV-D-460, IV-D-465, IV-D-474, IV-D-485, IV-D-486, IV-D-508, IV-D-516, IV-D-546, IV-D-633, IV-D-659, IV-D-735) felt that the ASARCO/Tacoma smelter should not be put in economic jeopardy. Commenters felt that the smelter should remain open and smelter workers should remain secure in their jobs because there is no proven link between smelter emissions and lung cancer.

One commenter (IV-D-322) said that until better information is available, EPA should remove the risk portions from its standards. Another (IV-D-376) said that EPA should not impose any more stringent regulations on arsenic emissions until a link between arsenic emissions and cancer is established. Similarly, another commenter (IV-F-3.50) suggested that it would be prudent to proceed with the proposed standards and the best available technology until more is known about the risk associated with arsenic. Some commenters (IV-D-588, IV-D-621-16.3, IV-F-3.39, IV-F-5.17) felt that actions to control emissions and provide an ample margin of safety must be based on irrefutable proof. Another (IV-D-542) did not support implementation of strict ambient standards for arsenic when there are no

scientific data to indicate its necessity for achievement of an ample margin of safety.

Commenters (IV-D-274, IV-D-466, IV-D-569) said that EPA should base its regulations on facts and not on theory or supposition. Others (IV-D-332, IV-D-388, IV-D-391, IV-D-763) said that actions to reduce industrial pollution must be based on objective studies and analysis, not emotion. One commenter (IV-D-278) stated that EPA should take time to run the necessary tests and give ASARCO time to prove what they are doing is working. Another (IV-D-489) said that community decisions which attempt to balance impacts of potential health hazards with jobs and other benefits should be based on fact, not perception. Another (IV-D-512) said the smelter should not be shut down because of false information or emotions.

Another (IV-D-538) stressed that it is essential that EPA make decisions based on accurate, empirical scientific data rather than misinformed, vague public concern. Another (IV-D-621-16.9) supported the use of the very best scientific methods for examining the problem and appraising the risks. The commenter felt the information should be verified and subject to peer revoew. Another commenter (IV-D-735) supported continuing research on health risks. Another (IV-D-729) felt that more information about the effects of arsenic exposure could and should have been obtained by EPA. He suggested epidemiologic studies and more accurate estimation of exposure and controls.

Another (IV-D-203) asked that EPA not let inaccurate data and faulty assumptions of a few panic the Agency into choosing to regulate this industry.

One commenter (IV-D-154) said that requiring ASARCO to spend millions because someone may get cancer (or maybe not) is unreasonable because one possible death (one maybe) is not significant.

Others (IV-D-155, IV-F-3.39) said that elimination of a source of income to the Pacific Northwest without reasonable documentation of health risk is unwise and inappropriate. One (IV-F-3.39) felt that to destroy a

major source of financial support to the community by demanding an operation so clean it cannot financially exist is a tough pill to swallow, especially when the current arsenic levels have not been proven to be a health hazard.

One commenter (IV-D-625) recommended that EPA should not contemplate the projected risks do, in fact, exist. He continued by stating that if the existing data fail to resolve this uncertainty, consideration should be given to conducting a more thorough epidemiological study. Another commenter (IV-D-545/IV-D-621-16.6/IV-F-4.24) felt that since the available scientific data are uncertain, more studies should be conducted to determine if arsenic is a no-threshold pollutant. He continued by saying that considering that more health data will be available and new control technologies will be developed, there should be a periodic review of the standard.

Some commenters (IV-D-338, IV-D-342) said that EPA needs to back off and look more closely at actual data over a longer time period. Two commenters (IV-D-144, IV-D-460) said that EPA cannot show emissions from ASARCO-Tacoma are harmful to the community. One (IV-D-460) concluded that EPA should drop its case against ASARCO. Two others (IV-F-1.1, IV-F-1.3) said if there are no demonstrated community health effects, regulation is not warranted. One commenter (IV-D-256) resented what he called EPA's attempt at baffling the public with unsupportable statements. He said that EPA should either find the true facts and be able to support them, or they should just keep quiet.

Response:

Many commenters believe that EPA should wait to regulate arsenic emissions until (1) the link between arsenic and lung cancer is established beyond any doubt, and (2) the effect of low ambient concentrations of arsenic on public health has been determined.

The current status of inorganic arsenic as a human and experimental animal carcinogen has been extensively and critically reviewed by public agencies such as the National Institutes of Occupational Safety and Health,

scientific bodies such as the National Academy of Science and the International Agency for Research on Cancer, and in a number of individual assessments. In addition, EPA's inorganic arsenic Health Assessment Document has been reviewed by the Science Advisory Board, a group of scientific experts from outside the Agency. At present, the collective evidence for an etiological role of inorganic arsenic in human cancers is strongest for cancers of the skin and lung. Cancer (and possible precancerous lesion) producing inorganic arsenic exposures have been demonstrated in both occupational populations, such as copper smelters, pesticide manufacturers and agricultural workers, and in non-occupational populations using arsenical drugs or consuming arsenic contaminated drinking water and/or food. (For further information see Chapter 2.)

However, the effect of low ambient concentrations of arsenic on public health has not been adequately determined. Very little information exists that can be used to extrapolate from high-exposure occupational studies to low environmental levels. For several practical reasons as mentioned earlier in this document, such low levels of risk cannot be readily measured either by animal experiments or by epidemiologic studies. The linear non-threshold model is used as the primary basis for risk extrapolation at low levels of exposure. The EPA considers this model to be a viable possibility for the true dose-response relationship (Health Assessment Document p. 7-89-90).

The Agency is not required to wait until irrefutable proof that arsenic causes cancer at low ambient concentrations is produced. It must be noted that section 112 of the Clean Air Act defines a toxic air pollutant as that which <u>may reasonably be anticipated</u> to result in an increase in mortality or an increase in serious irreversible, or incapacitating reversible illness.

<u>Comment</u>:

Many other commenters urged an approach which would err on the side of overprotection in the face of uncertain health results. Commenters urged immediate mitigation of potential health risks. One commenter (IV-D-150)

said that in the absence of clear knowledge of effects of arsenic on health and environment, EPA should close the smelter. Others (IV-D-420, IV-D-717, IV-D-722) advised that EPA take the same attitude and accused EPA of not acting until a serious amount of damage has occurred. One (IV-D-420) urged that EPA not take the gamble when cancer is at stake. One commenter (IV-D-13) asked how many cases of lung cancer are necessary to show that a health hazard exists? Similarly, another (IV-F-3.29) asked why a health hazard must be proven before any action is taken, when it would be obvious that a health hazard exists. One commenter (IV-F-4.11) reminded EPA that they are under a court order to proceed in a determination before all the facts are in. Another (IV-D-431) said that it makes no sense to risk public health with unknown consequences just for profit. Another (IV-D-11) asked EPA not to wait a generation to stop carcinogenic effects from arsenic emissions after the effects have materialized. He felt that enough is known right now to demonstrate the ill effects of Asarco's emissions on public health. Another (IV-F-5.15) said that until more is known about cancer, we cannot allow known carcinogens to be present in our environment at possibly hazardous levels.

Another commenter (IV-F-4.67) stated that the procrastination on this issue has gone too far. He did not intend to accept the absence of evidence for health hazards to be conclusive evidence of the safety of the emissions.

Two commenter (IV-F-3.53, IV-D-733) said that until there is proof that elevated urinary arsenics are safe, ASARCO should be required to control emissions to the point where local children's urinary arsenic levels are normal.

Another (IV-D-8) advised EPA to proceed with caution in the face of uncertainty. He said that EPA (society) cannot take the chance of being wrong. He argued that EPA must assume arsenic is creating a health hazard and must be prevented from entering the environment. Other commenters (IV-F-3.41, IV-F-4.4, IV-F-4.9, IV-F-4.31, IV-D-708a, IV-D-747) said that a safe exposure level may be difficult to determine, but EPA should err on the side of safety and adopt as stringent a standard as possible.

Two commenters (IV-F-3.55, IV-D-733), noting that nobody knows what the combined effect of arsenic is with other pollutants, stated that the only prudent thing to do is to reduce unnecessary exposure to all pollutants.

One (IV-D-628) recognized that certain actions must be taken to protect the environment and the public without adequate scientific support data. The commenter urged adoption of the proposed standards.

Another (IV-D-677.4) said further studies should be conducted, but they in no way should delay full enforcement of the proposed standards. Another (IV-D-621-16.1) said it would be prudent to minimize human exposure to arsenic by reducing arsenical emissions, especially low-level or fugitive emissions. Others (IV-D-545, IV-D-621-16.6, IV-F-4.24) urged timely adoption of the standards. They argued that given the doubts about the presently available data, further delay would only delay the time when reduction of present emissions could be accomplished.

One commenter (IV-F-3.21, IV-D-718) asked that EPA's proposed standards serve as interim controls until more conclusive results are in. Another (IV-F-4.24) recommended timely adoption of the proposed standards, stating that delay in doing so will only further delay the reduction of current emission levels. He also urged EPA to continue its research on the health effects of arsenic and suggested that there be periodic review of the standards as new evidence and/or technologies are developed. Another commenter (IV-F-4.3) said that the delay in setting standards is uncalled for - ASARCO should be allowed to install hoods immediately to eliminate unnecessary health risks. After these standards are met, EPA should go forward with further research. In the meantime, he said, EPA must assure those who perceive a health risk that everything that can be done is being done. Another commenter (IV-D-657, IV-D-733) urged adoption of the proposed standards until the arsenic question is further evaluated. Another (IV-D-368) felt that further study is advisable and that ASARCO should in the meantime be encouraged to cut down further on emissions if possible.

Response:

The EPA is following the prudent person policy advocated by many of these commenters, erring on the side of protecting public health. Section 112 requires that standards be set at levels which, in the Administrator's judgment, provide an ample margin of safety to protect public health. Thus, one factor EPA considers is the nature and relative magnitude of health hazards. Unfortunately, agencies can never obtain perfect data but have to make regulatory decisions on the basis of the best information available. So, EPA evaluates the potential detrimental effects to human health caused by pollutant exposure based on the best scientific information currently available.

The scientific uncertainties not resolved to date include the establishment of toxicity to humans based on extrapolation, using uncertain mathematical models from occupational exposure to low-dose public exposure at ambient air concentrations, and the identification of the appropriate level of emission controls for pollutants for which health effects thresholds have not been demonstrated. There also is uncertainty with exposure estimates because of difficulty in obtaining precise data on emission rates, atmospheric dispersion patterns and population concentrations around individual sources, and because of the lack of information on short-term and long-term movement (migration) of people and indoor versus outdoor toxic air pollutant concentration patterns (see exposure and risk determination section). Finally, there are uncertainties concerning possible additive effects of multiple sources or pollutants, synergistic or antagonistic health effects, and heightened susceptibilities to some cancers by some population groups. These factors make it difficult, if not impossible, to determine the absolute magnitude of the risk to human health based on the available data.

Comment:

One commenter (IV-F-4.6) said that in the Senate Report on Amendments to the Clean Air Act, the Senate stated: "Margins of safety are essential to any health-related environmental standards if a reasonable degree of protection is to be provided against hazards which research has not yet identified." Another commenter (IV-F-4.11) stated that a margin of safety

must be incorporated in the permissible dose to compensate for the degree of uncertainty in determining that dose. The less precise the determination of hazard, the larger must be the margin of safety.

Response:

Again, with cancer-causing agents, there appears to be no level at which an exposed individual is entirely safe (non-threshold pollutant). The question surrounding the decision is the uncertainty and acceptability of the risks which remain after the application of the selected control option. The term "margin of safety" are more commonly used with threshold pollutants and is not readily applied to inorganic arsenic.

6.5 JOBS VS. HEALTH

Comment:

Many commenters (IV-D-1, IV-D-32, IV-D-694, IV-D-43, IV-D-53, IV-D-61, IV-D-62, IV-D-94, IV-D-107, IV-D-116, IV-D-138, IV-D-144, IV-D-151, IV-D-158, IV-D-163, IV-D-224, IV-D-301, IV-D-375, IV-D-400, IV-D-426, IV-D-431, IV-D-632, IV-D-672, IV-D-582, IV-D-643, IV-D-644, IV-D-637, IV-D-670, IV-D-690, IV-D-241, IV-D-435, IV-D-437, IV-D-346, IV-D-674, IV-D-435, IV-D-710, IV-D-677-1, IV-D-677-6, IV-F-4.50, IV-F-4.68, IV-F-5.10, IV-F-5.15, IV-D-720, IV-D-730, IV-D-734, IV-D-783, IV-D-753, IV-F-9, IV-F-11, OAQPS 79-8/IV-D-4, IV-D-705, IV-F-10) felt that health concerns were more important than jobs or economic advantage. Several (IV-D-720, IV-D-778, IV-F-10, IV-D-20, IV-F-3.30, IV-D-551, IV-D-661, IV-D-56, IV-D-10, IV-D-55, IV-D-638, IV-D-224, IV-D-412, IV-D-421, IV-D-378, IV-D-87, IV-F-3.60, IV-F-4.43, IV-D-688, IV-D-415, IV-D-66, IV-F-5.18) said it is EPA's job to protect people's health from pollution, not to promote jobs and corporate profits.

One commenter (IV-F-4.50) said that health, in keeping with the intent of Congress, must be the one non-negotiable component. Another commenter (IV-F-4.15) said EPA should not defer to industry and wait for cancer deaths to occur. Another (IV-D-72) said that it is not the right of smelter workers to choose jobs over public health. Commenters (IV-D-671, IV-D-660,

IV-D-4, IV-F-4.35, IV-F-3.60) said that 600 jobs cannot justify even one additional death per year.

One commenter (IV-D-444) said that human life is of the greatest value and should not be compared to other things. He reasoned that a smelter could not be of greater value than the people who operate it or whose material needs brought it into existence in the first place. He further argued that personal income could not be of greater value than a person. Similarly, another (IV-F-4.13) stated that without life there can be no jobs. Another (IV-F-3.67) said that when comparing loss of jobs to loss of life, it must be taken into account that people can always get other jobs.

A commenter (IV-D-595) said that even if it were a choice between health and jobs, given the seriousness of the health risk, the number of residents adversely affected, and the number of jobs arguably at risk, the decision should clearly be to protect the public health. Another (IV-F-3.38) understood the profit motive of ASARCO, but did not think it should infringe on the public.

Some commenters (IV-D-78, IV-D-9, IV-D-710) objected to EPA weighing jobs v. health. The two commenters said that this was not allowable under the Clean Air Act.

Comment:

Some commenters (IV-D-361, IV-D-464, IV-D-489, IV-D-507, IV-D-508, IV-D-217) said that people of Tacoma need the jobs and money the smelter puts into their economy. Another (IV-D-467) asked EPA not to consider regulations that would put people out of business. Two (IV-D-278, IV-D-735) said the smelter should not be closed or cause loss of jobs unless there is solid proof of health risks. Still another (IV-D-162) said that jobs and economic considerations are more important than a health risk he perceived as minimal or nonexistent. Another (IV-D-395) said that the value of the smelter to the community far outweighs any danger to the health and well-being of people living in the area. Another (IV-D-550) said the economic significance

of the smelter far outweighs the potential community health hazards. Another (IV-D-349) said no more stringent standard should be proposed since this would create unreasonable economic conditions for ASARCO and the State of Washington. Other commenters (IV-D-483, IV-F-3.25) asked that EPA set reasonable standards and provide more than a reasonable length of time for ASARCO to reach those standards, especially in these tough economic conditions. One commenter (IV-D-713) attached a newspaper article to his letter which stated that "Residents and governmental organizations have told the Federal Environmental Protection Agency they will accept a small amount of cancer-causing arsenic in the air so a copper smelter can continue to operate".

Comment:

Several commenters (IV-F-4.3, IV-F-4.6, IV-F-4.68, IV-F-5.1, IV-F-5.2, IV-F-5.3, IV-F-5.4, IV-F-5.11, IV-D-623, IV-D-713, IV-D-737, IV-D-779, IV-D-756) said that the jobs v. health question should not exist - the community can have both. Another commenter (IV-F-4.4, IV-D-708a) stated that jobs v. health is not the issue - the real issue is jobs and health v. neither. One commenter saw the proposed standards as a way of protecting the public health and keeping an economic contributor to the community. Another (IV-D-75) said EPA should keep jobs and protect health. Others (IV-D-210, (IV-D-473, IV-D-168) said that people want to keep ASARCO in operation and at the same time keep the environment clean. One commenter (IV-F-5.1) said the public hearings gave residents the false impression they had to choose between jobs and health. Some commenters (IV-D-125, IV-D-514, IV-D-4.66, IV-D-5.2) said that to balance health and jobs is an unfair choice.

Another (IV-F-1.17) accused EPA of setting one part of the community against the other in asking them to choose between health risks and jobs. He said the people of Tacoma were being presented a false tradeoff.

Comment:

Some commenters (IV-D-4.61, IV-D-721, IV-D-781, IV-D-783, IV-D-753) said that the question of potential job loss probably does not exist,

because light industry will move into the area as the effects of the smelter diminish. This will more than offset any potential job loss from the smelter.

Comment:

One commenter (IV-D-144) said that balancing health risks and jobs is an appropriate tradeoff only when the people who bear the risks are the same as those who stand to receive the benefits. Another commenter (IV-D-74) said it is less dangerous to be unemployed than to be exposed to arsenic, cadmium, SO_2 and other hazardous pollutants. Another commenter (IV-D-4.4) stated that, although you cannot trade lives for dollars, you can compare the health risks associated with smelter emissions to health risks associated with unemployment.

Comment:

Several comments (IV-D-339, IV-D-252, IV-F-3.1, IV-F-3.31, IV-F-3.48, IV-F-4.39) urged that EPA proceed with reason, suggesting compromise between environmental and economic interests. One (IV-D-359) requested that EPA allow improvements to be made at a pace that ASARCO can afford. The commenter cited progress which has been made and is continuing to be made by ASARCO. One commenter (IV-D-386) said the smelter should be allowed to continue its operation with the understanding that it continue to improve control equipment as it becomes technologically and economically available, especially considering the state of the economy and the copper industry. One (IV-D-731) suggested as a general regulatory principle that if a company could prove that it cannot afford controls, it should be required to phase in such controls over a reasonable period of time, but should not be totally exempted from regulation.

Some commenters (IV-D-187, IV-D-255, IV-D-274, IV-D-395, IV-D-396, IV-D-344) urged both improvement of air quality and continued smelter at further reductions coupled with a decision to retain the ASARCO facility.

One commenter (IV-D-384) asked that EPA place viable limits on emissions but that the Agency not kill industry while trying to find what is viable.

One commenter (IV-D-279) said that we must as a nation be concerned and protective of our natural resources, but excluding the cost of controls, jobs, and standard of living from rulemaking is just as foolish as ignoring the consequences of contamination. Others (IV-F-4.2, IV-F-3.78) supported the use of the best scientific methods for examining the problem and assessing the risks, but stated that this information must then be weighed in reference to the economic burden of closing the smelter. Another commenter (IV-D-388) said EPA's decision must be based on both economics and the morality of placing others lives in potential danger.

Others, however (IV-F-3.7, IV-F-3.7), said that it is difficult to equate the costs of abatement with human life, as the health impact is somewhat immeasurable in terms of dollars.

Response:

The EPA recognizes as the principal objective under section 112 of the establishment of regulations to protect public health. The EPA does not interpret this objective as a requirement that risks must be totally eliminated. It is EPA's view that the intent of section 112 is to insure that health risks from significant sources of hazardous air pollutants are reduced to the maximum extent practicable.

The EPA's regulatory analysis includes evaluation of all major impacts of selected control alternatives, focusing on health impacts but including consideration of energy, environmental, and economic impacts. In its arsenic rulemaking, EPA has sought to reduce health risks to an acceptable level while minimizing adverse economic impacts.

6.5 Other

This section contains comments which could not be classified under any of the major categories presented in this document.

Comment:

One commenter (IV-F-1.18) requested that EPA create a mechanism by which funds would be set aside, so when bad years come for cyclical industries they cannot claim that they cannot afford pollution control requirements that are needed to protect public health.

When asked what legal authority EPA could use to establish such a mechanism, the commenter agreed to give some thought to the question and submit his suggestions to EPA.

Response:

The EPA can find no basis under the Clean Air Act or other legal ground which would permit them to establish such a mechanism.

Comment:

One commenter (IV-D-28) said that we desperately need a legal definition of what constitutes arsenic-induced cancer.

Response:

It would simplify liability claims procedures and regulatory development if a definition of arsenic-induced cancer could be developed. However, there is not currently, and is not likely to be in the future, any way to precisely define arsenic-induced cancer. Lung cancer can be caused by smoking, genetic predisposition, or exposure to numerious environmental pollutants. Therefore it is extremely difficult to establish that a particular case of cancer was caused by arsenic exposure, even though epidemiologic studies show that, statistically, increased arsenic exposure leads to increased lung cancer risk.

Comment:

One commenter (IV-F-3.38) compared statistical deaths to a lottery where, for instance, there is one death out of 400,000 people. One unlucky person will lose this lottery, while 399,999 people will win it. He added that no one knows who that unlucky person will be. Two commenters (IV-D-56, IV-D-784) objected to EPA's dealing with human life in statistical terms. Another (IV-D-144) felt that if victims could be identified it would be more likely that standards would be set.

Response:

The EPA must estimate risk using a mathematical model because, for reasons discussed in the above response and section 2.2, it is not possible to measure risk directly or to predict if a specific individual will contract arsenic-induced lung cancer. Statistical risk is the only estimate of risk available for use in setting standards. The EPA realizes that the risk model is not precise enough for use in predicting the actual number of deaths which may occur in Tacoma as a result of arsenic exposure.

Comment:

One commenter (IV-F-4.43) suggested regulatory options that may be used to reduce the health risk from the arsenic emissions associated with the ASARCO-Tacoma smelter. He lists these options in order of preference:

(i) The zero risk option.

(ii) Impose no greater risk than imposed on 14 other copper smelting communities.

(iii) Restrict arsenic emissions to a level no greater than that emitted by the 14 other copper smelters and apply the standard at all times.

(iv) Require state-of-the-art technology at ASARCO and consider costs to the community when developing best available technology.

Response:

These approaches were considered by EPA in regulating arsenic emissions. The zero risk approach is considered impractical because if this were applied to all hazardous pollutants the result would be wide-scale economic disruption. The zero risk approach is discussed in section 6.1.4. The commenter's second and third alternatives were also rejected as a regulatory policy. The reasons are contained in section 6.1.5 on comparative risks. The commenters last suggestion is similar to the BAT approach, which is discussed in section 6.2. As explained in that section, EPA's regulatory approach is to evaluate each control option in terms of health risk reduction and residual risk, as well as technical feasibility and economic impact. Considering these factors, the Administrator will select a control option. [The ASARCO-Tacoma plant has ceased copper smelting operations. The EPA has promulgated standards for the arsenic plant which may remain in operation.]

Comment:

One commenter (IV-D-710) said that an ambient air quality standard for a carcinogen would be inappropriate as a public health policy matter and unauthorized under the Clean Air Act. Others (IV-D-621-5, IV-D-621-15.5, IV-D-621-15.1) commented that EPA lacks authority under § 112 of the Clean Air Act to adopt an ambient standard for a hazardous air pollutant. One (IV-D-621-15.5) thought it an improper technique for reducing fugitive emissions. Commenter IV-F-3.5 said that setting an ambient standard for a carcinogen is tricky, since there will always be a health risk with any non-zero exposure. Others (IV-F-3.8, IV-D-708a) wanted to know how EPA would propose to set an acceptable ambient air standard in the absence of

established medical criteria for setting risk levels. He said that the Clean Air Act does not provide for adoption of an ambient air standard for arsenic in the circumstances which now exist.

Another commenter (IV-D-24) said that EPA should base its arsenic emission standards on ambient concentration. He said it was his understanding that EPA's position is one of not being able to set ambient air quality standards since there is no safe level, therefore no margin for protection of public health. He and another commenter (IV-D-708a) wanted EPA to set "action levels".

Response:

Since an enforceable ambient standard is not being established in the copper smelter standard, the comment that section 112 of the Clean Air Act does not give EPA the authority to set enforceable ambient standards is not pertinent to this rulemaking. The EPA agrees that an ambient standard cannot be established for inorganic arsenic based solely on health effects or risk estimates. The EPA does believe, however, that an enforceable ambient limit, which is an indicator of proper operation and maintenance of emission control systems is consistent with the goal of section 112 and may consider establishing such a limit at a later date.

Comment:

One commenter (IV-F-4.59) felt that, rather than attempting to control the methodology for emission reduction, EPA should just set an emission level and let industry decide how it wants to achieve that level economically and technologically.

Response:

In response to the first commenter, EPA's equipment standard is not intended to preclude the use of other secondary inorganic arsenic capture systems which may be as effective as an air curtain secondary hood. As specified in the Federal Register (48 FR 33134, July 20, 1983):

"Upon written application to EPA, the use of an alternative secondary inorganic arsenic capture system which has been demonstrated to EPA's satisfaction to be equivalent in terms of capture efficiency for inorganic arsenic systems may be approved."

Therefore, industry may decide what methods it wants to use to comply with the emission standard.

Comment:

One commenter (IV-F-1.13) said that funds available for environmental control measures are not unlimited. For this reason he said it is important to put the available money where it gives the maximum benefit. The commenter said that the most benefit appears to be gained by reducing the overall pollutant emissions and by improving conditions in plants rather than by spending money to bring down an already very low level (0.05 $\mu g/m^3$) of a single element (arsenic). Another (IV-D-616) emphasized that funds were not unlimited and stressed the need to put available money where it can do the most good. With this in mind, he saw little support for imposing strict regulations on low levels of a single compound based on lung cancer found in groups of people exposed to high levels of a large number of compounds.

Response:

The EPA realizes priorities must be set in controlling various pollutants in order to utilize the available resources efficiently. However, it is unclear how the first commenter derived the arsenic level of 0.05 $\mu g/m^3$. Furthermore, it is not the concentration of a pollutant per se, but the health risks associated with ambient concentrations which must be used in deciding whether to regulate a pollutant.

Comment:

Two commenters (IV-D-301, IV-D-778) said that because U. S. citizens are entitled under the 1st Amendment of the U.S. Constitution to life

itself, they are all entitled to a clean, safe environment. Others (IV-F-3.103, IV-D-732, IV-D-779) felt that it is the duty of government in our society to protect the rights of individuals from those who would infringe upon them, and the right to a healthy environment is fundamental and inalienable. Another (IV-D-598) said the laws, guidelines, and standards of EPA must comply with the principles set forth in the Constitution. The commenter saw only one remedy: elimination of the hazard immediately. The commenter found no other remedy suggested in the Constitution.

Response:

The EPA must rely on the directives of the Clean Air Act in setting standards for air pollutants. The EPA has followed the provisions of section 112 of the Act in setting arsenic standards.

Comment:

One commenter (IV-F-4.68/IV-F-4.71) said that 40 CFR (along with the Geneva and Nuremburg Codes) states that human subjects must be protected from involuntary or uninformed exposure. She further stated that if EPA does not set stringent standards, the Agency's failure to act could be construed as sanctioning an epidemiological project in which people are exposed involuntarily to uncertain risks. Such projects, the commenter said, would be a violation of these codes. In a situation such as this, true voluntary consent is not possible because people are afraid of the economic consequences of strict standards.

One commenter (IV-F-4.28) said that EPA was using the people of Tacoma as human guinea pigs; yet they have not received informed consent from the residents, nor have they guaranteed that the health care needs of people who are exposed to arsenic will be taken care of. Another (IV-F-3.58) stated that citizens living near polluters should not bear the burden of proving poisons are harmful by being used as unwilling research subjects. Another commenter (IV-D-661) said that EPA cannot permit continued emissions in a

large populous area where many do not consent and many more are not even aware of the risk.

Response:

The EPA is not using the citizens of Tacoma to conduct a research experiment. Citizens were informed of the potential risk and asked for their opinions on whether any further regulations should be applied. The EPA had considered these comments before ASARCO announced the shutdown of the copper smelter operations.

Comment:

One commenter (IV-D-51) said that the cost-benefit criterion where human life is at stake is of doubtful morality. Another (IV-D-67) said that placing the environment and jobs on a balancing scale is neither morally nor rationally defensible. Another commenter (IV-D-241) felt that someone up at the "top" playing God is saying, "We can afford to let between 10 and 150 people die so that industry can operate at a larger profit." This kind of attitude he found insensitive.

Response:

Section 112 of the Clean Air Act gives the Agency the authority to impose controls on hazardous air pollutant emissions. The Administrator believes that section 112 decisions that may or may not require further emissions controls must consider not only the potential health effects associated with such hazardous air pollutant emissions, but also the costs and other impacts (e.g., loss of jobs) on society. Admittedly, they may be at times difficult but the Clean Air Act requires that these decisions be made.

Comment:

Two commenters (IV-F-4.4, IV-D-708a) said that if the smelter closes, foreign smelters will get their business. This would mean, in effect, that

we would simply be transferring our health risks to another country which might have little or no pollution control, and he questioned the morality of this action.

Response:

Due to the lack of information, EPA did not consider the pollution impacts on foreign populations if the U.S. smelters close down as a result of the promulgation of section 112 air emissions standards.

7.0 QUALITY OF LIFE

Comment:

Several commenters urged EPA to consider the effects of emissions from ASARCO on the quality of life in the Tacoma area. One commenter (IV-D-164/IV-D-666) said that the public was entitled to "peace of mind," knowing that their families were located in safe places to live. The commenter complained that protecting quality of life did not seem to be a part of protecting public health. Others (IV-D-375, IV-D-732, IV-D-751, IV-F-11) said that their quality of life has been altered by the smelter. Still another (IV-F-3.7) said that health and welfare was a paramount concern, as well as the ability of people to enjoy life productively. Another (IV-D-639) said that EPA should put quality of life as its top priority. Another (IV-D-524) urged EPA to consider the effect of the emissions on the environment and its being a pleasant, comfortable, and safe place to live.

Response:

The EPA is aware that quality of life is an important concern. Some sections of the Clean Air Act, such as sections 108 and 109, incorporate quality of life considerations. Under these sections primary national ambient air quality standards for criteria pollutants are established to protect health, and secondary standards to protect "welfare." However, section 112 of the Act deals with hazardous air pollutants and is a completely health-based section. Welfare, or quality of life, is not mentioned. Hazardous pollutants are defined in section 112(a)(1) as those which may cause ". . .an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness." In section 112 (b)(1)(B) EPA is authorized to set standards which provide ". . .an ample margin of safety to protect public health for such hazardous air pollutant."

The Administrator realizes that reduction in inorganic arsenic emissions may have other beneficial effects on the "quality of life" for the community. On the other hand, commenters have also reminded EPA that, in general, plant closures will have some potentially severe adverse effects on the "quality of life" for the community, such as unemployment and loss of tax revenues. In making his decision, the Administrator considered these public comments and was mindful that the selection of the control option would have other effects, both positive and negative, on the surrounding communities.

8.0 VICTIM COMPENSATION

Comment:

Several commenters felt that ASARCO and/or EPA should assume some liability for their impact on the community. Commenters (IV-D-621-12.2, IV-F-3.43, IV-F-4.28, IV-D-28, IV-D-721, IV-F-3.103) said ASARCO should be required to post a bond, establish a health fund, or buy insurance to cover future claims against the company and compensate victims. One commenter (IV-D-520) said ASARCO should periodically replace residents' topsoil and be accountable for financial loss and health risks incurred from their emissions. Two commenters (IV-D-520, IV-F-4.66) said costs of victim compensation should be included in the economic assessment. One commenter (IV-F-5.16) said ASARCO should pay for health testing and monitoring of residents and their employees. Three commenters (IV-F-5.16, IV-F-3.46) said ASARCO or the government should buy people's homes near the smelter for fair market value. Three commenters (IV-F-4.28, IV-F-4.43, IV-D-710, IV-F-11) said EPA should give aid to ASARCO in bearing the burden of compensation, relocation of potential victims, or adjustment assistance for displaced employees and the city. Another (IV-D-530) said if jobs were lost, money should be spent on retraining and placing workers. He said workers should not have to bear the burden of ASARCO's polluting effects.

On the other hand, two commenters (IV-D-481, IV-D-28) expressed concern that some people would misuse victims' rights in the hope of getting large settlements to which they are not entitled.

Two commenters (IV-D-28, IV-D-719) said the problem of determining who the victims are and if or how they should be compensated has not been addressed very well. The one commenter (IV-D-28) presented positive and negative factors associated with a private insurance approach for compensating victims. He favored this approach over a Federal program for compensating victims.

Response:

The Clean Air Act has no provision that allows EPA to compensate victims or to require ASARCO to set up a program to do this. However, section 304 of the Act does provide for citizen suits against emission sources, governmental agencies, and the EPA Administrator. As detailed in section 304, suits can be filed if sources are in violation of emissions standards or permits or if the Administrator fails to perform his duty under the Clean Air Act.

8.0 VICTIM COMPENSATION

Comment:

Several commenters felt that ASARCO and/or EPA should assume some liability for their impact on the community. Commenters (IV-D-621-12.2, IV-F-3.43, IV-F-4.28, IV-D-28, IV-D-721, IV-F-3.103) said ASARCO should be required to post a bond, establish a health fund, or buy insurance to cover future claims against the company and compensate victims. One commenter (IV-D-520) said ASARCO should periodically replace residents' topsoil and be accountable for financial loss and health risks incurred from their emissions. Two commenters (IV-D-520, IV-F-4.66) said costs of victim compensation should be included in the economic assessment. One commenter (IV-F-5.16) said ASARCO should pay for health testing and monitoring of residents and their employees. Three commenters (IV-F-5.16, IV-F-3.46) said ASARCO or the government should buy people's homes near the smelter for fair market value. Three commenters (IV-F-4.28, IV-F-4.43, IV-D-710, IV-F-11) said EPA should give aid to ASARCO in bearing the burden of compensation, relocation of potential victims, or adjustment assistance for displaced employees and the city. Another (IV-D-530) said if jobs were lost, money should be spent on retraining and placing workers. He said workers should not have to bear the burden of ASARCO's polluting effects.

On the other hand, two commenters (IV-D-481, IV-D-28) expressed concern that some people would misuse victims' rights in the hope of getting large settlements to which they are not entitled.

Two commenters (IV-D-28, IV-D-719) said the problem of determining who the victims are and if or how they should be compensated has not been addressed very well. The one commenter (IV-D-28) presented positive and negative factors associated with a private insurance approach for compensating victims. He favored this approach over a Federal program for compensating victims.

Response:

The Clean Air Act has no provision that allows EPA to compensate victims or to require ASARCO to set up a program to do this. However, section 304 of the Act does provide for citizen suits against emission sources, governmental agencies, and the EPA Administrator. As detailed in section 304, suits can be filed if sources are in violation of emissions standards or permits or if the Administrator fails to perform his duty under the Clean Air Act.

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EPA-450/5-85-002
April 1985

Air

EPA

Inorganic Arsenic Risk Assessment For Primary and Secondary Lead Smelters, Primary Zinc Smelters, Zinc Oxide Plants, Cotton Gins, and Arsenic Chemical Plants

NESHAP

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EPA-450/5-85-002

Inorganic Arsenic Risk Assessment for Primary and Secondary Lead Smelters, Primary Zinc Smelters, Zinc Oxide Plants, Cotton Gins, and Arsenic Chemical Plants

Strategies and Air Standards Division

U.S. ENVIRONMENTAL PROTECTION AGENCY
Office of Air and Radiation
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

April 1985

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INORGANIC ARSENIC RISK ASSESSMENT FOR PRIMARY AND SECONDARY LEAD SMELTERS, PRIMARY ZINC SMELTERS AND ZINC OXIDE PLANTS, COTTON GINS AND ARSENIC CHEMICAL PLANTS

1 INTRODUCTION

1.1 Overview

The quantitative expressions of public cancer risks presented in this document are based on (1) a dose-response model that numerically relates the degree of exposure to airborne inorganic arsenic to the risk of getting lung cancer, and (2) numerical expressions of public exposure to ambient air concentrations of inorganic arsenic estimated to be caused by emissions from stationary sources. Each of these factors is discussed briefly below and details are provided in the following sections of this document.

1.2 The Relationship of Exposure to Cancer Risk

The relationship of exposure to the risk of contracting lung cancer is derived from epidemiological studies in occupational settings rather than from studies of excess cancer incidence among the public. The epidemiological methods that have successfully revealed associations between occupational exposure and cancer for substances such as asbestos, benzene, vinyl chloride, and ionizing radiation, as well as for inorganic arsenic, are not readily applied to the public sector, with its increased number of confounding variables, much more diverse and mobile exposed population, lack of consolidated medical records, and almost total absence of historical exposure data. Given such uncertainties, EPA considers it improbable that any association, short of very large increases in cancer, can be verified in the general population with any reasonable certainty by an epidemiological study. Furthermore, as noted by the National Academy of Sciences (NAS)[1], "...when there is exposure to a material, we are not starting at an origin

of zero cancers. Nor are we starting at an origin of zero carcinogenic agents in our environment. Thus, it is likely that any carcinogenic agent added to the environment will act by a particular mechanism on a particular cell population that is already being acted on by the same mechanism to induce cancers." In discussing experimental dose-response curves, the NAS observed that most information on carcinogenesis is derived from studies of ionizing radiation with experimental animals and with humans which indicate a linear no-threshold dose-response relationship at low doses. They added that although some evidence exists for thresholds in some animal tissues, by and large, thresholds have not been established for most tissues. NAS concluded that establishing such low-dose thresholds "...would require massive, expensive, and impractical experiments ..." and recognized that the U.S. population "...is a large, diverse, and genetically heterogeneous group exposed to a large variety of toxic agents." This fact, coupled with the known genetic variability to carcinogenesis and the predisposition of some individuals to some form of cancer, makes it extremely difficult, if not impossible, to identify a threshold.

For these reasons, EPA has taken the position, shared by other Federal regulatory agencies, that in the absence of sound scientific evidence to the contrary, carcinogens should be considered to pose some cancer risk at any exposure level. This no-threshold presumption is based on the view that as little as one molecule of a carcinogenic substance may be sufficient to transform a normal cell into a cancer cell. Evidence is available from both the human and animal health literature that cancers may arise from a single transformed cell. Mutation research with ionizing radiation in cell cultures indicates that such a transformation can occur as the result of interaction with as little as a single cluster of ion pairs. In reviewing the available data regarding carcinogenicity, EPA found no compelling scientific reason to abandon the no-threshold presumption for inorganic arsenic.

In developing the exposure-risk relationship for inorganic arsenic, EPA has assumed that a linear no-threshold relationship exists at and below the levels of exposure reported in the epidemiological studies of occupational exposure. This means that any exposure to inorganic arsenic is assumed to pose some risk of lung cancer and that the linear relationship between cancer risks and levels of public exposure is the same as that between cancer risks and levels of occupational exposure. EPA believes that this assumption is reasonable for public health protection in light of presently available information. However, it should be recognized that the case for the linear no-threshold dose-response relationship model for inorganic arsenic is not quite as strong as that for carcinogens which interact directly or in metabolic form with DNA. Nevertheless, there is no adequate basis for dismissing the linear no-threshold model for inorganic arsenic. Assuming that exposure has been accurately quantified, it is the Agency's belief that the exposure-risk relationship used by EPA at low concentrations represents only a plausible upper-limit risk estimate in the sense that the risk is probably not higher than the calculated level and could be much lower.

The numerical constant that defines the exposure-risk relationship used by EPA in its analysis of carcinogens is called the unit risk estimate. The unit risk estimate for an air pollutant is defined as the lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed throughout their lifetimes (about 70 years) to an average concentration of 1 $\mu g/m^3$ of the agent in the air which they breathe. Unit risk estimates are used for two purposes: (1) to compare the carcinogenic potency of several agents with each other, and (2) to give a crude indication of the public health risk which might be associated with estimated air exposure to these agents.

The unit risk estimate for inorganic arsenic that is used in this appendix was prepared by combining the five different exposure-risk numerical constants developed from four occupational studies.[2] The methodology used to develop the unit risk estimate from the four studies is described in Section 2 below.

1.3 Public Exposure

The unit risk estimate is only one of the factors needed to produce quantitative expressions of public health risks. Another factor needed is a numerical expression of public exposure, i.e., the numbers of people exposed to the various concentrations of inorganic arsenic. The difficulty of defining public exposure was noted by the National Task Force on Environmental Cancer and Health and Lung Disease in their 5th Annual Report to Congress, in 1982.[3] They reported that "...a large proportion of the American population works some distance away from their homes and experience different types of pollution in their homes, on the way to and from work, and in the workplace. Also, the American population is quite mobile, and many people move every few years." They also noted the necessity and difficulty of dealing with long-term exposures because of "...the long latent period required for the development and expression of neoplasia [cancer]..." The reader should note that the unit risk estimate has been changed from that value used in the inorganic NESHAP proposal as a result of EPA's analysis of several occupational epidemiological studies that have recently been completed.

EPA's numerical expression of public exposure is based on two estimates. The first is an estimate of the magnitude and location of long-term average ambient air concentrations of inorganic arsenic in the vicinity of emitting sources based on dispersion modeling using long-term estimates of source emissions and meteorological conditions. The second is an estimate of the number and distribution of people living in the vicinity of emitting sources based on 1980 Bureau of Census data which "locates" people by population

centroids in census tract areas. The people and concentrations are combined to produce numerical expressions of public exposure by an approximating technique contained in a computerized model. The methodology is described in Section 3 below.

1.4 Public Cancer Risks

By combining numerical expressions of public exposure with the unit risk estimate, two types of numerical expressions of public cancer risks are produced. The first, called individual risk, relates to the person or persons estimated to live in the area of highest concentration as estimated by the computer model. Individual risk is expressed as "maximum lifetime risk." As used here, the work "maximum" does not mean the greatest possible risk of cancer to the public. It is based only on the maximum annual average exposure estimated by the procedure used. The second, called aggregate risk, is a summation of all the risks to people estimated to be living within the vicinity (usually within 50 kilometers) of a source and is customarily summed for all the sources in a particular category. The aggregate risk is expressed as incidences of cancer among all of the exposed population after 70 years of exposure; for convenience, it is often divided by 70 and expressed as cancer incidences per year. These calculations are described in more detail in Section 4 below.

There are also risks of nonfatal cancer and other potential health effects, depending on which organs receive the exposure. No numerical expressions of such risks have been developed.

2. THE UNIT RISK ESTIMATE FOR INORGANIC ARSENIC[2]

The following discussion is summarized from a more detailed description of the Agency's derivation of the inorganic arsenic unit risk estimate as found in EPA's "Health Assessment Document for Inorganic Arsenic" (EPA-600/8-83-021F).

2.1 The Linear No-Threshold Model for Estimation of Unit Risk Based on Human Data (General)

The methodologies used to arrive at quantitative estimates of risk must be capable of being implemented using the data available in existing epidemiologic studies of exposure to airborne arsenic. This requires extrapolation from the exposure levels and temporal exposure patterns in these studies to those for which risk estimates are required. It is assumed that the age-specific mortality rate of respiratory cancer per year per 100,000 persons for a particular 5-year age interval, i, can be represented using the following linear absolute or additive risk model:

$$a_i(D) = a_i + 100{,}000a'D \qquad (1)$$

With this model, a_i is the age-specific mortality rate per year of respiratory cancer in a control population not exposed to arsenic, a' is a parameter representing the potential of airborne arsenic to cause respiratory cancer, and D is some measure of the exposure to arsenic up to the ith age interval. For example, D might be the cumulative dose in years-$\mu g/m^3$, the cumulative dose neglecting exposure during the last 10 years prior to the ith age interval, or the average dose in $\mu g/m^3$ over some time period prior to the ith age interval. The forms to be used for D are constrained by the manner in which dose was treated in each individual epidemiologic study. At low exposures the extra lifetime probability of respiratory cancer mortality will very correspondingly (e.g., linearly).

The dose-response data available in the epidemiologic studies for estimating the parameters in these models consists primarily of a dose measure D_j for the jth exposure group, the person-years of observation Y_j, the observed number of respiratory cancer deaths O_j, and the number E_j of these deaths expected in a control population with the same sex and age distribution as the exposure group. The expected number E_j is calculated as

$$E_j = \sum_i Y_{ji} a_i / 100{,}000 \qquad (2)$$

here Y_{ji} is the number of person-years of observation in the ith age category and the jth exposure group ($Y_j = \sum_i Y_{ji}$). This is actually a simplified representation, because the calculation also takes account of the change in the age-specific incidence rates with absolute time. The expected number of respiratory cancer deaths for the ith exposure group is

$$E(O_j) = \sum_i Y_{ji} \, (a_i + 100{,}000 a' D_j)/100{,}000$$

$$= E_j + a' Y_j D_j \qquad (3)$$

under the linear absolute risk model. Consequently, $E(O_j)$ can be expressed in terms of quantities typically available from the published epidemiologic studies.

Making the reasonable assumption that O_j has a Poisson distribution, the parameter a' can be estimated from the above equation using the method of maximum likelihood. Once this parameter is estimated, the age-specific mortality rates for respiratory cancer can be estimated for any desired exposure pattern.

To estimate the corresponding additional lifetime probability of respiratory cancer mortality, let $b_1,\ldots,b_{18}$ be the mortality rates, in the absence of exposure, for all cases per year per 100,000 persons for the age intervals 0-4, 5-9,..., 80-84, and 85+, respectively; let $a_1,\ldots,a_{18}$ represent the corresponding rates for malignant neoplasms of the respiratory system. The probability of survival to the beginning of the ith 5-year age interval is estimated as

$$\prod_{j=1}^{i-1} [1 - 5b_j/100{,}000] \qquad (4)$$

Given survival to the beginning of age interval i, the probability of dying of respiratory cancer during this 5-year interval is estimated as

$$5a_i/100{,}000 \qquad (5)$$

The probability of dying of respiratory cancer given survival to age 85 is estimated as a_{18}/b_{18}. Therefore, the probability of dying of respiratory cancer in the absence of exposure to arsenic can be estimated as:

$$P_0 = \sum_{i=1}^{17} [5a_i/100{,}000) \prod_{j=1}^{i-1} (1-5b_j/100{,}000)]$$
$$+(a_{18}/b_{18}) \prod_{j=1}^{17} (1 - 5b_j/100{,}000)$$

Here the mortality rates a_i apply to the target population for which risk estimates are desired, and consequently will be different from those in

(1)-(5), which applied to the epidemiologic study cohort. If the 1976 U.S. mortality rates (male, female, white, and non-white combined) are used in this expression, then $P_0 = 0.0451$.

To estimate the probability P_{EP} of respiratory cancer mortality when exposed to a particular exposure pattern EP, the formula (6) is again used, but a_i and b_i are replaced by $a_i(D_i)$ and $b_i(D_i)$, where D_i is the exposure measure calculated for the ith age interval from the exposure pattern EP. For example, if the dose measure used in (1) is cumulative dose to the beginning of the ith age interval in $\mu g/m^3$-years, and the exposure pattern EP is a lifetime exposure to a constant level of 10 $\mu g/m^3$, then $D_i = (i-1)(5)(10)$, where the 5 accounts for the fact that each age interval has a width of 5 years. The additional risk of respiratory cancer mortality is estimated as

$$P_{EP} - P_0 \qquad (7)$$

If the exposure pattern EP is constant exposure to 1 $\mu g/m^3$, then $P_{EP} - P_0$ is called the "unit risk."

This approach can easily be modified to estimate the extra probability of respiratory cancer mortality by a particular age due to any specified exposure pattern.

2.2 Unit Risk Estimates Derived from Epidemiologic Studies

Prospective studies of the relationship between mortality and exposure to airborne arsenic have been conducted for the Anaconda, Montana smelter and the Tacoma, Washington smelter. Table 1 summarizes the fit of the absolute linear model to dose-response data from 4 different studies at the two smelters. (See the "Health Assessment Document for Inorganic Arsenic",

Table 1
Summary of Quantitative Risk Analyses

Exposed Population	Study and Data Source	Model	Results of Goodness-of-Fit Test		
			x^2 (d.f.)	p-value	"unit" risk[a]
Anaconda smelter workers	Lee-Feldstein (heavy exposure omitted)	absolute risk	12.7(5)	0.025	2.80(-3)[b]
	Higgins et al.	absolute risk	1.2(3)	0.75	4.90(-3)
	Brown & Chu	absolute risk	7.01(7)	0.41	1.25(-3)
ASARCO smelter workers	Enterline & Marsh (zero lag)	absolute risk	5.5(4)	0.24	6.81(-3)
	Enterline & Marsh (10-year lag)	absolute risk	7.0(4)	0.14	7.60(-3)

[a]Additional lifetime risk of respiratory cancer mortality from lifetime environmental exposure to 1 μgm^3 arsenic.
[b]2.80 (-3) means 2.80×10^{-3}

Chapter 7, EPA-600/8-83-021F for detailed description of occupational studies.) Table 1 also displays the carcinogenic potencies a'. It should be noted that the potencies estimated from different models are in different units, and are therefore not comparable.

The estimated unit risk is presented for each fit for which the chi-square goodness-of-fit p-value is greater than 0.01. The unit risks derived from linear models--8 in all--range from 0.0013 to 0.0136. The largest of these is from the Ott et al. study, which probably is the least reliable for developing quantitative estimates, and which also involved exposures to pentavalent arsenic, whereas the other studies involved trivalent arsenic. The unit risks derived from the linear absolute-risk models are considered to be the most reliable; although derived from 5 sets of data involving 4 sets of investigators and 2 distinct exposed populations, these estimates are quite consistent, ranging from 0.0013 to 0.0076.

To establish a single point estimate, the geometric mean for data sets is obtained within distinct exposed populations, and the final estimate is taken to be the geometric mean of those values. This process is illustrated in Table 2.

Table 2
Combined Unit Risk Estimates for Absolute-Risk Linear Models

Exposure Source	Study	Unit Risk	Geometric Mean Unit Risk	Final Estimated Unit Risk
Anaconda smelter	Brown & Chu	1.25×10^{-3}		
	Lee-Feldstein	2.80×10^{-3}	2.56×10^{-3}	
	Higgins et al.	4.90×10^{-3}		4.29×10^{-3}
ASARCO smelter	Enterline & Marsh	6.81×10^{-3}		
		7.60×10^{-3}	7.19×10^{-3}	

3. QUANTITATIVE EXPRESSIONS OF PUBLIC EXPOSURE TO INORGANIC ARSENIC EMISSIONS

3.1 EPA's Human Exposure Model (HEM) (General)

EPA's Human Exposure Model is a general model capable of producing quantitative expressions of public exposure to ambient air concentrations of pollutants emitted from stationary sources. HEM contains (1) an atmospheric dispersion model, with included meteorological data, and (2) a population distribution estimate based on Bureau of Census data. The input data needed to operate this model are source data, e.g., plant location, height of the emission release point, and volumetric rate of release temperature of the off-gases. Based on the source data, the model estimates the magnitude and distribution of ambient air concentrations of the pollutant in the vicinity of the source. The model is programmed to estimate these concentrations for a specific set of points within a radial distance of 50 kilometers from the source. If the user wishes to use a dispersion model other than the one contained in HEM to estimate ambient air concentrations in the vicinity of a source, HEM can accept the concentrations if they are put into an appropriate format.

Based on the radial distance specified, HEM numerically combines the distributions of pollutant concentrations and people to produce quantitative expressions of public exposure to the pollutant.

3.1.1 Pollutant Concentrations Near a Source

The HEM dispersion model is a climatological model which is a sector-averaged gaussian dispersion algorithm that has been simplified to improve computational efficiency.[5]

Stability array (STAR) summaries are the principal meteorological input to the HEM dispersion model. STAR data are standard climatological frequency-of-occurence summaries formulated for use in EPA models and available for major U.S. meteorological monitoring sites from the National Climatic Center, Asheville, N.C. A STAR summary is a joint frequency-of-occurence of wind speed, atmospheric stability, and wind direction, classified according to Pasquill's categories. The STAR summaries in HEM usually reflect five years of meteorological data for each of 314 sites nationwide. The model produces polar coordinate receptor grid points consisting of 10 downwind distances located along each of 16 radials which represent wind directions. Concentrations are estimated by the dispersion model for each of the 160 receptors located on this grid. The radials are separated by 22.5-degree intervals beginning with 0.0 degrees and proceeding clockwise to 337.5 degrees. The 10 downwind distances for each radial are 0.2, 0.5, 1.0, 2.0, 5.0, 10.0, 20.0, 30.0, 40.0, and 50.0 kilometers. The center of the receptor grid for each plant is assumed to be the plant center. Concentrations at other points were calculated by using a log-linear scheme as illustrated in Figure 1.

3.1.2 Expansion of Analysis Area

At proposal, exposure and risk were estimated for people residing within 20 kilometers of the smelter. Some commenters pointed out that since people beyond 20 kilometers are exposed to some level of arsenic due to a source's emissions, EPA's proposal analysis underestimates the total exposure and risk. EPA agreed with the commenters and expanded its analysis out to 50 kilometers. When applying air dispersion models, the EPA's modeling guidelines recommend that, because of the increasing uncertainty of estimates with distance from the modeled source and because of the paucity of validation studies at larger distances, the impact may extend

out to 50 kilometers but the analysis should generally be limited to this distance from the source.[4] Such site-specific factors as terrain features (complex or flat), the objectives of the modeling exercise, and distance to which the model has been validated will determine the appropriate distance (whether greater than or less than the guideline distance) for which the Agency should apply the model.

3.2 Methodology for Reviewing Pollutant Concentrations

Before making HEM computer runs, EPA reviewed small-scale U.S. Geological Survey topographical maps (scale 1:24000) to verify locational data for each arsenic source. Plants were given accurate latitude and longitude values which were then incorporated into the HEM program.

After completing the HEM runs, nearby monitoring sites with ambient air quality data were identified by a computer search of EPA's National Aerometric Data Bank (NADB) (Table 3). At some sites, data collected over several years along with annual averages (based on different numbers of sample sizes for the years monitored) for each year were available. In these instances, weighted multi-year averages were calculated to provide an overall mean for each monitoring site. For purposes of annual mean calculations, values measured below mimimum detection limits were considered by EPA to be equal to one-half the detection limit. These ambient arsenic data were then compared to HEM predicted values in order to gauge the accuracy of the air dispersion model's estimates. As noted above, HEM predicted values were based on concentrations at 160 polar coordinate receptor grid points consisting of 10 downwind distances located along each of 16 radials which represented wind directions. Because the actual monitoring site locations identified in the NADB retrieval usually did not correspond to exact grid point locations, a log-linear interpolation scheme (Figure 1) was used to calculate an estimated concentration at the site.

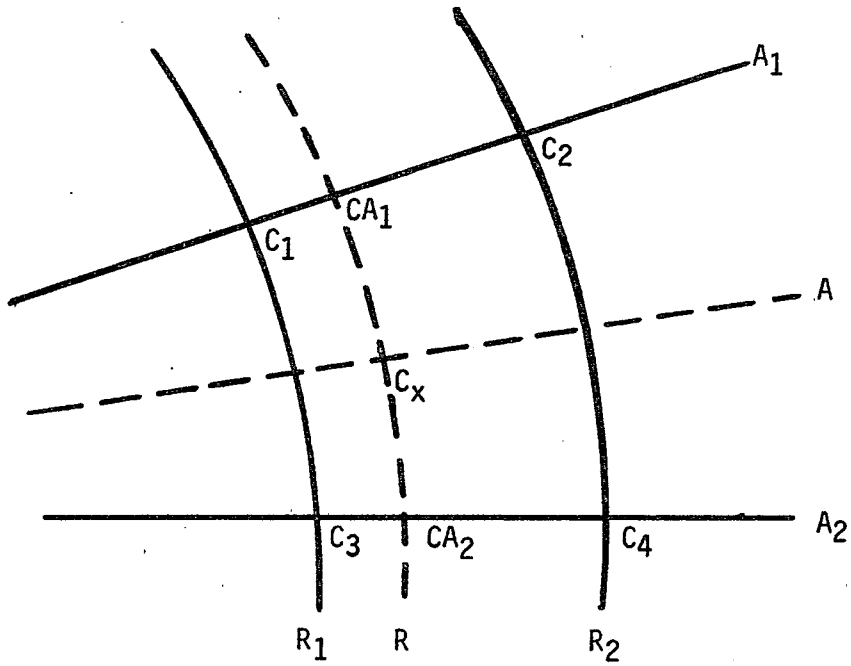
Table 3

Arsenic Concentrations Near ASARCO-East Helena
Primary Lead Smelter

Plant	# Obs.	Distance[1] (km)	Bearing	Predicted[2] (μg/m³)	Measured[3] (μg/m³)	MDL[4] (μg/m³)	Percentile[5]
ASARCO-East Helena	27	.5	119.6	0.230	0.108	0.02	30< % <50
	41	.7	11.5	0.078	0.151	0.02	<10
	137	.8	20.4	0.056	0.242	0.02	<10
	25	.9	343.9	0.050	0.161	0.02	<10
	31	1.4	45.3	0.076	0.078	0.02	30< % <50
	36	1.5	156.9	0.047	0.109	0.02	<10
	81	3.9	176.5	0.0159	0.031	0.02	70< % <90*
	23	4.7	270.4	0.005	0.025	0.02	50< % <70*
	20	7.2	273.4	0.003	0.030	0.0055	30< % <50*
Company Data	1460	1.1	275	0.024	0.059	--	--
	1460	1.3	5	0.050	0.24	--	--
	1460	1.3	145	0.077	0.078	--	--
	638	2.1	92	0.071	0.074	--	--
	1460	6.1	275	0.0037	0.024	--	--
	274	7.2	162	0.0084	0.028	--	--

* Indicates data point was disregarded; see Section 3.2.1
1 Distance from source to monitor (km)
2 Concentration predicted by Human Exposure Model (HEM). See Section 3.1.
3 The measured values are weighted averages. When the sampled arsenic concentrations were below the MDL, a value of 1/2 MDL was assumed for purposes of calculating the annual averages
4 Minimum detection limit
5 Percentile indicates percentage of data falling below minimum detectable levels.

Figure 1 Group 2 BG/ED Interpolation



Given:

A - The angle in radians subtended clockwise about the source from due south to the BG/ED centroid;

A1 - The angle from due south to the radial line immediately counter-clockwise of A, or passing through A if there is an exact match;

A2 - The angle from due south to the radial line immediately clockwise of A1 (A2 is 0 if it is due south);

R - The distance in km from the source to the BG/ED centroid;

R1 - The distance from the source to the largest circular arc of radius less than R;

R2 - The distance from the source to the smallest circular arc of radius greater than or equal to R;

C1 - The natural logarithm of the concentration value at (A1, R1);

C2 - The natural logarithm of the concentration value at (A1, R2);

C3 - The natural logarithm of the concentration value at (A2, R1);

C4 - The natural logarithm of the concentration value at (A2, R2);

then:

RTEMP - ln(R/R1)/ln(R2/R1);

ATEMP - (A-A1)/(A2-A1);

CA1 - exp(C1 + (C2-C1)xRTEMP);

CA2 - exp(C3 + (C4-C3)xRTEMP); and

CX - CA1 + (CA2-CA1)xATEMP,

where CX is the interpolated concentration at the BG/ED centroid.

3.2.1 Use of Ambient Data

Certain criteria were considered in review of ambient levels. Mean concentration values derived from sample sizes of less than 25 data points were disregarded. When reviewing the available monitoring data, it appeared that monitors situated at distances greater than 15 km from the arsenic source were considered too far from the source to gauge air dispersion results without interference from other arsenic sources. Furthermore, at distances greater than 15 km from the source, plant impacts were often predicted to be significantly lower than minimum detection limits. These data were not incorporated in the analyses. A third consideration in reviewing ambient data concerned the percentage of monitored data which fell below minimum detection limits. Although some monitoring sites registered data with over 90 percent of the values above minimum detection levels, many had about half the data points or more below such levels. Instances where more than 50 percent of the data were below MDL were disregarded. It should be noted that the various tables in subsequent sections display, in addition to company-collected data, all ambient monitoring data that were collected at sites within 15 kilometers of the source as identified by EPA's computer search although not all the data were used in the final analysis.

3.2.2 The People Living Near A Source

To estimate the number and distribution of people residing within 50 kilometers of the source, the HEM model uses the 1980 Master Area Reference File (MARF) from the U.S. Bureau of Census. This data base consists of enumeration district/block group (ED/BG) values. MARF contains the population centroid coordinates (latitude and longitude) and the 1980 population of each ED/BG (approximately 300,000) in the United States (50 states plus the District of Columbia). HEM identifies the population around each plant, by using the

geographical coordinates of the plant, and identifies, selects, and stores for later use those ED/BGs with coordinates falling within 50 kilometers of plant center.

3.2.3 Exposure[5]

The Human Exposure Model (HEM) uses the estimated ground level concentrations of a pollutant together with population data to calculate public exposure. For each of 160 receptors located around a plant, the concentration of the pollutant and the number of people estimated by the HEM to be exposed to that particular concentration are identified. The HEM multiplies these two numbers to produce exposure estimates and sums these products for each plant.

A two-level scheme has been adopted in order to pair concentrations and populations prior to the computation of exposure. The two level approach is used because the concentrations are defined on a radius-azimuth (polar) grid pattern with non-uniform spacing. At small radii, the grid cells are usually smaller than ED/BG's; at large radii, the grid cells are usually larger than ED/BG's. The area surrounding the source is divided into two regions, and each ED/BG is classified by the region in which its centroid lies. Population exposure is calculated differently for the ED/BG's located within each region. For ED/BG centroids located between 0.2 and 3.5 km from the emission source, populations are divided between neighboring concentration grid points. There are 64 (4 x 16) polar grid points within this range. Each ED/BG can be paired with one or many concentration points. The population associated with the ED/BG centroid is then divided among all concentration grid points assigned to it. The land area within each polar sector is considered in the apportionment.

For population centroids between 3.5 and 50 km from the source, a concentration grid cell, the area approximating a rectangular shape bounded by four receptors, is much larger than the area of a typical ED/BG. Since there is an approximate linear relationship between the logarithm of concentration and the logarithm of distance for receptors more than 2 km from the source, the entire population of the ED/BG is assumed to be exposed to the concentration that is logarithmically interpolated radially and arithmetically interpolated azimuthally from the four receptors bounding the grid cell. Concentration estimates for 96 (6 x 16) grid cell receptors at 5.0, 10.0, 20.0, 30.0, 40.0, and 50.0 km from the source along each of 16 wind directions are used as reference points for this interpolation.

In summary, two approaches are used to arrive at coincident concentration/population data points. For the 64 concentration points within 3.5 km of the source, the pairing occurs at the polar grid points using an apportionment of ED/BG population by land area. For the remaining portions of the grid, pairing occurs at the ED/BG centroids themselves through the use of log-log and linear interpolation. (For a more detailed discussion of the model used to estimate exposure, see Reference 5.)

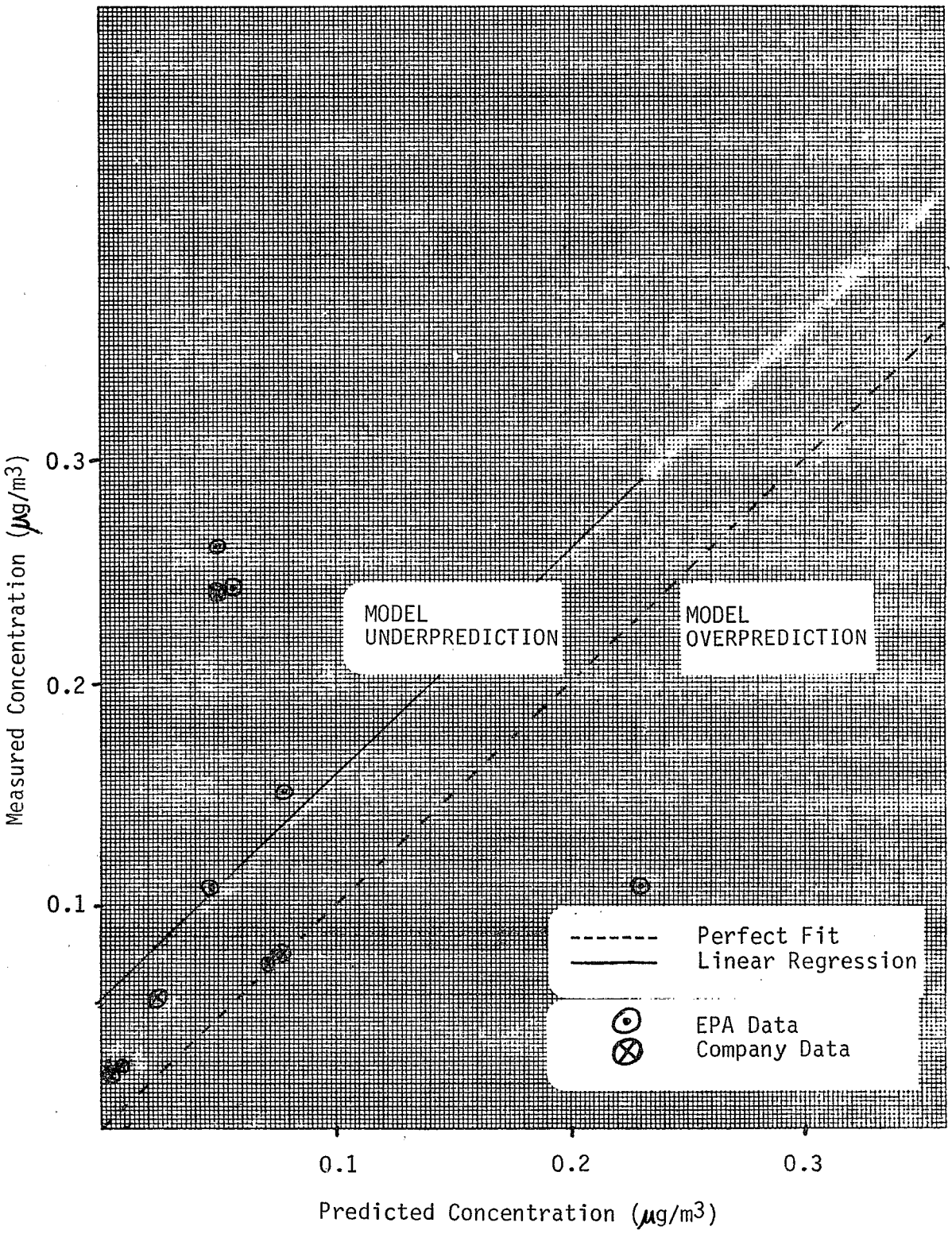
3.3 ASARCO-East Helena

Predicted (HEM) versus measured data were plotted (Figure 2) and a least squares weighted linear regression analysis was run based on thirteen data points (see Table 3). The least squares regression line (solid line) was determined on the basis of a comparison of National Aerometric Data Bank monitoring data (circumscribed dots) and ASARCO monitoring data (circumscribed Xs) with ambient concentrations predicted by the Human Exposure Model.

The reader should note that a perfect fit for the least squares regression analysis results in a line running through the origin at a 45° angle (dotted line on Figure 2). This means that if the HEM model predicts the measured data perfectly, then the data points would fall on the dotted line. In cases where the HEM model underpredicts concentrations, data points will be located above the 45° perfect fit line. Likewise, when the HEM model overpredicts concentrations, data points will be located below the perfect fit line. The regression line resulting from our comparison of predicted and monitored data runs nearly parallel to the perfect fit line but intersects the ordinate axis at a value of approximately 0.05 $\mu g/m^3$. This result is consistent with the expectation that air dispersion modeling would underpredict ambient concentrations. The air dispersion modeling did not consider other local sources of arsenic such as naturally-occurring arsenic in the windblown dust and reentrained arsenic particulate matter that had settled to the earth from past smelter emissions.

A study to determine source apportionment for particulate lead and total suspended particulates (TSP) in East Helena was completed in 1982. High volume TSP, low volume TSP, and dichotomous samplers were co-collected (same time period and same site) to permit differences in sample collection mass and chemistry to be understood. Analysis of hi-vol samples was carried out by the State of Montana and lo-vol and dichotomous samples were analyzed

FIGURE 2 Predicted Versus Measured
Inorganic Arsenic Ambient Concentrations
(ASARCO - East Helena, MT)



by NEA, Inc. In addition to particulate lead and TSP, samples were also measured in some cases for arsenic.[6]

At six locations where arsenic concentrations were measured using both lo-vol and hi-vol samplers, the ratio of lo-vol to hi-vol in percent ranged from 104 to 133 with a mean of 118%. This loss of arsenic compounds could have occurred in two areas: (1) the volatilization of the arsenic compounds from the hi-vol filter itself during sampling, and (2) the loss of volatile arsenic compounds during digestion and storage of samples prior to analysis. However, based on the data from the study, EPA concluded that the loss of arsenic on hi-vol filters was relatively minor in nature and within the overall accuracy goal of ± 15-20% considered adequate for most ambient air quality measurements.

3.3.1 Public Exposure to Inorganic Arsenic Emissions from Primary Lead Smelters

3.3.1.1 Source Data

Five primary lead smelters are included in the analysis. Table 4 lists the names and addresses of the plants considered, and Table 5 lists the plant data used as input to the Human Exposure Model (HEM).

3.3.1.2 Exposure Data

Table 6 lists, on a plant-by-plant basis, the total number of people encompassed by the exposure analysis and the total exposure. Total exposure is the sum of the products of number of people times the ambient air concentration to which they are exposed, as calculated by HEM. Table 7 sums, for the entire source category (5 plants), the numbers of people exposed to various ambient concentrations, as calculated by HEM. (Source-by-source exposure results are provided in the EPA docket numbered A-83-23.)

TABLE 4

IDENTIFICATION OF PRIMARY LEAD SMELTERS

Plant Number Code	Plant Name and Address
1	ASARCO East Helena, MT
2	ASARCO El Paso, TX
3	St. Joe Herculaneum, MD
4	ASARCO Glover, MO
5	Amax Boss, MO

Table 5 Input Data to Exposure Model Primary Lead Smelting Industry
(Assuming Baseline Controls)

Plant (Furnace)	Latitude (Degrees Minutes Seconds)	Longitude (Degrees Minutes Seconds)	Emission Rate (Kg/yr)	Emission Point Elevation (Meters)	Emission Point Diameter (Meters)	Emission* Point Cross Sectional Area (m^2)	Emission Point Gas Exit Velocity m/sec	Emission Point Gas Temp. (°K)	Emission Point Type
ASARCO-East Helena, MT	46-34-52	111-55-12	14700	128	2.7	100	16.5	352	Stack
			1680	130	3.0	100	19.4	330	Stack
			1680	122	3.4	100	11.7	375	Stack
			5040	0	--	10000	--	293	Fugitive
ASARCO-El Paso, TX	31-47-06	106-37-23	1680	186	4.3	100	17.0	345	Stack
			1680	91	4.9	100	3.0	330	Stack
			5040	0	--	10000	--	293	Fugitive
St. Joe-Herculaneum, MO	38-15-47	90-22-59	2772	107	6.1	100	13.5	353	Stack
			84	0	--	10000	--	293	Fugitive
ASARCO-Glover, MO	36-29-46	90-41-28	1680	24	1.5	100	18.3	294	Stack
			42	0	--	10000	--	293	Fugitive
Amax-Boss, MO	37-38-31	91-11-35	1680	61	4.6	100	10.0	355	Stack
			50.4	0	--	10000	--	293	Fugitive

TABLE 6 TOTAL EXPOSURE AND NUMBER OF PEOPLE EXPOSED
PRIMARY LEAD SMELTING INDUSTRY*

Plant	Total Number of People Exposed	Total Exposure (People - $\mu g/m^3$)
1	48,600	215
2	497,000	715
3	1,510,000	186
4	97,300	27
5	42,700	7

* A 50-kilometer radius was used for the analysis of primary lead smelting industry.

TABLE 7

PUBLIC EXPOSURE FOR PRIMARY LEAD SMELTING INDUSTRY
AS PRODUCED BY THE HUMAN EXPOSURE MODEL
(ASSUMING BASELINE CONTROLS)

Concentration Level (ug/m^3)	Population Exposed (Persons)*	Exposure (Persons - $\mu g/m^3$)**
0.437	<1	0
0.25	1	0
0.1	40	6
0.05	441	33
0.025	1240	62
0.01	7700	144
0.005	15900	199
0.0025	71700	398
0.001	340000	801
0.0005	545000	945
0.00025	657000	983
0.0001	1470000	1100
0.00005	2080000	1150
0.0000269	2190000	1150

*Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

**Column 3 displays the computed value of the cumulative exposure to the matching and higher concentation levels found in column 1.

3.4 Murph Metals-Dallas and Quemetco-Seattle

Predicted (HEM) versus measured data for Murph Metals-Dallas and Quemetco-Seattle were plotted (Figures 3 and 4) and a least squares weighted linear regression analysis was run based on a number of data points. The least squares regression line (solid line) was determined on the basis of a comparison of National Aerometric Data Bank monitoring data (circumscribed dots) and State agency monitoring data (circumscribed Xs) with ambient concentrations predicted by the Human Exposure Model.

The reader should note that a perfect fit for the least squares regression analysis results in a line running through the origin at a 45° angle (dotted lines in Figures 3 and 4). This means that if the HEM model predicts perfectly, then the data points would fall on the 45° line. In cases where the HEM model underpredicts concentrations, data points will be located above the 45° perfect fit line. Likewise, when the HEM model overpredicts concentrations, data points will be located below the perfect fit line. The regression line resulting from our comparison of predicted and monitored data lies above the perfect fit line, intersecting the ordinate axis at values of approximately 0.011 $\mu g/m^3$ and 0.026 $\mu g/m^3$ for Murph Metals and Quemetco respectively. This result is consistent with the expectation that air dispersion modeling would underpredict ambient concentrations. The air dispersion modeling did not consider other local sources of arsenic such as naturally-occurring arsenic in the windblown dust and re-entrained arsenic particulate matter that had settled to the earth from past smelter emissions.

FIGURE 3 Predicted Versus Measured
Inorganic Arsenic Ambient Concentrations
(Murph Metals - Dallas, TX)

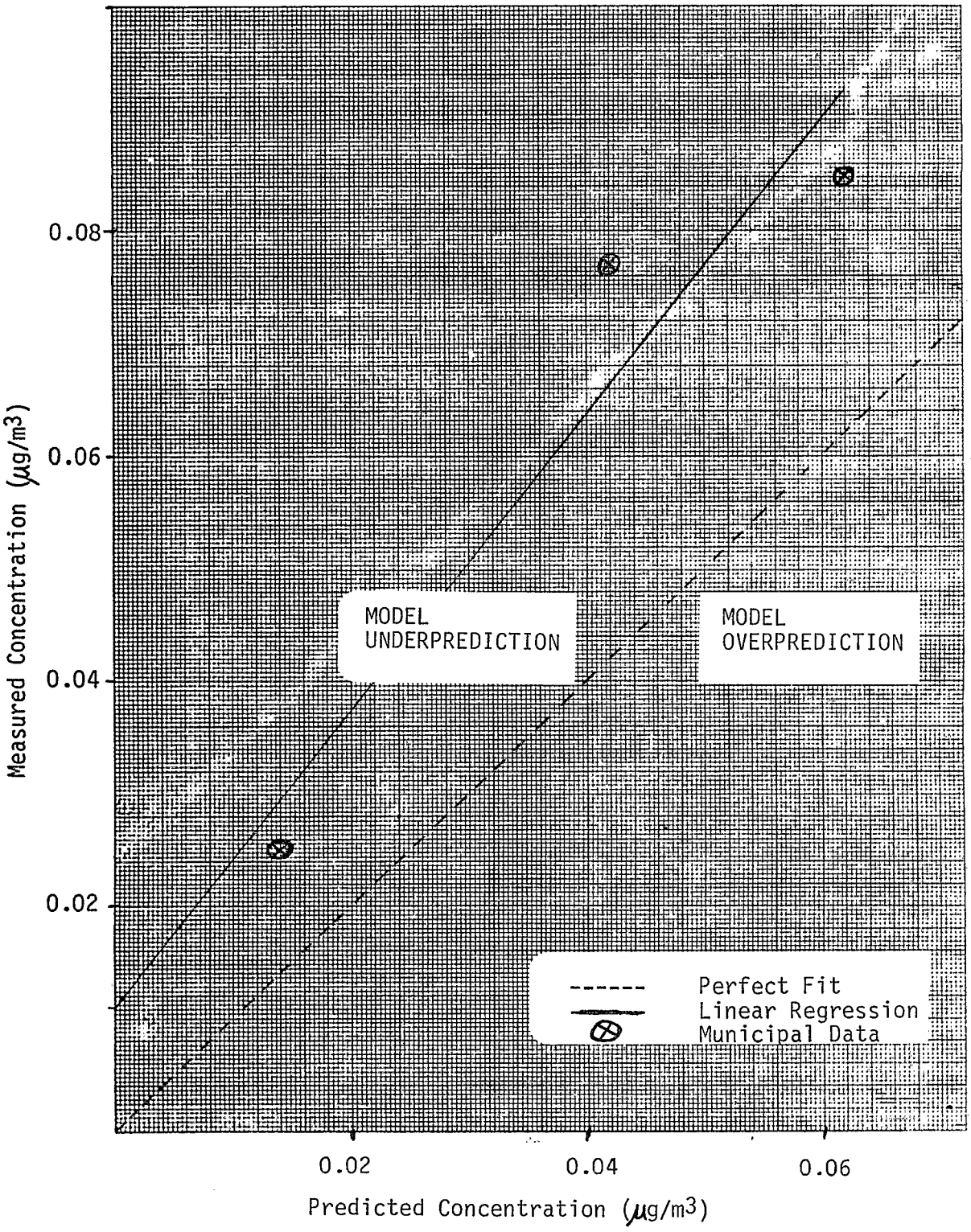
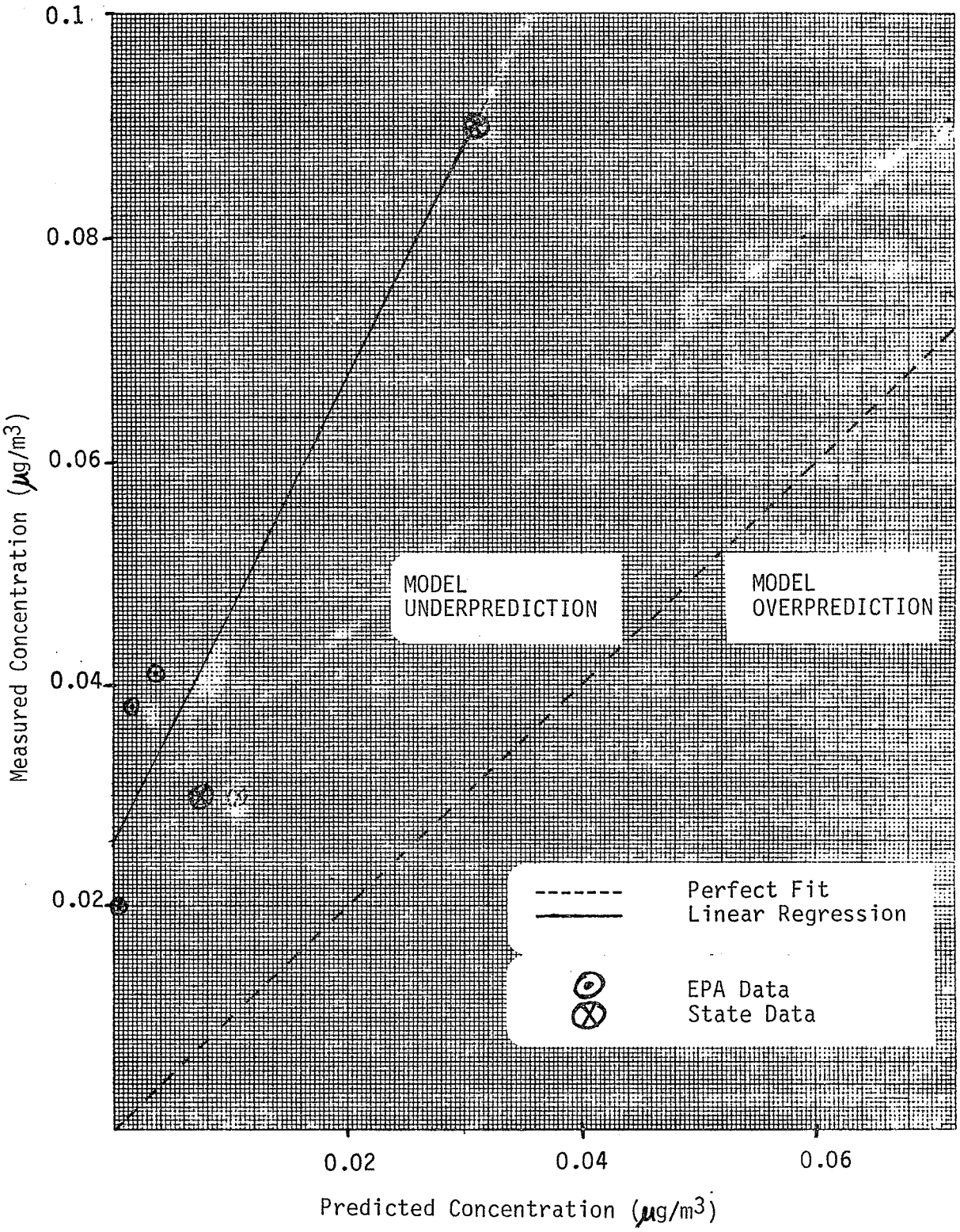


FIGURE 4 Predicted Versus Measured
Inorganic Arsenic Ambient Concentrations

(QUEMETCO - Seattle, WA)



3.4.1 Public Exposure to Inorganic Arsenic Emissions from Secondary Lead Smelters

3.4.1.1 Source Data

Thirty-five secondary lead smelters are included in the analysis. Table 8 lists arsenic concentrations near select secondary lead smelters. Table 9 lists the names and addresses of the plants considered, and Table 10 lists the plant data used as input to the Human Exposure Model (HEM).

3.4.1.2 Exposure Data

Table 11 lists, on a plant-by-plant basis, the total number of people encompassed by the exposure analysis and the total exposure. Total exposure is the sum of the products of number of people times the ambient air concentration to which they are exposed, as calculated by HEM. Table 12 sums, for the entire source category (35 plants), the number of people exposed to various ambient concentrations, as calculated by HEM. (Source-by-source exposure results are provided in the EPA docket numbered A-83-9.)

Table 8

Arsenic Concentrations Near Select
Secondary Lead Smelters

Plant	# Obs.	Distance[1] (km)	Bearing	Predicted[2] ($\mu g/m^3$)	Measured[3] ($\mu g/m^3$)	MDL[4] ($\mu g/m^3$)	Percentile[5]
General Battery, Reading, PA	29	5.1	189.5	0.00104	0.009	0.0055	30< % <50
Murph Metals-Dallas, TX	86	3.6	181.6	0.0024	0.028	0.05	90< % <95*
	21	3.7	181.5	0.0024	0.010	0.0055	30< % <50*
	93	7.6	311.6	0.00095	0.029	0.05	90, % <95*
	57	9.0	256.2	0.0004	0.025	0.05	>99*
Murph Metals-Dallas Texas Air Control Board Data	28	0.2	0	0.062	0.085	--	--
	31	0.2	337.5	0.042	0.077	--	--
	31	0.5	157.5	0.014	0.025	--	--
Quemetco-City of Industry, CA	29	17.8	314.0	0.00037	0.005	0.0055	70< % <90*
	25	22.8	281.9	0.000133	0.003	0.0055	>99*
	81	23.6	164.2	0.00018	0.003	0.0055	90< % <95*
	85	24.0	275.2	0.00023	0.006	0.0055	70< % <90*
	27	31.2	218.2	0.000083	0.003	0.0055	>99*
	47	32.2	161.0	0.000112	0.003	0.0055	70< % <90*
	30	35.1	300.0	0.000106	0.003	0.0055	>99*
	121	36.1	84.8	0.000113	0.005	0.0055	70< % <90*
	29	38.1	235.8	0.000066	0.004	0.0055	70< % <90*
Quemetco-Indianapolis, IN	64	12.5	78.2	0.00040	0.005	0.0055	50< % <70*
Quemetco-Seattle, WA	80	1.9	150.8	0.0036	0.041	0.0055	<10
	60	3.2	30.3	0.00183	0.038	0.0055	30< % <50
	72	13.5	2.6	0.00047	0.020	0.0055	30< % <50
Quemetco-Seattle Washington State Dept. of Ecology Data	60	0.2	157.5	0.031	0.09	--	--
	60	1.4	180	0.0075	0.03	--	--

*Indicates data point was disregardfed - see Section 3.3.1.1.
[1]Distance from source to monitor (km).
[2]Concentration predicted by Human Exposure Model (HEM). See Section 3.1.
[3]The measured values are weighted averages. When the sampled arsenic concentrations were below the MDL, a value of 1/2 MDL was assumed for purposes of calculating the annual averages.
[4]Minimum Detection Limit.
[5]Percentile indicates percentage of data falling below minimum detectable levels.

Table 9

Identification of Secondary Lead Smelters

Plant Number Code	Plant Name and Address
1	Alco Pacific Gardena, CA
2	Bergsoe St Helens, OR
3	Chloride Metals Columbus, GA
4	Chloride Metals Tampa, FL
5	Dixie Metals Dallas, TX
6	East Penn Lyons Station
7	Federated Metals San Fran, CA
8	General Battery Reading, PA
9	General Smelting College Grov, TN
10	Gopher Eugene, Minn
11	Gould Frisco, TX
12	Gould Vernon, CA
13	Gulf Coast Tampa, FL
14	Hyman Viener Richmond, VA
15	Interstate Lead Leeds, AL
16	Lancaster Lancaster, PA
17	Master Metals Cleveland, OH
18	Murph Metals Dallas, TX
19	National Smelting Atlanta, GA
20	National Smelting Pedricktown, NJ
21	Quemetco City of Industry, CA
22	Quemetco Indianapolis, IN
23	Quemetco Seattle, WA
24	Refined Metals Beach Grove, IN
25	Refined Metals Memphis, TN
26	Revere Wall, NY
27	Ross Metals Rossville, TN
28	Sanders Lead Troy, AI
29	Schuylkill Baton Rouge, LA
30	Schuylkill Forest City, MO
31	Standard San Antonio, TX
32	Taracorp Atlanta, GA
33	Taracorp Granite City, IL
34	Tonolli Nesquehoning, PA
35	USS Lead E. Chicago, IN

Table 10

Secondary Lead Industry Inputs to HEM Model
(Assuming Baseline Controls)

		Process					Process Fugitive					Area	
Plant	Latitude/Longitude (Degrees-Minutes-Seconds)	As Emission Rate kg/yr	Stack Ht. m	Stack Diam. m	Stack Vel. m/sec	Exit Temp. °K	Emission Rt. kg/yr	Stack Ht. m	Stack Diam. m	Stack Vel. m/sec	Exit Temp. °K	As Emission Rt. k/yr	Area m
Alco Pacific	33-50-20/118-18-07	7.6	31	0.62	29	331	.25	15.4	1.2	10.4	311	4.0	2,945
Bergsoe	45-50-58-122-49-3	86.6	31	0.92	25.9	400	2.6	15.4	1.2	13.3	311	0	5,862
Chloride Metals/GA	32-26-00/84-56-00	34.9	31	0.92	25.9	400	0.5	15.4	1.2	13.3	311	17.8	5,862
Chloride Metals/FL	27-54-5/82-24-12	34.9	31	0.92	25.9	400	0.5	15.4	1.2	13.3	311	13.8	5,862
Dixie Metals	32-44-49/96-46-37	52.3	31	0.92	25.9	400	0.75	15.4	1.2	13.3	311	168.9	5,862
East Penn	40-28-19/75-58-23	52.3	31	0.92	25.9	400	0.75	15.4	1.2	13.3	311	20.0	5,862
Federated Metals/CA	37-44-/122-23	14.5	31	0.62	29	400	0.21	15.4	1.2	10.4	311	4.0	2,945
General Battery	40-22-45/75-54-50	91.5	26	1.2	16.6	400	2.98	15.4	1.2	10.6	311	253.8	8,788
General Smelting	35-48-00/86-40-05	17.4	31	0.62	29	400	0.25	15.4	1.2	10.4	311	7.1	2,945
Gopher	40-50-/93-7-30	43.6	31	0.92	25.9	400	0.62	15.4	1.2	13.3	311	46.2	5,862
Gould/TX	33-08-38/96-49-44	87.2	31	0.92	25.9	400	1.24	15.4	1.2	13.3	311	13.8	5,862
Gould/CA	34-00-14/118-13-45	53.4	26	1.2	16.6	331	3.83	15.4	1.2	10.6	311	0	8,788
Gulf Coast	27-57-44/82-22-53	36.3	31	0.92	25.9	400	0.52	15.4	1.2	13.3	311	16.9	5,862
Hyman Viener	37-31-10/77-24-54	11.6	31	0.62	29	400	0.17	15.4	1.2	10.4	311	7.1	2,945
Interstate Lead	33-31-58/86-32-00	69.7	31	0.92	25.9	400	0.99	15.4	1.2	13.3	311	67.6	5,862
Lancaster	40-03-11/76-19-52	0.4	31	0.62	29	400	0.01	15.4	1.2	10.4	311	7.1	2,945
Master Metals	41-28-52/81-40-48	69.7	31	0.92	25.9	400	0.99	15.4	1.2	13.3	311	1.3	5,862
Murph Metals	32-46-40/96-52-21	174.3	26	1.2	16.6	400	2.48	15.4	1.2	10.6	311	692.1	8,788
National Smelting/GA	33-47-31/84-24-18	43.6	31	0.92	25.9	400	0.62	15.4	1.2	13.3	311	18.7	5,862
National Smelting/NJ	39-45-30/75-25-30	174.3	26	1.2	16.6	400	2.48	15.4	1.2	10.6	311	115.6	8,788

Table 10 (Continued)

Secondary Lead Industry Inputs to HEM Model
(Assuming Baseline Controls)

		Process					Process Fugitive					Area	
Plant	Lat/Long	As Emission Rate kg/yr	Stack Ht. m	Stack Diam. m	Stack Vel. m/sec	Exit Temp. °K	Emission Rt. kg/yr	Stack Ht. m	Stack Diam. m	Stack Vel. m/sec	Exit Temp. °K	As Emission Rt. k/yr	Area m
Quemetco/CA	34-01-30/117-58-58	61.0	26	1.2	16.6	331	1.99	15.4	1.2	10.6	311	692.1	8,788
Quemetco/IN	39-45-14/86-17-59	139.5	26	1.2	16.6	400	1.99	15.4	1.2	10.6	311	692.1	8,788
Quemetco/WA	47-34-44/122-21-04	69.7	31	0.92	25.9	400	0.99	15.4	1.2	13.3	311	461.4	5,862
Refined Metals/IN	39-42-36/86-03-5 4"	52.3	31	0.92	25.9	400	0.75	15.4	1.2	13.3	311	76.9	5,862
Refined Metals/TN	35-05-13/90-04-10	87.2	31	0.92	25.9	400	1.24	15.4	1.2	13.3	311	76.9	5,862
Revere.NY	41-27-37/74-21-35	139.5	26	1.2	16.6	400	1.99	15.4	1.2	10.6	311	692.1	8,788
Ross Metals	35-02-42/89-34-30	27.9	31	0.62	29	400	0.39	15.4	1.2	10.4	311	0	2,945
Sanders Lead	31-47-28/85-58-16	116.2	31	0.92	25.9	400	1.66	15.4	1.2	13.3	311	29.3	5,862
Schuylkill/LA	30-58-08/91-14-40	232.4	26	1.2	16.6	400	3.31	15.4	1.2	10.6	311	20.9	8,788
Schuylkill/MO	40-01-59/95-13-59	104.6	31	0.92	25.9	400	1.49	15.4	1.2	13.3	311	13.8	5,862
Standard	29-20-00/98-29-38	23.5	31	0.62	29	400	0.34	15.4	1.2	10.4	311	7.6	2,945
Taracorp/GA	33-47/84-22	87.2	31	0.92	25.9	400	1.24	15.4	1.2	13.3	311	13.8	5,862
Taracorp/IL	38-42-05/90-08-37	244.1	26	1.2	16.6	400	3.48	15.4	1.2	10.6	311	23.1	8,788
Tonolli	40-51-03/75-52-46	57.2	26	1.2	16.6	331	1.86	15.4	1.2	10.6	311	69.3	8,788
USS Lead	41-36-58/87-27-47	58.1	31	0.92	25.9	400	0.83	15.4	1.2	13.3	311	61.3	5,862

Table 11

Total Exposure and Number of People Exposed
Secondary Lead Smelting Industry*

Plant	Total Number of People Exposed	Total Exposure (People - $\mu g/m^3$)
1	8,450,000	32
2	1,120,000	15
3	314,000	17
4	1,670,000	19
5	2,350,000	228
6	1,310,000	16
7	3,370,000	22
8	1,260,000	250
9	679,000	3
10	58,200	1
11	1,800,000	17
12	8,900,000	189
13	1,690,000	32
14	766,000	15
15	844,000	41
16	1,160,000	6
17	2,530,000	59
18	2,560,000	668
19	1,920,000	51
20	4,210,000	197
21	8,860,000	2300
22	1,150,000	460
23	2,060,000	576
24	1,180,000	67
25	927,000	115
26	948,000	249
27	902,000	4
28	92,000	11
29	143,000	4
30	149,000	2
31	1,050,000	24
32	1,920,000	83
33	2,190,000	159
34	934,000	21
35	5,280,000	103

* A 50-kilometer radius was used for the analysis of secondary lead smelters.

Table 12

Public Exposure for Secondary Lead Smelters
as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons - $\mu g/m^3$)**
0.101	<1	0
0.1	<1	0
0.05	256	16
0.025	2880	104
0.01	16000	300
0.005	53100	543
0.0025	152000	878
0.001	743000	1770
0.0005	1940000	2590
0.00025	4510000	3480
0.0001	12800000	4750
0.00005	21700000	5390
0.000025	31100000	5720
0.00001	44000000	5930
0.000005	55700000	6010
0.0000025	64100000	6050
0.000001	70800000	6060
0.0000005	73800000	6060
0.00000025	74700000	6060

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

3.5 Public Exposure to Inorganic Arsenic Emissions from Primary Zinc Smelters

3.5.1 Source Data

Five primary zinc smelters are included in the analysis. Table 13 lists ambient arsenic concentrations near select primary zinc smelters. Table 14 lists the names and addresses of the plants considered, and Table 15 lists the plant data used as input to the Human Exposure Model (HEM).

3.5.2 Exposure Data

Table 16 lists, on a plant-by-plant basis, the total number of people encompassed by the exposure analysis and the total exposure. Total exposure is the sum of the products of number of people times the ambient air concentration to which they are exposed, as calculated by HEM. Table 17 sums, for the entire source category (5 plants), the numbers of people exposed to various ambient concentrations, as calculated by HEM. (Source-by-source exposure results are provided in the EPA docket numbered A-83-23.)

Table 13

Arsenic Concentrations Near
Select Primary Zinc Smelters

Plant	# Obs	Distance[1] (km)	Bearing	Predicted[2] (μg/m^3)	Measured[3] (μg/m^3)	MDL[4] (μg/m^3)	Percentile[5]
St. Joe-Monaca, PA	No data within 15 km						
ASARCO-Corpus Christi, TX	36	3.0	262.8	0.000024	0.025	0.05	100*
	299	3.8	252.1	0.0000149	0.026	0.05	95< % <99*
	33	5.5	299.4	0.0000156	0.025	0.05	100*
	83	6.2	289.7	0.0000125	0.029	0.05	95< % <99*
	319	6.9	173.0	0.0000067	0.026	0.05	95< % <99*
	26	14.9	187.7	0.0000029	0.008	0.0055	30< % <50*
	190	14.9	187.7	0.0000029	0.028	0.05	90< % <95*
Amax-Sauget, IL	27	4.1	314.4	0.000042	0.007	0.0055	50< % <70*
Jersey Miniere Zinc Co-Clarksville, TN	No data within 15 km						
National Zinc-Bartlesville, OK	No data within 15 km						

* Indicates data point was disregarded; see Section 3.1.
1 Distance from source to monitor (km).
2 Concentration predicted by Human Exposure Model (HEM).
3 The measured values are weighted averages. When the sampled arsenic concentrations were below the MDL, a value of 1/2 MDL was assumed for purposes of calculating the annual averages.
4 Minimum detention limit.
5 Percentile indicates percentage of data falling below minimum detectable levels.

Table 14

Identification of Primary Zinc Smelters

Plant Number Code	Plant Name and Address
1	St. Joe - Monaca, PA
2	ASARCO - Corpus Christi, TX
3	Amax - Sauget, IL
4	Jersey Miniere Zinc Co - Clarksville, TN
5	National Zinc - Bartlesville, OK

Table 15

Input Data to Exposure Model
Primary Zinc Smelting Industry
(Assuming Baseline Controls)

Plant	Latitude (Degrees Minutes Seconds)	Longitude (Degrees Minutes Seconds)	Emission Rate (Kg/yr)	Emission Point Elevation (Meters)	Emission Point Diameter (Meters)	Emission Point Cross Sectional Area (m^2)	Emission Point Gas Exit Velocity m/sec	Emission Point Gas Temp. (°K)	Emission Point Type
St. Joe - Monaca, PA	40-40-12	80-20-10	109	61	3.4	100	7.0	325	Stack
			8.4	31	1.8	100	1.8	325	Stack
			67.7	37	2.1	100	10.7	336	Stack
			8.4	32	2.0	100	1.1	325	Stack
ASARCO - Corpus Christi, TX	27-48-00	97-23-46	8.4	92	2.0	100	2.7	389	Stack
Amax - Sauget, IL	38-36-07	90-10-16	23.5	46	1.5	100	9.7	373	Stack
Jersey Miniere Zinc Comp. Clarksville, TN	36-30-54	87-24-14	25.2	61	1.8	100	6.3	334	Stack
National Zinc Bartelsville, OK	36-44-24	95-58-59	11.3	31	1.3	100	5.7	325	Stack

Table 16

Total Exposure and Number of People Exposed
Primary Zinc Smelter*

Plant	Total Number of People Exposed	Total Exposure (People - $\mu g/m^3$)
1	2,000,000	47
2	336,000	2
3	2,200,000	16
4	235,000	3
5	120,000	2

* A 50-kilometer radius was used for the analysis of primary zinc smelters.

Table 17

Public Exposure for Primary Zinc Smelters
as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons - $\mu g/m^3$**
0.00182	7	0
0.001	109	0
0.0005	2350	2
0.00025	19400	8
0.0001	91700	18
0.00005	212000	27
0.000025	450000	35
0.00001	1870000	55
0.000005	3170000	65
0.0000025	3960000	68
0.000001	4650000	69
0.0000005	4800000	69
0.00000025	4850000	69
0.0000001	4870000	69
0.00000005	4890000	69

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

3.6 Public Exposure to Inorganic Arsenic Emissions from Zinc Oxide Plants

3.6.1 Source Data

Two zinc oxide plants are included in the analysis. Table 18 lists ambient arsenic concentrations near select zinc oxide plants. Table 19 lists the names and addresses of the plants considered, and Table 20 lists the plant data used as input to the Human Exposure Model (HEM).

3.6.2 Exposure Data

Table 21 lists on a plant-by-plant basis, the total number of people encompassed by the exposure analysis and the total exposure. Total exposure is tHe sum of tHe products of number of people times the ambient air concentration to which they are exposed, as calculated by HEM. Table 22 sums, for the entire source category (2 plants), the numbers of people exposed to various ambient concentrations, as calculated by HEM. (Source-by-source exposure results are provided in the EPA docket numbered A-83-11.)

Table 18

Arsenic Concentrations Near
Select Zinc Oxide Plants

Plant	# Obs	Distance[1] (km)	Bearing	Predicted[2] ($\mu g/m^3$)	Measured[3] ($\mu g/m^3$)	MDL[4] ($\mu g/m^3$)	Percentile[5]
ASARCO-Columbus, OH	127	3.8	206.0	0.0000124	0.006	0.0055	70< % <90*
New Jersey Zinc-Palmerton, PA	No data within 15 km						

* Indicates data point was disregarded; see Section 3.5.1.1.
1 Distance from source to monitor (km).
2 Concentration predicted by Human Exposure Model (HEM).
3 The measured values are weighted averages. When the sampled arsenic concentrations were below the MDL, a value of 1/2 MDL was assumed for purposes of calculating the annual averages.
4 Minimum detection limit.
5 Percentila indicates percentage of data falling below minimum detectable levels.

TABLE 19

Identification of Zinc Oxide Plants

Plant Number Code	Plant Name and Address
1	ASARCO-Columbus, OH
2	New Jersey Zinc Palmerton, PA.

Table 20

Input Data to Exposure Model Zinc Oxide Plants
(Assuming Baseline Controls)

Plant	Latitude (Degrees Minutes Seconds)	Longtitude (Degrees Minutes Seconds)	Emission Rate (Kg/yr)	Emission Point Elevation (Meters)	Emission Point Diameter (Meters)	Emission* Point Cross Sectional Area (m^2)	Emission Point Gas Exit Velocity m/sec	Emission Point Gas Temp. (°K)	Emission Point Type
ASARCO-Columbus, OH	39-59-53	82-58-48	11.3	61	1.2	100	7.5	333	Stack
New Jersey Zinc-Palmerton, PA	40-49-41	75-35-22	3155	24	5	100	7.3	411	Stack
			2656	18	1.8	100	7.9	364	Stack
			2754	9	1.2	100	17.4	466	Stack

Table 21

Total Exposure and Number of People Exposed

(Zinc Oxide Plants)*

Plant	Total Number of People Exposed	Total Exposure (People - ug/m^3)
1	1,210,000	8
2	907,000	1260

* A 50 kilometer radius was used for the analysis of zinc oxide plants.

Table 22

Public Exposure for Zinc Oxide Plants
as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons - $\mu g/m^3$)**
0.269	2	0
0.25	2	0
0.1	138	17
0.05	1160	79
0.025	3990	180
0.01	11700	300
0.005	21300	366
0.0025	54000	474
0.001	392000	921
0.0005	732000	1200
0.00025	883000	1250
0.0001	908000	1260
0.00005	913000	1260
0.000025	976000	1260
0.00001	1160000	1260
0.000005	1360000	1260
0.0000025	1610000	1270
0.000001	1840000	1270
0.0000005	1960000	1270
0.00000025	2110000	1270

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

3.7 Methodology for Reviewing Pollutant Concentrations - Cotton Gins

A total of 320 cotton gins were identified as processors of arsenic desiccated cotton. Due to the large number of gins, EPA determined that it was impractical to obtain the location data necessary for arsenic risk assessment. Based on information regarding the range of processing rates possible, four model plants operating at 4, 7, 12 and 20 bales/hour were designed that are representative of the operations and emissions of the gin population.[7] These were located at each of three sites typical of the areas in which the gins are located. Of the 320 gins, it was assumed that 32 processed 4 bales per hour, 96 processed 7 bales per hour, 160 processed 12 bales per hour, and 32 processed 20 bales per hour. The Human Exposure Model was run for each scenario to establish a range of exposure and risk estimates for individual sources. To provide data for validating the model plant exposure estimates, two operating gins in south central Texas were chosen for test sites over a one year period. Monitors were arranged in a fan-like array of sites positioned at distances of 100, 200 and 400 meters downwind of the gin. Upwind sites were placed at 400m (one gin only) and 100m. This configuration provided a total of 13 sampling sites. The study was conducted over a period of one year with intense sampling (4 hour intervals) for 15 days during the short ginning season followed by 6 day interval sampling for the remainder of the year.

Data from these two gins were compared to Human Exposure Model calculated values (Table 23). The comparison was hampered somewhat by the large number of monitored values which fell below minimum detection limits -- only 298 measurements out of 708 were above the MDL of 0.05 ug/m^3. To circumvent this problem, a range of mean measured values was developed. At one end, all values below MDL were considered as zero values, and at the other end, all such values were considered equal to the MDL of 0.05 ug/m^3.

Table 23

Arsenic Concentrations Near Two Texas Cotton Gins

Plant	Distance (km)[1]	Predicted (HEM) ($\mu g/m^3$)	Measured[2] ($\mu g/m^3$)
A (≃ 9 bales/hr)	0.1	--	0.083-0.088
	0.2	0.011	--
	0.2	0.011	0.051-0.060
	0.1	--	0.12 -0.12
B (≃ 12 bales/hr)	0.1	--	0.015-0.024
	0.2	0.011	0.013-0.022
	0.1	--	0.013-0.022

[1] Distance from source to monitor.

[2] Weighted mean concentrations for one calendar year. Lesser value represents weighted mean concentration calculated with values less than minimum detection limit set equal to zero. Greater value represents weighted mean concentration calculated with values less than minimum detection limit set equal to MDL (0.0065 $\mu g/m^3$).

When comparing the measured arsenic values to the predicted concentrations from the appropriate model gin exposure analysis, EPA found that the predicted values were reasonably close to concentrations measured very near the gins. The monitoring study data also showed that the arsenic concentrations fell off very rapidly with distance from the gins. This result suggests that people living at some distance from the gins are not being significantly exposed to the gins' emissions. Such a result, coupled with the observation that many gins are in rural areas supports the Agency's conclusion that the aggregate risks for this source category are low.

3.7.1 Public Exposure to Inorganic Arsenic Emissions from Cotton Gins

3.7.1.1 Source Data

Four model cotton gins at each of three geographic locations are included in the analysis. Table 24 lists the names and addresses of the plants considered, and Table 25 lists the plant data used as input to the Human Exposure Model (HEM).

3.7.1.2 Exposure Data

Tables 26 - 37 sum, for the entire source category (12 plants), the numbers of people exposed to various ambient concentrations, as calculated by HEM. (Model plant-by-model plant exposure results are provided in the EPA docket numbered A-83-10.)

Table 24

Identification of Model Cotton Gins

Model Plant Location	Model Plant Production
Hutto, TX	4 Bales/Hour
	7 Bales/Hour
	12 Bales/Hour
	20 Bales/Hour
Buckholtz, TX	4 Bales/Hour
	7 Bales/Hour
	12 Bales/Hour
	20 Bales/Hour
Itasca, TX	4 Bales/Hour
	7 Bales/Hour
	12 Bales/Hour
	20 Bales/Hour

Table 25

Input Data to Exposure Model Cotton Gins
(Assuming Baseline Controls)

Plant	Latitude (Degrees Minutes Seconds)	Longitude (Degrees Minutes Seconds)	Emission Rate (Kg/yr)	Emission Point Elevation (Meters)	Emission Point Diameter (Meters)	Emission Point Cross Sectional Area (m^2)	Emission Point Gas Exit Velocity m/sec	Emission Point Gas Temp. (°K)	Emission Point Type
Hutto, TX	30-33-00	97-33-00							
4 Bales/Hour			1.0	9	0.3	25	20.4	298	Stack
			1.0	5	--	12	--	298	Fugitive
7 Bales/Hour			2.7	9	0.4	25	20.4	298	Stack
			2.7	5	--	12	--	298	Fugitive
12 Bales/Hour			4.6	9	0.4	25	20.4	298	Stack
			4.6	5	--	27	--	298	Fugitive
20 Bales/Hour			10.2	10	0.4	25	20.4	298	Stack
			10.2	5	--	27	--	298	Fugitive
Buckholts, TX	30-52-00	97-08-00							
4 Bales/Hour			1.0	9	0.3	25	20.4	298	Stack
			1.0	5	--	12	--	298	Fugitive
7 Bales/Hour			2.7	9	0.4	25	20.4	298	Stack
			2.7	5	--	12	--	298	Fugitive
12 Bales/Hour			4.6	9	0.4	25	20.4	298	Stack
			4.6	5	--	27	--	298	Fugitive
20 Bales/Hour			10.2	10	0.4	25	20.4	298	Stack
			10.2	5	--	27	--	298	Fugitive
Itasca, TX	32-10-00	97-09-00							
4 Bales/Hour			1.0	9	0.3	25	20.4	298	Stack
			1.0	5	--	12	--	298	Fugitive
7 Bales/Hour			2.7	9	0.4	25	20.4	298	Stack
			2.7	5	--	12	--	298	Fugitive
12 Bales/Hour			4.6	9	0.4	25	20.4	298	Stack
			4.6	5	--	27	--	298	Fugitive
20 Bales/Hour			10.2	10	0.4	25	20.4	298	Stack
			10.2	5	--	27	--	298	Fugitive

Table 26

Public Exposure for 4 Bales/Hour Model Cotton Gin
(Hutto,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.00263	1	0
0.0025	1	0
0.001	1	0
0.0005	6	0
0.00025	23	0
0.0001	112	0
0.00005	177	0
0.000025	433	0
0.00001	1810	0
0.000005	1810	0
0.0000025	3390	0
0.000001	46800	0
0.0000005	285000	0
0.00000025	506000	0

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 27

Public Exposure for 7 Bales/Hour Model Cotton Gin
(Hutto,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.00682	1	0
0.005	1	0
0.0025	1	0
0.001	8	0
0.0005	28	0
0.00025	112	0
0.0001	282	0
0.00005	799	0
0.000025	1810	0
0.00001	2420	0
0.000005	7220	0
0.0000025	55400	0
0.000001	448000	1
0.000000528	506000	1

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 28

Public Exposure for 12 Bales/Hour Model Cotton Gin (Hutto,TX) as Produced by the Human Exposure Model (Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.011	1	0
0.01	1	0
0.005	1	0
0.0025	5	0
0.001	25	0
0.0005	102	0
0.00025	161	0
0.0001	523	0
0.00005	1810	0
0.000025	1810	0
0.00001	3820	0
0.000005	39500	1
0.0000025	232000	1
0.000001	506000	2

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 29

Public Exposure for 20 Bales/Hour Model Cotton Gin (Hutto,TX) as Produced by the Human Exposure Model (Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.0234	1	0
0.01	1	0
0.005	5	0
0.0025	23	0
0.001	102	0
0.0005	169	0
0.00025	433	0
0.0001	1810	1
0.00005	1810	1
0.000025	3390	1
0.00001	46800	1
0.000005	300000	3
0.0000025	506000	4

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 30

Public Exposure for 4 Bales/Hour Model Cotton Gin
(Buckholts,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.00263	<1	0
0.0025	<1	0
0.001	<1	0
0.0005	2	0
0.00025	10	0
0.0001	49	0
0.00005	77	0
0.000025	190	0
0.00001	1050	0
0.000005	1050	0
0.0000025	4020	0
0.000001	10600	0
0.0000005	86700	0
0.00000025	129000	0
0.000000196	131000	0

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 31

Public Exposure for 7 Bales/Hour Model Cotton Gin
(Buckholts,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.00682	<1	0
0.005	<1	0
0.0025	<1	0
0.001	3	0
0.0005	12	0
0.00025	49	0
0.0001	124	0
0.00005	269	0
0.000025	1050	0
0.00001	1050	0
0.000005	6020	0
0.0000025	15500	0
0.000001	121000	0
0.000000528	131000	0

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 32

Public Exposure for 12 Bales/Hour Model Cotton Gin (Buckholts,TX) as Produced by the Human Exposure Model (Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.011	<1	0
0.01	<1	0
0.005	<1	0
0.0025	2	0
0.001	11	0
0.0005	45	0
0.00025	71	0
0.0001	230	0
0.00005	1050	0
0.000025	1050	0
0.00001	6020	0
0.000005	10100	0
0.0000025	81500	0
0.000001	131000	1

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 33

Public Exposure for 20 Bales/Hour Model Cotton Gin
(Buckholts,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.0234	<1	0
0.01	<1	0
0.005	2	0
0.0025	10	0
0.001	45	0
0.0005	74	0
0.00025	190	0
0.0001	1050	0
0.00005	1050	0
0.000025	4020	0
0.00001	11300	1
0.000005	89500	1
0.0000025	129000	1
0.000002	131000	1

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 34

Public Exposure for 4 Bales/Hour Model Cotton Gin
(Itasca,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.0011	1	0
0.001	1	0
0.0005	5	0
0.00025	19	0
0.0001	57	0
0.00005	153	0
0.000025	489	0
0.00001	1280	0
0.000005	2140	0
0.0000025	2660	0
0.000001	6520	0
0.0000005	38900	0
0.00000025	107000	0
0.0000001	156000	0
0.0000000634	162000	0

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 35

Public Exposure for 7 Bales/Hour Model Cotton Gin
(Itasca,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.00285	1	0
0.0025	1	0
0.001	7	0
0.0005	23	0
0.00025	70	0
0.0001	167	0
0.00005	587	0
0.000025	1280	0
0.00001	2140	0
0.000005	3870	0
0.0000025	6520	0
0.000001	65200	0
0.0000005	120000	0
0.00000025	159000	0
0.000000171	162000	0

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 36

Public Exposure for 12 Bales/Hour Model Cotton Gin
(Itasca,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.00461	1	0
0.0025	4	0
0.001	22	0
0.0005	42	0
0.00025	118	0
0.0001	489	0
0.00005	948	0
0.000025	1980	0
0.00001	2660	0
0.000005	4970	0
0.0000025	20300	0
0.000001	114000	0
0.0000005	153000	0
0.000000293	162000	0

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

Table 37

Public Exposure for 20 Bales/Hour Model Cotton Gin
(Itasca,TX) as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.0097	1	0
0.005	4	0
0.0025	14	0
0.001	46	0
0.0005	146	0
0.00025	489	0
0.0001	1280	0
0.00005	2140	0
0.000025	2660	0
0.00001	6520	1
0.000005	40100	1
0.0000025	109000	1
0.000001	156000	1
0.0000006 49	162000	1

* Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

** Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

3.8 Public Exposure to Inorganic Arsenic Emissions from Arsenic Plants

3.8.1 Source Data

Eight arsenic chemical plants are included in the analysis. Table 38 lists ambient arsenic concentrations near select arsenic chemical plants. Table 39 lists the names and addresses of the plants considered, and Table 40 lists the plant data used as input to the Human Exposure Model (HEM).

3.8.2 Exposure Data

Table 41 lists, on a plant-by-plant basis, the total number of people encompassed by the exposure analysis and the total exposure. Total exposure is the sum of the products of numbers of people times the ambient air concentration to which they are exposed, as calculated by HEM. Table 42 sums, for the entire source category (8 plants), the numbers of people exposed to various ambient concentrations, as calculated by HEM. (Source-by-source exposure results are provided in the EPA docket numbered A-83-23.)

Table 38

Arsenic Concentrations Near Select
Arsenic Chemical Plants

Plant	# Obs	Distance[1] (km)	Bearing	Predicted[2] (μg/m³)	Measured[3] (μg/m³)	MDL[4] (μg/m³)	Percentile[5]
Diamond Shamrock-Greens Bayou, TX	302	1.6	291.1	0.00000042	0.027	0.05	90< % <95*
	20	5.5	229.7	0.0000000042	0.026	0.05	>99*
	37	5.9	186.3	0.000000051	0.008	0.0055	50< % <70*
	143	6.2	197.3	0.000000029	0.027	0.05	95< % <99*
	261	8.3	70.3	0.0000000118	0.025	0.05	>99*
	45	8.7	62.2	0.0000000105	0.026	0.05	95< % *
	26	11.5	195.9	0.0000000121	0.025	0.05	100 *
	30	12.0	143.0	0.0000000090	0.025	0.05	100 *
Koppers Co.-Conley, GA	76	13.0	336.1	0.0000000079	0.008	0.0055	70< % <90*
Koppers Co.-Valparaiso, IN		No data within 15 km					
Mineral R&D-Concord, NC		No data within 15 km					
Osmose Wood Preserving Co.-Memphis, TN	32	5.9	27.7	0.000099	0.005	0.0055	70< % <90*
	72	9.3	20.8	0.000054	0.004	0.0055	50< % <70*
Pennwalt Inc.-Bryan, TX	107	0.1	296.4		0.026	0.05	95< % <99*
Vineland Chemical-Vineland, NJ		No data within 15 km					
Voluntary Purchasing Group-Bonham,TX		No data within 15 km					

* Indicates data point was disregarded; see Section 3.7.1.1.
1 Distance from source to monitor (km).
2 Concentration predicted by Human Exposure Model (HEM).
3 The measured values are weighted averages. When the sampled arsenic concentrations were below the MDL, a value of 1/2 MDL was assumed for purposes of calculating the annual averages.
4 Minimum detection limit.
5 Percentile indicates percentage of data falling below minimum detectable levels.

Table 39

Identification of Arsenic Chemical Plants

Plant Number Code	Plant Name and Address
1	Diamond Shamrock - Greens Bayou, TX
2	Koppers Co. - Conley, GA
3	Koppers Co., - Valparaiso, IN
4	Mineral Research & Development Co. - Concord, NC
5	Osmose Wood Preserving Co., - Memphis, TN
6	Pennwalt Inc. - Bryan, TX
7	Vineland Chemical - Vineland, NJ
8	Voluntary Purchasing Group - Bonham, TX

Table 40

Input Data to Exposure Model Arsenic Chemical Plants
(Assuming Baseline Controls)

Plant	Latitude (Degrees Minutes Seconds)	Longitude (Degrees Minutes Seconds)	Emission Rate (Kg/yr)	Emission Point Elevation (Meters)	Emission Point Diameter (Meters)	Emission Point Cross Sectional Area (m^2)	Emission Point Gas Exit Velocity m/sec	Emission Point Gas Temp. (°K)	Emission Point Type
Diamond Shamrock-Greens Bayou,TX	29-45-58	95-12-22	0.030	13	0.38	3000	11.9	298	Stack
Koppers Co.-Conley, GA	33-38-42	84-19-34	0.027	31	0.61	3000	0.1	298	Stack
Koppers Co.-Valparaiso,IN	41-28-34	87-04-40	0.054	9	0.76	3000	1.6	298	Stack
Mineral R&D Co.-Concord, NC	35-24-29	80-34-44	0.022	11	0.50	3000	8.8	298	Stack
Osmose Wood Preserving Co.-Memphis,TN	35-05-13	90-04-19	51.3	5	0.36	3000	14.3	298	Stack
Pennwalt Inc.-Bryan, TX	30-40-30	96-22-12	0.019	10	0.53	3000	8.9	298	Stack
Vineland Chemical-Vineland, NJ	39-55-59	74-44-53	0.001	13	0.38	3000	0.1	298	Stack
Voluntary Purchasing Group-Bonham,TX	33-34-41	96-10-41	0.019	10	0.53	3000	8.9	298	Stack

Table 41

Total Exposure and Number of People Exposed
(Arsenic Chemical Plants)*

Plant	Total Number of People Exposed	Total Exposure (People - $\mu g/m^3$)
1	2,680,000	0
2	1,900,000	0
3	1,190,000	0
4	813,000	0
5	927,000	68
6	138,000	0
7	4,230,000	0
8	152,000	0

* A 50-kilometer radius was used for the analysis of arsenic chemical plants.

Table 42

Public Exposure for Arsenic Chemical Plants
as Produced by the Human Exposure Model
(Assuming Baseline Controls)

Concentration Level ($\mu g/m^3$)	Population Exposed (Persons)*	Exposure (Persons-$\mu g/m^3$)**
0.0541	31	2
0.05	31	2
0.025	62	3
0.01	472	10
0.005	1440	15
0.0025	2720	19
0.001	8130	28
0.0005	16700	34
0.00025	30900	38
0.0001	105000	49
0.00005	210000	56
0.000025	374000	61
0.00001	653000	66
0.000005	852000	68
0.0000025	908000	68
0.000001	935000	68
0.0000005	952000	68
0.00000025	984000	68
0.0000001	1040000	68
0.00000005	1100000	68
0.000000025	1320000	68
0.00000001	2230000	68
0.000000005	3670000	68
0.0000000025	5880000	68
0.000000001	7750000	68
0.0000000005	7890000	68
0.00000000025	8320000	68
0.0000000001	9910000	68
0.00000000005	11900000	68
0.0000000000395	12000000	68

*Column 2 displays the computed value, rounded to the nearest whole number, of the cumulative number of people exposed to the matching and higher concentration levels found in column 1. For example, 0.5 people would be rounded to 0 and 0.51 people would be rounded to 1.

**Column 3 displays the computed value of the cumulative exposure to the matching and higher concentration levels found in column 1.

4 QUANTITATIVE EXPRESSIONS OF PUBLIC CANCER RISKS FROM INORGANIC ARSENIC EMISSIONS

4.1 Methodology (General)

4.1.1 The Two Basic Types of Risk

Two basic types of risk are dealt with in the analysis. "Aggregate risk" applies to all of the people encompassed by the particular analysis. Aggregate risk can be related to a single source, to all of the sources in a source category, or to all of the source categories analyzed. Aggregate risk is expressed as incidences of cancer among all of the people included in the analysis, after 70 years of exposure. For statistical convenience, it is often divided by 70 and expressed as cancer incidences per year. "Individual risk" applies to the person or persons estimated to live in the area of the highest ambient air concentrations and it applies to the single source associated with this estimate as estimated by the dispersion model. Individual risk is expressed as "maximum lifetime risk" and reflects the probability of getting cancer if one were continuously exposed to the estimated maximum ambient air concentration for 70 years.

4.1.2 The Calculation of Aggregate Risk

Aggregate risk is calculated by multiplying the total exposure produced by HEM (for a single source, a category of sources, or all categories of sources) by the unit risk estimate. The product is cancer incidences among the included population after 70 years of exposure. The total exposure, as calculated by HEM, is illustrated by the following equation:

$$\text{Total Exposure} = \sum_{i=1}^{N} (P_i C_i)$$

where

Σ = summation over all grid points where exposure is calculated

P_i = population associated with grid point i,

C_i = long-term average inorganic arsenic concentration at grid point i,

N = number of grid points to 2.8 kilometers and number of ED/BG centroids between 2.8 and 50 kilometers of each source.

To more clearly represent the concept of calculating aggregate risk, a simplified example illustrating the concept follows:

EXAMPLE

This example uses assumptions rather than actual data and uses only three levels of exposure rather than the large number produced by HEM. The assumed unit risk estimate is 4.29×10^{-3} at 1 $\mu g/m^3$ and the assumed exposures are:

ambient air concentrations	number of people exposed to given concentration
2 $\mu g/m^3$	1,000
1 $\mu g/m^3$	10,000
0.5 $\mu g/m^3$	100,000

The probability of getting cancer if continuously exposed to the assumed concentrations for 70 years is given by:

concentration		unit risk		probability of cancer
2 $\mu g/m^3$	x	4.29×10^{-3} $(\mu g/m^3)^{-1}$	=	9×10^{-3}
1 $\mu g/m^3$	x	4.29×10^{-3} "	=	4×10^{-3}
0.5 $\mu g/m^3$	x	4.29×10^{-3} "	=	2×10^{-3}

The 70 year cancer incidence among the people exposed to these concentrations is given by:

probability of cancer at each exposure level		number of people at each exposure level		after 70 years of exposure
9×10^{-3}	x	1,000	=	9
4×10^{-3}	x	10,000	=	40
2×10^{-3}	x	100,000	=	200
			TOTAL =	249

The aggregate risk, or total cancer incidence, is 249 and, expressed as cancer incidence per year, is 249 ÷ 70, or 3.6 cancers per year. The total cancer incidence and cancers per year apply to the total of 111,000 people assumed to be exposed to the given concentrations.

4.1.3 The Calculation of Individual Risk

Individual risk, expressed as "maximum lifetime risk," is calculated by multiplying the highest concentration to which the public is exposed, as reported by HEM, by the unit risk estimate. The product, a probability of getting cancer, applies to the number of people which HEM reports as being exposed to the highest listed concentration. The concept involved is a simple proportioning from the 1 $\mu g/m^3$ on which the unit risk estimate is based to the highest listed concentration. In other words:

$$\frac{\text{maximum lifetime risk}}{\text{highest concentration to which people are exposed}} = \frac{\text{the unit risk estimate}}{1\ \mu g/m^3}$$

4.2 Risks Calculated for Emissions of Inorganic Arsenic

The explained methodologies for calculating maximum lifetime risk and cancer incidences were applied to each plant, assuming a baseline level of emissions. A baseline level of emissions means the level of emissions after

the application of controls either currently in place or required to be in place to comply with current state or Federal regulations but before application of controls that would be required by a NESHAP.

Tables 43-49 summarize the calculated risks for each source category. To understand the relevance of these numbers, one should refer to the analytical uncertainties discussed in section 5 below. Note that the annual incidence is not calculated for cotton gins. As mentioned earlier in this document, it was impractical to identify and locate all the gins handling arsenic-acid-desiccated cotton ($\simeq$ 300 gins). The Agency does not have enough available data to provide an estimate of annual cancer incidence that would be comparable in accuracy to the other source category estimates. As outlined in Section 3.7, three model gins operating at each of four production rates were used to establish a range of exposure and risk estimates for individual sources. Likewise, two operating gins in south central Texas were chosen for ambient air monitoring in order to validate the model plant exposure estimates. Maximum lifetime risk estimates were calculated for each of the three model plants (Table 47) and for the two operating gins (Table 48).

Table 43
Maximum Lifetime Risk and Cancer Incidence for Primary Lead Smelters
(Assuming Baseline Controls)

Plant	Maximum Lifetime Risk	Cancer Incidences Per Year
1	2×10^{-3}	0.013
2	4×10^{-5}	0.044
3	2×10^{-5}	0.011
4	1.3×10^{-5}	0.0016
5	4×10^{-5}	0.0004

Table 44
Maximum Lifetime Risk and Cancer Incidence for Secondary Lead Smelters
(Assuming Baseline Controls)

Plant	Maximum Lifetime Risk	Cancer Incidences Per Year
1	5×10^{-6}	0.0019
2	4×10^{-6}	0.0009
3	8×10^{-6}	0.0010
4	8×10^{-6}	0.0011
5	8×10^{-5}	0.014
6	1.1×10^{-5}	0.0010
7	5×10^{-6}	0.0013
8	1.1×10^{-4}	0.015
9	2×10^{-7}	0.0002
10	1.6×10^{-5}	<0.0001
11	9×10^{-6}	0.0010
12	4×10^{-6}	0.011
13	1.1×10^{-5}	0.0019
14	4×10^{-6}	0.0009
15	6×10^{-5}	0.0024
16	9×10^{-6}	0.0004
17	4×10^{-6}	0.0035
18	3×10^{-4}	0.040
19	8×10^{-6}	0.0031
20	4×10^{-5}	0.012
21	4×10^{-4}	0.14
22	2×10^{-4}	0.028
23	4×10^{-4}	0.035
24	2×10^{-5}	0.0040
25	7×10^{-5}	0.0069
26	3×10^{-4}	0.015
27	2×10^{-6}	0.0002
28	1.2×10^{-5}	0.0007
29	1.7×10^{-6}	0.0002
30	1.1×10^{-6}	0.0001
31	1.5×10^{-5}	0.0015
32	6×10^{-6}	0.0050
33	1.5×10^{-5}	0.0095
34	3×10^{-5}	0.0013
35	2×10^{-5}	0.0062

Table 45

Maximum Lifetime Risk and Cancer Incidence for Primary Zinc Smelters
(Assuming Baseline Controls)

Plant	Maximum Lifetime Risk	Cancer Incidences Per Year
1	8×10^{-6}	0.0029
2	1.9×10^{-7}	0.0001
3	1.1×10^{-6}	0.0010
4	9×10^{-7}	0.0002
5	3×10^{-6}	0.0001

Table 46

Maximum Lifetime Risk and Cancer Incidence for Zinc Oxide Plants
(Assuming Baseline Controls)

Plant	Maximum Lifetime Risk	Cancer Incidences Per Year
1	4×10^{-7}	0.005
2	1.2×10^{-3}	0.077

Table 47

Maximum Lifetime Risk and Cancer Incidence for Model Cotton Gins

(Assuming Baseline Controls)

Model Plant	Maximum Lifetime Risk	Cancer Incidences Per Year
Hutto, TX		
4 Bales/Hr	1.1×10^{-5}	<0.0001
7 Bales/Hr	3×10^{-5}	0.0001
12 Bales/Hr	5×10^{-5}	0.0001
20 Bales/Hr	1.0×10^{-4}	0.0002
Buckholts,TX		
4 Bales/Hr	1.1×10^{-5}	<0.0001
7 Bales/Hr	3×10^{-5}	<0.0001
12 Bales/Hr	5×10^{-5}	<0.0001
20 Bales/Hr	1.0×10^{-4}	0.0001
Itasca,TX		
4 Bales/Hr	5×10^{-6}	<0.0001
7 Bales/Hr	1.2×10^{-5}	<0.0001
12 Bales/Hr	2×10^{-5}	<0.0001
20 Bales/Hr	4×10^{-5}	0.0001

Table 48

Lifetime Risk for Two Texas Cotton Gins
(Assuming Baseline Controls)

Plant	Maximum Lifetime Risk
A	5×10^{-4}*
B	1.0×10^{-4}

* Represents final risk estimate as incorporated by EPA.

Table 49

Maximum Lifetime Risk and Cancer Incidence for Arsenic Chemical Plants
(Assuming Baseline Controls)

Plant	Maximum Lifetime Risk	Cancer Incidences Per Year
1	4×10^{-8}	<0.0001
2	7×10^{-9}	<0.0001
3	3×10^{-8}	<0.0001
4	3×10^{-8}	<0.0001
5	2×10^{-4}	0.0042
6	3×10^{-8}	<0.0001
7	9×10^{-10}	<0.0001
8	3×10^{-8}	<0.0001

5 ANALYTICAL UNCERTAINTIES APPLICABLE TO THE CALCULATIONS OF PUBLIC HEALTH RISKS CONTAINED IN THIS DOCUMENT

5.1 The Unit Risk Estimate

The procedure used to develop the unit risk estimate is described in reference 2. The model used and its application to epidemiological data have been the subjects of substantial comment by health scientists. The uncertainties are too complex to be summarized sensibly in this appendix. Readers who wish to go beyond the information presented in the reference should see the following Federal Register notices: (1) OSHA's "Supplemental Statement of Reasons for the Final Rule", 48 FR 1864 (January 14, 1983); and (2) EPA's "Water Quality Documents Availability" 45 FR 79318 (November 28, 1980).

The unit risk estimate used in this analysis applies only to lung cancer. Other health effects are possible; these include skin cancer, hyperkeratosis, peripheral neuropathy, growth retardation and brain dysfunction among children, and increase in adverse birth outcomes. No numerical expressions of risks relevant to these health effects is included in this analysis.

Although the estimates derived from the various studies are quite consistent, there are a number of uncertainties associated with them. The estimates were made from occupational studies that involved exposures only after employment age was reached. In estimating risks from environmental exposures throughout life, it was assumed through the absolute-risk model that the increase in the age-specific mortality rates of lung cancer was a function only of cumulative exposures, irrespective of how the exposure was accumulated. Although this assumption provides an adequate description of

all of the data, it may be in error when applied to exposures that begin very early in life. Similarly, the linear models possibly are inaccurate at low exposures, even though they provide reasonable descriptions of the experimental data.

The risk assessment methods employed were severely constrained by the fact that they were based only upon the analyses performed and reported by the original authors--analyses that had been performed for purposes other than quantitative risk assessment. For example, although other measures of exposure might be more appropriate, the analyses were necessarily based upon cumulative dose, since that was the only usable measure reported. Given greater access to the data from these studies, other dose measures, as well as models other than the simple absolute-risk model, could be studied. It is possible that such wide analyses would indicate that other approaches are more appropriate than the ones applied here.

5.2 Public Exposure

5.2.1 General

The basic assumptions implicit in the methodology are that all exposure occurs at people's residences, that people stay at the same location for 70 years, that the ambient air concentrations and the emissions which cause these concentrations persist for 70 years, and that the concentrations are the same inside and outside the residences. From this it can be seen that public exposure is based on a hypothetical premise. It is not known whether this results in an over-estimation or an underestimation of public exposure.

5.2.2 The Public

The following are relevant to the public as dealt with in this analysis:

1. Studies show that all people are not equally susceptible to cancer. There is no numerical recognition of the "most susceptible" subset of the population exposed.

2. Studies indicate that whether or not exposure to a particular carcinogen results in cancer may be affected by the person's exposure to other substances. The public's exposure to other substances is not numerically considered.

3. Some members of the public included in this analysis are likely to be exposed to inorganic arsenic in the air in the workplace, and workplace air concentrations of a pollutant are customarily much higher than the concentrations found in the ambient, or public air. Workplace exposures are not numerically approximated.

4. Studies show that there is normally a long latent period between exposure and the onset of lung cancer. This has not been numerically recognized.

5. The people dealt with in the analysis are not located by actual residences. As explained previously, people are grouped by census districts and these groups are located at single points called the population centroids. The effect is that the actual locations of residences with respect to the estimated ambient air concentrations are not known and that the relative locations used in the exposure model may have changed since the 1980 census. However, for the population sectors estimated to be at highest risk, U.S.

Geological Survey topographical maps were checked to verify that people did live or could live in locations near the sources as modeled predictions estimated. Maps in certain instances were old and the possibility could not be excluded that additional areas near sources have been developed since publication of the maps.

6. Many people dealt with in this analysis are subject to exposure to ambient air concentrations of inorganic arsenic where they travel and shop (as in downtown areas and suburban shopping centers), where they congregate (as in public parks, sports stadiums, and schoolyards), and where they work outside (as mailmen, milkmen, and construction workers). These types of exposures are not numerically dealt with.

5.2.3. The Ambient Air Concentrations

The following are relevant to the estimated ambient air concentrations of inorganic arsenic used in this analysis:

1. Flat terrain was assumed in the dispersion model. Concentrations much higher than those estimated would result if emissions impact on elevated terrain or tall buildings near a plant.

2. The estimated concentrations do not account for the additive impact of emissions from plants located close to one another.

3. The increase in concentrations that could result from re-entrainment of arsenic-bearing dust from, e.g., city streets, dirt roads, and vacant lots, is not considered.

4. Meteorological data specific to plant sites are not used in the dispersion model. As explained, HEM uses the meteorological data from the STAR station nearest the plant site. Site-specific meteorological data could result in significantly different estimates, e.g., the estimated location of the highest concentrations.

5. In some cases, the arsenic emission rates are estimates that are based on assumptions rather than on measured data.

6 REFERENCES

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