EXPERT REPORT OF

RICHARD A. PARENT, PHD, DABT, FATS, RAC, ERT

IN THE MATTER OF

[redacted]

VS.

[redacted]

AND

[redacted]

IN THE

[redacted]

[redacted]

STATE OF LOUISIANA, DIVISION D

CIVIL ACTION FILE NO. [redacted]

CONSULTOX, LIMITED

DAMARISCOTTA, MAINE

[redacted]

TABLE OF CONTENTS

QUALIFICATIONS

I, Richard A. Parent, PhD, DABT, FATS, RAC, ERT, am a board certified toxicologist with over 26 years' experience in the field of industrial toxicology and 19 years' experience in litigation support for both the plaintiff and defense. I have testified in local and federal courts as an expert in toxicology and have given expert testimony in the disciplines of toxicology and chemistry. During my career, I have spent 10 years in research on organic chemicals at American Cyanamid Company. In the field of toxicology, I have initiated and carried out an active program in product safety relating to toxicology for the Xerox Corporation. I have directed two contract toxicology laboratories: Food and Drug Research Laboratories, Inc. and Gulf South Research Institute, Life Sciences Division. In 1984 I established Consultox, Limited, a toxicology consulting firm, and have since consulted in product safety for various industries and have designed toxicology studies to assess the safety of materials being considered for use in various products. For litigants, I have provided toxicological support and have addressed causation issues for the plaintiff as well as the defense. I present myself to the Court as an expert in the fields of toxicology and chemistry. For the Court's information, I offer my curriculum vitae in Appendix A.

INTRODUCTION

In preparing to evaluate this case, I have reviewed medical records of [redacted] dating from 7/11/84 to 2/22/02. In addition, I have read and summarized a medical narrative report on Mr. [redacted] in the form of a letter from [redacted], MD, ENT to [redacted] dated June 18, 2001 and a deposition of Dr. [redacted] given in this matter and relating to Mr. [redacted] office visits and treatments both before and after the incident of April 7, 1999. In addition, I have reviewed in detail, the "Emergency Response Report for Diamond Services Molten Sulfur Tank Fire" dated June 4, 1999 and describing the fire which began between 1200 and 1500 hours on April 7, 1999 in Gibson, Louisiana and the deposition of [redacted], taken on Aug 22nd, 2001.

From the above- referenced "Emergency Response" report, I understand that a tank car containing sulfur caught fire on the opposite side of the Bayou from the Kiva Construction and Engineering Company and that during the incident, the wind was blowing from the southwest which caused the smoke plume to drift across Bayou Black toward KIVA Construction and Engineering. It is my understanding that Mr. [redacted] was exposed to this smoke plume in the vicinity of KIVA for a period of about two and one half hours and that he suffered damage to his upper respiratory system as a result of this exposure and as described by his physician. It is my further understanding that sulfur dioxide exposure levels were measured as high as 35 ppm in the air at the KIVA dock and that this measurement was not taken at the peak of the incident. To my knowledge, no measurements of hydrogen sulfide were attempted.

MATERIALS REVIEWED

- Deposition of [redacted] taken on August 22nd, 2001.
- Letter from [redacted], MD, ENT to [redacted] dated June 18th, 2001, a narrative report on Mr. [redacted]
- Emergency Response Report for Diamond Services Molten Sulfur Tank Fire in Gibson, Louisiana, on June 4th, 1999 by Ecology and Environment to the EPA.
- Medical Records of Mr. [redacted] from July 11th, 1984 to February 22nd, 2002(summarized below).
- Deposition of Dr. [redacted]
- Peer-reviewed published literature relating to the effects of inhaled sulfur oxides on human health.

SUMMARY OF MEDICAL RECORDS OF LARRY WEAVER

REDACTED

DISCUSSION

It should be noted that burning sulfur will produce high levels of sulfur dioxide and hydrogen sulfide and that the sulfur dioxide will become oxidized in the environment producing some sulfur trioxide which, when combined with moisture in the air, will be converted to a sulfuric acid aerosol.[1-4] Thus, [redacted] was most probably exposed to a mixture of mainly sulfur dioxide, with hydrogen sulfide, sulfur trioxide and sulfuric acid mist. All of these chemicals are highly irritating to the upper respiratory system and sulfur dioxide has resulted in several cases of human deaths at exposures involving elevated concentrations.[2,5,6]

Inhalation of sulfur dioxide gas, has been shown to produce all degrees of respiratory tract irritation ranging from irritation of mucous membranes of the upper respiratory system to pulmonary edema and death,[6-11] depending on the exposure scenario.[2,12] Sulfur dioxide and the other components of the mixture to which Mr. [redacted] was exposed are highly soluble in water and are consequently rapidly absorbed by the mucosa of the nose and upper respiratory system following inhalation exposure[2,5,13-15] and this has been reported to stimulate hypersecretion of mucus.[16] It is interesting to note that a single exposure to a high concentration of sulfur dioxide can result in bronchial hyperreactivity resulting from epithelial cell injury and destruction, airway mucosal edema/inflammation and airway smooth muscle bronchospasm, a condition referred to as Reactive Airways Disease Syndrome(RADS).[17,18,19] In this syndrome, bronchial epithelial damage results in increased sensitization and non-specific hypersensitivity to a wide range of other irritant stimuli.[9,17-19] The pulmonary hyperreactivity which results from acute exposures to sulfur dioxide may persist for several years[9,20] as demonstrated by positive histamine challenge testing showing bronchial hyperreactivity.[20] Increased bronchial reactivity has also been reported from exposure to

sulfuric acid aerosols.[21] In one report, exposure of copper mine workers to >40ppm sulfur dioxide resulted in burning of the nose and throat, dyspnea, and severe airway obstruction that was only partially reversed after two years.[11]

Asthmatics are particularly susceptible to the effects of sulfur dioxide[15,22-26] and tend to exhibit bronchoconstriction[27] and progressive lower respiratory symptoms such as wheezing, chest tightness, dyspnea, and cough.[24] Death rates in exposed populations have been correlated with increased concentrations of sulfur dioxide in the air.[28]

Exposure to very low concentrations of sulfur dioxide have been shown to produce definitive effects in exposed individuals.[2,15,29] For example: exposure to 0.5 ppm sulfur dioxide for three minutes resulted in increased airway resistance, wheezing, chest tightness, dyspnea, and bronchoconstriction;[30-32] exposure to from 0.6 to 0.8 ppm for 5 minutes produced increased airway resistance;[33] exposure to 4 ppm for 20 minutes resulted in changes in the defense capabilities of the lung;[34] exposure to 5 ppm for 10 minutes resulted in increased airway resistance;[35] exposure to from 1 to 8 ppm for 10 minutes resulted in increased tidal volume, respiratory rate and pulse;[36] exposure to 5 ppm for 10-30 minutes resulted in increased flow resistance after one minute, cough, and irritation.[37] Exposures to from 6 to 12 ppm may cause nasal and throat irritation, to 10 ppm may cause upper respiratory irritation and nosebleeds, to 20 ppm may cause definite eye irritation, chronic respiratory symptoms and exposures in excess of 100 ppm are considered to be an immediate danger to life.[5] Long-term, low-level exposure to sulfur dioxide is also reported to be related to the development of COPD.[38-41] Asthmatics are particularly susceptible to the effects of sulfur dioxide[42] while healthy non-asthmatics can show pulmonary function changes following inhalation exposures to as little as 1ppm sulfur dioxide.[2,21,26,43-47]

Animal studies have demonstrated the effects of sulfur dioxide on the nasal mucosa including a progression from normal ciliated epithelium to cuboidal epithelium with complete disappearance of cilia to squamous stratified epithelium.[48] A study in rats exposed to sulfur dioxide produced respiratory mucus cell hyperplasia resulting in hypersecretion of mucus and accumulation of mucus throughout the respiratory tract, a condition similar to that observed in human bronchitis.[16,49,50] Mice exposed to sulfur dioxide were reported to exhibit edema, loss of cilia, epithelial thinning and epithelial desquamation in olfactory epithelial tissue.[51] Rabbits exposed to sulfur dioxide were used as models for human bronchitis[52] and exposed dogs showed increased bronchial responsiveness.[53,54] Hypertrophy and hyperplasia of the submucosal mucous glands in exposed rat tracheas have also been reported.[55] Another study in rats exposed to as little as 5ppm sulfur dioxide demonstrated glutathione depletion in the respiratory tract.[56] Mice exposed to 10 ppm sulfur dioxide for 24 hours showed lesions in the nasomaxillary turbinates (nasal area) consisting of edema, necrosis and desquamation of the respiratory and olfactory epithelium.[57] Another study reported edema, loss of cilia, epithelial thinning and epithelial desquamation in olfactory epithelia of mice exposed to sulfur dioxide.[51] Loss of cilia in response to acute sulfur dioxide exposure appears to constitute the initial injury event which compromises the upper respiratory immune system[58,59] while at higher concentrations, general exfoliation of the epithelium has been reported in dogs.[6] After long-term exposure to sulfur dioxide, dogs have been reported to exhibit bronchoconstriction,[60] chronic bronchitis[41] and nasopharyngeal changes including

epithelial proliferation, loss of secretory material and moderate mononuclear cell infiltration.[61] Mucociliary clearance disruption has been demonstrated in guinea pig tracheas[59,62] and in exposed ferrets.[63] Also of interest is the initiation of sinusitis in rabbits by exposure to sulfur dioxide[64] and the similar effect noted in humans.[65] Allergic rhinitis in children has been noted to be related to sulfur dioxide concentrations in the atmosphere.[66]

One of the more insidious aspects of acute exposures to sulfur dioxide, sulfur trioxide or sulfuric acid is the destruction of the ciliated epithelial and mucosal tissue resulting in a major compromise of the defense capability of the nasal and upper respiratory system resulting in persistent infections.[5] Inhalation of sulfur dust has been shown to result in asthma or exacerbation of asthma as well as maxillary and frontal sinusitus but usually bilateral and pansinusitis.[5] Hydrogen sulfide also produces irritation of mucosal membranes but is, in addition, a neurotoxin. However, it is flammable and would probably not survive a fire in any significant amount since it would be converted to sulfur dioxide. Inhalation of small amounts of hydrogen sulfide does cause rapid onset of olfactory fatigue resulting in loss of the sense of smell.[67]

CONCLUSIONS AND OPINIONS

It is my understanding that, prior to the incident of 04/07/1999, Mr. [redacted] had chronic sinus problems for which he was being treated by [redacted], MD, ENT. In addition, he was a heavy smoker and was probably suffering from COPD, was a diabetic, was suffering from gastrointestinal problems and had a chronic perforation of the left tympanic membrane.

On the day following his exposure to toxic fumes, Mr. [redacted] sought treatment from his family physician, Dr. [redacted] who observed congestion, cough, expiratory wheezing, and bronchitis and related some of these symptoms to a "toxic inhalation". Subsequent visits describe nasal irritation and swollen nasal mucosa. Mr. [redacted] subsequently went to see Dr. [redacted] on 04/16/1999 and 05/10/1999 and many times subsequently. Dr. [redacted] found Mr. [redacted]'s nasal mucosa to be dry, red and scabby with an infection with enterobactra aerogenes, a bacteria commonly found in burn injuries. In subsequent comments after continuing treatment, Dr. [redacted] described the injury as "chronic rhinitis secondary to an inhalation burn and injury" and indicated that the injury is most likely permanent.

Considering the medical records indicated above, the exposure scenario described and the nature of the toxic fumes to which Mr. [redacted] was exposed, I opine that Mr. [redacted]'s chronic allergic rhinitis and reactive airways presenting as asthma was most probably caused by his exposure to greater than 35 ppm sulfur oxides emanating from the sulfur fire which occurred on April 7, 1999. I further believe that Mr. [redacted] chronic sinusitis, while not being caused by his exposure to the fumes from the sulfur fire, was most probably exacerbated by the exposure incident.

CONSULTOX, LIMITED

Date