DE #1

SUPERIOR COURT OF NEW JERSEY
LAW DIVISION: MIDDLESEX COUNTY
DOCKET NO. L-060148-87

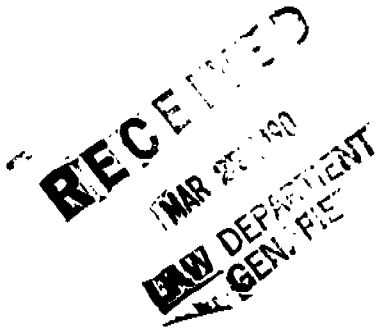
JOHN PETERSON and SHIRLEY MAE PETERSON,

Plaintiffs,

vs.

UNION CARBIDE CORPORATION,

Defendants.



DEPOSITION DIGEST OF
SAMUEL EPSTEIN
DECEMBER 16, 1989

DIGESTED BY: NANCY K. DOCHERTY
LEGAL ASSISTANT

Page	Lines	Subject Matter	Digest of Testimony
		DIRECT EXAMINATION CONTINUED BY ROBERT L. HOLLINGSHEAD (Defendant)	
4 to 15	13-24 1-7	**Exhibits Epstein 5-17**	Extensive discussion held re documents Mr. Hollingshead requested from witness during his 12/1/89 deposition. **Epstein 5** is a copy of witness' C.V. **Epstein 6** is the transcript of witness' deposition from the *Strader v. Franklin Electric* case involving meat wrappers asthma. **Epstein 7** is witness' transcript from *Starling v. McDonnell Douglas* case involving nasal sinus cancer following multiple exposures including asbestos. **Epstein 8** is a letter from an attorney by the name of Ellis E. Neder, Jr., dated 5/14/82. **Epstein 9** is a letter and attachment from the law firm of Brown, Connery, et al, dated 2/9/81. The attachment is a report on John Grasso prepared by witness on 1/21/81. The case *Grasso v. BF Goodrich* involved VC exposure. **Epstein 10** is a letter dated 1/16/87 from the law firm of Brown, Tyrrel, et al, to witness with a 1-page report on Andrew Ski attached. The case involved asbestos exposure and Ski was a co-worker of Starling. **Epstein 11** is a letter dated 10/26/84 from the law firm of Philo, Atkinson, et al, to witness re *Earle v. Cryovac* and involved meat wrappers asthma. **Epstein 12** is a letter dated 2/3/84 from the law firm of Philo, Atkinson, et al, to witness re *Wall v. Hobart* involving meat wrappers asthma. **Epstein 13** is a letter dated 12/9/83 from the law firm of Watkins, Boulware, et al, to witness re *Strader v. Franklin*. **Epstein 14** is a letter

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			dated 12/1/86 from the law firm of Philo, Atkinson, et al, re *Sutkaitis v. Goodyear* with an attachment of a 2-page report by witness re Mrs. Sutkaitis. It was another meat wrappers asthma case. **Epstein 15** is a letter dated 1/15/85 from the law firm of Smith & Goldstein, re *Ferrara v. Tenneco Chemicals* with an attachment of a 1-page report dated 11/30/85 by witness re Mrs. Ferrara. The case involved VC. **Epstein 16** is a letter dated 7/9/81 from the law firm of Bogus & Bogus re *Mikyska v. BF Goodrich* involving PVC/VC. **Epstein 17** is a letter dated 4/6/79 from the law firm of Stanley Rosenblatt to witness re *Ellias v. Parke Davis and Sprayon* with an attachment of witness' 4-page affidavit. The case involved VC. (Witness requested that the deposition transcripts in the *Strader* and *Starling* cases be returned to him after being copied by Mr. Hollingshead.)
15 16 17	8-24 1-24 1-21	**Exhibit Epstein 18**	**Epstein 18** are several pages from the exhibit that had previously been marked as **Epstein 4** (witness' preliminary report). The pages had been updated since witness' previous deposition. The update involved the inclusion of six new studies (Byren; Bufler; International Agency For Research On Cancer, 1979; Infante, 1981; International Agency For Research On Cancer, 1987 and Doll, 1988).
17 18	22-24 1-15	**Exhibit Epstein 19**	**Epstein 19** is a document entitled State of the Art on Residual CV and PVC Prior to 1975.
18 19	16-24 1-4	**Exhibit Epstein 20**	**Epstein 20** is a 1-page document entitled Infante, et al, 1981.

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20 21	9-24 1-4	**Exhibits Epstein 21 and 22**	**Epstein 21** is the new 2-page document entitled State of the Art on VC/PVC Toxicology Prior to 1974. **Epstein 22** is the 5-pag document entitled Illustrative Literature on the Toxic and Carcinogenic Effects of VC/PVC in the Respiratory Tract of Exposed Animals. (For the record: **Epstein 18** and **Epstein 22** are the same with the exception that **Epstein 18** contains witnesses notations.)
21 22 23	5-24 1-24 1-8		Extensive attorney discussion held re the fact witness continually submits supplementals to his preliminary report.
23 24 25	9-24 1-24 1-2		(Mr. Hollingshead requested a copy of the info Levinson supplied to witness re Peterson's work history and exposure.)
25 26	12-24 1-18	Asbestos/Silica Exposure At Lean Manufacturing Company	Witness stated that there was no indication in Peterson's occupational history that Peterson was exposed to asbestos/silica at Lean Manufacturing Company from 1962-67. Literature suggests there is a relation between asbestos exposure and laryngeal cancer but not to silica exposure.
28 29	16-24 1-19	Tabershaw Supports PVC-Laryngeal Cancer	Witness stated that although there are a substantial number of reports linking PVC exposure to respiratory tract cancers, there is no breakdown citing how many are laryngeal and how many are lung cancers. The only literature support for the proposition that laryngeal cancer had been related to exposure to PVC are the Tabershaw papers.
30 31 32	17-24 1-24 1-2	Peterson's PVC Concentrations Unknown	Witness stated due to the lack of monitoring data, Peterson's actual PVC/VC exposure at ATC is

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			unknown. It is known that Peterson's exposure to PVC occurred largely in the bagging area of ATC.
32 33	6-24 1	Thermal Degradation Of PVC	Witness explained that the thermal degradation of PVC would result in HCL in combination with benzene, carbon monoxide and phosgene, among other materials, when dealing with PVC film. When dealing with PVC pellets and dust, the major source of exposure would be VC.
33 to 37	2-24 1-9		Extensive discussion was held re witness' reference to Davidson's expertise re specifics of PVC thermal degradation.
37 38	10-24 1-5	Difference Between PVC Film/Pellets	Witness explained that PVC film produces higher levels of residual VC because the VC will constantly evaporate from the film and the film is so thin that the measured levels of VC are minuscular. He further stated that no matter how thermal degradation of PVC occurs, whether it is from the heat sealing process or the welding process, the amount of VC given off and to what extent it is given off remains the same.
38 39 40	7-24 1-24 1-16		Extensive discussion held re various tables witness included in his preliminary report.
40 41 42	17-24 1-24 1-6	**Exhibit Epstein 23**	Epstein 23 is witness' addition to his chart entitled Illustrative Literature on the Toxic and Carcinogenic Effects of VC/PVC in the Respiratory Tract of Experimental Animals. The addition was a report by Cornish & Abar of 1969 dealing with rats' inhalation of fumes from heated PVC producing interstitial edema and focal

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			hemorrhages in the lungs.
42 43	17-24 1-3	Fibrosis/ Granuloma/ Adenoma	Witness stated that the disease of fibrosis is basically just scar The disease of granuloma is a chronic inflammatory reaction generally characterized by formation of nodules and multinucleate giant cells associated with fibrosis. Adenoma is a tumor of the lung which hasn't progressed to malignancy.
43	4-22	Any Dust Can Produce Pneumoconiosis	Witness stated that pneumoconiosis is a chronic inflammatory condition of the lung generally associated with fibrosis and granular nodulation and granuloma formation. Any form of dust in high concentrations can potentially cause pneumoconiosis.
43 44	23-24 1-8	Frongia Study Of 1974	Witness stated that the Frongia Study of 1974 involved rats and guinea pigs being exposed in the areas where workers were bagging PVC. The animals, like the workers, developed fibrosis nodulation granulomas.
44	10-20	Agarwal & Feron Studies	Witness stated that although the overwhelming majority of PVC test were inhalation studies, two were not. The Agarwal Study of 1978 involved intra-trachial injection. The Feron Study of 1981 was a gavage study which involved the incorporation of the PVC in the diet.
46 to 50	7-24 1-21	Shortcomings Of Doll Study	Extensive discussion held re the shortcomings of the Doll Study. Witness stated that the shortcomings involved Doll's failure to cite critical references re respiratory tract cancers, and although Doll recognizes lung cancer he didn't reflect his source of

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			research support. Witness stated that Doll performed a highly selective review of the literature, but did not conduct any original research to come to Doll's conclusions. Doll also stated that there was no positive evidence of non-malignant disease despite the vast body of documentation to prove otherwise.
51 52	23-24 1-18	Byren Study	Witness stated that the Byren Study was one of many studies confirming the excess of lung cancer in VC/PVC workers. He further stated that the study was biologically but not statistically significant.
52 53	19-24 1-6	Bufler Study	Witness stated that the Bufler Study of 1979 was statistically significant because once the study was balanced to take into account smokers, there was an excess of respiratory tract cancers in VC worker.
53 54	7-24 1-18	IARC Report	Witness stated that the International Agency for Research on Cancer (IARC) did a report in 1979 depicting a review of the literature prior to 1978 on VC/PVC in animals and humans. The IARC stated that VC is a human carcinogen whose target organs are the liver, brain, lungs and haemolymphopoietic system. The report did not include whether or not the larynx was a target organ for VC.
55 56	7-24 1-4		Witness stated that if any agency, such as the IARC, was aware of the existence of an unpublished study, they would not include it in their report because they only cite published data from the literature which is available to the scien-

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			tific community. Not all published studies are subject to peer review.
56 57 58	5-24 1-24 1-7	Infante Report	Witness stated that the Infante Report of 1981 is a review of eight studies involving the carcinogenic effects of VC in humans. Seven of the eight studies showed excess risk of lung cancers ranging from 7 to 200%. He explained that the one study that was negative showed a short duration of follow-up limiting the validity of any inferences that could be developed. He further explained that Infante stated that the carcinogenic effects of VC in humans extended beyond the liver and that the brain and lungs should be considered target organs. He stated that respiratory system cancers include laryngeal cancers. Laryngeal cancer is a relatively rare cancer with a figure of 8.5 per hundred thousand where the incidence for lung cancer is more than ten times that.
58	8-18	IARC 1987 Report	Witness stated that the IARC 1987 Report updated the IARC 1979 Report. The report stressed that exposure to PVC dust and VC was associated with an increased incidence of lung tumors.
59 to 66	20-24 1-7	Loomis' Report Of 11/2/89	Witness described the various sections of Loomis' report of 11/2/89 that he considered "scientific rubbish." He felt Loomis' statement that PVC is toxicologically safe and produced only minimal, non-specific, primary effects during industrial exposure to be nonsense. He did not agree with Loomis' statement that laryngeal cancer had never been documented to have been causally related to

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			VC/PVC exposure. Witness stated that any individual acting as an expert to industry received unpublished data.
67 68 69	2-21 1-24 1-23	Austin 1982-Laryngeal Cancer	Witness referred to a reference book that was published in 1982 authored by Schottenfeld and Fraumeni which include a chapter on laryngeal cancer that was authored by Austin, which he included as a reference for his preliminary report. Witness explained that Austin would not have access to the unpublished studies of Tabershaw of 1974-75 because Austin did not work specifically in the VC/PVC field. He further explained that Austin's particular chapter re laryngeal cancer is devoid of any comment about VC/PVC.
69 70 71	24 1-24 1-24	Asbestos-Cancer	Witness stated that he agreed with Austin re the fact asbestos is a risk factor for cancer including laryngeal cancer. He explained that Austin discussed a wide range of occupations in which excessive laryngeal cancer is found. They included chemical exposures to unspecified chemicals. Other occupational factors included tobacco, alcohol, nickel and mustard gas exposures. No where in Austin's paper is VC mentioned. Witness went on to say that Austin found elevated PMR in meat cutters.
72 to 75	16-24 1-2	PS Present At ATC	Witness stated that the pellet form of polystyrene (PS) was present at ATC. Due to fragmentation of the handling of the pellets, dust would be generated thereby causing pneumoconiosis. In order to induce pneumoconiosis, it would depend on the exposure

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			and the size of the dust fragments. Particles of 2 microns are 100% respirable, 5 microns are 20 to 30% respirable and 10 microns are non-respirable. Witness stated that he was not aware of any study re a causal relationship between exposure to PS and laryngeal cancer. He further explained that the epidemiological study of Hobson and Jones showed an association between styrene exposure and laryngeal cancer.
75 76 77	20-24 1-24 1-6	IARC Rankings Of Carcinogens	Witness explained the IARC listing of carcinogens. Group I is when clear-cut epidemiological data is available for humans. Group II-A is when reasonable epidemiological data is available for humans. Group II-B is when excellent animal data is available. Group III is when animal data with no epidemiological data is available and Group IV is basically non-carcinogenic or questionable validity of animal data. IARC frequently stated that valid animal data creates a strong presumption of human cancer risk. Witness further explained that EPA rankings, in general, reflect the IARC ranking and sometimes EPA refers to the IARC rankings.
77 78 79	7-24 1-24 1-20	Degassing Of Styrene	Witness stated that the styrene monomer is degassed from the PS pellet the same way that VC is degassed from PVC. Additionally, when you heat-treat PS, you will liberate styrene. Witness further stated that PS contained up to 1% of the volatile residual styrene monomer. Higher concentrations of unreacted styrene are released from fresh PS rather than aged PS. He further explained that

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			when you degas in the air, you will have a progressive reduction in levels of the monomer in the polymer. When you degas in a closed system, there is a migration from within the polymer to the closed dead air space and with the secondary absorption on the surface of the polymer.
80 to 83	8-24 1-16	Peterson's Exposures At ATC	Witness stated that because of the lack of monitoring data by ATC, he was not able to quote what percentage of exposures Peterson had to any resin in the ATC plant. He is unable to substantiate his assumption that the majority of Peterson's time was spent in the presence of PVC. Witness agreed that one of the reasons that he's assumed that PVC was the main chemical to which Peterson was exposed to, was due to Levinson's original letter dated 4/88 re PVC pellets.
83 to 86	17-24 1-2	Shell Report- Bisphenol A Resins	Witness discussed various sections of the Shell Report of 1982. He stated that the isopropylidene bisphenol resins are potent primary irritants and sensitizers. Various components of the resins that cause this reaction are epichlorohydrin, phenolics and the glycidol ethers. He stated that isopropylidene bisphenol resins and particularly 4.4 isopropylidene diphenyl are the same as bisphenol A resins. He is not aware of any literature linking bisphenol A resins and epichlorohydrin with laryngeal cancer. There is literature that linked them with nasal cancer, lung cancer and respiratory tract cancers and the "respiratory tract includes the larynx."

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86 87	3-24 1-5	Interline Reports	Witness explained the conclusions of the three Interline Reports. He stated that the data on studies based on experimental or occupational studies and on a single exposure would understate risk to particular target organs when you expose workers to a wide range of different carcinogens.
87 88	6-24 1-20	Peterson's Exposure To Welding Fumes/Emissions	Witness stated that it is not known what percentage of welding fumes/emissions Peterson was exposed to at ATC but it was his opinion that they played a contributory role to Peterson's laryngeal cancer.
89 90	6-24 1-10	"Attendant Complications"	Witness explained that the term "attendant complications" referred to various complications that can occur from one disease entity.
90 91 92	18-24 1-24 1-16	"Substantially Increased Risk"	Witness stated that although he felt that Peterson was at a "substantially increased risk" for developing other cancers from his occupational exposures at ATC, he could not quantitate it. He felt he was at excess risk for respiratory tract cancers because Peterson was exposed to a wide range of pulmonary carcinogens and the fact Peterson had already had cancer in the respiratory tract, his chances of getting another cancer in that same system are substantially increased.
92 to 95	17-24 1-10	Peterson's Cancer Risk	Witness stated that due to Peterson's chronic obstructive lung disease, Peterson has a substantial increase with relation to cancer above and beyond the general population's one and three risk of getting cancer and one and four risk of dying from cancer.

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95 96 97	11-24 1-24 1-14	Austin Report	Witness stated that the Austin Report studied the effects of smoking and alcohol upon laryngeal cancer. Witness did not consider Peterson's smoking in relation to his development of laryngeal cancer because Peterson had stopped smoking one year prior to his employment at ATC.
97 98	15-24 1-5	Selikoff Report	Witness stated that the Selikoff Report studied the synergistic effect of cigarette smoking and exposure to asbestos. Witness had not done researh re Selikoff's claim.
98	10-24	US Surgeon General's Report	Witness stated that the US Surgeon General's Report summarized that there is a progressive reduction in the risk of developing laryngeal cancer following cessation of smoking.
101 102 103	2-24 1-24 1-9	Smoking/Laryngeal Cancer	Witness stated that due to the fact Peterson developed his laryngeal cancer at a much earlier age than the general population's anticipated age of 60-plus, that would tend to minimize the role of Peterson's smoking and maximize the role of his occupational factors. He further explained that according to the Surgeon General's Report, smoking and laryngeal cancer is strongly dependent on the numbers and due to the fact Peterson smoked less than one pack a day, he would be considered a light smoker which decreased the relationship between his smoking and laryngeal cancer.
104 105	17-24 6-15	Latency Period-Cigarette Smoking	Witness stated that although he does not mention specifically in his report about latency period of contracting any cancer as a result

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			of exposure to cigarette smoking, he does acknowledge the fact that the longer you follow the VC/PVC workers and the more heavily they were exposed, the incidence of respiratory tract cancer is higher.
105 to 108	16-24 1-4	No Observable Effect Level For Carcinogens	Witness stated there is no way of setting safe levels for chemical carcinogens because, from a standpoint, one molecule on one receptor site can create genetic damage which can theoretically be associated with carcinogen effects.
108 109	5-24 1-5		Witness stated that the risk of Peterson's laryngeal cancer is the same as that of a non-smoker.
109 to 112	6-24 1-8	Early V. Late Stage Carcinogens	Witness explained the difference between an early stage and late stage carcinogen. The overwhelming majority of carcinogens are considered early stage, the longer you wait, the higher their risk is. Other carcinogens, such as tobacco and estrogen, depend upon continuing exposure. Once removed from that exposure, their risk decrease progressively. It has been noted by the Consumer Products Safety Commission that one hour of exposure to VC can lead to the development of tumors later in life.
113 114 115	12-24 1-24 1-9	Difference In Latency Periods	Witness explained various descriptions of latency periods. Generally, it is the time of initial exposure to the time of diagnosis. Other descriptions include the time of initial exposure to the time of death and "sometimes latency is defined more sloppily from the mid-point of exposure."

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115	14-24	VC Latency Period	Witness stated that it is not possible to say what VC's latency period is because most of the epidemiological studies have had an inadequate follow-up. He considers the latency period for VC to commence with the first exposure by the individual.
116	1-24		
117	1-4		

Deposition Concluded