United States District Court

for the

NORTHERN DISTRICT OF FLORIDA
PENSACOLA DIVISION

REDACTED, individually and on behalf of all those so unfortunate as to be similarly situated as a result of suffering physiological injury associated with their exposure to the dioxin contaminated phenoxy herbicides and the cacodylates used at Eglin Air Force Base, particularly at *Site C–52A,* during the War in Southeast Asia from 1962 through 1970, and as a result thereof are now in danger of death from systemic diseases resulting from such exposure, and

REDACTED and on behalf of all those so unfortunate as to be similarly situated as a result of the untimely death of their spouse systemic diseases associated with their suffering physiological injury associated with their exposure to the dioxin contaminated phenoxy herbicides and the cacodylates used at Eglin Air Force Base, particularly at *Site C–52A,* during the War in Southeast Asia from 1962 through 1970, and

REDACTED individually and on behalf of all the other landowners and residents of the Eglin Air Force Base Regional Hydrologic System so unfortunate as to be similarly situated as a result of suffering physiological injury associated with their exposure to the dioxin contaminated phenoxy herbicides and the cacodylates used at Eglin Air Force Base, particularly at *Site C–52A,* during the War in Southeast Asia from 1962 through 1970, and as a result thereof are now in danger of death from systemic diseases resulting from such exposure,

all jointly and severally, individually and collectively, on behalf of all those so unfortunate as to be similarly afflicted as a result of exposure to the environmental toxicants contained in products manufactured, advertised, promoted, marketed, and sold by the Defendant war contractors, jointly and severally, individually and collectively, for deployment as chemical defoliants during the War in Southeast Asia,

Plaintiffs

*–**against**–*

BAE SYSTEMS PLC, successor in interest to GEC PLC, successor in interest to TRACOR SYSTEMS TECHNOLOGIES, successor in interest to TRACOR, which purchased GK TECHNOLOGIES, successor in interest to GENERAL CABLE CORPORATION, successor in interest to AUTOMATION INDUSTRIES (Vitro Engineering Corp.), successor in interest to VITRO CORP. OF AMERICA, the civilian contractor conducting tests of herbicides at *Site C–52A,*

Defendant

–and–

BAYER AG, successor in interest by acquisition to **MONSANTO COMPANY**,

VALERO ENERGY CORPORATION and **OCCIDENTAL PETROLEUM**, as their several interest may appear to the extent they are the successors in interest to ULTRAMAR DIAMOND SHAMROCK, as successor in interest to ULTRAMAR as successor in interest to DIAMOND SHAMROCK CORPORATION, successor in interest to DIAMOND ALKALI which had acquired NATIONAL OIL PRODUCTS COMPANY and NOPCO CHEMICAL,

DowDuPont successor in interest by merger and acquisition to THE **DOW CHEMICAL COMPANY**,

ASHLAND INC. as successor in interest to HERCULES, INC.,

SCA COMPAGNIE GÉNÉRALE DES ÉTABLISSEMENTS MICHELIN, successor in interest to UNIROYAL INC.,

HARCROS CHEMICALS, INC., successor in interest to HARRISONS AND CROSFIELD PLC**,** successor in interest to NORTH AMERICAN PHILIPS, which acquired THOMPSON-HAYWARD CHEMICAL COMPANY,

KONINKLIJKE PHILIPS ELECTRONICS N.V., which is the parent of Philips Electronics North America Corporation, which is the parent of THAN, which is the successor in interest, and

THOMPSON CHEMICAL COMPANY,

each jointly and severally, individually and collectively, as manufacturers of dioxin contaminated phenoxy herbicides and the cacodylates or components thereof or constituent chemical raw materials thereof to which the Plaintiffs were exposed at Eglin Air Force Base, including site C–52A,

TCDD Defendants

–and–

JOHNSON CONTROLS INTERNATIONAL PLC which merged with TYCO INTERNATIONAL LTD, successor in interest to WORMALD INTERNATIONAL which acquired ANSUL FIRE PROTECTION which was the renamed ANSUL COMPANY which was the renamed ANSUL CHEMICAL COMPANY which manufactured organic arsenicals designated *Agent Blue* or components therefore or constituent chemical raw materials thereof to which the Plaintiffs were exposed at Eglin Air Force Base, including site C–52A,

Cacodylate Defendant.

VERIFIED CLASS ACTION COMPLAINT

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VERIFIED CLASS ACTION COMPLAINT

INTRODUCTION

This is a civil action brought by civilian employees of government contractors and their surviving family members, seeking damages from the Defendants, jointly and severally, individually and collectively, for causing those employees and their families physiological injury, systematic disease, genetic damage and death by allowing the unprotected exposure of their employees to dioxin contaminated phenoxy herbicides and the cacodylates tested at Eglin Air Force Base during the War in Southeast Asia.

The claims arise out of the manufacture, supply and use of dioxin contaminated phenoxy herbicides and the cacodylates by the Defendants.

This action is a mass toxic tort where the physiological injury, systematic disease, genetic damage and death afflicting the Plaintiffs and attributable to the toxic effects of their exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base has resulted in catastrophic damages to the Plaintiffs.

PLAINTIFFS DEMAND A JURY TRIAL

JURISDICTION

1. OMITTED FROM THIS ABRIDGED VERSION

VENUE

7. OMITTED FROM THIS ABRIDGED VERSION

OMITTED FROM THIS ABRIDGED VERSION

JOINDER OF PLAINTIFFS IN THIS ACTION

52. There are multiple questions of fact and law common to each of the Plaintiffs who have joined together to bring this action, including, but not limited to the following.

53. Whether the Plaintiffs were exposed to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base.

54. Whether the Defendants, individually or collectively, jointly or severally, knew or should have known that the phenoxy herbicides they manufactured, sold, distributed, deployed, or used were contaminated with 2,3,7,8–tetrachloro-dibenzo-*p*-dioxin (TCDD or dioxin).

55. Whether the phenoxy herbicides manufactured by one or more of the Defendants were contaminated with 2,3,7,8–tetrachloro-dibenzo-*p*- dioxin (TCDD or dioxin).

56. Whether the Defendants, individually or collectively, jointly or severally, knew or should have known of the dangers to human health associated with direct human contact and exposure to dioxin contaminated phenoxy herbicides and the cacodylates.

57. Whether the Defendants, individually or collectively, jointly or severally, should have disclosed their knowledge of and about the dangers to human health associated with exposure to dioxin contaminated phenoxy herbicides and the cacodylates.

58. Whether the Defendants, individually or collectively, jointly or severally, had a duty to warn the Plaintiffs before they were

exposed to dioxin contaminated phenoxy herbicides and the cacodylates.

59. Whether the Defendants, individually or collectively, jointly or severally, had a duty to protect Plaintiffs from exposure to dioxin contaminated phenoxy herbicides and the cacodylates.

60. Whether repeated exposure to dioxin contaminated phenoxy herbicides and the cacodylates during at Eglin Air Force Base, site C–52A, during the war with Southeast Asia caused physiological injury to Plaintiffs.

61. Whether the Defendants, individually or collectively, jointly or severally, are entitled to immunity for their actions as "government contractors."

CLASS ACTION ELEMENTS

62. OMITTED FROM THIS ABRIDGED VERSION

Commonality Fed.R.Civ.P. 23(a)(2)

66. There are multiple questions of fact and law common to members of the class including, but not limited to the following.

67. Whether the Plaintiffs were exposed to dioxin contaminated phenoxy herbicides at Eglin Air Force Base.

68. Whether the Defendants, individually or collectively, jointly or severally, knew or should have known that the phenoxy herbicides they manufactured, sold, distributed, deployed, or used were contaminated with 2,3,7,8–tetrachloro-dibenzo-*p*-dioxin (TCDD or dioxin).

69. Whether the phenoxy herbicides manufactured by one or more of the Defendants were contaminated with 2,3,7,8–tetrachloro-dibenzo-*p*- dioxin (TCDD or dioxin).

70. Whether the Defendants, individually or collectively, jointly or severally, knew or should have known of the dangers to human health associated with direct human contact and exposure to dioxin contaminated phenoxy herbicides.

71. Whether the Defendants, individually or collectively, jointly or severally, should have disclosed their knowledge of and about the dangers to human health associated with exposure to dioxin contaminated phenoxy herbicides.

72. Whether the Defendants, individually or collectively, jointly or severally, had a duty to warn the Plaintiffs before they were exposed to dioxin contaminated phenoxy herbicides.

73. Whether the Defendants, individually or collectively, jointly or severally, had a duty to protect Plaintiffs from exposure to dioxin contaminated phenoxy herbicides.

74. Whether repeated exposure to dioxin contaminated phenoxy herbicides during at Eglin Air Force Base, site C–52A, during the war with Southeast Asia caused physiological injury to Plaintiffs.

75. Whether the Defendants, individually or collectively, jointly or severally, are entitled to immunity for their actions as "government contractors."

76. Defendants, jointly and severally, individually and collectively, can be expected to raise common defenses to these claims, including denying that FDC is expected to raise common defenses to the claims of the Representative Plaintiffs.

OMITTED FROM THIS ABRIDGED VERSION

THE PLAINTIFF CLASS OF CIVILIAN EMPLOYEES

91. Each of the Plaintiffs was an employee of the civilian contractors participating in the tests of chemical defoliants including dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base, or a spouse, or child of such employee.

92. Each of the Plaintiff employees of the civilian contractors participating in the tests of chemical defoliants including dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base was exposed without protection to dioxin contaminated phenoxy herbicides and the cacodylates during their employment at Eglin Air Force Base during the War in Southeast Asia from 1962 through 1970.

93. Each of the Plaintiff employees of the civilian contractors participating in the tests of chemical defoliants including dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base has suffered physiological injury and is, or if they are already dead, was, afflicted with systematic disease as a result of such exposure.

94. Each of the Plaintiffs working on the ground during the tests of chemical defoliants including dioxin contaminated phenoxy herbicides and cacodylates at Eglin Air Force Base was routinely sprayed with the herbicides without the protection of any safety gear, and none of the Plaintiffs was never advised, much less warned of the dangers associated with exposure to dioxin contaminated phenoxy herbicide and organic arsenicals, the cacodylates.

THE PLAINTIFF CLASS OF REGIONAL RESIDENTS

95. The members of this class are the landowners and residents of the Eglin Air Force Base Regional Hydrologic System so unfortunate as to be similarly situated as a result of suffering

physiological injury associated with their exposure to the dioxin contaminated phenoxy herbicides and the cacodylates used at Eglin Air Force Base, particularly at *Site C–52A,* during the War in Southeast Asia from 1962 through 1970, and as a result thereof are now in danger of death from systemic diseases resulting from such exposure.

96. All the landowners and residents of this Plaintiff class were exposed to dioxin contaminated phenoxy herbicides and the cacodylates from direct contact or through inhalation during the period these chemicals were applied by aerial spraying at Eglin Air Force Base and later through ingestion or contact with herbicide contaminated water from the Eglin Air Force Base Regional Hydrologic System

97. All the landowners and residents of this Plaintiff class are or have been residents of the Eglin Air Force Base Regional Hydrologic System and ingested or have been exposed to ground and surface waters contaminated with dioxin contaminated phenoxy herbicides and the cacodylates.

98. All the landowners and residents of this Plaintiff class have lived and worked within the Eglin Air Force Base Regional Hydrologic System since the aerial application of dioxin contaminated phenoxy herbicides and the cacodylates began 1n 1962.

99. All the landowners and residents of this Plaintiff class have been exposed on a daily basis to dioxin contaminated phenoxy herbicides and the cacodylates throughout the time they have lived within the Eglin Air Force Base Regional Hydrologic System.

THE REPRESENTATIVE PLAINTIFFS

100. OMITTED FROM THIS ABRIDGED VERSION

DEFENDANTS

Defendant BAE Systems PLC

132. BAE Systems PLC, is the successor in interest to GEC PLC, which was the successor in interest to TRACOR SYSTEMS TECHNOLOGIES, which was the successor in interest to TRACOR, which purchased GK TECHNOLOGIES, which was the successor in interest to GENERAL CABLE CORPORATION, which was the successor in interest to AUTOMATION INDUSTRIES (Vitro Engineering Corp.), which was the successor in interest to VITRO CORP. OF AMERICA, which, upon information and belief, was the civilian contractor conducting tests of dioxin contaminated phenoxy herbicides and the cacodylates to which the Plaintiffs were exposed at Eglin Air Force Base, particularly, Site C–52A.

Vitro Corp. of America

133. Vitro was incorporated in 1950 as the Vitro Manufacturing Company. Its main product was slide transparencies for overhead projectors. For some time prior to incorporation, the founders had produced tinted lighting gels for the theater and stage industry and were known for their leadership in the production of gels used to replicate the lighting characteristics of the outdoors on a sunny day. These gels had this quality because of the admixture of salts of uranium, which are bright yellow. This business had positioned Vitro as a ready source of uranium during US military and scientific research efforts into nuclear energy during the Second World War.

134. In 1950 Vitro acquired the Kellex Corporation, a company involved in the development of the nuclear bomb. In 1953 the company reorganized as the Vitro Corp. of America.

135. One of Vitro's earliest customers was the United States Navy, a relationship which continued into the 1990s.

136. In 1968 Vitro was acquired by Automation Industries Inc and renamed Vitro Engineering Corp.

137. In 1978 Automation Industries and its Vitro subsidiary were acquired by General Cable Corp, which renamed the new company GK Technologies.

138. In 1981 GK Technologies (including Vitro) was acquired by the Penn Central Corporation. Penn Central had emerged from bankruptcy without its failing rail businesses (acquired by Federal government) and diversified into defense and other businesses. By the early 1990s Penn Central had begun to focus on financial services and in 1992 announced it was divesting Vitro.

139. In 1993 Tracor completed its purchase of Vitro for $94 million. The Vitro acquisition almost doubled the size of Tracor. Vitro merged with Tracor Applied Sciences to form Tracor Systems Technologies, Tracor's largest subsidiary.

140. In July 1998 the British electronics conglomerate GEC PLC purchased Tracor.

141. In November 1999 GEC merged its defense arm (including Tracor) with British Aerospace to form BAE Systems.

BAE Systems PLC

142. BAE Systems PLC is a British multinational defense, security and aerospace company. It has operations worldwide. It was ranked as the third-largest defense company based on applicable 2015 revenues.

143. Its BAE Systems Inc. subsidiary is one of the six largest suppliers to the US Department of Defense.

144. The company was formed on 30 November 1999 by the £7.7 billion merger of two British companies: Marconi Electronic Systems (MES), the defense electronics and naval shipbuilding subsidiary of the General Electric Company PLC (GEC), and British Aerospace (BAe), an aircraft, munitions and naval systems manufacturer.

145. Marconi Electronic Systems was the defense subsidiary of British engineering firm The General Electric Company (GEC), dealing largely in military systems integration, as well as naval and land systems. Marconi dates back to Guglielmo Marconi's Wireless Telegraph & Signal Company, founded in 1897. GEC purchased English Electric which included Marconi in 1968 and thereafter used the Marconi brand for its defense businesses as GEC-Marconi and later Marconi Electronic Systems.

146. In June 1998, MES acquired Tracor, a major American defense contractor, for £830 million (approximately US$1.4 billion *c.* 1998).

147. BAE Systems is the successor to various aircraft, shipbuilding, armored vehicle, armaments and defense electronics companies, including the Marconi Company, the first commercial company devoted to the development and use of radio; A.V. Roe and Company, one of the world's first aircraft companies; de Havilland, manufacturer of the Comet, the world's first commercial jet airliner; British Aircraft Corporation, co-manufacturer of the Concorde supersonic transport; Supermarine, manufacturer of the Spitfire; Yarrow Shipbuilders, builder of the Royal Navy's first destroyers; Fairfield Shipbuilding and Engineering Company, pioneer of the triple-expansion engine and builder of the world's first battlecruiser; and Vickers Shipbuilding and Engineering, builder of the Royal Navy's first submarines. Since its formation it has made a number of acquisitions, most notably

of United Defense and Armor Holdings of the United States, and sold its shares in Airbus, Astrium, AMS and Atlas Elektronik.

148. BAE Systems is involved in several major defense projects, including the Lockheed Martin F-35 Lightning II, the Eurofighter Typhoon, the *Astute*-class submarine and the *Queen Elizabeth*-class aircraft carriers. BAE Systems is listed on the London Stock Exchange and is a constituent of the FTSE 100 Index.

Defendant Bayer AG

149. Defendant **Bayer AG** is a German multinational pharmaceutical and life sciences company and one of the largest pharmaceutical companies in the world.

150. Bayer is headquartered in Leverkusen, Germany.

151. Bayer's areas of business include human and veterinary pharmaceuticals; consumer healthcare products; agricultural chemicals, seeds and biotechnology products.

152. Bayer is a component of the Euro Stoxx 50 stock market index.

153. Founded in Barmen in 1863 as a dyestuffs factory.

154. Bayer's first and best-known product was aspirin.

155. In 1898 Bayer trademarked the name *heroin* for the drug diacetylmorphine and marketed it as a cough suppressant and non-addictive substitute for morphine until 1910.

156. Bayer also introduced phenobarbital; prontosil, the first widely used antibiotic and the subject of the 1939 Nobel Prize in Medicine; the antibiotic Cipro (ciprofloxacin); and Yaz (drospirenone) birth control pills.

157. In 1925 Bayer was one of six chemical companies that merged to form IG Farben, the world's largest chemical and pharmaceutical company.

158. The Allied Control Council seized IG Farben after World War II, because of its role in the Nazi war effort and involvement in the Holocaust, which included using slave labor from concentration camps.

159. IG Farben was split into its original six constituent companies in 1951, then split again into three: BASF, Bayer and Hoechst.

160. Bayer played a key role in the *Wirtschaftswunder* in post-war West Germany, quickly regaining its position as one of the world's largest chemical and pharmaceutical corporations.

161. In 2006 Bayer acquired Schering.

162. In 2014 Bayer acquired the consumer business of Merck & Co., with brands such as Claritin, Coppertone and Dr. Scholl's.

163. Bayer CropScience develops genetically modified crops and pesticides.

Monsanto Company

164. Defendant **Monsanto Company** was an American agrochemical and agricultural biotechnology corporation that existed from 1901 until 2018 when it was acquired by Bayer as part of its crop science division.

165. In September 2016, Bayer announced its intent to acquire Monsanto for US$66 billion. After gaining US and EU regulatory approval, the sale was completed on June 7, 2018.

166. In an effort to receive regulatory clearance for the deal, Bayer announced the sale of significant portions of its current agriculture businesses, including its seed and herbicide businesses, to BASF.

167. The deal was approved by the European Union on March 21, 2018 and approved in the United States on May 29, 2018. The sale closed on June 7, 2018; Bayer announced its intent to

discontinue the Monsanto name, with the combined company operating solely under the Bayer brand.

168. Monsanto Company was a manufacturer of dioxin contaminated phenoxy herbicides to which the Plaintiffs were exposed at Eglin Air Force Base, including site C–52A.

169. Monsanto was one of the top 10 U.S. chemical companies until it divested most of its chemical businesses between 1997 and 2002, through a process of mergers and spin-offs that focused the company on biotechnology.

170. Monsanto once manufactured DDT, PCBs, Agent Orange and recombinant bovine growth hormone.

171. In September 2016 Monsanto agreed to accept the offer of Bayer to purchase the company for $66 billion, pending regulatory approval.

172. In 1901 Monsanto was founded in St. Louis, Missouri, as a chemical company. The founder was John Francis Queeny. He used his wife's maiden name for the company. The company's first products the artificial sweetener saccharin, caffeine and vanillin.

173. Monsanto expanded to Europe in 1919 in a partnership with Graesser's Chemical Works at Cefn Mawr, Wales. The venture produced vanillin, aspirin and its raw ingredient salicylic acid, and later rubber processing chemicals.

174. In the 1920s, Monsanto expanded into basic industrial chemicals including sulfuric acid and PCBs.

175. In 1926 the company founded and incorporated a town called Monsanto in Illinois. It was formed to provide minimal regulation and low taxes for Monsanto plants at a time when local jurisdictions had most of the responsibility for environmental rules. It was renamed in honor of Leo Sauget, its first village president.

176. Edgar Monsanto Queeny took over the company in 1928.

177. In 1935, Monsanto bought the Swann Chemical Company in Anniston, Alabama, and entered the business of producing PCBs.

178. In 1936, Monsanto acquired Thomas & Hochwalt Laboratories in Dayton, Ohio, to acquire the expertise of Charles Allen Thomas and Carroll A. Hochwalt. The acquisition became Monsanto's Central Research Department. Thomas spent the rest of his career at Monsanto, serving as President (1951–60) and Board Chair (1960–65). He retired in 1970.

179. In 1943, Thomas was called to a meeting in Washington, DC, with Leslie Groves, commander of the Manhattan Project, and James Conant, president of Harvard University and chairman of the National Defense Research Committee (NDRC).

180. The National Defense Research Committee (NDRC) was an organization created "to coordinate, supervise, and conduct scientific research on the problems underlying the development, production, and use of mechanisms and devices of warfare" in the United States from June 27, 1940, until June 28, 1941. It was superseded by the Office of Scientific Research and Development in 1941 which was eventually terminated during 1947.

181. Thomas declined to become co-director of the Manhattan Project at Los Alamos with Robert Oppenheimer, but he joined the NDRC, and the Monsanto Central Research Department began to conduct related research. Monsanto operated the Dayton Project, and later Mound Laboratories, and assisted in the development of the first nuclear weapons.

182. Monsanto began manufacturing DDT in 1944.

183. In 1946, Monsanto developed and marketed *All* laundry detergent, which they sold to Lever Brothers in 1957.

184. In 1947, the Monsanto styrene factory was destroyed in the Texas City Disaster.

185. In 1949, Monsanto acquired American Viscose from Courtaulds.

186. In 1954, Monsanto partnered with German multinational chemical giant Bayer to form Mobay and market polyurethanes in the United States.

187. In the mid-1960s, chemists at Monsanto developed the Monsanto process for making acetic acid, which until 2000 was the most widely used production method.

188. In 1964, Monsanto chemists invented *AstroTurf* which was originally marketed as *ChemGrass*.

189. In the mid-1960s, William Standish Knowles and his team invented a way to selectively synthesize enantiomers via asymmetric hydrogenation, the first method for the catalytic production of pure chiral compounds. Knowles' team designed the first industrial process to chirally synthesize L-dopa, which is used to treat Parkinson's disease. In 2001, Knowles and Ryōji Noyori won the Nobel Prize in Chemistry.

190. In 1968, Monsanto became the first company to start mass production of visible light emitting diodes (LEDs), using gallium arsenide phosphide.

191. In the 1960s and 1970s, Monsanto was a major producer of Agent Orange.

192. In 1977, Monsanto stopped producing PCBs.

193. Monsanto scientists were among the first to genetically modify a plant cell, publishing their results in 1983. Five years later the company conducted the first field tests of genetically modified crops.

194. Increasing involvement in agricultural biotechnology by Monsanto dates from the installment of Richard Mahoney as CEO in 1983, leading ultimately to the disposition of product lines unrelated to agriculture.

195. In 1985, Monsanto acquired G. D. Searle & Company, a life sciences company that focused on pharmaceuticals, agriculture and animal health for $2.7 billion. Searle's aspartame business became a separate subsidiary, the NutraSweet Company. CEO of NutraSweet, Robert B. Shapiro, served as CEO of Monsanto from 1995 to 2001.

196. In 1993, its Searle division filed a patent application for Celebrex, which in 1998 became the first selective COX-2 inhibitor to be approved by the U.S. Food and Drug Administration (FDA). Celebrex was often mentioned as a key reason for Pfizer's acquisition of Monsanto's pharmaceutical business in 2002.

197. In 1994, Monsanto introduced a recombinant version of bovine somatotropin, brand-named *Posilac*. Monsanto later sold this business to Eli Lilly and Company.

198. In 1996, Monsanto purchased Agracetus, the biotechnology company that had generated the first transgenic cotton, soybeans, peanuts and other crops, and from which Monsanto had been licensing technology since 1991.

199. In 1996, Monsanto acquired a majority interest in Calgene, creators of the *Flavr Savr* tomato.

200. Monsanto entered the maize seed business when it purchased 40% of Dekalb purchasing the remainder of the corporation in 1998.

201. In 1997, Monsanto spun off its industrial chemical and fiber divisions into Solutia.

202. In 1997, Monsanto announced the purchase for $925 million of Holden's Foundations Seeds, a privately held seed business making Monsanto the largest American producer of "foundation" corn, the parent seed from which hybrids are made.

203. In 1998, Monsanto purchased Cargill's international seed business, which gave it access to sales and distribution facilities in 51 countries.

204. In 1999, Monsanto sold off NutraSweet Co.

205. In 1999, Monsanto merged with Pharmacia and Upjohn. The agricultural division became a wholly owned subsidiary of the "new" Pharmacia.

206. In 2000, Pharmacia spun off its Monsanto subsidiary into a new company, the "new Monsanto" and Monsanto agreed to indemnify Pharmacia against potential liabilities from judgments against Solutia. Pharmacia was bought by Pfizer in 2003.

207. In 2005, Monsanto acquired Emergent Genetics and its *Stoneville* and *NexGen* cotton brands. Emergent was the third largest U.S. cotton seed company, with about 12% of the U.S. market. Monsanto stated that its goal was to obtain "a strategic cotton germplasm and traits platform."

208. Seminis, Inc.

209. In 2005, Monsanto finalized the purchase of Seminis Inc., a leading global vegetable and fruit seed company, comprised of several well-known local and regional seed brands, some dating back to the 1860s, for $1.4 billion, becoming the world's largest conventional seed company.

210. In 1865 Seminis' oldest brand, Asgrow Vegetable Seeds was established.

211. In 1868 Royal Sluis was established in Holland and later becomes a leading European vegetable seed company.

212. In 1906 The global seed industry begins. Royal Sluis becomes the first European vegetable seed company to grow seed crops in both Europe and the Americas. The company also opens its first branch office in the U.S.

213. In 1936 In Korea, Hungnong Seed Company was established. Hungnong was the leading provider of radish and hot pepper seeds, as well as many Asian vegetables.

214. In 1946 Bruinsma (est 1934), a specialist in greenhouse and protected culture, introduces the first commercial tomato hybrid in Europe. Korean seed company, Choong Ang, was established.

215. In 1950 Petoseed was founded in California. Petoseed led the hybridization of hot peppers and tomatoes in the 1970s and 80s; the company built a strong presence in Mexico and the U.S. with disease-resistant seed varieties.

216. In 1967 The first Brazilian plant breeding company, Horticeres, was founded.

217. In 1994 Seminis was founded through the acquisition of Asgrow Seed Company, as well as the Bruinsma and Genecorp brands, specializing in greenhouse and lettuce, respectively.

218. In 1995 Seminis acquires Petoseed Company and Royal Sluis.

219. In 1998 Seminis purchases Korean firms Hungnong Seed Company and Choong Ang Seed Company to enhance product lines in Asia. Seminis purchases Horticeres, the vegetable division of Sementes Agreceres SA, strengthening its presence in South America and product lines for tropical climates.

220. In 2001 Seminis introduces the Seminis Vegetable Seeds brand.

221. In 2002 Seminis completes the integration of its legacy systems.

222. In 2004 In China, Seminis opens an operations center and its third research facility.

223. In 2003-04 Seminis adds four research stations in India, opens an operations center and significantly expands commercial activities. Seminis launches innovative products such as Bambino®, a personal watermelon and Lettuce Jammers®, a new type of lettuce for wraps.

224. In 2005 Seminis becomes a wholly owned subsidiary of Monsanto Company.

225. In 2007 Monsanto acquires Western Seed and Poloni Semences and creates International Seed Group, Inc. (ISG), an investment holding company that provides specialized, regional vegetable and fruit seed companies with access to capital and technology.

226. In 2008 Monsanto acquires Peotec as part of ISG, and De Ruiter Seeds, specialized in protected cultures. Seminis continues to focus on open field crops.

227. In 2007, Monsanto and BASF (*Badische Anilin und Soda Fabrik*) announced a long-term agreement to cooperate in the research, development, and marketing of new plant biotechnology products.

228. In 2007, Monsanto purchased Delta and Pine Land Company, a major cotton seed breeder, for $1.5 billion. As a condition for approval from the Department of Justice, Monsanto was obligated to divest its *Stoneville* cotton business, which it sold to Bayer, and to divest its *NexGen* cotton business, which it sold to Americot. Monsanto also exited the pig-breeding business by selling Monsanto Choice Genetics to Newsham Genetics LC in November, divesting itself of “any and all

swine-related patents, patent applications, and all other intellectual property".

229. In 2008, Monsanto purchased Dutch seed company De Ruiter Seeds for €546 million.

230. Monsanto developed and sold recombinant bovine somatotropin (also known as rBST and rBGH), a synthetic hormone that increases milk production by 11–16% when injected into cows. In October 2008, Monsanto sold this business to Eli Lilly for $300 million plus additional considerations.

231. In 2009 Michael R. Taylor, former Monsanto VP for Public Policy became a Senior Advisor to the United States FDA Commissioner.

232. Monsanto is a member of the Washington D.C based Biotechnology Industry Organization (BIO), the world's largest biotechnology trade association, which provides "advocacy, business development, and communications services." Between 2010 and 2011 BIO spent a total of $16.43 million on lobbying.

233. In 2012, for $210 million, Monsanto purchased Precision Planting Inc., a company that produced computer hardware and software designed to enable farmers to increase yield and productivity through more precise planting.

234. In 2013, Monsanto purchased San Francisco-based Climate Corp for $930 million. Climate Corp. makes local weather forecasts for farmers based on data modeling and historical data and insures their forecasts.

235. As of November 2013, Monsanto was associated with 9 "active" Superfund sites and 32 "archived" sites in the United States according to the EPA's Superfund database.

236. In 2015 Monsanto was the world's biggest supplier of seeds, controlling 26% of the global seed market. Du Pont was second with 21%).

237. Until it ended production in 1977, Monsanto was the source of 99% of the polychlorinated biphenyls (PCBs) used by United States industry. They were sold under brand names such as *Aroclor* and *Santotherm*. The name *Santotherm* is still used for non-chlorinated products. PCB production was banned by the U.S. Congress in 1979 and by the *Stockholm Convention on Persistent Organic Pollutants* in 2001.

238. In the late 1960s, the Monsanto plant in Sauget, Illinois, was the nation's largest producer of PCBs, which remained in the water along Dead Creek there.

239. In 2003, Solutia and Monsanto agreed to pay $700 million to settle claims by over 20,000 Anniston residents who, in a 2002 lawsuit provided evidence showing that the local Monsanto factory knowingly discharged both mercury and PCB-laden waste into local creeks for over 40 years.

240. In 1969 Monsanto dumped 45 tons of PCBs into Snow Creek, a feeder for Choccolocco Creek, which supplies much of the drinking water for the area, and buried millions of pounds of PCB manufacturing wastes in open-pit landfills located on hillsides above the plant and surrounding neighborhoods.

241. Monsanto manufactured Agent Orange during the War in Southeast Asia.

242. Monsanto is the only manufacturer of white phosphorus for military use in the US.

243. Individuals associated with Monsanto policy often move from or to the Company from or to positions in the United States government.

244. Earle H. Harbison, Jr., once Central Intelligence Agency Deputy Director, served as President, Chief Operating Officer, and Director of Monsanto from 1986 to 1993.

245. Michael A. Friedman, MD—FDA deputy commissioner.

246. Linda J. Fisher was EPA assistant administrator, then Monsanto VP from 1995 to 2000 and then EPA deputy administrator.

247. Michael R. Taylor, assistant to the FDA commissioner, then attorney for King & Spalding, then FDA deputy commissioner for policy on food safety between 1991 and 1994, then became VP for Public Policy at Monsanto and then became Senior Advisor to the FDA Commissioner.

248. Supreme Court Justice Clarence Thomas who worked as an attorney for Monsanto in the 1970s.

249. Mickey Kantor, US trade representative became a Monsanto board member.

250. William D. Ruckelshaus, EPA Administrator, then acting Director of the Federal Bureau of Investigation, then Deputy Attorney General of the United States, then EPA administrator, then Monsanto Board member.

251. Current member of the Board of Directors for Monsanto are: Dwight M. "Mitch" Barns, Chief Executive Officer, Nielsen Holdings PLC; Gregory H. Boyce, Retired Chairman and Chief Executive Officer of Peabody Energy Corporation; David L. Chicoine, Professor of Economics at South Dakota State University; Janice L. Fields, Former President of McDonald's USA, LLC, a subsidiary of McDonald's Corporation, Hugh Grant, Chairman and Chief Executive Officer of Monsanto Company; Arthur H. Harper, Managing Partner of GenNx360 Capital Partners; Laura K. Ipsen, Senior Vice President & General Manager of Oracle Marketing Cloud, for Oracle

Corporation; Marcos M. Lutz, Chief Executive Officer of Cosan Ltd.; C. Steven McMillan, Retired Chairman of the Board and CEO of Sara Lee Corporation; Jon R. Moeller, Chief Financial Officer of The Procter & Gamble Company; George H. Poste, Ph. D., D.V.M., Chief Executive of Health Technology Networks; Robert J. Stevens, Retired Chairman of the Board and Chief Executive Officer of Lockheed Martin Corporation; Patricia D. Verduin, Chief Technology Officer of Colgate-Palmolive Company.

252. In an undated press release issued sometime in the late 2000s and posted at http://www.monsanto.com/newsviews/pages/agent-orange-background-monsanto-involvement.aspxhttp://www.monsanto.com/newsviews/pages/agent-orange-background-monsanto-involvement.aspx Monsanto admits that from 1965 to 1969, the former Monsanto Company manufactured Agent Orange for the U.S. military as a wartime government contractor.

253. According to the press release, the current Monsanto Company has maintained responsibility for this product since it was spun-off as a separate, independent agricultural company in 2002.

Defendant Valero Energy Corporation

254. Valero Energy Corporation is the successor in interest to Ultramar Diamond Shamrock which was the successor in interest to ULTRAMAR which was the successor in interest to Diamond Shamrock Corporation which was the successor in interest to Diamond Alkali which had acquired National Oil Products Company and NOPCO Chemical either or both of which were involved in the manufacture of dioxin contaminated phenoxy herbicides and the cacodylates to which

the Plaintiffs were exposed at Eglin Air Force Base, particularly, Site C–52A.

255. Valero Energy Corporation is a Fortune 500 international manufacturer and a marketer of transportation fuels, other petrochemical products, and power.

256. The company owns and operates 16 refineries throughout the United States, Canada, the United Kingdom, and the Caribbean with a combined throughput capacity of approximately 3 million barrels (480,000 m3) per day, 11 ethanol plants with a combined production capacity of 1.2 billion US gallons (4,500,000 m^3) per year, and a 50-megawatt wind farm.

257. Before the 2013 spinoff of CST Brands, Valero was one of the United States' largest retail operators with approximately 6,800 retail and branded wholesale outlets in the United States, Canada, the United Kingdom, and the Caribbean under the Valero, Diamond Shamrock, Shamrock, Ultramar, Beacon, and Texaco brands.

258. Valero was created on January 1, 1980, as a spinoff of Coastal States Gas Corporation. At the time, it was the largest corporate spinoff in U.S. history. Valero took over the natural gas operations of the LoVaca Gathering Company, a defunct subsidiary of Coastal States Gas. The name Valero comes from Misión San Antonio de Valero.

259. Valero acquired a small oil refinery in Corpus Christi, Texas in 1981, and began refining operations in 1984.

260. In 1997, Valero spun off its refinery and retail divisions into a separate company, which kept the Valero name. At the same time, the remaining divisions, which consisted primarily of natural gas operations, were acquired by the Pacific Gas and Electric Company. Later that year, the firm acquired Basis Petroleum, which left it with four refineries in Texas and

Louisiana. In 1998, it then acquired a Paulsboro, New Jersey refinery.

261. In 2000, Valero purchased the ExxonMobil refinery in Benicia, California, and interest in 350 Exxon-branded service stations in California, mainly in the San Francisco Bay Area. The company also began retailing gasoline under the Valero brand.

262. In June 2001, Valero acquired the Huntway Refining Company, along with two asphalt plants on the West Coast.

263. On December 31, 2001, Valero completed its acquisition of Ultramar Diamond Shamrock. The merger left Valero with over 4,700 Ultramar, Diamond Shamrock, and Beacon retail sites in the United States, Canada, and the Caribbean. With this acquisition, Valero also received ownership of Shamrock Logistics L.P., which was renamed Valero L.P. In 2006, the division was spun off as NuStar Energy. The acquisition also includes all past Diamond Shamrock assets, including the former Sigmor Petroleum assets.

264. On April 25, 2005, Valero agreed to buy Premcor, Inc., for $8 billion in cash and stock to become the largest U.S. refinery.

265. On June 30, 2005, Valero announced that it was beginning a two-year process of converting Diamond Shamrock stations to the Valero brand. In the next year, on May 5, 2008, Valero agreed to buy 72 Albertsons gas stations.

266. Valero laid off 500 employees at its refinery in Delaware City, Delaware on November 20, 2009, due to profitability concerns. It was reported the refinery had lost $1 million per day since the beginning of that year.

267. In 2013, Valero spun off its retail operations into a new publicly traded company, CST Brands. Under long-term supply agreements, Valero continues to supply fuel to over

7,400 retail locations, many of which use brand names formerly owned by Valero.

268. According to media reports, Valero Energy Corp. has been awarded multiple multimillion-dollar contracts by the U.S. Defense Energy Support Center (DESC) in order to provide fuel to Israel.

269. Valero Energy reached an agreement to sell the assets of its terminal operation in Delaware City to the wholly owned subsidiaries of PBF Energy, Delaware City Refining and Delaware Pipeline, for approximately $220 million in 2010.

270. Shortly after the divestiture of Delaware City, the company sold its refinery at the Port of Paulsboro to PBF Energy, as well. The sale concluded Valero's refinery ownership on the East Coast.

271. On August 1, 2011, Valero acquired the Pembroke Refinery from Chevron, as well as the marketing and logistics assets, for $730 million, excluding working capital, which was valued at approximately $1 billion. The Pembroke plant is one of the largest and most complex refineries in Western Europe with a total throughput capacity of 270,000 barrels (43,000 m3) per day and a Nelson complexity index rating of 11.8.

272. Owning a total of 15 refineries and 2.9 million barrels (460,000 m^3) per day of throughput capacity overall, Valero is the largest independent refinery in the world.

273. Valero also purchased ownership interest in four major pipelines and eleven fuel terminals, a 14,000-barrel (2,200 m^3)-per-day aviation fuel business, and a network of more than 1,000 Texaco-branded wholesale sites, which is the largest branded dealer network in the United Kingdom and the second largest in Ireland. Valero has continued with the Texaco brand in these markets.

274. Valero retails gasoline branded as Valero, Shamrock, Diamond Shamrock, Ultramar, Beacon, and Total, the last under license from Total S.A. The Beacon and Shamrock brands are used by retailers as a low-cost alternative to the premium Valero brand. The Shamrock brand is based on the former Shamrock Oil and Gas Company, which merged with Diamond Alkali in 1967 to form Diamond Shamrock. The name Ultramar, while being eliminated in the United States, continued as Valero's brand name in Canada.

275. In 2013, Valero completed the spinoff of the retail operations as CST Brands. Valero no longer owns retail operations using the Valero, Diamond Shamrock, Shamrock, Beacon, Ultramar, or Texaco names, but Valero continues to supply fuel.

276. Valero issues its own private label credit cards for its stations through its credit card-only subsidiary, DSRM National Bank. The initials stand for "Diamond Shamrock Refining & Marketing", the unit of Diamond Shamrock which created it before being purchased by Valero.

Ultramar

277. Ultramar is an Eastern Canadian gas and home fuel retailer, with head office in Montreal. It used to have oil refining and marketing operations known as *Golden Eagle* or *Aigle d'or*. It was a subsidiary of Valero Energy Corporation, an American company that acquired Ultramar's parent company, Ultramar Diamond Shamrock.

278. Ultramar has been a trademark of CST Brands Inc. after it was spun off from Valero in May 2013.

279. Today, Ultramar operates gas stations and home fuel delivery in Ontario, Quebec, and Atlantic Canada. It also operates ValuMax, a cashback program for fuel purchases.

280. The Company was formed by Ultramar PLC, a British Group, who established operations in Canada in 1961 Its refinery in Lévis, Quebec was built ten years later.

281. From 1979 to 1996, Ultramar grew by acquiring stations from several other companies, including Canadian fuel marketers Texaco Canada, Gulf Canada, Sergaz, Sunoco and Spur.

282. In 1981, Ultramar acquired Hanford, California-based Beacon Oil Company. It retained the name.

283. In 1990, Ultramar acquired the Dartmouth, Nova Scotia refinery with the purchase of other assets of Texaco Canada.

284. In 1991, Lasmo, a British oil company, bought Ultramar PLC and in 1992 Lasmo spun off the North American refining and marketing operations which became known as *Ultramar Corporation*.

285. In 1994, Ultramar acquired Sergaz which had been founded in 1971 by André Ducharme and Sunoco's Quebec gas stations.

286. In 1996, Ultramar Corporation merged with Diamond Shamrock to form *Ultramar Diamond Shamrock*.

287. In 1997, the Sunoco name was withdrawn from Quebec, and all stations converted to the Ultramar brand.

288. The refinery in Lévis was renamed in honour of retired Ultramar Diamond Shamrock CEO Jean Gaulin in 2001.

289. On December 31, 2001, Valero Energy Corporation completed its acquisition of Ultramar Diamond Shamrock.

290. On May 1, 2013, Ultramar was spun off from Valero Energy Corporation to become a trademark of CST Brands Inc.

Diamond Shamrock

291. Diamond Shamrock Corp. or Diamond Shamrock Refining and Marketing was an oil refinery and gas station company in the United States.

292. The origins of Diamond Shamrock can be traced back to three foundation companies: Diamond Alkali, Shamrock Oil and Gas, and Sigmor Corporation.

293. **Diamond Alkali** was founded in 1910 by a group of glass manufacturers in Pittsburgh, Pennsylvania to produce soda ash, a key ingredient in glass production. A factory was built in Painesville, Ohio in 1912 to produce soda ash.

294. After World War II, in 1946, a new plant was built to produce Chlorine and Caustic soda in Deer Park, Houston, Texas.

295. In 1948 the company moved its headquarters from Pittsburgh to Cleveland, Ohio.

296. During the 1950s a third plant was constructed in Muscle Shoals, Alabama, helping the company continue to enlarge its range of products, expanding to produce plastics and chemicals for agriculture.

297. During the 1960s A facility was opened in Delaware City, Delaware and additional chemical companies were purchased, including Chemical Process Company of Redwood City, California and the Nopco Chemical Company of New Jersey.

298. Between 1951 and 1969, Diamond Alkali in Newark produced approximately 700,000 US gallons (2,600,000 l; 580,000 imp gal) of the herbicide Agent Orange.

299. A number of accidents occurred at the plant which seriously contaminated the *Ironbound* section of Newark, New Jersey and the nearby Passaic River with toxic chemicals.

300. The *Ironbound* plant produced the phenoxy herbicides most contaminated with 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin

301. The Company frequently dumped "bad" batches of phenoxy herbicides into the nearby Passaic River.

302. In 1967 Diamond Alkali merged with Shamrock Oil and Gas of Amarillo. At the time of the merger the company produced about 20% oil and gas and 80% chemicals.

303. From 1968 through the mid-1980s the business name "Diamond Shamrock" was associated with the oil and gas and chemical businesses originating from the merger in 1967 of Diamond Alkali Company, a chemical business headquartered in Cleveland, Ohio, with The Shamrock Oil & Gas Corporation, headquartered in Amarillo, Texas.

304. The surviving corporation was named Diamond Shamrock Corporation and continued to operate two core businesses: chemicals and oil and gas.

305. In 1978 Diamond Shamrock moved its headquarters to Dallas.

306. By 1980 Diamond Shamrock had about 12,400 employees in thirty-seven countries.

307. In 1983, the businesses were reorganized to create separate business corporations to own and hold the different core businesses, chemicals and oil and gas, as well as others.

308. On July 19, 1983, a new corporation was formed to become the ultimate parent holding company and as soon as the reorganization was completed, this new corporation was renamed Diamond Shamrock Corporation.

309. The "New" Diamond Shamrock Corporation became the ultimate parent of all the Diamond Shamrock subsidiaries and also became the publicly traded entity.

310. On August 30, 1983, Diamond Shamrock Refining and Marketing Company was incorporated as a Delaware corporation and became the owner of substantially the same assets is held at the time of the affidavit in 1993.

311. Shortly thereafter, Diamond Chemicals Company, the old Diamond Shamrock Corporation, changed its name to Diamond Shamrock Chemicals Company.

312. After the reorganization in 1983, the structure of the "entity" was Diamond Shamrock Corporation, the new holding company organized in 1983, acting as the parent of Diamond Shamrock Chemicals Company, the old Diamond Shamrock Corporation, and the parent of Diamond Shamrock Refining and Marketing Company.

313. On January 26, 1984, Diamond Shamrock Chemicals Company assigned all of the stock of DSRMC to Diamond Shamrock International Energy Company, a wholly owned subsidiary of the new Diamond Shamrock Corporation.

314. The new Diamond Shamrock Corporation was the main holding company with two direct subsidiaries: Diamond Shamrock International Energy Company which owned and conducted the energy businesses; and Diamond Shamrock Chemicals Company which owned and conducted various chemicals businesses. DSRMC was a wholly-owned subsidiary of Diamond Shamrock International Energy Company.

315. The former *Ironbound* plant property and adjoining portions of the Lower Passaic River were declared a Superfund site in 1984.

316. In 1986, the Diamond Shamrock Corporation agreed to pay $150,000 for a canvas tarpaulin to cover 3 acres (12,000 m^2) of the contaminated area.

317. On September 4, 1986, Diamond Shamrock Chemicals Company sold its chemical businesses to Occidental Petroleum Corporation and certain of its subsidiaries.

318. Remediation efforts at Diamond Alkali began in 2000 and ecological investigation, dredging, and other cleanup activities were still underway as of 2012.

319. **Shamrock Oil and Gas** was founded on August 9, 1929 by John Sheerin who named the company after the symbol of his country of origin. The company was financed by the Fownes family of Pennsylvania and headquartered in Amarillo.

320. In 1933 Shamrock built its first refinery and its first gas station, both in Sunray, Moore County, Texas. James Harold Dunn joined the company in 1938 as a vice president and general manager, having previously been an engineer at the Lone Star Gas Corporation.

321. During 1939–1940 Lone Star and Shamrock cooperated on the construction of a plant at Murchison in Henderson County whose aim was to recycle natural gas.

322. In 1943 the company paid its first dividend, and by 1944 the company was listed on the NYSE. In 1959 Shamrock opened its first catalytic cracking unit in Sunray.

323. In 1960 Shamrock purchased a large number of gas stations from the chain of Sigmor.

324. **Sigmor** was founded by Thomas E. Turner, founder of TETCO Inc. (the Sigmor brand name was the initials of Sigfried Moore, Turner's former employer) which merged with Shamrock Oil and Gas in 1960 forming Sigmor Shamrock which was merged into Diamond Shamrock in 1982. Turner later repurchased Mission Petroleum Carriers when it was acquired by Diamond Shamrock and the business portfolio of the former National Convenience Stores, which was acquired by Diamond Shamrock in November 1995.

325. Sigmor had been operated during the 1930s and 1940s by Sigfried (Sig) Moore.

326. In 1943, Moore loaned Thomas E. Turner, an employee, money to launch his own business. Turner decided to use the name Sigmor for the chain of stores he established during the 40s and 50s.

327. In 1952 Sigmor was incorporated.

328. In 1959 a restructuring took place which allowed each separate gas station to incorporate separately.

329. In 1960 most of the chain was purchased by Shamrock and then leased back to Turner, who continued to lead the company.

330. In 1978 Sigmor purchased its stations back from Diamond Shamrock and continued to market DS products. By 1983 Sigmor was one of the largest independent service-station chains in the USA.

331. The merger gave Diamond Shamrock 600 retail outlets, plus the Three Rivers oil refinery which was built by Sigmor during the 1970s.

332. In 1987 The Diamond Shamrock Refining and Marketing Company severed ties with Diamond Shamrock Corporation, which was the parent company, and became independent with its headquarters in San Antonio.

333. As part of the reorganization, Diamond Shamrock Corporation became Maxus Energy Corporation, severing all legal ties to the Diamond Shamrock Refining and Marketing Company.

334. In 1990 Diamond Shamrock Refining and Marketing Company shortened its name to Diamond Shamrock, Incorporated.

335. On December 3, 1986 T. Boone Pickens offered to buy Diamond Shamrock for $2 billion.

336. In 1988 the company's annual refinery sales were $1.8 billion. Diamond Shamrock owned and operated 529 stores. 423 of

them were in Texas, with 94 of them in Greater Houston. The company owned an additional 64 stores in Colorado.

337. In 1988 the company bought from investor F. Philip Handy 80 Tenneco gasoline stations, with 30 of them in Houston.

338. In 1995 Diamond Shamrock had 2,000 stores, with most of them in Texas, Colorado, New Mexico, and Louisiana. Of them, over 170 stores were in Houston. That year, Diamond Shamrock bought the National Convenience Stores Stop N Go chain for $260 million. The plans called for the combined company to be headquartered in San Antonio. The combined company was to have two refineries in Texas, 11,000 employees, and 2,600 stores.

339. In 1996, Canadian company Ultramar bought Diamond Shamrock for $1.96 billion in stock and assumed debt. The combined company was renamed "Ultramar Diamond Shamrock".

340. Valero Energy Corporation acquired Ultramar Diamond Shamrock in 2001.

Defendant Occidental Petroleum

341. Occidental Petroleum Corporation (Oxy) is a multinational oil and gas exploration and production company with operations in the United States, the Middle East, and Latin America, headquartered in Houston, Texas.

342. Occidental Petroleum Corporation conducts its business through two major subsidiaries, Occidental Oil and Gas Corporation and Occidental Chemical Corporation.

343. Occidental Oil and Gas, representing the larger of the two business segments, operates in the United States, the Middle East, and Latin America. Domestically, the company ranks as the largest natural gas producer in California and the largest oil producer in Texas.

344. Overseas assets in oil and gas are located in Oman, Qatar, Yemen, Columbia, Ecuador, Russia, and Pakistan.

345. Occidental Chemical manufactures vinyls, chlorine, and caustic soda, relying on 24 manufacturing sites in the United States, two facilities in Canada, and one plant in Chile.

346. Occidental, the governing entity for the oil and gas and chemical operations, derives roughly $7.5 billion in annual sales from its oil and gas interests and $3.6 billion from its chemicals business.

347. As of December 31, 2016, Occidental had 2.406 billion barrels of oil equivalent (1.472×10^{10} GJ) of oil equivalent net proved reserves, of which 56% was petroleum, 17% was natural gas liquids, and 27% was natural gas.

348. In 2016, the company had production of 630 thousand barrels of oil equivalent (3,900,000 GJ) per day.

349. Occidental Chemical Corporation (OxyChem), a wholly owned subsidiary of Occidental Petroleum, based in Dallas, is a leading chemical manufacturer.

350. Among its interests are basic chemicals, vinyls and performance chemical products. Oxy's wholly owned chemical subsidiaries include Occidental Chemical Corporation (OxyChem), OxyVinyls, and INDSPEC Chemical Corporation.

351. OxyChem manufactures polyvinyl chloride (PVC) resins, chlorine and caustic soda used in plastics, pharmaceuticals and water treatment chemicals.

352. Other products manufactured by the company include caustic potash, chlorinated organics, sodium silicates, chlorinated isocyanurates and calcium chloride.

353. OxyChem has manufacturing facilities in the United States, Canada and Chile.

354. In a joint venture with Church & Dwight, OxyChem owns Armand Products Company, which sells potassium carbonate and potassium bicarbonate.

355. In October 2013, OxyChem and Mexichem announced the formation of a 50/50 joint venture, Ingleside Ethylene LLC, to build a 1.2-billion-pound per year capacity ethylene cracker at the OxyChem plant in Ingleside, Texas, along with pipelines and storage at Markham, Texas.

356. The most successful of Oxy's operations during the mid-to late 1980s was its chemical branch, Occidental Chemical (Oxychem).

357. The chemical operations were built largely through the acquisitions of other companies.

358. Occidental purchased holdings from Diamond Shamrock Chemicals in 1986 and from Du Pont and Shell Chemical in 1987, among others.

359. In the five-year period from 1983 through 1987, Oxychem almost doubled its sales to nearly $3 billion.

360. In 1988 Occidental, spending $2.2 billion to purchase Cain Chemical, moved up to become the nation's sixth largest chemical producer, with sales accounting for almost 25 percent of Oxy's total.

361. Cain Chemical then became known as Oxy Petrochemicals Inc.

362. In February 1988 Oxy was found liable for cleaning up the toxic wastes at the country's most infamous landfill, Love Canal in Niagara Falls, New York. After eight years of deliberations, a federal judge ruled that Occidental was responsible for the improper disposal by Hooker Chemical of more than 21,000 tons of chemicals on the site, during the 1940s and 1950s.

363. Before the ruling, Oxy had paid $20 million in damages to 1,300 former Love Canal residents, but nothing toward the cleanup of the site. Total cleanup costs were expected to exceed $100 million.

364. In September 2014 Occidental Petroleum Corp.'s chemical subsidiary has agreed to pay New Jersey $190 million to resolve liability for damages to the Passaic River, as the successor to Diamond Shamrock Chemicals Co., which operated a facility that polluted the river.

365. Occidental Petroleum — formerly based in California — relocated its corporate headquarters to Houston earlier this year. The settlement is with Occidental Chemical Corp., a wholly owned subsidiary of Occidental Petroleum.

366. In 2011, a court ruled that Occidental was liable for certain cleanup costs because of its role as successor to Diamond Shamrock Chemicals Co. New Jersey had accused that company of dumping carcinogens known as dioxins into the Passaic River for decades from its Newark plant which manufactured pesticides and herbicides — including Agent Orange— from the 1940s until the 1960s, Hoffman's office said in a statement.

367. New Jersey initiated litigation against Occidental and other companies affiliated with the Diamond Shamrock site eight years ago.

368. Occidental Petroleum sought reimbursement from Maxus Energy Corp. which it claims is financially responsible.

369. Occidental said it purchased Diamond Shamrock Chemical's stock from Maxus in 1986 — 17 years after the plant closed — and said the alleged misconduct occurred while Maxus companies owned and operated the site.

370. Maxus Energy and New-Jersey based Tierra Solutions, Inc., are affiliates of YPF SA, an Argentinean energy company.

Defendant DowDuPont

371. Defendant DowDuPont Inc. is an American company formed after the merger of Dow Chemical and DuPont on August 31, 2017.

372. DowDuPont is the world's largest chemical company in terms of sales.

373. Within 18 months the company was split into three publicly-traded companies with focuses on the following: agriculture, materials science, and specialty products.

374. The agriculture division is named Corteva Agriscience, the materials science spin-off is named Dow Inc., and the specialty products division is named DuPont.

375. The merger has been reported to be worth an estimated $130 billion.

376. DowDuPont ranked No. 47 in the 2018 Fortune 500 list of the largest United States corporations by total revenue.

Defendant The Dow Chemical Company

377. The Dow Chemical Company, commonly referred to as Dow, is an American multinational chemical corporation which manufactured dioxin contaminated phenoxy herbicides to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A.

378. As of 2014, Dow ranked third in chemical production after BASF and Sinopec, and as of 2015, was the third largest chemical company in the world by revenue after Sinopec and BASF.

379. Dow was also the largest chlorine producer in the world.

380. Dow was ranked as the world's largest plastics manufacturer during 2008.

381. Dow was the largest manufacturer of polyalkylene glycols in 2013.

382. Dow employs approximately 53,000 people worldwide.

383. Dow sales in 2014 sales totaled approximately $58.2 billion.

384. Most of Dow sales are to other industries rather than end users, although Dow sells directly to end users primarily in the human and animal health, agriculture, and consumer products markets.

385. Dow was founded in 1897 by Canadian-born chemist Herbert Henry Dow, who invented a new method of extracting the bromine that was trapped underground in brine at Midland, Michigan.

386. Dow originally sold only bleach and potassium bromide, achieving a bleach output of 72 tons a day in 1902.

387. In 1905, German bromide producers began dumping bromides at low cost in the U.S. in an effort to prevent Dow from expanding its sales of bromides in Europe. Instead of competing head-on with the German producers, Dow bought the cheap German-made bromides and shipped them back to Europe, undercutting his German competitors.

388. Within twenty years, Dow had become a major producer of agricultural chemicals, elemental chlorine, phenol and other dyestuffs, as well as magnesium metal.

389. During World War I, Dow supplied many war materials which the U.S. had previously imported from Germany. Dow produced magnesium for incendiary flares, monochlorobenzene and phenol for explosives, and bromine for medicines and tear gas. By 1918, 90% of Dow production was geared towards the

war effort and Dow created the diamond logo that is still used by the company.

390. After the war, Dow continued research in magnesium, and developed refined automobile pistons that were used heavily in racing vehicles. The 1921 winner of the Indianapolis 500 used the Dow metal pistons in his vehicle.

391. In the 1930s, Dow began producing plastic resins. The first plastic products were ethylcellulose, made in 1935, and polystyrene, made in 1937.

392. From 1940 to 1941, Dow built its first plant at Freeport, Texas, in order to produce magnesium extracted from seawater rather than underground brine, marking the first time that humans had mined the ocean for metal.

393. The Freeport plant is one of the largest integrated chemical manufacturing sites in the world.

394. In 1942, Dow began its foreign expansion with the formation of Dow Chemical of Canada in Sarnia, Ontario to produce styrene for use in styrene-butadiene synthetic rubber. The Canadian government invited Dow to build a plant there and Dow then built a polystyrene plant in 1947. Up to the early 1990s, the Chemical Valley site contained numerous plants.

395. During WWII, Dow and Corning began their joint venture, Dow Corning, to produce silicones for military and, later, civilian use.

396. Dow found its first overseas subsidiary in Japan in 1952 as Asahi-Dow Limited.

397. Dow opened a consumer products division beginning with Saran wrap in 1953.

398. Dow sales exceeded $1 billion in 1964, $2 billion in 1971, and $10 billion in 1980.

399. From 1951 to 1975, Dow managed the Rocky Flats nuclear weapons production facility that produced plutonium triggers for hydrogen bombs near Denver, Colorado. Rocky Flats is now the Rocky Flats National Wildlife Refuge.

400. During the Vietnam War, Dow was one of several manufacturers who began producing the napalm B compound under government contract from 1965 at its Torrance, California plant. After experiencing protests and negative publicity, the other suppliers discontinued manufacturing the product, leaving Dow as the sole provider. Dow continued to manufacture napalm B until 1969.

401. Dow manufactured Agent Orange, a chemical defoliant contaminated with dioxin in New Plymouth, New Zealand and in the United States for use by the British military during the Malayan Emergency and the U.S. military during the Vietnam War.

402. In the early 1990s, Dow reorganized; moving from geographically based regional presidents reporting directly to the overall company president and CEO to a new organization which combined the same businesses from different sites all over the world.

403. In 1999, Dow agreed to purchase Union Carbide Corp. for $9.3 billion in stock. At the time, the combined company was the second largest chemical company, behind DuPont.

404. Union Carbide became a subsidiary of Dow in 2001.

405. Until the late 1970s, Dow produced DBCP (1,2-dibromo-3-chloropropane), a soil fumigant, and nematicide, sold under the product names *Nemagon* and *Fumazone*.

406. In November 2006, areas along the Tittabawassee River which runs within yards of Dow's main plant in Midland, Michigan were found to contain elevated levels of dioxin. In July 2007,

Dow reached an agreement with the Environmental Protection Agency to remove 50,000 cubic yards (38,000 m^3) of sediment from three areas of the riverbed and levees of the river, In November 2008, the Dow Chemical Company along with the United States Environmental Protection Agency and Michigan Department of Environmental Quality agreed to establish a Superfund to address dioxin cleanup of the Tittabawassee River, Saginaw River and Saginaw Bay.

407. On August 31, 2006, Dow announced that it planned to close facilities at five locations: Sarnia, Ontario, Dow's first manufacturing site in Canada; one plant at its site in Barry (South Wales), a triple string STR styrene polymer production unit; one plant at its site in Porto Marghera (Venice), Italy; and two plants at its site in Fort Saskatchewan, Alberta, Canada.

408. On November 2, 2006, Dow and Izolan, the leading Russian producer of polyurethane systems, formed the joint venture Dow-Izolan and built a manufacturing facility in the city of Vladimir.

409. In 2006, Dow formed the Business Process Service Center (BPSC).

410. In 2007, Dow announced it planned to exit the automotive sealers business in 2008 or 2009.

411. In 2007, Dow announced the formation of a joint venture, K-Dow, with Petrochemical Industries Co. (PIC), a subsidiary of Kuwait Petroleum Corporation. In exchange for $9.5 billion, the agreement included Dow selling 50% of its interest in five global businesses: polyethylene, polypropylene and polycarbonate plastics, and ethylenamines and ethanolamines. The agreement was terminated by PIC on December 28, 2008, citing the global recession that began in the latter half of 2008.

412. On July 10, 2008, Dow agreed to purchase all of the common equity interest of Rohm and Haas Co. for $15.4 billion. The buyout was to be financed with equity investments of $3 billion by Berkshire Hathaway Inc. and $1 billion by the Kuwait Investment Authority. However, on January 26, 2009 the company informed Rohm and Haas that it would be unable to complete the transaction by the agreed upon deadline, citing a deteriorated credit market and the collapse of the K-Dow Petrochemical deal as reasons for failing to timely close the merger.

413. After negotiating the sale of preferred stock with Rohm and Hass' two largest stockholders and extending their one-year bridge loan an additional year, the company purchased Rohm and Haas for $15 billion on March 9, 2009. The transaction to purchase the outstanding interest of Rohm and Haas closed on April 1, 2009.

414. On December 8, 2008, Dow announced that due to the 2008 economic crisis, it would accelerate job cuts resulting from its reorganization; closing 20 facilities, temporarily idling 180 plants, and eliminating 5,000 full-time jobs, about 11% of its workforce, and 6,000 contractor positions.

415. On February 12, 2009, the company cut its quarterly dividend for the first time in the dividend's 97-year history.

416. In 2011, Dow and Saudi Arabian Oil Company (Saudi Aramco) developed the joint venture, Sadara Chemical Company, worth $20 billion with initial plants to come on stream in 2015.

417. In February 2013 a federal court rejected two tax shelter transactions entered into by Dow that created approximately $1 billion in tax deductions between 1993-2003. The Court termed the transactions "schemes that were designed to exploit perceived weaknesses in the tax code and not designed for legitimate business reasons." The schemes were created by

Goldman Sachs and the law firm of King & Spalding and involved creating a partnership that Dow operated out of its European headquarters in Switzerland.

418. In the fourth quarter of 2014, Dow announced it would give further support to its end-market orientation and increase its alignment to Dow's key value chains, ethylene and propylene.

419. Dow's new propane dehydrogenation (PDH) facility in Freeport, Texas, is expected to come online in 2015, with a first 750000 metric tonne per year unit.

420. On March 27, 2015, Dow and Olin Corporation announced that the boards of directors of both companies unanimously approved a definitive agreement under which Dow will separate a significant portion of its chlorine business and merge that new entity with Olin in a transaction that will create an industry leader, with revenues approaching $7 billion. Olin, the new partnership, became the largest chlorine producer in the world.

421. On December 11, 2015, Dow announced that it had reached a deal to acquire Corning Incorporated's stake in their joint venture Dow Corning for $4.8 billion in cash and a roughly 40% stake in Hemlock Semiconductor Corporation.

Defendant Ashland, Inc.

422. Ashland Inc. is the successor in interest to Hercules, Inc. which manufactured dioxin contaminated phenoxy herbicides and the cacodylates to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A.

423. Ashland Inc. is an American Fortune 500 company which operates in more than 100 countries. Headquartered in Lexington, Kentucky, in the United States, the company traces its roots back to Ashland, Kentucky (for which it is named).

424. Ashland was founded in 1924 as Ashland Refining Company of Ashland, Kentucky, by Paul G. Blazer who had been hired by J. Fred Miles of the Swiss Oil Company of Lexington, Kentucky. to locate, purchase and be general manager of a refinery in eastern Kentucky.

425. With funds supplied by Swiss Oil, Blazer arranged to buy, at a price of $212,500, the 1,000 barrel per day refinery of Great Eastern Refining Company built in 1916 in northeastern Kentucky, where the Big Sandy River joins the Ohio River. It had been owned by coal operators out of Huntington, West Virginia.

426. On February 2, 1924, Blazer and three Swiss Oil executives incorporated Ashland Refining Company, with a paid in capital of $250,000. They took over the operations of the Catlettsburg Refinery which had twenty-five employees who were working seven days per week and twelve hours per day. Blazer moved from Lexington to Ashland. The only member of the Swiss Oil organization to come to Ashland with Blazer was Ashland Refining Company's first treasurer, William Waples.

427. Ashland's refinery operations were successful from the very first month. Wages were increased and the hours of work were reduced. After making repairs and purchasing some new equipment, the refinery soon had output of 500,000 barrels a year and sales of $1,300,000. Within a few years, Ashland Refining Company, the small subsidiary of Swiss Oil formed solely to facilitate the purchase of some eastern Kentucky oil producing properties, began showing larger earnings than the parent company.

428. Ashland Refining Co. grew rapidly through both internal expansion and acquisitions including Union Gas and Oil Company (1925), Tri-State Refining Company (1930), and Cumberland Pipeline Company (1931).

429. By 1933, Ashland Refining Company owned more than 1,000 wells, 800 miles of pipelines, bulk distribution plants in twelve cities, service stations, river transportation terminals and river equipment.

430. In 1936, the company ownership changed from Swiss Oil to the Ashland Oil and Refining Company shareholder group in Ashland, Kentucky. Blazer was appointed chief executive officer of the company. Blazer's success as manager was recognized by major stockholders. They gave him the power to run Ashland as his own operation, though at no point during his tenure as chief executive officer (1936–1957) did he own a controlling interest in the company.

431. Two of his early changes were offering employees' sick leave with full pay and in 1947 the introduction of an employee profit-sharing plan, which made Ashland Oil and Refining Company one of the first companies in the region to offer such benefits. Blazer also started the well-known tradition of having local Greenup County educator and internationally acclaimed author Jesse Stuart open each annual meeting with a story, a poem, or a bit of humor. He also was a pastor at his local church.

432. After World War II, Ashland teamed with Sperry Corporation to develop the introduction of radar on commercial river vessels and teamed with various shipyards to develop the integrated tow. The "jumbo" tank barge of 195 ft. by 35 ft. became the industry standard.

433. Ashland grew from a small Eastern Kentucky refinery into a Forbes 500 company by relying on barges to bring in crude oil and deliver refined products to independent marketers.

434. Ashland operated the nation's largest inland towing fleet and in 1953 the Port of Huntington-Tristate exceeded Pittsburgh as the busiest port on the Ohio River.

435. Following World War II, Ashland Oil & Refining Company acquired Allied Oil Company (1948), Cleveland and Lakeland Tankers (1948), Aetna Oil Company (1949), Freedom-Valvoline Company (1950), Frontier Oil of Buffalo (1950) and National Refining Company (1950).

436. By 1953, Ashland Oil and Refining Company had 3,518 miles of crude oil pipelines, 252 miles of product lines, six refineries processing an average of 124,000 barrels a day, operated nine tow boats on the inland waterways, and owned over 100 barges. Although still involved as chairman of Ashland's Finance Committee and Executive Committee, Blazer stepped down as chief executive officer in 1957.

437. Louisville Refining Company was purchased in 1959. United Carbon was purchased in 1963.

438. In 1966, Ashland Oil and Refinery Company Inc. sales had grown to $699,000,000.

439. Diversification continued with the purchase of Warren Brothers in 1966, which later was to become Ashland Paving and Construction.

440. In 1967 the company purchased ADM Chemical Group.

441. In 1969, the company reorganized to form Ashland Petroleum, as well as entering into a joint venture in Coal mining under the name Arch Mineral.

442. In the 1980s and early part of the 1990s, Ashland continued to expand, buying The Permian Corporation and merging it with Scurlock Oil Company in 1991 to form a subsidiary known as Scurlock Permian Corporation.

443. In 1992, most of Unocal's chemical distribution business was acquired, making Ashland the top chemical distributor in North America. At this time, the Industrial Chemicals & Solvents (IC&S) division was established.

444. The company's name was changed from "Ashland Oil" to the present "Ashland Inc." in 1995.

445. In 1998, the petroleum division merged with Marathon Oil to form Marathon Ashland Petroleum, LLC (MAP).

446. In 1999, Ashland agreed to sell its Scurlock Permian subsidiary to Plains All American Pipeline.

447. In 2005, when Ashland sold its shares of the Marathon Ashland Petroleum joint venture to Marathon Oil, effectively dissolving the remnants of their petroleum division. After the sale, the company was no longer involved in the refining or marketing of fuels. The original oil refinery in Catlettsburg, Kentucky, is still in operation today and is owned and operated by Marathon.

448. In 2006, Ashland sold APAC (the paving and construction division) to the Oldcastle Materials subsidiary of Oldcastle Inc. of Dublin, Ireland.

449. Ashland purchased the adhesive and emulsions divisions of Air Products & Chemicals in 2008.

450. Ashland announced plans to acquire Hercules Inc. on July 11, 2008, for $3.3B. On November 13, 2008, the transaction was completed.

451. In July 2010 Ashland merged its foundry chemicals activity with Süd-Chemie of Munich, Germany, to form ASK Chemicals L.P. with headquarters in Dublin, Ohio.

452. In November 2010 Ashland announced plans to sell its distribution business known as Ashland Distribution to TPG Capital for $930 Million. The Ashland Distribution business had been a part of Ashland since 1969. With revenues of $3.4 billion, the Ashland Distribution business had approximately 2,000 employees across North America and Europe and entered the China plastics market in 2009. The sale was

finalized April 1, 2011, with a final sale price of US$979 million. The new privately held company is named Nexeo Solutions.

453. In May 2011 Ashland announced that it had bought the privately-owned company International Specialty Products Inc. (ISP) for $3.2 billion. ISP is a supplier of specialty chemicals and performance-enhancing products for consumer and industrial markets.

454. Valvoline Instant Oil Change moved into the Southern California area in March 2012 when the EZ Lube franchise was purchased by Henley Enterprises, VIOC's largest franchisee.

455. In 2014, Ashland Water Technologies was sold to a private equity buyer.

Defendant Hercules, Inc.

456. During the War in Southeast Asia it manufactured dioxin contaminated phenoxy herbicides and the cacodylates to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A.

457. Until it was acquired by Ashland Inc. in 2008, Hercules, Inc. manufactured specialty chemicals and materials used in the pulp and paper, food, pharmaceuticals, personal care, paints and adhesives, and construction materials industries.

458. Hercules Powder Company was formed in 1882 by DuPont and Laflin & Rand Powder Company to finance construction of a dynamite plant on land adjacent to San Francisco Bay owned by DuPont subsidiary California Powder Works.

459. Hercules Inc. was a manufacturer of chemicals and munitions based in Wilmington, Delaware, established in 1912 by T.W. Bacchus as the Hercules Powder Company.

460. The Hercules Powder Company was one of the companies created from the breakup of the E.I. du Pont de Nemours "powder trust" in 1911 as the result of a ruling by the Supreme Court of the United States decision.

461. Hercules, Inc., was one of the major producers of smokeless powder for warfare in the United States during the 20th century.

462. At the time of its spin-off, the DuPont Corp. retained the processes and patents for the production of "single-base" nitrocellulose gun powders, whereas Hercules, Inc., was given the patents and processes for the production of "double-base" gun powders that combined nitrocellulose and nitroglycerine.

463. Hercules Powder Company ranked 65th among United States corporations in the value of World War II military production contracts.

464. Richard F. Heck, recipient of the 2010 Nobel Prize in Chemistry, gained experience with transition metal chemistry while working at Hercules in 1957.

465. Beginning in 1959, Hercules, Inc., became a primary producer of large solid-fuel rocket motors these, especially for the U.S. Department of Defense, U.S. Air Force, U.S. Navy, and U.S. Army, and to a lesser degree for the civilian National Aeronautics and Space Administration (NASA). One of its major solid-fuel rocket products was the third-stage engine for the three-stage solid-fueled Minuteman intercontinental ballistic missile (ICBM) for the U.S. Air Force, of which 1,000 were made and deployed at Air Force Bases in several northern states during the 1960s.

466. Hercules, Inc., also produced the solid-fueled rocket motors for the two-stage Polaris missile system of intermediate-range ballistic missiles (IRBMs) for the U.S. Navy.

467. During the 1960s, Hercules, Inc., also made solid-fuel rocket motors for hundreds of the U.S. Army's Honest John missile, a mobile tactical missile for carrying tactical nuclear weapons for U.S. Army divisions mostly deployed with the U.S. Seventh Army in West Germany as part of the American commitment to NATO to defend Western Europe against aggression from the Warsaw Pact, using nuclear weapons on Eastern Europe, if necessary.

468. For space exploration and satellite launches by the U.S. Air Force and NASA, Hercules, Inc., developed and manufactured the two large, strap-on solid-fueled booster rockets for the otherwise liquid-fueled, and huge, Titan III and Titan IV rockets. These strap-on rockets were used on the Titan IIIC, Titan IIID, and Titan IIIE rockets, and on all of the Titan IV rockets that were ever produced.

469. In 1995 the aerospace division of Hercules, including its solid motor line, was acquired by the American defense contractor ATK.

470. After the end of production and firing of NASA's huge Saturn IB and Saturn V rockets, the Titan IV was the largest and heaviest unmanned rocket available anywhere in the world. The Titan IV-Centaur was used for special launches of heavy space probes into the Solar System, such as the Cassini-Huygens mission to Saturn which was launched in 1997. The Titan IV is no longer manufactured, and the last one of these was fired during a launch in October 2005.

471. By the end of the 1990s, Hercules Inc. had sold off a significant number of its divisions.

472. In its later years, Hercules, Inc., manufactured and marketed worldwide specialist chemicals that were used in a wide variety of industrial, home, and office markets, and it and had over 4,500 employees. Hercules was composed of two major

divisions: the Paper Technologies and Ventures (PTV) division and the Aqualon division. In 2005, the former accounted for 49% of its sales and 35% of its operating profits, with the latter producing 37% and 68% respectively.

473. On November 13, 2008, Hercules, Inc. was acquired by Ashland Inc. for $3.3B.

Defendant SCA *Compagnie Générale des Établissements Michelin*

474. *SCA Compagnie Générale des Établissements Michelin*, is the successor in interest to Uniroyal Inc., which manufactured dioxin contaminated phenoxy herbicides and the cacodylates or components thereof or constituent chemical raw materials therefore to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A.

475. Michelin s one of the three largest tire manufacturers in the world along with Bridgestone and Goodyear. In addition to the Michelin brand, it also owns the BFGoodrich, Kleber, Tigar, Riken, Kormoran and Uniroyal (in North America) tire brands.

476. Michelin manufactures tires for space shuttles, aircraft, automobiles, heavy equipment, motorcycles, and bicycles. In 2012, the Group produced 166 million tires at 69 facilities located in 18 countries.

477. In 1889 two brothers, Édouard Michelin and André Michelin incorporated the Company on 28 May 1889.

478. In 1891 Michelin took out its first patent for a removable pneumatic tire which was used by Charles Terront to win the world's first long distance cycle race, the 1891 Paris–Brest–Paris.

479. In the 1920s and 1930s, Michelin operated large rubber plantations in Vietnam, "French Indochina," where the French community of about 40,000 lived in the European quarters,

and the indigenous population lived on plantations working to produce the colony's exports of rice, tin, tea, and rubber. Conditions at these plantations led to the labor movement Phu Rieng Do.

480. In 1934, Michelin introduced a tire which, if punctured, would run on special foam lining; a design now known as a run-flat tire (self-supporting type).

481. In 1946, Michelin developed and patented the radial tire.

482. Michelin bought the then-bankrupt Citroën in the 1930s.

483. In 1968, Michelin opened its first North American sales office. By 1989 the company had a 10% market share for OEM tires purchased by American automobile makers.

484. In 1989, Michelin acquired the recently merged tire and rubber manufacturing divisions of the American firms B.F. Goodrich Company, founded in 1870, and Uniroyal, Inc., founded in 1892 as the United States Rubber Company. Uniroyal Australia had already been bought by Bridgestone in 1980. This purchase included the Norwood, North Carolina manufacturing plant which supplied tires to the U.S. Space Shuttle Program.

485. Michelin also controls 90% of Taurus Tire in Hungary, as well as Kormoran, a Polish brand.

486. In January 1991, Michelin Group closed the historic Eau Claire, Wisconsin, plant, eliminating 1,350 positions. Later in 1991 it closed the tire-cord manufacturing plant in Lindsay, Ontario, with 74 workers on August 30, 1991, due to high cost and two tire factories with 1,000 jobs in Kitchener, Ontario, citing overcapacity.

487. As of 1 September 2008, Michelin again became the world's largest tire manufacturer after spending two years as number two behind Bridgestone.

488. Michelin produces tires in France, Spain, Germany, the USA, the UK, Canada, Brazil, Thailand, Japan, Italy and several other countries.

489. Michelin publishes a variety of road maps, mostly of France but also of other European countries, Africa, Thailand and the United States.

490. The Michelin roadmaps were reproduced in England for the Allied invasion during World War II.

491. ViaMichelin is a wholly owned subsidiary of Michelin Group, and was started in 2001, to represent Michelin's digital mapping services. As of August 2008, ViaMichelin generates 400 million maps and routes per month on its main website.

United States Rubber Company (Uniroyal)

492. The United States Rubber Company (Uniroyal) is an American manufacturer of tires and other synthetic rubber-related products, as well as variety of items for military use, such as ammunition, explosives, and operations and maintenance activities (O&MA) at government-owned contractor-operated facilities.

493. United States Rubber Company became Uniroyal, Inc., as part of creating a unified brand for its products and subsidiaries in 1961.

494. Uniroyal, Inc., manufactured dioxin contaminated phenoxy herbicides and the cacodylates or components thereof or constituent chemical raw materials therefore to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A

495. In 1990, Uniroyal was acquired by French tire maker Michelin and ceased to exist as a separate business. The Company's long-lived advertising slogan was "*United States Tires are Good Tires*."

496. United States Rubber Company was founded in Naugatuck, Connecticut, in 1892 and was one of the original 12 stocks in the Dow Jones Industrial Average.

497. By 1892, there were many rubber manufacturing companies in Naugatuck, Connecticut, as well as elsewhere in Connecticut. Nine companies consolidated their operations in Naugatuck to become the United States Rubber Company. One of the nine, Goodyear's India Rubber Glove Mfg. Co., named Litchfield Rubber Co. until 1847, which manufactured rubber gloves for telegraph linemen was the only company in which Charles Goodyear, inventor of the rubber vulcanization process, is known to have owned stock.

498. From 1892 to 1913, the rubber footwear divisions of U.S. Rubber manufactured their products under 30 different brand names, including the Wales-Goodyear Shoe Co. The company consolidated these footwear brands under one name, Keds, in 1916, and were mass-marketed as the first flexible rubber-sole with canvas-top "sneakers" in 1917.

499. On May 26, 1896, Charles Dow created the Dow Industrial average of twelve industrial manufacturing stocks, which included among them U.S. Rubber Company. When the average expanded to a list of 20 stocks in 1916, U.S. Rubber remained, however the listing expanded to 30 stocks in 1928 and U.S. Rubber was dropped.

500. In 1931, U.S. Rubber Company bought a substantial portion of the Gillette Safety Tire Company. The company was founded in 1916 by Raymond B. Gillette and its primary manufacturing plant was located in Eau Claire, Wisconsin. The Gillette plant held large contracts with the General Motors Corporation and with the addition of U.S. Rubber products, became one of the world's largest suppliers of

original equipment tires. U.S. Rubber produced tires under the Gillete, Ward, Atlas, U.S. Rubber and U.S. Royal brands.

501. In 1940, U.S. Rubber purchased the remainder of the Gillette Safety Tire Company.

502. During World War II, U.S. Rubber factories produced military truck and airplane tires, as well as the canvas-top, rubber-soled Jungle boot for soldiers and marines serving in tropical and jungle environments. U.S. Rubber ranked 37th among United States corporations in the value of wartime production contracts.

503. In 1942, the United States government restricted the sale of scarce rubber products for civilian use and the company sold the Eau Claire plant to the government, which then converted it for the manufacture of small caliber ammunition and renamed it the Eau Claire Ordnance plant.

504. By December 31, 1943, the need for tires outweighed the need for ammunition. U.S. Rubber repurchased the plant from the government for more than US$1 million and converted it back to synthetic rubber tire production. The company began an expansion and modernization program at the plant that lasted through 1951. When it ended, the Eau Claire plant was the fifth largest tire facility in the United States. The company again expanded the plant in 1965 to produce tires for construction machinery, and for many years it was the largest private employer in Eau Claire and the second largest in neighboring Chippewa Falls before it was closed in 1991.

505. In late 1943, U.S. Rubber engineer **Dr. Louis Marick** developed a propeller de-icing system in which a rubber boot was fitted onto the leading edge of a propeller. The boot contained wires that conducted electricity to heat the edge and break-up ice.

506. In 1958, Uniroyal entered into a partnership with the Englebert tire company of Liège, Belgium, which became known as Uniroyal Englebert Deutschland AG. In 1963, the name was shortened to Uniroyal-Englebert, and in 1967 it became Uniroyal along with all company divisions. Uniroyal sold this division with its four factories in Belgium, Germany, France and Scotland to Continental AG in 1979. Continental continues to market tires under the Uniroyal brand in Belgium, the Czech Republic, Germany, Poland and the United Kingdom.

507. Uniroyal operations in Canada were carried out under the name Dominion Rubber Company for a number of decades.

508. Dominion started operations as Brown, Hibbard and Bourne, established in 1854. In 1866, the company registered as the Canadian Rubber Company of Montreal Limited manufacturing waterproof cloth, rubber footwear and machinery belts. It began to produce auto tires in 1906 in its Montreal factory and through a series of mergers with other companies in Ontario and Quebec became the Canadian Consolidated Rubber Company Limited.

509. After another series of mergers, the company became the Dominion Rubber Company Limited in 1926. It produced footwear under a variety of brand names, coated upholstery fabrics, mechanical parts, industrial chemicals and vehicle tires.

510. n 1966, after four decades as Dominion, the company was renamed Uniroyal Ltd., along with all other company holdings, and in 1981, it was sold to Waterville Ltd.

511. In 1939, Mark Lodge and Harold Hill established S. A. Rubber Mills Pty. Ltd. in Edwardstown, South Australia. The company grew until 1963 when U.S. Rubber purchased 25% of the business. By 1980, Uniroyal owned sixty percent of what

was now Uniroyal Brazil which it sold to Bridgestone. The subsidiary which operated Australian plants under the name Uniroyal Tyre Company became Bridgestone Australia Ltd. and was traded on the Australian stock exchange as a majority-owned subsidiary of Japan's tire company. On 20 March 2007 stockholders of Bridgestone Australia Ltd. voted to make the company a wholly owned subsidiary of Bridgestone Corporation of Japan.

512. In 1961, the company became **Uniroyal, Inc.** The Uniroyal name was applied to all its operating components and products by 1967, creating a unified brand.

513. In 1985, Uniroyal was taken private by its management and the New York investment firm of Clayton & Dubilier to prevent a hostile takeover by financier Carl C. Icahn. At the time, Uniroyal was the fifth-largest tire company in the country. To help pay the nearly $1 billion in debt incurred in the leveraged buyout, the company sold its Uniroyal Chemical subsidiary to Avery Inc., a producer of agricultural chemicals, industrial chemical additives and specialized rubber and plastic products, for $760 million in May 1986.

514. The remaining tire operation was merged with that of B.F. Goodrich Company, a S&P 500-listed tire and rubber fabricator that made high-performance replacement tires. The joint venture partnership became the Uniroyal Goodrich Tire Company and B.F. Goodrich Company held a 50% stake in the new tire company.

515. The new Uniroyal Goodrich Tire Company established its headquarters at the former B. F. Goodrich corporate headquarters, within its 27-building downtown complex in Akron, Ohio, which contained Goodrich's original factory. In the fall of 1987, the B.F. Goodrich Company closed several manufacturing operations at the site, and most of the complex

remained vacant until February 1988, when B.F. Goodrich announced plans to sell the vacant portions of the complex to the Covington Capital Corporation, a New York developer.

516. In June 1988, B.F. Goodrich sold its 50% stake to a group of investors led by Clayton & Dubilier, Inc. for US$225 million. At the same time, B.F. Goodrich also received a warrant to purchase indirectly up to 7% of the equity in Uniroyal Goodrich Tire Company.

517. Also in June 1988 as part of the sale deal, the new privately held tire company acquired publicly held debt of $415 million. The Uniroyal Goodrich Tire Company offered the debt securities in two parts through underwriters led by Drexel Burnham Lambert Inc. The two instruments were a US$250 million issue of 14-⅛% notes due in 1998, and a US$165 million issue of 14-½% subordinated debentures due in 2000.

518. For the year 1988, Uniroyal Goodrich Tire posted sales revenue of US$2.2 billion, while profit declined to less than US$12 million, which included an extraordinary credit of nearly US$2 million from the purchase of Canadian annuity pension obligations, and also a charge of over US$16 million from the June 1988 recapitalization resulting from the selloff by B.F. Goodrich.

519. Also in 1988, Michelin Group, a subsidiary of the French tire company Michelin et Cie (Euronext: ML) proposed to acquire the Uniroyal Goodrich Tire Company and began acquiring a stake.

520. In 1989, Uniroyal Goodrich Tire Company posted sales revenue that was up to almost US$2.3 billion, but profit was down by 90% to just over US$1 million but included over US$9 million extraordinary credit that year for the ongoing Canadian annuity pension obligation purchase. 1989 year-end net income results were also hurt by increased interest

expense of nearly US$31 million on the June 1988 debt recapitalization, and a US$29 million charge for deferred employee compensation related to the proposed purchase of the company by Michelin Group.

521. By May 1990, Michelin Group completed its purchase of Uniroyal Goodrich Tire Company from Clayton & Dubilier of New York. The deal was valued at about US$1.5 billion. B.F. Goodrich surrendered its 7% warrant to Michelin Group for US$32.5 million.

522. With the sale, B.F. Goodrich then exited the tire business and became the Goodrich Corporation to focus on building its chemicals and aerospace businesses through reinvestment and acquisitions. Michelin Group continued to operate the Uniroyal Goodrich Tire Company as its tire manufacturing unit in the United States and Canada.

523. Also in 1991, the Uniroyal tiger returned to national television after a 10-year hiatus, featured in a new 30-second spot created by Wyse Advertising of Cleveland, Ohio. The animated Uniroyal tiger had been a television advertising icon for the company through the 1970s. The new commercial appeared on ESPN and CNN sports-related programming, and also was run by Uniroyal dealers in local markets.

524. By 1993, Michelin North America employed 28,000 people at 18 plants, in South Carolina, Alabama, Oklahoma, Indiana, Nova Scotia and Ontario. In mid-1993, Michelin North America cut 2,500 of those jobs, which represented about 9% of its work force in the United States and Canada. As of 2010, the Uniroyal Goodrich Tire unit continued to operate with about 1,000 workers at its tire plant in Woodburn, Indiana, and another plant in Tuscaloosa, Alabama. The plant in Opelika, Alabama, was closed in 2009.

Defendant Harcros Chemicals, Inc.

525. Harcros Chemicals, Inc., is the successor in interest to Harrisons and Crosfield PLC**,** which was the successor in interest to North American Philips, which, in 1961, acquired Thompson-Hayward Chemical Company which manufactured dioxin contaminated phenoxy herbicides and the cacodylates or components thereof or constituent chemical raw materials therefore to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A .

526. Harcros Chemicals Inc. is a major distributor and manufacturer of industrial and specialty chemicals. Privately held since 2001, the company has a long-standing history in the chemical industry.

527. Harcros began business in 1917 as Thompson, Munro and Robins, and changed its name to Thompson-Hayward Chemical Company (THCC) in 1923.

528. North American Philips acquired THCC in 1961.

529. In 1981, Harrisons and Crosfield PLC purchased the bulk of the business from North American Philips.

530. The company name was changed to Harcros Chemicals, Inc. in 1988.

531. In 2001 a management buyout resulted in the privatization of Harcros.

532. The Company is employee owned effective January 2014 through the creation of an ESOP.

533. Harcros operates twenty-nine distribution locations in nineteen states.

534. The Harcros Manufacturing Division is a niche producer of surfactants, emulsifiers, defoamers, and a myriad of specialty products, custom blends, and reaction chemistries.

Defendant Koninklijke Philips Electronics N.V.

535. *Koninklijke Philips Electronics N.V.* is the parent of Philips Electronics North America Corporation, which is the parent of THAN, which is the successor in interest to T.H. Agriculture and Nutrition Co., which manufactured dioxin contaminated phenoxy herbicides and the cacodylates or components thereof or constituent chemical raw materials therefore to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A.

536. Philips' Electronics North American headquarters is based in Andover, Massachusetts.

537. Philips Lighting has its corporate office in Somerset, New Jersey, with manufacturing plants in Danville, Kentucky, Dallas, Salina, Kansas and Paris, Texas and distribution centers in Mountain Top, Pennsylvania, Ontario, California and Memphis, Tennessee.

538. Philips Healthcare is headquartered in Andover, Massachusetts. The North American sales organization is based in Bothell, Washington. There are also manufacturing facilities in Andover, Massachusetts, Bothell, Washington, Baltimore, Maryland, Cleveland, Ohio, Foster City, California, Gainesville, Florida, Milpitas, California and Reedsville, Pennsylvania. Philips Healthcare also formerly had a factory in Knoxville, Tennessee.

539. Philips Consumer Lifestyle has its corporate office in Stamford, Connecticut. Philips Lighting has a Color Kinetics office in Burlington, Massachusetts.

540. Philips Research North American headquarters is in Cambridge, Massachusetts.

541. THCC was originally incorporated as Thompson-Munro-Robbins Chemical Co. in January 1917.

542. THCC did not mine asbestos or manufacture asbestos-containing products. However, on behalf of Carey-Canadian Mines, Ltd., THCC distributed various grades of chrysotile asbestos fibers to portions of the United States between April 1, 1960, and June 1, 1980.

543. THAN also distributed laundry products and vermiculite that may have contained asbestos.

544. T.H. Agriculture & Nutrition Company (THAN) was a California chemical company. It operated a pesticide-formulating facility from 1962 until 1981 that is now a Superfund site.

545. THAN was formed in 1998 and is the successor by merger to TH Agriculture and Nutrition, Inc., formerly known as Thompson-Hayward Chemical Company ("THCC"), which manufactured dioxin contaminated phenoxy herbicides and the cacodylates or components thereof or constituent chemical raw materials therefore to which the Plaintiffs were exposed at Eglin Air Force Base, including site C–52A.

546. THAN, the Thompson-Hayward Chemical Company, is a limited liability company organized under the laws of Delaware with its principal place of business in Lenexa, Kansas.

547. THAN is a subsidiary of Philips Electronics North America Corporation ("PENAC"), which in turn is a subsidiary of *Koninklijke Philips Electronics N.V.* ("Philips"), a Dutch corporation.

548. On November 24, 2008 THAN filed a voluntary petition for relief under Chapter 11 of the United States Bankruptcy Code.

Defendant Thompson Chemical Company

549. Upon information and belief, Defendant Thompson Chemical Company was a manufacturer of dioxin contaminated phenoxy

herbicides and the cacodylates to which the Plaintiffs were exposed at Eglin Air Force Base, including site-C52A involved in and did make a contribution, directly or by an insurance carrier, to the settlement of MDL 381, *in re Agent Orange Litigation* in 1984.

Defendant Johnson Controls International PLC

550. Johnson Controls is an American multinational conglomerate producing automotive parts such as batteries and electronics and HVAC equipment for buildings.

551. Johnson Controls employs 170,000 people in more than 1,300 locations across six continents.

552. As of 2012 Johnson Controls was listed as 67th in the Fortune 500 and 251st in Global 500.

553. In January 2016, Johnson Controls announced its merger with Tyco International to form Johnson Controls International PLC. based in Ireland. The merger was completed on September 9, 2016 and will save the company about $150-million per year in taxes.

554. On October 31, 2016, the former Johnson Controls Automotive Experience division was spun off as a separate, publicly-traded company, Adient plc, and began trading on the New York Stock Exchange.

555. The Johnson Controls plant in Lakeshore, Ontario closed in late March 2010 and the property was sold.

556. In 1982, Johnson Controls enacted what it called a "fetal protection policy," which denied women the right to work on the battery production line because of the potential harm to a fetus they might conceive. Women were allowed to work on the production line only if they could prove that "...their inability to bear children had been medically documented."

557. Johnson Controls operations are segmented into four business units: Building Efficiency, Global WorkPlace Solutions, Power Solutions and Automotive Experience.

558. The Building Efficiency business unit designs, produces, installs and services heating, ventilation and air conditioning systems, industrial refrigeration, building management systems, fire and security systems and mechanical equipment for commercial and residential buildings. The brands produced under this business unit are York, TempMaster, Metasys, Panoptix, Frick and Sabroe.

559. Building Efficiency is the company's longest-running business unit, dating to 1885 when Johnson founded the Johnson Electric Service Company after patenting the electric thermostat in 1883. As of 2012, the business unit operated from 700 branch offices in more than 150 countries.

560. The Global WorkPlace Solutions business unit provides outsourced facilities management services globally and manages corporate real estate on behalf of its customers including acquiring and disposing of property, administering leases, and managing building related projects such as equipment replacements. On September 23, 2015, CBRE, Inc. purchased the Global Workplace Solutions business unit, retaining the name "Global Workplace Solutions".

561. The Power Solutions business unit designs and manufactures automotive batteries for passenger cars, heavy and light duty trucks, utility vehicles, motorcycles, golf carts and boats. It supplies more than one third of the world's lead-acid batteries to automakers and aftermarket retailers. This part of the company also manufactures Lithium-ion cells and complete battery systems to power hybrid and electric vehicles such as the Ford Fusion and Daimler's S-Class 400. It also manufactures Absorbent Glass Matt (AGM) and Enhanced

Flooded Batteries (EFB) batteries to power Start-Stop vehicles such as the Chevy Malibu and Ford Fusion. As of 2012, the business unit operated from 60 locations worldwide.

562. The Automotive Experience business unit supplies automotive seating, interiors and electronics to automakers and is one of the largest suppliers of car interiors in the world.

563. Johnson Controls designs and manufactures seating components, including mechanisms, tracks, structures foams, fabrics and trim. It is the largest automotive seat supplier in the world. A separate interiors division produces overhead systems, headliners, door panels, instrument panels, and overhead and floor consoles for automotive interiors. An electronics division designs and manufactures analog and digital instrument clusters, infotainment systems and hands-free electronics. As of 2012, the business unit operated from 240 locations worldwide.

564. On January 12, 2016 Johnson Controls announced that its remaining Automotive Experience holdings will spin-off and become Adient. On October 3, 2016, Adient began to be publicly traded on the NYSE as ADNT.

565. Johnson Controls-Saft Advanced Power Solutions (JCS) was a joint venture between Johnson Controls and French battery company Saft Groupe S.A. officially launched in January 2006.

566. Varta established a JCS development center at its German HQ, following the setting-up of Varta-SAFT joint venture.

567. In May 2011, Johnson Controls and Saft dissolved the joint venture. Johnson Controls paid Saft 145 million dollars for its shares in the joint venture as well as for the right to use certain technology developed by the joint venture. Johnson Controls retained the Michigan facility built by the partnership. The French joint facility was transferred to Saft.

568. Brookfield Johnson Controls is a joint venture with Brookfield Properties to provide commercial property management services in Canada. Established in 1992, it was known as Brookfield LePage Johnson Controls or BLJC until May 2015. In 2013, Johnson Controls and Brookfield Asset Management formed a similar joint venture for properties in Australia and New Zealand.

569. Diniz Johnson Controls is a joint venture with Diniz Holding in Turkey building complete automotive seats for major OEMs.

570. Amara Raja Batteries of India signed a joint venture with Johnson Controls in December 1997 to manufacture automotive batteries in India, under the brand name "Amaron".

571. On June 6, 2015, Johnson Controls exited from the automotive seating business to concentrate on the core business of building ventilation systems and automotive batteries.

572. On September 9, 2016, Johnson Controls merged with Tyco International Ltd. combining all businesses of Tyco and Johnson Controls as Johnson Controls International PLC.

Tyco International Ltd.

573. Tyco International PLC was a security systems company incorporated in the Republic of Ireland, with operational headquarters in Princeton, New Jersey, United States (Tyco International (US) Inc.).

574. Founded by Arthur J. Rosenberg in 1960, Tyco, Inc. was formed as an investment and holding company with two segments: Tyco Semiconductors and The Materials Research Laboratory.

575. In 1962, the business was incorporated in Massachusetts and refocused on high-tech materials science and energy conservation products. Two years later in 1964, the company

went public and began to fill gaps in its development and distribution network by acquiring Mule Battery Products, the first of Tyco's 16 acquisitions in the next four years.

576. Following aggressive acquisitions through the 1970s, Tyco divided the company into three business segments: Fire Protection, Electronics, and Packaging.

577. In the later part of the 1980s, Tyco acquired Grinnell Corporation, Allied Tube and Conduit, and the Mueller Company and reorganized its subsidiaries into four segments: Electrical and Electronic Components, Healthcare and Specialty Products, Fire and Security Services and Flow Control.

578. In 1992, Dennis Kozlowski became CEO of Tyco International, and, for the next several years, the company again adopted an aggressive acquisition strategy, eventually acquiring, by some accounts, over 1,000 other companies between 1991 and 2001.

579. Major acquisitions in the 1990s included Wormald International Limited, which owned Ansul, and many other companies.

580. In 1996, Tyco was added to the Standard & Poor's S&P 500 Composite Index, which consists of the 500 publicly traded companies in the United States with the largest market capitalization.

581. In 1997, Tyco International acquired AT&T Submarine Systems, gaining research and development and fleet assets, along with the manufacturing capability to produce repeaters and transmission equipment. These additional capabilities, combined with cable manufacturing at Tyco Integrated Cables Systems in Newington, New Hampshire, established Tyco Telecommunications as the world's first vertically integrated global optical network supplier, capable of developing the

technology and manufacturing the components, to designing, building and maintaining systems.

582. In July 1997, Tyco merged by reverse takeover with a smaller publicly traded security services company named ADT Limited, controlled by Michael Ashcroft. As part of the deal, Tyco International Ltd. of Massachusetts became a wholly owned subsidiary of ADT Limited, and simultaneously ADT changed its name to Tyco International Ltd., retaining the former Tyco stock symbol, TYC. The merger moved Tyco's incorporation to Bermuda, a tax haven, where it was headquartered in the colonial capital of Hamilton. A new subsidiary named ADT Security Services was also formed out of the merger.

583. In 1999, Tyco acquired two S&P 500 companies in a buyout. They acquired the electronics connector manufacturer AMP Inc., for $12.22 billion and a materials science company, Raychem Corp., for $1.4 billion.

584. In 2000, Tyco closed the year spinning off a deep-sea fiber-optic cable-laying division it had purchased from AT&T as Tyco Submarine Systems in an initial public offering.

585. In the fiscal 2001 year, Tyco acquired Mallinckrodt Inc. and Simplex Time Recorder Company which it later merged in January 2002 with Grinnell Fire Protection to form an indirect wholly owned subsidiary, SimplexGrinnell LP, the world's largest fire protection company.

586. In 2005, Tyco was awarded the largest statewide public safety communications project in the United States in 2004 when one of Tyco Electronics' businesses, M/A-COM Technology Solutions, signed a contract to maintain the New York Statewide Wireless Network (SWN). The contract was worth approximately $2 billion and would last for 20 years.

587. In 2007, Tyco spun off the Electrical and Healthcare segments to create three publicly independent companies: Covidien Ltd., formerly Tyco Healthcare, TE Connectivity Ltd., formerly Tyco Electronics Ltd., and Tyco International Ltd., formerly Tyco Fire & Security and Tyco Engineered Products & Services (TFS/TEPS).

588. In 2012, Tyco split once again, resulting in creation of Tyco which focused on fire protection and electronic security products, installation and services worldwide; ADT Corporation, focused on residential and small business security installation and services in North America; and Flow Control, focused on water and fluid solutions, valves and controls, and equipment protection products worldwide, which merged with and is now part of Pentair Ltd.

589. In 2014, Tyco International sold its New Zealand based security company Armourguard Security limited to Evergreen International.

590. On September 9, 2016, January 25, 2016, Johnson Controls merged with Tyco combining all businesses of Tyco and Johnson Controls as Johnson Controls International PLC.

Wormald International

591. The founders of the company were brothers Joseph Dawson Wormald and Henry Percy Wormald. Born in Edinburgh, Joseph arrived in Australia in 1889 when brother Sir John Wormald, of the Manchester-based engineering firm, Mather & Platt, sought to distribute the company's Grinnell-licensed sprinklers in that country.

592. In partnership with Stanley Russell, Russell & Wormald was formed to manufacture and import the fire sprinklers, as well as Simplex fire extinguishers and fire doors. With Mr. Russell retiring in 1896 to join Mather & Platt as the country manager of France, Harry, who migrated to in Australia in 1890, joined

the company as a sub-manager two years later and was admitted into partnership in 1900, Russell & Wormald becoming Wormald Bros., based in Melbourne.

593. In May 1949, Wormald Bros. became a public company, Wormald Brothers Industries Limited, with head offices in Sydney.

594. As well as their core fire protection department, its other operating divisions included Metalbilt, manufacturing doors, including fire doors, and roller shutters; Steelbilt, manufacturers of storage equipment such as steel cabinets, shelving and cupboards and the license for the manufacture of products from the Kirsch Company, "the world's largest manufacturer of Drapery hardware and Venetian Blinds."

595. In 1974, the company, now known as Wormald International Limited, acquired Mather & Platt, whose fire-protection products began Wormald's business nearly a century earlier.

596. In 1990, Wormald International, valued at US$1 billion, was itself purchased by Princeton, New Jersey-based Tyco International, becoming part of its Boca Raton, Florida-based fire protection division that included Grinnell Mechanical Products, the company whose fire sprinklers Mather & Platt had licensed to be distributed by Wormald in 1889.

597. On December 4, 2015, Tyco International's fire-protection business in Australia made up of Wormald, National Fire Solutions, GAAM Emergency Products, Exegard and Simplex Time Solutions, was acquired by New York City-based private equity firm Evergreen Capital, LLC.

598. Evergreen Capital had formed an Australasian business relationship with Tyco, having previously acquired Tyco's fire and security operations in Fiji, including the rights to the Wormald brand there, and purchasing Armourguard, Tyco's New Zealand security division, for a nominal NZ$1 in 2014.

599. Evergreen returned Wormald to local management.

Ansul

600. Ansul is a corporation headquartered in Marinette, Wisconsin that manufactures fire suppression systems, extinguishers, and offers fire training.

601. The company changed its name over the years, but always kept Ansul as part of its name. From 1915–1963 it was the Ansul Chemical Company, from 1963–1981: the Ansul Company and 1981–1995 Ansul Fire Protection. The current name has been used since 1995.

602. *Ansul* is the registered trade name for Ansul products.

603. Ansul was created by Francis "Frank" G. Hood from the bankrupt Bastol Company, an enterprise started in Marinette in 1912.

604. Ansul's initial activities included production of cattle feed, refrigerants and selected specialty chemicals. The name Ansul comes from ANhydrous SULfur dioxide (SO_2), which was sold to dye works and fruit preservers, and later as a refrigerant.

605. Production of fire suppression chemicals began in 1934.

606. Virginia Chemicals, Inc., now part of Celanese Corporation, acquired the Refrigeration and Air Conditioning Products Division of The Ansul Company in 1967.

607. By 1983 Ansul had discontinued all other production at the facility in Marinette, Wisconsin.

608. Ansul remained independent until 1978, when it was acquired by Wormald International, an Australian corporation.

609. Wormald was in turn acquired by Tyco International in 1990.

610. Ansul now exists as a brand of the Tyco Fire Protection segment of Tyco International which has become part of Johnson Controls International PLC.

611. Ansul Manufactured arsenic-based herbicides adjacent to the Menominee River which flows into Green Bay, Wisconsin, a part of Western Lake Michigan.

612. These arsenic-based herbicides were the principal component of *Agent Blue* to which the Plaintiffs were exposed at Eglin Air Force Base designated ranges, including site C–52A.

613. The river bottom and ground water are heavily contaminated with arsenic compounds that were released by Ansul from 1957 to 1977 as a result of herbicide manufacture.

614. Certain areas of Menominee River sediment contain levels as high as 11,000 ppm arsenic.

615. From 1960–1966, arsenic-laden wastewater was discharged directly into the Menominee River.

616. More than 95,000 tons of arsenic salt were at one time stored on site.

617. St. Thomas Aquinas Academy High School, formerly known as Marinette Catholic Central High School until 2004, is adjacent to Ansul's property, and next to it is the Marinette Middle School, formerly Marinette High School until 1973.

HERBICIDES

618. Herbicides arc chemical compounds used to kill or damage plants. They can be used to dry foliage or to stimulate or inhibit growth by modifying physiological processes in plants and are classified as contact herbicides or as systemic herbicides, depending on the way they affect plants.

619. Contact herbicides or desiccants are used to damage plant tissue by local action at the point of application and show little or no movement throughout the plant. After being sprayed with a contact herbicide, susceptible plants dry rapidly; causing leaves to fall from some plant species, and to shrivel but remain on others.

620. Systemic herbicides. Systemic herbicides are absorbed at the point of application and moved by the sap stream to other parts of the plant. These chemicals are growth regulators and usually act more slowly than contact herbicides. They damage plants by both local and systemic action, causing leaves to fall and, finally killing the plants when the dose has been adequate. Plants treated with sublethal doses frequently recover partially or completely. Some plant species are highly resistant to this class of chemicals and show very little outward change. Systemic herbicides are most effective on woody plants and broad-leaved vegetation in an active state of growth.

The phenoxy herbicides

621. Phenoxy herbicides are synthetic chemical analogues of hormones found in plants that regulate the rate and pattern of plant growth. These herbicides cause aberrant growth or death of certain plant species.

622. 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) is an herbicide that kills plants by artificially overstimulating the growth hormone.

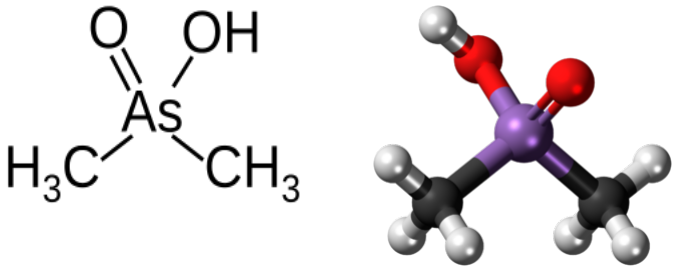
623. Agent Orange was a 50-50 mixture of the esters, or inorganic salts, of 2,4,5-T and another herbicide, 2,4-Dichlorophenoxyacetic acid, or 2,4-D.

624. The reported sites at which dioxin contaminated phenoxy herbicides and the cacodylates were manufactured and stored in large quantities are located on the following map.

625. Reported sites for dioxin contaminated phenoxy herbicides

Cacodylic acid and the cacodylates

626. Cacodylic acid also known as dimethylarsinic acid, Hydroxy-dimethylarsine oxide, *Arsecodile, Ansar, Silvisar, Phytar 560, DMAA,* and *UN 1572* is the chemical compound with the formula $(CH_s)_2AsO_2H$.



627. Derivatives of cacodylic acid, the cacodylates, were frequently used as herbicides.

628. "Agent Blue," one of the defoliant chemicals used during the Vietnam War, is a mixture of cacodylic acid and sodium cacodylate.

629. Sodium cacodylate is frequently used as a buffering agent in the preparation and fixation of biological samples for electron microscopy.

630. Significant early research into cacodyls was done by Robert Bunsen at the University of Marburg. Bunsen said of the compounds, “the smell of this body produces instantaneous tingling of the hands and feet, and even giddiness and insensibility...It is remarkable that when one is exposed to the smell of these compounds the tongue becomes covered with a black coating, even when no further evil effects are noticeable”.

631. Cacodyl oxide, ((CH3)2As)$_2$O, is often considered the first organometallic compound to be prepared synthetically.

632. Cacodylic acid and its salts were incorporated into herbicides by large variety of manufacturers under numerous brand names. The variety used in Vietnam (as Agent Blue) was Phytar 560G.

633. In the 18th century it was known that combining As_2O_3 and four equivalents of potassium acetate (CH_3CO_2K) yielded a product called “Cadet’s fuming liquid” which contains cacodyl oxide, $((CH_3)_2As)_2O$ and cacodyl, $((CH_3)_2As)_2$.

634. Cacodylic acid can be reduced to dimethylarsine (III) derivatives, which are versatile intermediates for the synthesis of other organoarsenic compounds.

635. Cacodylic acid is highly toxic by ingestion, inhalation, or skin contact. It has been shown to be teratogenic in rodents, most often causing cleft palate but also fetal fatality at high doses.

636. It has been shown to be genotoxic in human cells, causing apoptosis and also decreased DNA production and shorter DNA strands.

637. Cacodylic acid promotes tumors in the presence of carcinogens in organs such as the kidneys and liver.

MILITARY APPLICATIONS OF HERBICIDES

638. The Rainbow Herbicides are the group of "tactical use" chemicals used by in Southeast Asia during the Vietnam War.

639. According to a December 1971 United States Army Field Manual, entitled *Tactical Employment of Herbicides*, "Various commercial chemical compounds used as herbicides have been adapted for use in military operations. Authority to use these chemicals must be obtained through the proper channels." And "Defoliation of heavily vegetated areas by the use of herbicides is the primary means of obtaining visual observation of enemy forces, facilities, roads, ambush sites, infiltration routes, and other enemy locations from the air, ground, or water."

United States military policy on the use of herbicides

640. According to a December 1971 United States Army Field Manual, entitled Tactical Employment of Herbicides, in the Section Entitled, "COMMAND AND CONTROL OF HERBICIDE OPERATIONS," it was stated that

> "National policy will govern the use of herbicides in a theater of operations. … The employment of herbicides for military purposes must be judiciously controlled. Many unforeseen and undesirable problems may arise unless the user is thoroughly familiar with the socioeconomic and political implications, the type of vegetation to be attacked, the best herbicide to use, and the most efficient mode of dissemination. The user must know which chemicals will produce the desired level of defoliation on vegetation in a particular target area."

and later,

> Close staff coordination and planning are essential to enable the subordinate commander to make the proper decision. A key staff element is the GS/civil military operations section, which performs the dual functions of civil affairs and psychological operations (PSYOP). The civil affairs function includes all activities that

might affect the relationship between the military, the civil government, and the people of the area. As for PSYOP, every action taken by the government and its military forces has a psychological impact on the populace and must be considered in all planning activities. … When such operations involve civilians and enemy forces, PSYOP can assist by informing the target population of what can be expected in the area, and of instructions and actions that will minimize structural, crop, and plant damage and nonmilitary casualties. To achieve national objectives, there will be situations where short range tactical advantages and expediencies should be sacrificed in favor of long-range goals. For example, firepower must be used with discretion to minimize non- combatant casualties. Employment of herbicides capable of causing widespread crop and plant damage can have a disastrous effect on civilian support and attitudes, post hostility, rehabilitation, and economic recovery."

641. The Department of Defense Advanced Research Project Agency ("ARPA") *Project Agile* was instrumental in the United States' development of herbicides as a military weapon, an undertaking inspired by the British use of 2,4,5-T to destroy jungle-grown crops during the insurgency in Malaya in the 1950s.

642. ARPA supported tests on combinations and concentrations of herbicides; calibration studies of the spray delivery system to achieve the desired $281ha^{-1}$ (3 gallons/acre) rate; and experiments on optimal conditions to minimize spray drift.

643. Between 1961 and 1971, herbicide mixtures—nicknamed by the colored identification band painted on their 208-litre storage barrels—were used by the United States and Republic of Vietnam ("RVN") forces to defoliate forests and mangroves, to clear perimeters of military installations and to destroy "unfriendly" crops, as a tactic for decreasing enemy armed forces protective cover and food supplies.

644. Agent Green, a 100% n-butyl ester of 2,4,5-T, was used prior to 1963.

645. Agent Pink, a 100% 2,4,5-T mixture of 60% n-butyl ester 2,4,5-T, and 40% iso-butyl ester of 2,4,5-T, was used prior to 1964.

646. Agent Purple, a mixture of 50% 2,4,5-T containing 30% n-butyl ester of 2,4,5-T, and 20% isobutyl ester of 2,4,5-T, and 50% n-butyl ester of 2,4-D was used from 1961–65

647. Agent Blue (Phytar 560G), an organic arsenical made up of cacodylic acid (Ansar 138) and its sodium salt sodium cacodylate was used from 1962–71 as powder and in water solution.

648. Agent White (Tordon 101), a commercial product of The Dow Chemical Company contained 21.2% on an acid weight basis of triisopropanolamine salts of 2,4-D and 5.7% picloram. It was used from 1966–71.

United States use of military herbicides in Vietnam 1961–1971)

Name	Chemical constituents	Concentration active ingredient	Years used	Estimated quantities sprayed (liters)
Agent Pink	60%-40% n-Butyl; isobutyl ester of 2,4,5-T	961-1,081 gl^{-1} acid equivalent	1961; 1965	50,312 sprayed; 413,852 additional on procurement records
Agent Green	n-Butyl ester 2,4,5-T	(Should have same acid equivalent as Agent Pink)	(Unclear but within time frame for Agent Pink)	31,026 shown on procurement records
Agent Purple	50% n-Butyl ester 2,4-D; 30% n-butyl ester 2,4,5-T; 20% isobutyl ester 2,4,5-T	1,033 gl^{-1} acid equivalent	1962-1965	1,892,773
Agent Orange	50% n-Butyl ester 2,4-D; 50% n-butyl ester 2,4,5-T	1,033 gl^{-1} acid equivalent	1965-1970	45,677,937 (may include Agent Orange II)

Agent Orange II	50% n-Butyl ester 2,4-D; 50% isooctyl ester 2,4,5-T	910 gl^{-1} acid equivalent	After 1968 (?)	Unknown but at least 3,591,000 shipped
Agent White	Acid weight basis: 21.2% tri-isopropanolamine salts of 2,4-D and 5.7% picloram	By acid weight: 240.2 gl^{-1} 2,4-D and 64.9 gl^{-1} picloram	1966-1971	20,556,525
Agent Blue (powder)	Cacodylic acid (dimethylarsinic acid) and sodium cacodylate	Acid: 65% active ingredient; salt: 70% active ingredient	1962-1964	25,650
Agent Blue (H_20 solution)	21% sodium cacodylate + cacodylic acid to yield at least 26% total acid equivalent by weight	Acid weight: 360.3 gl^{-1}	1964-1971	4,715,731

649. Agent Orange or Herbicide Orange, (HO) was a mixture of 50% n-butyl ester 2,4-D and 50% n-butyl ester 2,4,5-T used from 1965–70.

650. Agent Orange II was a mixture of 50% n-butyl ester 2,4-D and 50% isooctyl ester 2,4,5-T used after 1968.

651. Agent Orange III was a mixture of 66.6% n-butyl 2,4-D and 33.3% n-butyl ester 2,4,5-T.

652. Enhanced Agent Orange, Orange Plus, or Super Orange (SO), or DOW Herbicide M-3393 was standardized Agent Orange mixture of 2,4-D and 2,4,5-T combined with an oil-based mixture of picloram, a proprietary DOW Chemical product called Tordon 101, an ingredient of Agent White.

Agent Orange

According to a December 1971 United States Army Field Manual, entitled *Tactical Employment of Herbicides*, "ORANGE is a systemic herbicide that defoliates a wide variety of woody and broad-leaved herbaceous plants. It affects grasses, bamboos, and similar plants less. Agent

ORANGE is absorbed by a plant at the point of application within a few hours, and the chemical is translocated."

"The components of ORANGE are rapidly decomposed by soil micro–organisms and the chemical usually disappears from soils within 1 to 3 months following application. Lateral distribution of the agent due to volatility alone is negligible."

"ORANGE is low in toxicity to man, fish, and wildlife; but it will cause slight skin irritation and minor inhalation effects."

"ORANGE will defoliate adequately forest vegetation in temperate and tropical regions. … Treated grasses and bamboos may exhibit brown fo1iage and partial top-kill, but they recover rapidly. The typical response of tropical, woody vegetation to systemic defoliants is progressive. …

Agent Blue

653. According to a December 1971 United States Army Field Manual, entitled *Tactical Employment of Herbicides*, "Agent BLUE currently in use is a commercial, liquid formulation of sodium cacodylate called Phytar 560G."

"BLUE is a fast-acting contact herbicide that is effective againstbroad-leaved herbaceous root or tuber crops."

"BLUE is a fast-acting contact herbicide that is effective against broad-leaved herbaceous or woody plants or grassy vegetation. It causes rapid browning and drying with accompanying shriveling and falling of leaves in some woody species. BLUE is exceedingly effective in the top-kill of grassy plants to include perennials during any season. At rates of application used for defoliation the chemical exhibits little or no systemic action within the plant."

"In contact with soil, BLUE is quickly deactivated by surface absorption; it is non-volatile and is not affected by light."

"This agent is readily absorbed through the skin, and prolonged absorption may cause a distinct garlic odor on the breath. BLUE has a very low toxicity to animals."

"When applied to susceptible vegetation at the recommended application rate, noticeable browning or discoloration is

evident in 1 day and maximum defoliation usually occurs in 2 to 4 weeks. However, the desired level of defoliation is of relatively short duration when compared to that of Systemic agents. In dense forests with multilayered canopies, applications of BLUE can be repeated in 2 to 4 weeks after the initial treatment to ensure penetration to lower vegetation layers and to extend the period of defoliation. Regrowth of some perennial grasses, such as elephant grass, wild cane, or cogon grass, is likely to occur within 1 to 2 months after treatment. This necessitates repeated spray applications."

Agent White (*Tordon* 101)

654. "WHITE is readily absorbed by foliage and the root system and is quickly transported throughout the plant.

655. Since soil microorganisms have little effect on the components of WHITE, this agent is more persistent in soils than ORANGE or BLUE and losses from soils occur principally by leaching. In sparsely vegetated areas, when applied at rates used for defoliation, WHITE may persist in soils for as long as 1 year. It is subject to only limited decomposition by sunlight and ultraviolet radiation.

656. Tests indicate that a single direct exposure to a spray of WIIITE of normal concentration would not constitute a percutaneous or inhalation hazard. This chemical is considered nontoxic and not hazardous to humans, animals, or fish.

657. WHITE is effective principally on broad-leaved herbaceous plants and particularly on woody plants. However, effects of the agent develop slowly on woody plants and full defoliation may not occur for several months after spray-application. Temperate zone conifers are also susceptible to WHITE but defoliation is delayed. Most grasses and monocotyledonous plants, including nipa palm, are resistant to WHITE.

658. WHITE is not recommended for use on crops because of its persistence in soils.

Military acquisition of the "Rainbow" Herbicides

659. In the early 1960s, the United States government, pursuant to the *Defense Production Act of 1950*, entered into a series of fixed-price production or procurement contracts with the defendants.

660. The government characterized delivery of Agent Orange as part of the prosecution of military action, which enabled the Defendant manufacturers to procure otherwise scarce materials and equipment necessary to produce it.

661. The government bought as much of Agent Orange as defendants were able to produce.

662. The contracts instructed the defendants not to label the contents of the fifty-gallon herbicide containers except by a color-coded three-inch band, in accordance with the type of herbicide.

663. The first large-scale United States military defoliation took place in Camp Drum, New York, in 1959, using Agent Purple (a 50-50 mixture of 2,4-D and 2,4,5-T) and a spray system which was the model for those used in Vietnam.

664. In 1961, under Project AGILE the US Department of Army Plant Science Laboratories at Fort Detrick, Maryland, was given the responsibility to determine the technical feasibility of defoliating jungle vegetation in South Vietnam.

665. Success with Project AGILE field tests with herbicides in South Vietnam in 1961 led to the formal herbicidal program Trail Dust, the predecessor to Operation Ranch Hand.

666. The first major herbicide shipment arrived in RVN in January 1962.

667. Defoliation targets were sprayed with Agent Purple during September and October 1962; crop destruction targets were sprayed with Agent Blue in November 1962.

668. Systematic testing of herbicides and calibration of herbicide delivery systems continued for several years.

669. United States Air Force ("USAF") operations, codenamed Operation Ranch Hand, dispersed more than 95% of all herbicides used in Operation Trail Dust, the overall herbicide program.

670. Other branches of the United States armed services and RVN forces, generally using hand sprayers, spray trucks (Buffalo turbines), helicopters and boats, sprayed much smaller quantities of herbicide.

671. Crop destruction required White House approval until 1963, after which final approval was delegated to the United States Ambassador to the RVN.

672. Herbicide tests were run from August to December 1961 in the Republic of Vietnam (RVN) using dinoxol and trinoxol. An insecticide test series was also undertaken.

673. From 1962 to 1965, small quantities of Agents Purple, Pink, and Green were used.

674. From 1965 to 1970, Agents Orange, White, and Blue were employed.

675. From 1970 to 1971, only Agents White and Blue were used in the defoliation program.

676. The herbicides were applied aerially at a rate of approximately 3 gallons per acre. According to military records of Operation Ranch Hand, from August 1965 to February 1971, a total of 17.6 million gallons of herbicide was sprayed over approximately 3.6 million acres in Vietnam.

677. Agent Orange was the most extensively used herbicide in Vietnam; it consisted of a 50:50 mixture by weight of the *n*-butyl esters of two phenoxy acids: 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).

678. The military use of 2,4,5-T was suspended by the Department of Defense in April 1970 following notice by Dow that it was contaminated with 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin (TCDD or dioxin) and that TCDD was highly toxic.

679. In early 1970, the AAAS set up a commission, the Herbicide Assessment Commission (HAC), to assess the effects of large-scale use of herbicides on the environment and population of Vietnam. In August 1970, they traveled to Vietnam on an inspection field trip to examine the extent to which the herbicides had destroyed the vegetation and local food crops in areas where they had been sprayed.

680. After returning from Vietnam, HAC members wrote a report on the defoliation of Vietnam in which they found that "crops had been sprayed in an area with an estimated population of 180 persons per square kilometer and that nearly all of the food being destroyed would have been used by mountain-dwelling Montagnard civilians instead of by enemy troops." The commission maintained that the military use of herbicides had been considerably more destructive than previously imagined—half of the mangrove forests had been destroyed and there were indications of serious health effects. The HAC members documented reports of stillbirths and birth defects in Vietnamese, noting that these adverse reproductive effects were possibly associated with 2,4,5-T and its contaminant, TCDD.

681. On December 26, 1970, President Richard M. Nixon announced that it was initiating an orderly yet rapid phase out of the herbicide operation. The AAAS council adopted a

resolution commending the U.S. government for its intention to phase out the use of herbicides in Vietnam.

682. United States use of chemical defoliants in Vietnam ended in 1971.

683. Johnston Atoll, also known as Kalama Atoll to Native Hawaiians, is an unincorporated territory of the United States currently administered by the United States Fish and Wildlife Service.

684. For nearly 70 years, the atoll was under the control of the American military. In that time it was used as a bird sanctuary, as a naval refueling depot, as an airbase, for nuclear and biological weapons testing, for space recovery, as a secret missile base, and as a chemical weapon and Agent Orange storage and disposal site.

685. These activities left the area environmentally contaminated and remediation and monitoring continue.

686. The Environmental Protection Agency (EPA) reported that 1,800,000 gallons of Herbicide Orange were stored at Johnston Island in the Pacific and that an additional 480,000 gallons which had been stored at Gulfport, Mississippi was brought to Johnston Atoll for destruction.

687. After Agent Orange spraying by the United States ended, the USAF was required to dispose of those very large stockpiles of surplus herbicide.

688. TCDD concentrations ranged from 6.2 to 14.3 ppm. and averaged 13.25 ppm. in samples drawn for incineration-effluent modelling studies from 28 different barrels chosen by the USAF as representative of the seven manufacturers contributing to the stockpile.

689. In other samples drawn from the stockpile, the TCDD range was about 0.05 to 13.3 ppm (weighted average 1.77 ppm).

690. After the termination of the Vietnam War, unused Agent Orange was removed to Johnston Atoll from South Vietnam and Gulfport, Mississippi in 1972 under Operation Pacer IVY and stored on the northwest corner of the island known as the Herbicide Orange Storage site but dubbed the “Agent Orange Yard”.

691. Leaking barrels during the storage and spills during re-drumming operations contaminated both the storage area and the lagoon with herbicide residue and its toxic contaminant 2,3,7,8-tetrachlorodibenzodioxin.

692. The Agent Orange stored on Johnston Island was eventually destroyed during Operation Pacer HO on the Dutch incineration ship MT Vulcanus in the Summer of 1977.

A photo of some of the barrels of Agent Orange, circa 1973

Selection and deployment of aircraft for Vietnam

693. Following World War II, the USAF assumed responsibility for the operations of the Special Aerial Spray Flight Unit, a military unit that provided control of insect pests through the use of aerial applications of insecticides.

694. In 1960, the Special Aerial Spray Flight phased out the C-47 aircraft and selected the Fairchild-built C-123B *Provider* as its replacement.

695. The scientists at Fort Detrick selected the candidate herbicides and assigned the Special Aerial Spray Flight the selection of aircrews and the initial spray equipment that was used in the early years of operations in Vietnam.

696. The C-123B aircraft was a high-wing, twin-engine assault transport with excellent low-speed maneuverability. The aircraft was ideal for the aerial dissemination of herbicides because the high-mounted wings allowed convenient positioning of wing spray booms, and the large cargo compartment and load capacity were ideal to receive a large spray system for internal carriage.

697. The initial spray system was the MC-1 Hourglass System, a system previously developed, but not used, for the Korean Conflict.

698. Refinement and modifications of this system occurred at Eglin AFB, and during the early years of deployment to Vietnam.

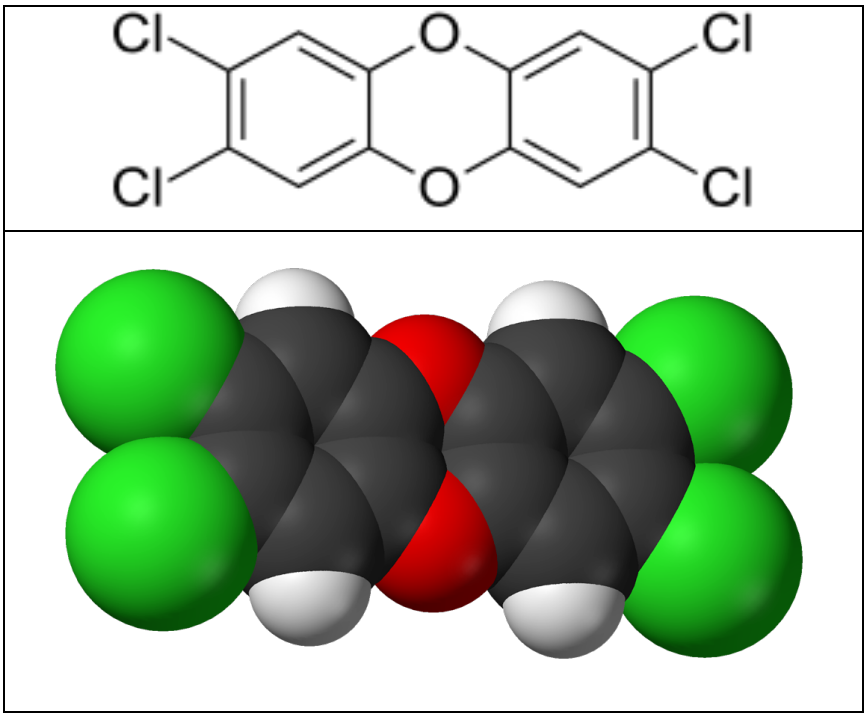
699. Beginning in July 1965, this spray system was replaced with the N A 45Y-1 Internal Defoliant Dispenser. This modular system consisted of a 3,785-liter tank, pump, and engine, which were mounted on a frame pallet for easy installation and removal from-the aircraft. Wing booms were mounted from the outboard engine nacelles toward the wing tips, and short tail boom was positioned centrally near the aft cargo door.

700. The UC-123 series aircraft became the “work horse” of Operation *Ranch Hand*. During the peak activity of operations in Vietnam from 1968-1969, approximately thirty UC-123K

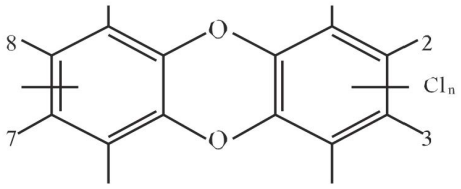
aircraft were employed (U designating spray aircraft and K, a jet booster modification).

2,3,7,8-TETRACHLORO-DIBENZO-P-DIOXIN (TCDD OR DIOXIN)

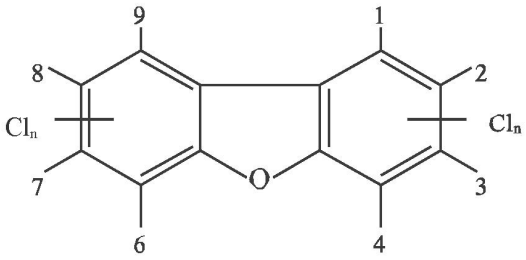
701. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is a polychlorinated dibenzo-p-dioxin, sometimes shortened inaccurately to simply "dioxin", with the chemical formula $C_{12}H_4Cl_4O_2$.



702. The term "dioxins" usually refers to polychlorinated dibenzo-dioxins (PCDDs)



703. and polychlorinated dibenzo-furans (PCDFs).



704. In their pure form, dioxins are colorless solids or crystals, but they enter the environment as mixtures containing a variety of individual components and impurities.

705. Dioxins formed by combustion are bound to particles such as ash. Small particles can be transported to much longer distances from the emission source.

706. Dioxins are hydrophobic and strongly lipophilic, and their solubility in organic solvents increases with chlorine content.

707. TCDD is a persistent environmental contaminant usually present in a complex mixture of dioxin-like compounds and is a carcinogen.

708. In the aquatic environment most of the dioxins attach strongly to any material with high organic content, especially to microscopic plants and animals (plankton) which are consumed by larger animals, circulating over time throughout

many trophic levels of complex food webs with and without biomagnification.

709. TCDD decays slowly in the soil under normal environmental conditions, which indicates that "its potential hazards may be very persistent" according to the National Academy of Sciences.

710. 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin (TCDD) arises during the hydrolysis of tetrachlorobenzene to form 2,4,5-trichlorophenol, the industrial precursor of 2,4,5-T.

711. According to accounts in Dow documents, TCDD is an impurity formed under conditions of high temperature in the production of trichlorophenol.

712. Trichlorophenol is used to make 2,4,5-trichlorophenol, which is used to make 2,4,5-Trichlorophenoxycetic acid, or 2,4,5-T.

713. In the early 1960s, dioxin produced in making trichlorophenol ended up in the 2,4,5-T and at concentrations of up to 140 parts per million in one company's product.

714. Later, Dow, which had been producing some of the cleanest 2,4,5-T, recommended that the military not use any product with more than 1 part per million.

715. 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), informally known as dioxin. is a contaminant introduced during the manufacture of 2,4,5-T depending on the temperatures and pressures used in the reactors.

716. Levels of TCDD contamination in Agent Orange ranged from less than 0.05 to almost 50 parts per million, with a mean of about 2 parts per million (NAS, 1974).

717. An estimated 368 pounds of dioxin was sprayed in Vietnam over a six-year period.

The industrial history of the dioxins

718. In 1872, two German chemists prepared the first chlorinated dioxin, but its structure was not understood until much later.

719. Dioxins have no commercial use or economic value. Most are formed and released as by-products of human activities, especially of industrial processes and incomplete combustion processes

720. There were a number of dioxin-related accidents during the 20th century.

721. On March 8, 1949, a massive explosion occurred at the Monsanto plant in Nitro, West Virginia, when a pressure valve failed on a reaction vessel involved in the manufacture of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T).

722. Within days, workers experienced skin eruptions. Many were soon diagnosed with chloracne. A confidential medical report at the time said the explosion "caused a systemic intoxication in the workers involving most major organ systems." Court records indicate that 226 plant workers became ill.

723. Monsanto downplayed the impact, stating that the contaminant affecting workers was "fairly slow acting" and caused "only an irritation of the skin."

724. In the meantime, the Nitro plant continued to produce herbicides, rubber products, and other chemicals. In the 1960s, the factory manufactured Agent Orange, creating TCDD as a by–product.

725. Some of the dioxin-contaminated wastes from the Nitro plant were burned in incinerators, some dumped in landfills or storm drains, some allowed to run into streams.

726. Monsanto stopped producing chemicals at the Nitro plant in 1969.

727. Many products manufactured by Monsanto were contaminated with dioxins, including the widely used household disinfectant Lysol, and the known defoliant Agent Orange, used in the Vietnam War.

728. In the 1970s at Times Beach, Missouri, oil used for spraying streets for dust control had been highly contaminated with dioxins. The US Environmental Protection Agency (EPA), eventually ordered the town to be evacuated and the area cleaned. During the process, more than 265,000 tons of dioxin-contaminated soil was incinerated and the waste ash buried on site. Today, that place is a state park commemorating the historic Route 66.

729. In 1976, an explosion occurred in a 2,4,5-trichlorophenol reactor of the ICMESA chemical plant in Seveso, 25 km north of Milan, Italy. Thousands of people were exposed to substantial quantities of 2,3,7,8-TCDD.

730. In 1999, 500 tons of fodder contaminated with 50 kg of PCBs and 1 g of dioxins, were distributed to animal farms mostly in Belgium, but also in the Netherlands, France, and Germany. After a few months, the first signs of toxicity started to appear at chicken farms. All poultry and derived products were removed from the market and most of them were destroyed, but studies showed that the body burden was tripled in people exposed to contaminated food.

731. There was an attempt to assassinate Ukrainian president Viktor Yushchenko with TCDD in 2004. His blood serum level of TCDD was 50,000 times the level in general population. He suffered severe health consequences and visible disfiguration typical of acute chloracne.

Mechanism of physiological and pharmacological action

732. Once dioxins enter the human body, a part is metabolized and eliminated and the other part is stored in body fat, a process known as bioaccumulation.

733. To be eliminated from the body, dioxins first have to be converted to polar derivatives because they are not water-soluble and cannot be readily eliminated directly by the kidneys.

734. Metabolism of TCDD in the liver is not "normal." Because TCDD is chemically stable, lipophilic, and resists degradation it tends to bioaccumulate.

735. When metabolism of TCDD does occur, the rate it is slow enough to allow accumulation and persistence in lipid tissue. Storage of TCDD in fatty tissue creates a constant or chronic source of exposure which leads to more damaging effects then occur with a nonfatal single or acute exposure.

736. Biological half-life differs between congeners. The half-life of 2,3,7,8-TCDD is between five and ten years. Elimination depends on dose, age, and quantity of body fat.

Molecular mechanism of TCDD action

737. The cytochrome P450 superfamily of enzymes plays a critical role in the oxygenation of xenobiotics such as TCDD.

738. Oxygenation is the first step in their conversion to polar substrates, which can be excreted from the body. The liver microsomal P450 enzymes involved in xenobiotic biotransformation belong to three main P450 gene families: CYP1, CYP2, and CYP3.

739. P450 1A1, encoded by the CYP1A1 gene, oxygenates lipophilic chemicals such as dioxins and is induced as a result of increased transcription of the CYP1A1 gene.

TCDD and the aryl hydrocarbon (AH) receptor (AhR)

740. TCDD and dioxin-like compounds act via the aryl hydrocarbon (AH) receptor, a specific receptor present in all cells.

741. AhR is a cytosolic receptor that binds to different environmental pollutants, including dioxins, and mediates their carcinogenic action.

742. AhR is a ligand-activated nuclear transcription factor which mediates cellular response in terms of expression regulation of a large number of genes.

743. AhR upregulates a number of xenobiotic metabolizing enzymes such as cytochrome P450 1A1 (CYP1A1), P450 1A2 (CYP1A2), and P450 1B1 (CYP1B1) as well as the phase II enzymes, glutathione S-transferase A1 (GST-A1) and UDP-glucuronosyltransferases (UGT1-06). However, CYP1A1 is the most potently induced gene following AhR activation.

744. The AH receptor is a transcription factor which is involved in expression of genes including those activating the breakdown of foreign and often toxic compounds.

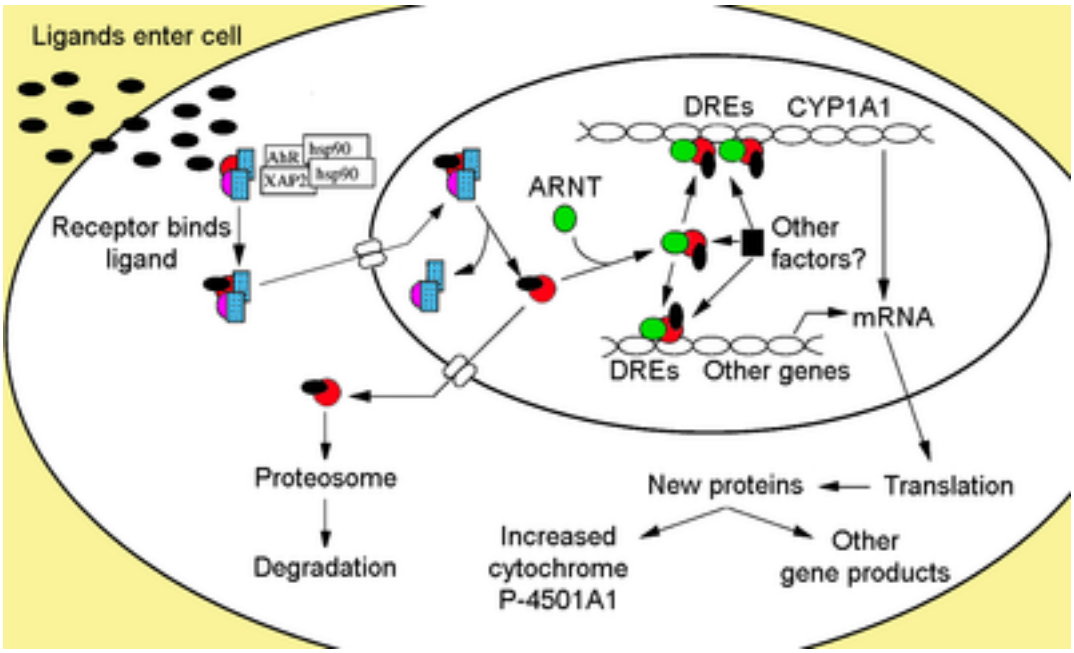
745. Phylogenetically, the AH receptor is highly conserved transcription factor found in all vertebrates over at least the last 500 million years.

746. The physiological function of the AH receptor is to increase the activity of enzymes breaking down foreign chemicals or normal chemicals of the body as needed and there may be other functions, related to growth of various organs or other regulatory functions.

747. A basal degree of AH receptor activation is necessary to achieve normal physiological function.

748. The following graphic representation of the AhR Signaling Pathway is a true and accurate copy from Denison MS, Nagy SR (2003). “Activation of the aryl hydrocarbon receptor by

structurally diverse exogenous and endogenous chemicals". Annu. Rev. Pharmacol. Toxicol. 43: 309–34.



749. After TCDD exposure, AhR binds to a ligand, then translocates into the nucleus, where it forms an active heterodimer with aromatic hydrocarbon nuclear translocator (ARNT).

750. The cytosolic aryl hydrocarbon receptor (AhR) mediates the carcinogenic action of TCDD. It binds to dioxin, translocates to nucleus and together with hydrocarbon nuclear translocator (ARNT) and xenobiotic responsive element (XRE) increases the expression of CYP1A1.

751. This AhR/Arnt heterodimer binds to a specific xenobiotic responsive element (XRE) located upstream in the promoter region of the target gene, resulting in increased expression of the gene (37-39). A study which investigated the effects of PAHs and dioxins on 1152 genes in waste incineration workers occupationally exposed to dioxin reported upregulation of five genes involved in oxidative stress, including GSTA1 (40).

752. Activated AhR also interacts with other signaling proteins involved in the regulation of the cell cycle and apoptosis. It can alter cell function such as growth and differentiation. In addition, dioxins can induce responses caused by other signaling pathways.

753. Although AhR signaling is the first step of dioxin toxicity, there can be a variety of biochemical and toxicological responses to dioxin exposure.

754. There may also be an AhR-independent pathway of dioxin-induced toxicity.

755. Excessive and persistent stimulation of AH receptor , however, leads to a multitude of adverse effects.

756. The endocrine disrupting activities of TCDD seem to be anti-estrogenic when estrogen is present or in high concentration in the body, and estrogenic in the absence of estrogen.

757. TCDD may in some conditions potentiate the carcinogenic effects of other compounds. An example is benzo(a)pyrene that is metabolized in two steps, oxidation and conjugation. Oxidation produces epoxide carcinogens that are rapidly detoxified by conjugation, but some molecules may escape to the nucleus of the cell and bind to DNA causing a mutation, resulting in cancer initiation.

758. When TCDD increases the activity of oxidative enzymes more than conjugation enzymes, the epoxide intermediates may increase, increasing the possibility of cancer initiation. Thus a beneficial activation of detoxifying enzymes may lead to deleterious side effects.

759. Male-mediated toxicological effects from either the long-term effects of past exposure to TCDD or from the continuing presence of physiologically active levels of TCDD within the bodies of those exposed can occur through decreased quality of

sperm resulting from gonadal lesion during spermatogenesis, altered hormonal regulation, or transmittable defects in heritable characteristics of sperm; potentiation of xenobiotic metabolism in gonadal tissue; transfer of TCDD into seminal fluid and subsequent transfer to the womb.

760. The presence of TCDD prevents the normal action of the AhR signaling pathway in the maintenance of homeostasis and induces changes in the expression of genes and promotes the production of toxic metabolites.

761. Complex and intricate interactions between the AhR and ER (estrogen receptor) signaling pathways, suggesting that dioxin may also have indirect effects on some ER-mediated endpoints via AhR signaling.

TCDD is physiologically active at low doses

762. In 2001, the National Toxicology Program (NTP) defined low-dose effects as any biological changes occurring in the range of typical human exposures or occurring at doses lower than those typically used in standard testing protocols, i.e. doses below those tested in traditional toxicology assessments.

763. Other definitions of low dose include a dose below the lowest dose at which a biological change or damage for a specific chemical has been measured in the past, i.e. any dose below the lowest observed effect level or lowest observed adverse effect level (LOAEL).

764. A dose administered to an animal that produces blood concentrations of that chemical in the range of what has been measured in the general human population, not exposed occupationally is often referred to as an environmentally relevant dose because it creates an internal dose relevant to concentrations of the chemical measured in humans and takes into account differences in chemical metabolism and pharmacokinetics, the absorption, distribution, and excretion

of the chemical, across species and reduces the importance of route of exposure by directly comparing similar blood or other tissue concentrations across model systems and experimental paradigms.

765. The definition of a non-monotonic dose-response curve (NMDRC) is based upon the mathematical definition of non-monotonicity: that the slope of the dose-response curve changes sign from positive to negative or vice versa at some point along the range of doses examined. NMDRCs need not span from true low doses to high, pharmacologically relevant, doses. The observation of non-monotonicity makes no assumptions about the range of doses tested.

766. NMDRCs challenge the traditional approaches in toxicology taken from the early days of Industrial Hygiene prior to the DDT litigation in the 1960s which assume that the dose-response curve is monotonic. This assumption justifies using high-dose testing as the standard for assessing chemical safety, but when a compound has a NMDRC, high-dose testing regimes cannot be used to assess the safety of low doses.

767. For all monotonic responses, the observed effects may be linear or nonlinear, but the slope does not change sign. This assumption justifies using high-dose testing as the standard for assessing chemical safety. When it is violated, high-dose testing regimes cannot be used to assess the safety of low doses.

768. Estimations of beneficial or adverse effects cannot be ascertained from the direction of the slope of a dose-response curve, however, any biological effect, whether it is observed to follow linear relationships with administered dose or not, provides conclusive evidence that a chemical has biological activity, and other biological effects are likely to be present but may remain undetected or unexamined.

769. The phenoxy herbicides and the cacodylates were designed to have biological effects—kill plants. The question of whether these chemicals have biological effects is answered unequivocally in their design; the question is what other effects are induced by these biologically active agents, not whether they exist.

TCDD is an endocrine disruptive chemical (EDC)

770. TCDD is one of a small group of chemicals which target a number of endocrine endpoints including many that act as estrogens and anti-androgens as well as others that affect the metabolism, secretion, or synthesis of a number of hormones and manifest low-dose effects.

771. Low level effects associated with TCDD involve complex physiological processes including include spermatogenesis, immune function and oxidative stress, tooth and bone development, female reproduction, mammary gland functions, and behavior.

772. TCDD has a half-life of approximately 10 yrs, and additional factors influence TCDD pharmacokinetics including the amount of body fat present.

773. TCDD binds to the aryl hydrocarbon receptor (AhR), and there are also important non-genomic pathways affected by TCDD that are mediated by AhR. Because other adverse effects, including those related to the endocrine-disrupting activities of TCDD, occur at similar doses or body burdens across animal species, AhR affinity alone cannot predict all the effects of TCDD.

774. The mechanisms responsible for many of the endocrine-disrupting activities of TCDD are poorly understood.

775. Teratogenic effects of TCDD have been documented involving most organs and systems in the body. High doses that did not

produce lethality caused severe weight loss, intestinal hemorrhaging, alopecia, chloracne, edemas, and severe liver damage.

776. TCDD is lipophilic. It is concentrated in the fat component of breast milk and therefore passed in large quantities from a nursing mother to her infant.

777. Prenatal exposure to low doses of TCDD affects sperm-related endpoints in adulthood.

778. TCDD affects the weight of the adult prostate and the timing of puberty.

779. Low doses of TCDD affect a multitude of endpoints in animals, altering immune function, indicators of oxidative stress, bone and tooth development, female reproduction and timing of puberty, mammary gland development and susceptibility to cancers, and behaviors.

Adverse health effects attributable to TCDD

780. Of 210 dioxin and dibenzofuran congeners, only 17 are considered toxic.

781. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is the best known and the most toxic dioxin. It has the highest toxic potential, TEF (toxic equivalent factor). The toxic potentials of the other PCDDs and PCDFs are defined in comparison to TCDD.

782. In September 1971, an early account of research on the appearance of TCDD in trace quantities in samples of 2,4,5-T was presented at a session on the origin and fate of chlorodioxins at the American Chemical Society meeting.

783. TCDD was defined to be the most toxic of all chlorodibenzodioxins studied at that time. Further accounts of dioxin's toxicity were presented at a meeting on "Perspectives on Chlorinated Dibenzodioxins and Dibenzofurans" sponsored

by the National Institute of Environmental Health Sciences in North Carolina in April 1973.

784. The major findings indicated "… that there was a variation of sensitivity among species, the liver was the target organ, the toxic effects were delayed after absorption, and the mechanism of teratogenesis was still incompletely understood … patterns of absorption and of distribution among organs were beginning to emerge".

785. Dr. K. Diane Courtney, the principal investigator in the Study concluded that all species of pregnant animals treated or exposed to TCDD responded with abortions, fetal death, fetal toxicities or malformations. The routes of exposure were oral, diet or dermal.

786. TCDD can cause male-mediated transmittable damage manifested by birth defects, miscarriages, and other untoward pregnancy outcomes when the intensity and duration exposure to TCDD is appropriately paired with the timing of that exposure in relationship to spermatogenesis and the participation of the male in reproduction.

787. In 1974, the National Academy of Sciences' Committee on the Effects of Herbicides in Vietnam reported that "TCDD is extremely toxic to some laboratory animals. … TCDD has been found to be teratogenic in mice; results with other laboratory animals have not been conclusive. The lethal dose in humans is not known, nor is that required to cause birth defects, if indeed there is such an activity. TCDD is strongly implicated as the main cause of chloracne, a disease that has affected employees in some plants manufacturing 2,4,5-T or its precursor, 2,4,5-trichlorophenol."

788. In 2000, the Expert Group of the World Health Organization considered developmental toxicity as the most pertinent risk of dioxins to human beings.

789. The most sensitive population to dioxin exposure is the fetuses and infants, especially those exposed to high levels of dioxins through mothers' milk.

790. Short-term exposure to high levels of dioxins damage liver function and cause chloracne.

791. Long-term exposures to dioxins are associated with disturbances in the nervous, immune, reproductive, and endocrine system.

792. TCDD's persistence in the body can cause atherosclerosis, hypertension, diabetes, and nervous system damage.

793. The International Agency for Research on Cancer (IARC) and the WHO classified TCDD as a "known human carcinogen."

794. Exposure to TCDD is associated with increases in blood cholesterol and triglyceride levels.

795. Exposure to TCDD initiates enzyme induction and disturbance of porphyrin metabolism in the liver.

796. TCDD is immunosuppressive and individuals chronically exposed to TCDD are subject to increased occurrence of infections, neoplastic processes (cancer), and systemic diseases.

797. TCDD can be teratogenic, mutagenic, fetotoxic, embryotoxic and gonadotoxic.

798. Developmental effects occur at very low doses in animals. They include frank teratogenicity such as cleft palate and hydronephrosis. Very low doses perturb the development of sexual organs in rodents and the development of teeth in rats.

799. After the Seveso incident, there was an increased incidence of gastrointestinal, lymphatic, and hematopoietic cancers and soft-tissue sarcomas. Studies conducted 10 to 20 years after the accident all found increased death rate from all cancer

types, especially in the male population. A significant increase in lymphohaemopoietic neoplasms was found in both sexes, as well as in non-Hodgkin's lymphoma (NHL) and myeloid leukemia. Mortality due to diabetes mellitus was substantial in women, while chronic circulatory and respiratory diseases were slightly higher than in the general population. Tooth deformities were also after the Seveso accident.

800. TCDD can cause male-mediated transmittable damage manifested by birth defects, miscarriages, and other untoward pregnancy outcomes when the intensity and duration exposure to TCDD is appropriately paired with the timing of that exposure in relationship to spermatogenesis and the participation of the male in reproduction.

801. Gestational exposure to TCDD produces fetotoxic responses in most laboratory mammals such as decreased fetal growth, prenatal mortality, and nervous system changes.

802. Increased metabolism in pregnant women and mobilization of accumulated dioxins in fat tissue present a threat to fetuses and infants.

803. Dioxins can pass through the placenta and reach the fetus and exposure continues in infants through breastfeeding.

804. Prenatal exposure could be even more relevant than postnatal exposure.

THE "PRESUMPTIVE" DISEASES AND SYNDROMES

805. The Veterans Administration of the United States (VA) has recognized certain cancers and other diseases and syndromes as presumptively associated with exposure to dioxin contaminated phenoxy herbicides and the cacodylates.

806. At this time the "Presumptive" diseases and syndromes are: AL Amyloidosis, Chronic B-cell Leukemias, Chloracne or similar acneform disease, Diabetes Mellitus Type 2, Hodgkin's

Disease, Ischemic Heart Disease, Multiple Myeloma, Non-Hodgkin's Lymphoma, Parkinson's Disease, Early-Onset Peripheral Neuropathy, Porphyria Cutanea Tarda, Prostate Cancer, Respiratory Cancers including lung cancer, Cancers of the lung, larynx, trachea, and bronchus, Soft Tissue Sarcomas other than osteosarcoma, chondrosarcoma, Kaposi's sarcoma, or mesothelioma.

Chloracne and similar acneform disease

807. Chloracne is an acne-like eruption of blackheads, cysts, and pustules associated with over-exposure to certain halogenated aromatic compounds, such as chlorinated dioxins and dibenzofurans. The lesions are most frequently found on the cheeks, behind the ears, in the armpits and groin region.

808. The condition was first described in German industrial workers in 1897 by Siegfried Bettmann and was initially believed to be caused by exposure to chlorine, hence the name "chloracne." The substances that may cause chloracne are now collectively known as chloracnegens.

809. In 1957, Dr. W. Sandermann of the Institute of Wood Chemistry in Hamburg published results of his synthesis of TCDD. While working on the synthesis, his laboratory assistant was exposed to the substance being tested when some of it blew into his face. He soon developed skin lesions over his entire face and decided to seek treatment from Dr. Karl Schulz, a dermatologist who treated chemical workers and had observed chloracne in some of them. After examining Sandermann's laboratory assistant, Schulz identified the skin lesions on his face as chloracne. When the laboratory assistant explained that the compound he was synthesizing was TCDD, Schulz was the first to correlate the presence of chloracne with exposure to dioxin. To further confirm this assumption, Schulz

applied a TCDD solution to the skin of his forearm and noted that chloracne appeared.

810. Chloracne is particularly linked to toxic exposure to dioxins which are byproducts of many chemical processes, including the manufacture of phenoxy herbicides and is considered a clinical sign of dioxin exposure.

811. Chloracnegens are lipid-soluble and persist in the body fat for a very long period following exposure. Chloracne is a chronic inflammatory condition that results from this persistence, in combination with the toxin's chemical properties.

812. In rodent models, the toxin activates a series of receptors promoting macrophage proliferation, inducing neutrophilia, leading to a generalized inflammatory response in the skin which may also be augmented by induction of excess tumor necrosis factor in the blood serum.

813. The inflammatory processes lead to the formation of keratinous plugs in skin pores, forming yellowish cysts and dark pustules. The associated pus is usually greenish in color. The skin lesions occur mainly in the face, but in more severe cases they involve the shoulders and chest, the back, and the abdomen. In advanced cases, the lesions appear also on the arms, thighs, legs, hands and feet.

814. Chloracne is very often seen in combination with hyperhidrosis, clammy, sweaty skin, and porphyria cutanea tarda, a skin condition characterized by increased pigmentation, hair coarsening, and blistering.

AL Amyloidosis

815. Amyloid light-chain (AL) amyloidosis, primary systemic amyloidosis (PSA) or just primary amyloidosis is the most common form of systemic amyloidosis in the US. The disease is caused when a person's antibody-producing cells do not

function properly and produce abnormal protein fibers made of components of antibodies called light chains. These light chains come together to form amyloid deposits in different organs which can cause serious damage to these organs. Abnormal light chains in blood and urine are sometimes referred to as “Bence Jones protein”.

816. AL amyloidosis can affect a wide range of organs, and consequently present with a range of symptoms. The kidneys are the most commonly affected organ in AL amyloidosis. Symptoms of kidney disease and renal failure can include fluid retention, swelling, and shortness of breath.

817. In addition to kidneys, AL amyloidosis may affect the heart, peripheral nervous system, gastrointestinal tract, blood, lungs and skin. Heart complications, which affect more than a third of AL patients, include heart failure and irregular heartbeat.

818. Other symptoms can include stroke, gastrointestinal disorders, enlarged liver, diminished spleen function, diminished function of the adrenal and other endocrine glands, skin color change or growths, lung problems, bleeding and bruising problems, fatigue and weight loss.

Chronic B-cell Leukemias

819. B-cell chronic lymphocytic leukemia (B-CLL), also known as chronic lymphoid leukemia (CLL), is the most common type of leukemia, a type of cancer of the white blood cells in adults. CLL affects B cell lymphocytes, which originate in the bone marrow, develop in the lymph nodes, and normally fight infection by producing antibodies.

820. In CLL, B cells grow in an uncontrolled manner and accumulate in the bone marrow and blood, where they crowd out healthy blood cells. CLL is a stage of small lymphocytic lymphoma (SLL), a type of B-cell lymphoma, which presents

primarily in the lymph nodes. CLL and SLL are considered the same underlying disease, just with different appearances.

821. Complications include Richter's syndrome, hypogammaglobulinemia leading to recurrent infection, warm autoimmune hemolytic anemia in 10–15% of patients, and transformation to high-grade lymphoma.

822. Chronic lymphocytic leukemia may transform into Richter's syndrome, the development of fast-growing diffuse large B cell lymphoma, prolymphocytic leukemia, Hodgkin's lymphoma, or acute leukemia in some patients.

823. Gastrointestinal (GI) involvement can occur with chronic lymphocytic leukemia. Some of the reported manifestations include intussusception, small intestinal bacterial contamination, colitis, and others. Usually, GI complications with CLL occur after Richter transformation.

Diabetes Mellitus Type 2

824. Diabetes mellitus type 2, also known as type 2 diabetes, noninsulin-dependent diabetes mellitus (NIDDM), and adult-onset diabetes is a long-term metabolic disorder that characterized by high blood sugar levels resulting from the body's inability to respond properly to the hormone insulin. Long-term complications from high blood sugar include heart disease, strokes, diabetic retinopathy which can result in blindness, kidney failure, and poor blood flow in the limbs which may lead to amputations. The sudden onset of hyperosmolar hyperglycemic state may occur; however, ketoacidosis is uncommon.

825. Type 2 diabetes is typically a chronic disease associated with a ten-year-shorter life expectancy partly due to a number of complications with which it is associated, including: two to four times the risk of cardiovascular disease, including ischemic heart disease and stroke; a 20-fold increase in lower

limb amputations, and increased rates of hospitalizations. It has also been associated with an increased risk of cognitive dysfunction and dementia through disease processes such as Alzheimer's disease and vascular dementia. Other complications include acanthosis nigricans, sexual dysfunction, and frequent infections.

826. Type 2 diabetes is due to insufficient insulin production from beta cells in the setting of insulin resistance, the inability of cells to respond adequately to normal levels of insulin. It occurs primarily within the muscles, liver, and fat tissue. In the liver, insulin normally suppresses glucose release. However, in the setting of insulin resistance, the liver inappropriately releases glucose into the blood. The proportion of insulin resistance versus beta cell dysfunction differs among individuals, with some having primarily insulin resistance and only a minor defect in insulin secretion and others with slight insulin resistance and primarily a lack of insulin secretion.

827. Other potentially important mechanisms associated with type 2 diabetes and insulin resistance include: increased breakdown of lipids within fat cells, resistance to and lack of incretin, high glucagon levels in the blood, increased retention of salt and water by the kidneys, and inappropriate regulation of metabolism by the central nervous system. However, not all people with insulin resistance develop diabetes, since an impairment of insulin secretion by pancreatic beta cells is also required.

Hodgkin's Disease

828. Hodgkin's lymphoma (HL) is a malignant lymphoma (cancer) characterized by progressive enlargement of the lymph nodes, liver, and spleen, and by progressive anemia.

829. There are two major types of Hodgkin lymphoma: classical Hodgkin lymphoma and nodular lymphocyte-predominant Hodgkin lymphoma.

830. Hodgkin lymphoma may be treated with chemotherapy, radiation therapy, and stem cell transplant. The choice of treatment often depends on how advanced the cancer is and whether or not it has favorable features.

831. It was named after the English physician Thomas Hodgkin, who first described the condition in 1832 although Hodgkin noted that perhaps the earliest reference to the condition was provided by Marcello Malpighi in 1666.

832. Hodgkin's lymphoma was one of the first cancers which could be treated using radiation therapy and, later, it was one of the first to be treated by combination chemotherapy.

Ischemic Heart Disease

833. Ischemic heart disease, also known as atherosclerotic heart disease, atherosclerotic and cardiovascular disease, coronary heart disease and coronary artery disease (CAD) is a group of diseases that includes: stable angina, unstable angina, myocardial infarction, and sudden cardiac death.

834. Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the myocardial cells. Myocardial cells may die from lack of oxygen and this is called a myocardial infarction (commonly called a heart attack). It leads to heart muscle damage, heart muscle death and later myocardial scarring without heart muscle regrowth. Chronic high-grade stenosis of the coronary arteries can induce transient ischemia which leads to the induction of a ventricular arrhythmia, which may terminate into ventricular fibrillation leading to death.

835. Typically, coronary artery disease occurs when part of the smooth, elastic lining inside a coronary artery which supplies blood to the heart muscle develops atherosclerosis and the lining of the artery becomes hardened, stiffened, and swollen with calcium deposits, fatty deposits, and abnormal inflammatory cells which form a plaque. Deposits of calcium phosphates (hydroxyapatites) in the muscular layer of the blood vessels appear to play not only a significant role in stiffening arteries but also for the induction of an early phase of coronary arteriosclerosis. This can be seen in a so-called metastatic mechanism of calciphylaxis as it occurs in chronic kidney disease and haemodialysis.

Multiple Myeloma

836. Multiple myeloma (MM), also known Plasma cell myeloma, myelomatosis, or Kahler's disease is a cancer of plasma cells, a type of white blood cell normally responsible for producing antibodies. The word myeloma is from the Greek myelo- meaning "marrow" and -oma meaning "tumor".

837. Complications may include amyloidosis.

838. The underlying mechanism involves abnormal plasma cells producing abnormal antibodies which can cause kidney problems and overly thick blood. The plasma cells can also form a mass in the bone marrow or soft tissue. When only one mass is present, it is known as a plasmacytoma while more than one is known as multiple myeloma.

839. Multiple myeloma is considered treatable but generally incurable. Without treatment, typical survival is seven months. With current treatments, survival is usually 4–5 years. Remissions may be brought about with steroids, chemotherapy, thalidomide or lenalidomide, and stem cell transplant. Bisphosphonates and radiation therapy are sometimes used to reduce pain from bone lesions.

840. Multiple myeloma develops in B lymphocytes after they have left the part of the lymph node known as the germinal center. The normal cell line most closely associated with MM cells is generally taken to be either an activated memory B cell or the precursor to plasma cells, the plasmablast.

841. B lymphocytes start in the bone marrow and move to the lymph nodes. As they progress, they mature and display different proteins on their cell surface. When they are activated to secrete antibodies, they are known as plasma cells.

842. The immune system keeps the proliferation of B cells and the secretion of antibodies under tight control. When chromosomes and genes are damaged, often through rearrangement, this control is lost. Often, a promoter gene moves or translocates to a chromosome where it stimulates an antibody gene to overproduction.

843. A chromosomal translocation between the immunoglobulin heavy chain gene on chromosome 14, locus q32 and an oncogene, often 11q13, 4p16.3, 6p21, 16q23 and 20q11, is frequently observed in patients with multiple myeloma. This mutation results in dysregulation of the oncogene which is thought to be an important initiating event in the pathogenesis of myeloma. The result is a proliferation of a plasma cell clone and genomic instability that leads to further mutations and translocations. The chromosome 14 abnormality is observed in about 50% of all cases of myeloma. Deletion of (parts of) chromosome 13 is also observed in about 50% of cases.

844. Production of cytokines (especially IL-6) by the plasma cells causes much of their localized damage, such as osteoporosis, and creates a microenvironment in which the malignant cells

thrive. Angiogenesis, the creation of new blood vessels, is increased.

845. The produced antibodies are deposited in various organs, leading to kidney failure, polyneuropathy, and various other myeloma-associated symptoms.

Non-Hodgkin's Lymphoma

846. Non-Hodgkin lymphoma (NHL) is a group of blood cancers that includes all types of lymphoma except Hodgkin's lymphomas. Some forms are slow growing while others are fast growing.

847. Lymphomas are types of cancer that develops from lymphocytes, a type of white blood cell. Risk factors include poor immune function, autoimmune diseases, Helicobacter pylori infection, hepatitis C, obesity, and Epstein-Barr virus infection.

848. The World Health Organization (WHO) classifies lymphomas into five major groups, including one for Hodgkin's lymphoma. Within the four groups for NHL there are over 60 specific types of lymphoma.

849. Treatment depends on if the lymphoma is slow or fast growing and if it is in one area or many areas. Treatments may include chemotherapy, radiation, immunotherapy, targeted therapy, stem cell transplantation, surgery, or watchful waiting. If the blood becomes overly thick due to antibodies, plasmapheresis may be used. Radiation and some chemotherapy, however, increase the risk of other cancers, heart disease or nerve problems over the subsequent decades.

Parkinson's Disease

850. Parkinson's Disease, also known as idiopathic or primary Parkinsonism, hypokinetic rigid syndrome, and paralysis agitans is a long-term degenerative disorder of the central

nervous system that mainly affects the motor system. The main motor symptoms the most obvious of which are shaking, rigidity, slowness of movement, and difficulty with walking, are collectively called "parkinsonism" or a "parkinsonian syndrome." The disease is named after the English doctor James Parkinson, who published the first detailed description in *An Essay on the Shaking Palsy*, in 1817.

851. The cause of Parkinson's disease is generally unknown, but believed to involve both genetic and environmental factors. The motor symptoms of the disease result from the death of cells in the substantia nigra, a region of the midbrain. This results in not enough dopamine in these areas. The reason for this cell death is poorly understood, but involves the build-up of proteins into Lewy bodies in the neurons.

852. There is no cure for Parkinson's disease. Initial treatment is typically with the antiparkinson medication L-DOPA (levodopa), with dopamine agonists being used once levodopa becomes less effective. As the disease progresses and neurons continue to be lost, these medications become less effective while at the same time they produce a complication marked by involuntary writhing movements.

853. The average life expectancy following diagnosis is between 7 and 14 years. Death from aspiration pneumonia is twice as common in individuals with PD as in the healthy population.

Peripheral Neuropathy, Early-Onset

854. Peripheral neuropathy (PN) is damage to or disease affecting nerves, which may impair sensation, movement, gland or organ function, or other aspects of health, depending on the type of nerve affected. In conventional medical usage, the word *neuropathy* (neuro-, "nervous system" and -pathy, "disease of") without modifier usually means *peripheral neuropathy*.

855. Neuropathy affecting just one nerve is called "mononeuropathy" and neuropathy involving multiple nerves in roughly the same areas on both sides of the body is called "symmetrical polyneuropathy" or simply "polyneuropathy." When two or more (typically just a few, but sometimes many) separate nerves in disparate areas of the body are affected it is called "mononeuritis multiplex," "multifocal mononeuropathy," or "multiple mononeuropathy."

856. Peripheral neuropathy may be chronic (a long-term condition where symptoms begin subtly and progress slowly) or acute (sudden onset, rapid progress, and slow resolution).

857. Neuropathy may cause painful cramps, fasciculations (fine muscle twitching), muscle loss, bone degeneration, and changes in the skin, hair, and nails. Additionally, motor neuropathy may cause impaired balance and coordination or, most commonly, muscle weakness; sensory neuropathy may cause numbness to touch and vibration, reduced position sense causing poorer coordination and balance, reduced sensitivity to temperature change and pain, spontaneous tingling or burning pain, or skin allodynia (severe pain from normally nonpainful stimuli, such as light touch); and autonomic neuropathy may produce diverse symptoms, depending on the affected glands and organs, but common symptoms are poor bladder control, abnormal blood pressure or heart rate, and reduced ability to sweat normally.

Porphyria Cutanea Tarda

858. *Porphyria cutanea tarda* (PCT) is the most common subtype of porphyria. PCT is a chronic condition. The disease is named because it is a porphyria that often presents with skin manifestations, onycholysis and blistering of the skin in areas that receive higher levels of exposure to sunlight.

859. In addition to the symptomatic manifestation of the disease in the skin, chronic liver problems are extremely common, including hepatic fibrosis, cirrhosis, and inflammation.

860. The primary cause of this disorder is a deficiency of uroporphyrinogen decarboxylase (UROD), a cytosolic enzyme that is a step in the enzymatic pathway that leads to the synthesis of heme. Uroporphyrinogen decarboxylase occurs in nature as a homodimer of two subunits. It participates in the heme synthesis pathway and is active in the cytosol. This enzymatic conversion results in coproporphyrinogen III as the primary product. This is accomplished by the clockwise removal of the four carboxyl groups present in the cyclic uroporphyrinogen III molecule. Therefore, a deficiency in this enzyme causes the buildup of uroporphyrinogen and hepta-carboxylic porphyrinogen, and to a lesser extent hexa-carboxylic porphyrinogen, and penta-carboxylic porphyrinogen in the urine.

861. The dermatological symptoms of PCT that include blistering and lesions on sun-exposed areas of the skin are caused by a buildup of porphyrin compounds, specifically uroporphyrinogen, close to the surface of the skin that have been oxidized by free radicals or sunlight. The oxidized porphyrins initiate degranulation of dermal mast cells which release proteases that catabolize the surrounding proteins. This begins a cell mediated positive feedback loop which matches the description of a type 4 delayed hypersensitivity reaction. The resulting blisters therefore do not appear immediately but begin to show up 2–3 days after sun exposure. Due to the highly conjugated structure of porphyrins involving alternating single and double carbon bonds, these compounds exhibit a deep purple color, resulting in the discoloration observed in the skin.

Prostate Cancer

862. Prostate cancer is classified as an adenocarcinoma, or glandular cancer, that begins when normal semen-secreting prostate gland cells mutate into cancer cells. The region of prostate gland where the adenocarcinoma is most common is the peripheral zone. Initially, small clumps of cancer cells remain confined to otherwise normal prostate glands, a condition known as carcinoma in situ or prostatic intraepithelial neoplasia (PIN). Although there is no proof that PIN is a cancer precursor, it is closely associated with cancer. Over time, these cancer cells begin to multiply and spread to the surrounding prostate tissue, the stroma, forming a tumor. Eventually, the tumor may grow large enough to invade nearby organs such as the seminal vesicles or the rectum, or the tumor cells may develop the ability to travel in the bloodstream and lymphatic system.

863. Prostate cancer is considered a malignant tumor because it is a mass of cells that can invade other parts of the body, a process called metastasis. Prostate cancer most commonly metastasizes to the bones, lymph nodes, and may invade rectum, bladder and lower ureters after local progression. The route of metastasis to bone is thought to be venous as the prostatic venous plexus draining the prostate connects with the vertebral veins.

864. The prostate glands require male hormones, androgens, to function properly. Androgens include testosterone, which is made in the testes; dehydroepiandrosterone, made in the adrenal glands; and dihydrotestosterone, which is converted from testosterone within the prostate itself.

865. The prostate is a zinc-accumulating, citrate-producing organ. The protein ZIP1 is responsible for the active transport of zinc into prostate cells. One of the zinc's important roles is to

change the metabolism of the cell in order to produce citrate, an important component of semen. The process of zinc accumulation, alteration of metabolism, and citrate production is energy inefficient, and prostate cells sacrifice enormous amounts of energy (ATP) in order to accomplish this task. Prostate cancer cells are generally devoid of zinc. This allows prostate cancer cells to save energy not making citrate and utilize the new abundance of energy to grow and spread. The absence of zinc is thought to occur via a silencing of the gene that produces the transporter protein ZIP1. ZIP1 is now called a tumor suppressor gene product for the gene SLC39A1. Strategies which transport zinc into transformed prostate cells effectively eliminate these cells in animals. Zinc inhibits NF-κB pathways, is anti-proliferative and induces apoptosis in abnormal cells.

866. Loss of cancer suppressor genes, early in prostatic carcinogenesis, have been localized to chromosomes *8p*, *10q*, *13q*, and *16q*.

867. RUNX2 is a transcription factor that prevents cancer cells from undergoing apoptosis thereby contributing to the development of prostate cancer.

868. The PI3k/Akt signaling cascade works with the transforming growth factor beta/SMAD signaling cascade to ensure prostate cancer cell survival and protection against apoptosis. X-linked inhibitor of apoptosis (XIAP) is hypothesized to promote prostate cancer cell survival and growth and is a target of research because if this inhibitor can be shut down then the apoptosis cascade can carry on its function in preventing cancer cell proliferation. Macrophage inhibitory cytokine-1 (MIC-1) stimulates the focal adhesion kinase (FAK) signaling pathway which leads to prostate cancer cell growth and survival.

869. The androgen receptor helps prostate cancer cells to survive.

870. Prostate specific membrane antigen (PSMA) stimulates the development of prostate cancer by increasing folate levels for the cancer cells to use to survive and grow. PSMA increases available folates for use by hydrolyzing glutamated folates.

Respiratory Cancers

871. Respiratory cancers are cancers of the lung, larynx, trachea, and bronchus

872. Most cancers that start in the lung, known as primary lung cancers, are carcinomas. The two main types are small-cell lung carcinoma (SCLC) and non-small-cell lung carcinoma (NSCLC).

873. Lung cancer is initiated by activation of oncogenes or inactivation of tumor suppressor genes. Carcinogens cause mutations in these genes which induce the development of cancer.

874. Mutations in the K-ras proto-oncogene are responsible for 10–30% of lung adenocarcinomas. About 4% of non-small-cell lung carcinomas involve an EML4-ALK tyrosine kinase fusion gene.

875. Epigenetic changes—such as alteration of DNA methylation, histone tail modification, or microRNA regulation—may lead to inactivation of tumor suppressor genes.

876. The epidermal growth factor receptor (EGFR) regulates cell proliferation, apoptosis, angiogenesis, and tumor invasion. Mutations and amplification of EGFR are common in non-small-cell lung carcinoma and provide the basis for treatment with EGFR-inhibitors. Her2/neu is affected less frequently. Other genes that are often mutated or amplified are c-MET, NKX2-1, LKB1, PIK3CA, and BRAF.

877. The cell lines of origin are not fully understood. The mechanism may involve abnormal activation of stem cells. In

the proximal airways, stem cells that express keratin 5 are more likely to be affected, typically leading to squamous-cell lung carcinoma. In the middle airways, implicated stem cells include club cells and neuroepithelial cells that express club cell secretory protein. Small-cell lung carcinoma may be derived from these cell lines or neuroendocrine cells and may express CD44.

878. Metastasis of lung cancer requires transition from epithelial to mesenchymal cell type. This may occur through activation of signaling pathways such as Akt/GSK3Beta, MEK-ERK, Fas, and Par6.

879. Primary lung cancers themselves most commonly metastasize to the brain, bones, liver and adrenal glands.

Soft Tissue Sarcomas

880. A soft-tissue sarcoma is a form of sarcoma that develops in connective tissue, muscle, fat, blood and lymph vessels, though the term is sometimes applied to elements of the soft tissue that are not currently considered connective tissue.

881. Soft-tissue sarcomas are relatively uncommon cancers. They account for less than 1% of all new cancer cases each year.

882. Soft tissue sarcomas commonly originate in the upper body, in the shoulder or upper chest.

883. The most common site to which soft tissue sarcoma spreads is the lungs.

Bladder cancer

884. Nearly all bladder cancers start as a neoplastic process in the urothelium or lining of the urinary bladder, a hollow organ in the lower pelvis with flexible, muscular walls that can stretch to hold urine and squeeze to send it out of the body.

885. The wall of the bladder has many several layers. Each layer of the bladder wall is made up of different kinds of cells.

886. Most bladder cancers start in the innermost lining of the bladder, which is called the urothelium or transitional epithelium. As the cancer grows into or through the other layers in the bladder wall, it has a higher stage, becomes more advanced, and can be harder to treat.

887. Urothelial carcinoma, previously called transitional cell carcinoma, occurs in the urothelial cells that line the inside of the bladder and expand and contract as the bladder fills and empties. These same cells line the inside of the ureters which carry urine from the kidneys to the bladder and the urethra which carries the urine away from the bladder.

888. Squamous cell carcinoma is associated with chronic irritation of the bladder, and is rare in the United States.

889. Adenocarcinoma begins in cells that make up mucus-secreting glands in the bladder and is also rare in the United States.

890. Non-invasive cancers are only in the inner layer of cells (the transitional epithelium). They have not grown into the deeper layers.

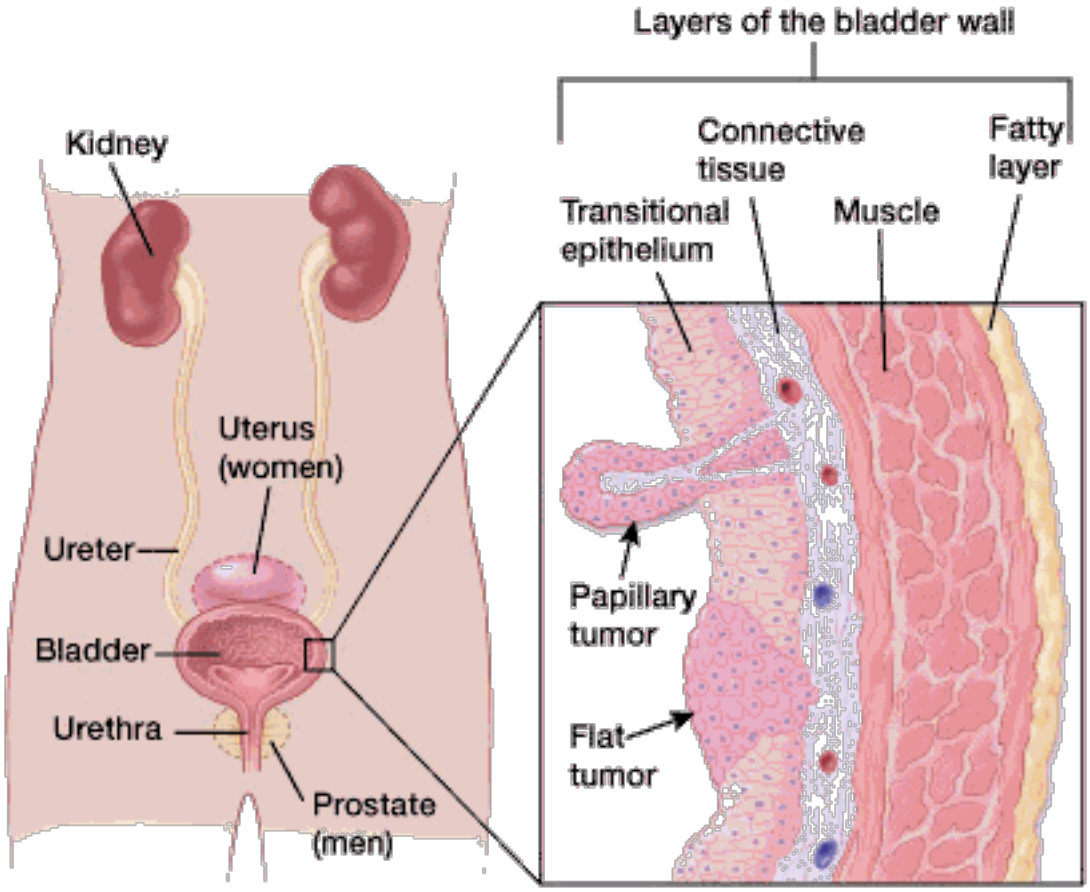
891. Invasive cancers have grown into deeper layers of the bladder wall. These cancers are more likely to spread and are harder to treat.

892. A bladder cancer can also be described as superficial or non-muscle invasive. These terms include both non-invasive tumors as well as any invasive tumors that have not grown into the main muscle layer of the bladder.

893. Bladder cancers are also divided into two subtypes, papillary and flat, based on how they grow.

894. Papillary carcinomas grow in slender, finger-like projections from the inner surface of the bladder toward the hollow center of the bladder without growing into the deeper bladder

layers—non-invasive papillary cancers. Very low-grade, slow growing, non-invasive papillary cancer is sometimes called papillary urothelial neoplasm of low-malignant potential (PUNLMP).



895. Flat carcinomas do not grow toward the hollow part of the bladder at all. If a flat tumor is only in the inner layer of bladder cells, it's known as a non-invasive flat carcinoma or a flat carcinoma in situ (CIS).

896. If either a papillary or flat tumor grows into deeper layers of the bladder, it's called an invasive urothelial or transitional cell carcinoma.

897. Over time, bladder cancer might grow outside the bladder and when bladder cancer spreads, it tends to appear in distant lymph nodes, the bones, the lungs, or the liver.

898. The staging system most often used for bladder cancer is the American Joint Committee on Cancer (AJCC) **TNM** system, which is based on 3 key pieces of information:

T describes how far the main (primary) **tumor** has grown through the bladder wall and whether it has grown into nearby tissues.

N indicates any cancer spread to lymph **nodes** near the bladder. Lymph nodes are bean-sized collections of immune system cells, to which cancers often spread first.

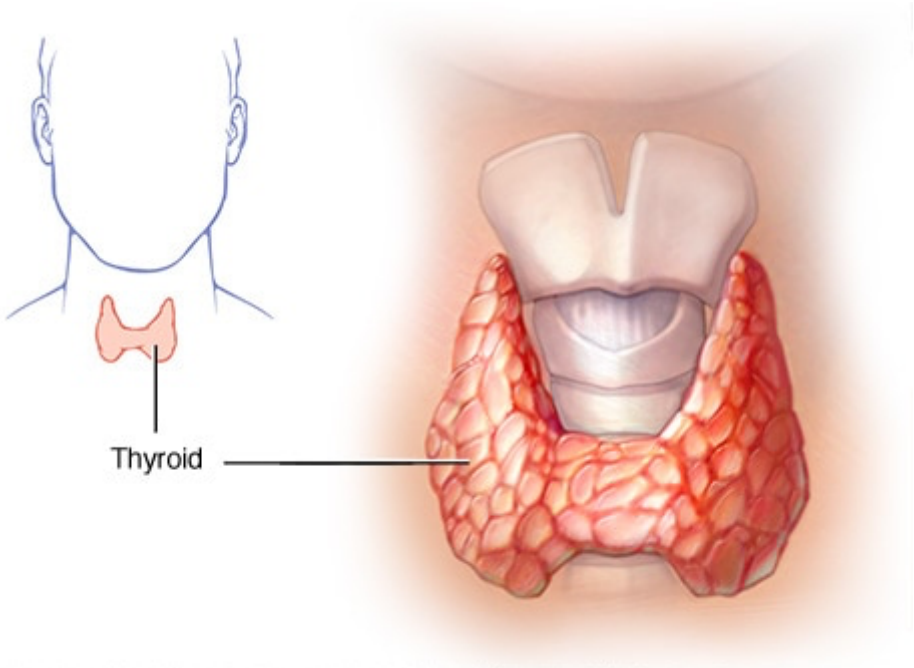
M indicates if the cancer has spread (**metastasized**) to distant sites, such as other organs, like the lungs or liver, or lymph nodes that are not near the bladder.

899. Numbers or letters after T, N, and M provide more details about each of these factors. Higher numbers mean the cancer is more advanced. The earliest stage cancers are called stage 0 (or carcinoma in situ), and then range from stages I (1) through IV (4).

Hypothyroidism

900. Hypothyroidism is a condition in which the thyroid gland doesn't produce enough of certain crucial hormones.

901. The thyroid gland is a small gland, normally weighing less than one ounce, located in the front of the neck, made up of two halves, called lobes, that lie along the windpipe (trachea) and are joined together by a narrow band of thyroid tissue, known as the isthmus.



902.

903. The function of the thyroid gland is to convert iodine into thyroid hormones: thyroxine (T4) and triiodothyronine (T3). Thyroid cells are the only cells in the body which can absorb iodine. These cells combine iodine and the amino acid tyrosine to make T3 and T4. T3 and T4 are then released into the blood stream and are transported throughout the body where they control metabolism.

904. Every cell in the body depends upon thyroid hormones for regulation of their metabolism. The normal thyroid gland produces about 80% T4 and about 20% T3, however, T3 possesses about four times the hormone "strength" as T4.

905. The thyroid gland is under the control of the pituitary gland. When the level of thyroid hormones (T3 & T4) drops too low, the pituitary gland produces Thyroid Stimulating Hormone (TSH) which stimulates the thyroid gland to produce more hormones. Under the influence of TSH, the thyroid will manufacture and secrete T3 and T4 thereby raising their blood levels.

906. The pituitary senses this and responds by decreasing its TSH production.

907. The pituitary gland itself is regulated by another gland, the hypothalamus which produces TSH Releasing Hormone (TRH) which tells the pituitary gland to stimulate the thyroid gland (release TSH).

908. The diagnosis of hypothyroidism is based almost exclusively upon measuring the amount of thyroid hormone in the blood.

Hypertension

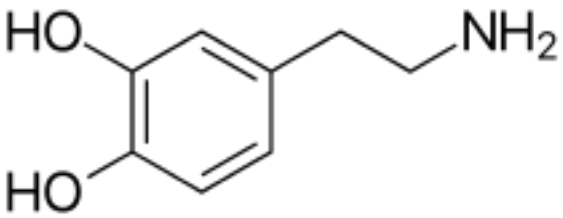
909. Hypertension (HTN or HT), also known as high blood pressure (HBP), is a long-term medical condition in which the blood pressure in the arteries is persistently elevated and becomes a major risk factor for coronary artery disease, stroke, heart failure, atrial fibrillation, peripheral vascular disease, vision loss, chronic kidney disease, and dementia.

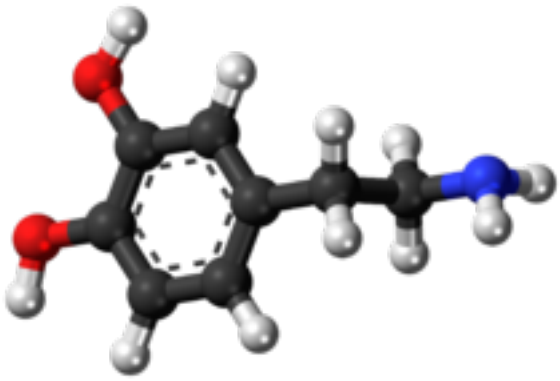
Parkinson's-like symptoms

910. is a long-term degenerative disorder of the central nervous system that mainly affects the motor system. Early in the disease, the most obvious symptoms are shaking, rigidity, slowness of movement, and difficulty with walking. Thinking and behavioral problems may also occur.

911. The motor symptoms of the disease result from the death of cells in the *substantia nigra*, a region of the midbrain which results in not enough dopamine in these areas.

912. Dopamine (3,4-dihydroxyphenethylamine) is an organic chemical of the catecholamine and phenethylamine families.

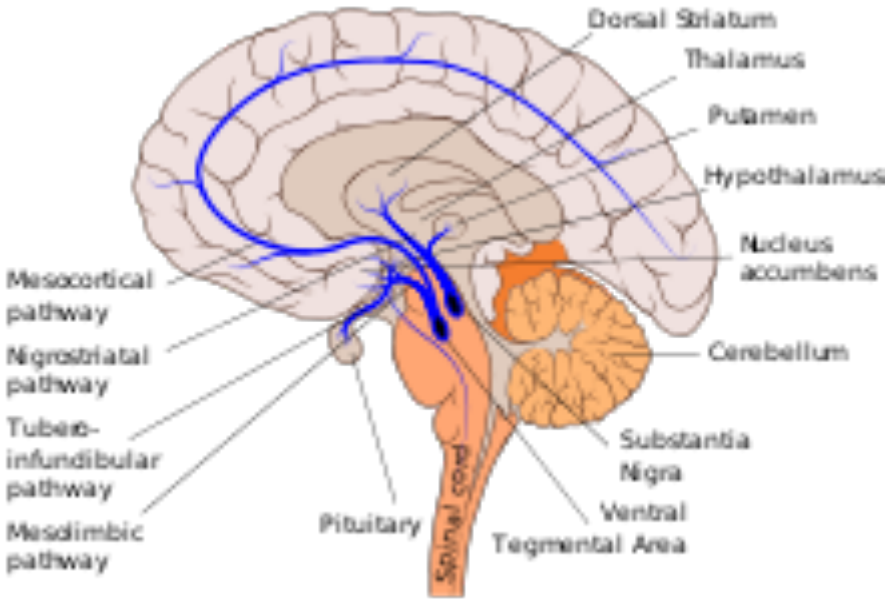




913. Dopamine functions both as a hormone and a neurotransmitter.

914. Dopamine is an amine synthesized by removing a carboxyl group from a molecule of its precursor chemical L-DOPA, which is synthesized in the brain and kidneys. Dopamine is also synthesized in plants and most animals.

915. In the brain, dopamine functions as a neurotransmitter—a chemical released by neurons to send signals to other nerve cells. The brain includes several distinct dopamine pathways.



916. Some brain dopamine pathways are involved in motor control and in controlling the release of various hormones. These pathways and cell groups form a dopamine system which is neuromodulatory.

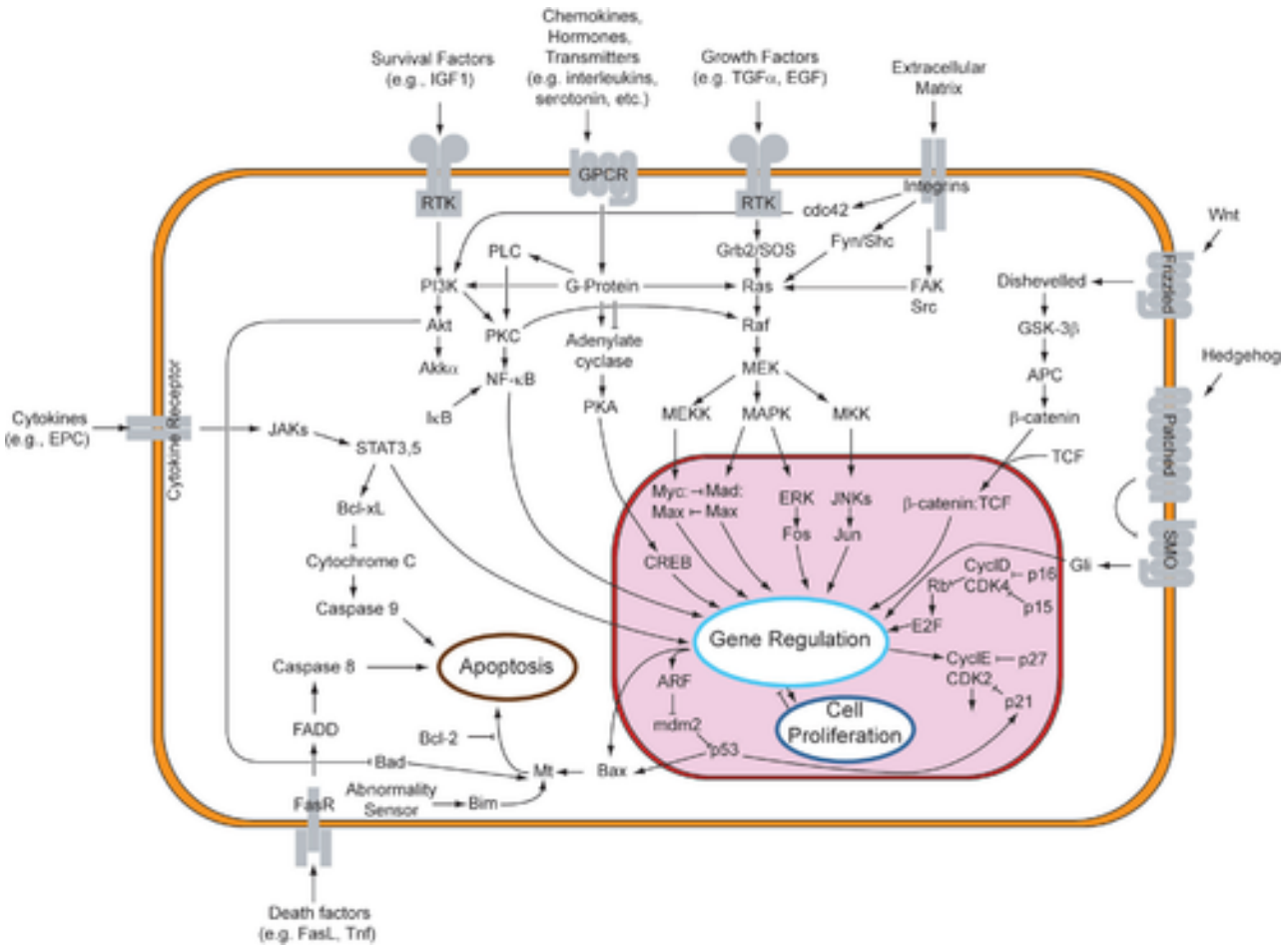
917. Neuromodulation is the physiological process by which a given neuron uses one or more chemicals to regulate diverse populations of neurons in contrast to synaptic transmission in which an axonal terminal secretes neurotransmitters to target fast-acting receptors of only one particular partner neuron.

918. In neuromodulation, the receptors are typically G-protein coupled receptors while in classical chemical neurotransmission, they are ligand-gated ion channels.

919. Dopamine confers motivational salience a cognitive process which regulates the intensity of behaviors that facilitate the attainment of a particular goal, the amount of time and energy that an individual is willing to expend to attain a particular goal, and the amount of risk that an individual is willing to accept while working to attain a particular goal.

920. Outside the central nervous system, dopamine functions primarily as a local paracrine messenger.

921. An overview of signal transduction pathways.



922. In blood vessels, dopamine inhibits norepinephrine release and acts as a vasodilator at normal concentrations; in the kidneys, it increases sodium excretion and urine output; in the pancreas, it reduces insulin production; in the digestive system, it reduces gastrointestinal motility and protects intestinal mucosa; and in the immune system, it reduces the activity of lymphocytes. With the exception of the blood vessels, dopamine in each of these peripheral systems is synthesized locally and exerts its effects near the cells that release it.

923. There is no single laboratory test or radiological exam that will provide a definitive diagnosis of Parkinsons disease.

GENERAL CAUSATION

924. The Plaintiffs were exposed to one or more toxic substances, including dioxin contaminated phenoxy herbicides and cacodylates, in herbicides manufactured, marketed, promoted, and sold by the Defendants, jointly and severally, individually and collectively, at Eglin Air Force Base more likely than not caused, or contributed to, or aggravated their physiological injury, systematic disease, genetic damage and death.

925. That the only known exposure of the Plaintiffs to dioxin contaminated phenoxy herbicides and the cacodylates and cacodylates was during their employment at Eglin Air Force Base.

926. There is no known safe level for TCDD in human beings.

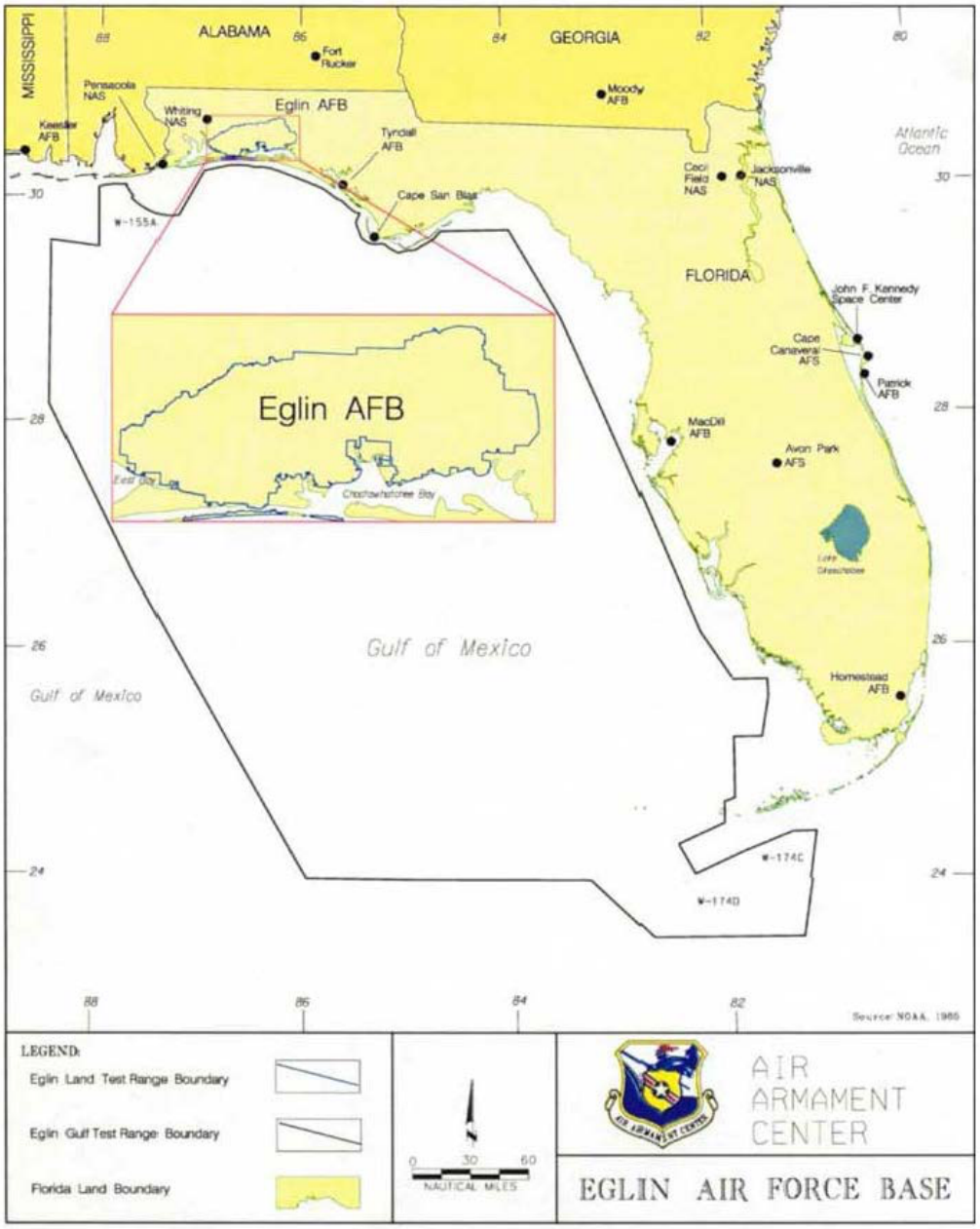
927. Since data from human experiments is universally recognized among civilized peoples as immoral and illegal, there can be no "controlled" study of TCDD effects on human beings, however, a fair preponderance of available scientific evidence population clearly establishes that unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A can be a probable cause of the physiological injury, systematic disease, genetic damage and death of the Plaintiffs.

928. The unprotected exposure of the Plaintiffs to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A is the only identifiable proximate cause of the physiological injury, systematic disease, genetic damage and death suffered by the Plaintiffs and was a substantial factor, or an aggravating factor resulting in injury and death sooner than would have occurred otherwise.

929. The actions of the Defendants, jointly and severally, individually and collectively, complained of in this Complaint were substantial factors and material and contributing factors

in the physiological injury, systematic disease, genetic damage and death of the Plaintiffs.

THE EGLIN AIR FORCE BASE REGION



Source: Eglin AFB 2000d

930. Eglin Air Force Base (AFB) (IATA: VPS, ICAO: KVPS, FAA LID: VPS) is a United States Air Force base located approximately 3 miles (5 kilometers) southwest of Valparaiso, Florida in Okaloosa County.

931. Eglin Air Force Base covers 463,128 acres (1,874.2 km² / 723.6 sqm). The land area of Okaloosa County is 930 square miles (2,400 km²)

EGLIN AIR FORCE BASE: THE ENVIRONMENT

932. Eglin Air Force Base was created from the Choctawhatchee National Forest which was established by President Theodore Roosevelt on November 27, 1908.

933. On June 27, 1940, Congress transferred 340,890 acres (1380 km²) of the Choctawhatchee from the Forest Service to the War Department and subsequently to the United States Department of the Air Force for military purposes. The law provided that the land may be restored to national forest status by proclamation or order of the President when it was no longer needed for military purposes. It has been home to Eglin Air Force Base since. The forest is located in parts of southern Okaloosa, Walton, and Santa Rosa counties.

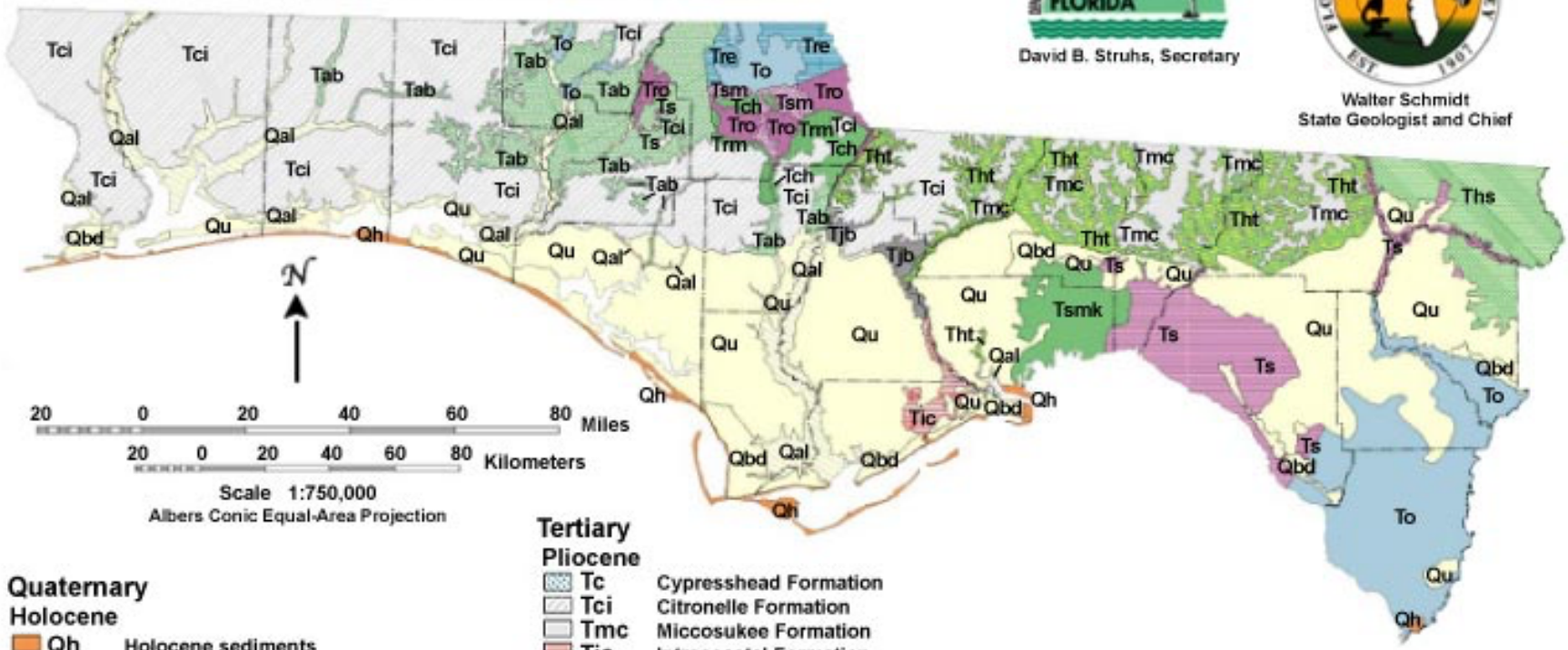
934. The forests and shores of Eglin Air Force Base are at the center of one of the most biodiverse locations in North America. Over 50 species threatened in Florida are found on the base, including sea turtles that nest on its white-sand beaches and red-cockaded woodpeckers that thrive in its longleaf pine forests. Longleaf pine forest, a forest type reduced to 5% of its former range in the last few centuries, covers 200,000 acres (810 km2) of the base. Part of this forest, 6,795 acres (27.50 km²), is old growth, making the base home to one of the most extensive old-growth longleaf pine forests in the world.

Geologic Map of the State of Florida - Panhandle

by Thomas M. Scott, P. G. #99, Kenneth M. Campbell, Frank R. Rupert, Jonathan D. Arthur, Thomas M. Missimer, Jacqueline M. Lloyd, J. William Yon, and Joel G. Duncan

David B. Struhs, Secretary

Walter Schmidt
State Geologist and Chief



Scale 1:750,000
Albers Conic Equal-Area Projection

Quaternary

Holocene

- Qh — Holocene sediments

Pleistocene/Holocene

- Qal — Alluvium
- Qbd — Beach ridge and dune
- Qu — Undifferentiated sediments

Pleistocene

- Qa — Anastasia Formation
- Qk — Key Largo Limestone
- Qm — Miami Limestone
- Qtr — Trail Ridge sands

Tertiary/Quaternary

Pliocene/Pleistocene

- TQsu — Shelly sediments of Plio-Pleistocene age
- TQu — Undifferentiated sediments
- TQd — Dunes
- TQuc — Reworked Cypresshead sediments

Tertiary

Pliocene

- Tc — Cypresshead Formation
- Tci — Citronelle Formation
- Tmc — Miccosukee Formation
- Tic — Intracoastal Formation
- Tt — Tamiami Formation
- Tjb — Jackson Bluff Formation

Miocene/Pliocene

- Thcc — Hawthorn Group, Coosawhatchie Formation, Charlton Member
- Thp — Hawthorn Group, Peace River Formation
- Thpb — Hawthorn Group, Peace River Formation, Bone Valley Member

Miocene

- Trm — Residuum on Miocene sediments
- Tab — Alum Bluff Group
- Th — Hawthorn Group
- Thc — Hawthorn Group, Coosawhatchie Formation
- Ths — Hawthorn Group, Statenville Formation
- Tht — Hawthorn Group, Torreya Formation
- Tch — Chatahoochee Formation
- Tsmk — St. Marks Formation

Oligocene/Miocene

- Tha — Hawthorn Group, Arcadia Formation
- That — Hawthorn Group, Arcadia Formation, Tampa Member

Oligocene

- Tro — Residuum on Oligocene sediments
- Ts — Suwannee Limestone
- Tsm — Suwannee Limestone - Marianna Limestone undifferentiated

Eocene

- Tre — Residuum on Eocene sediments
- To — Ocala Limestone
- Tap — Avon Park Formation

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THE NORTHWEST FLORIDA MANAGEMENT DISTRICT

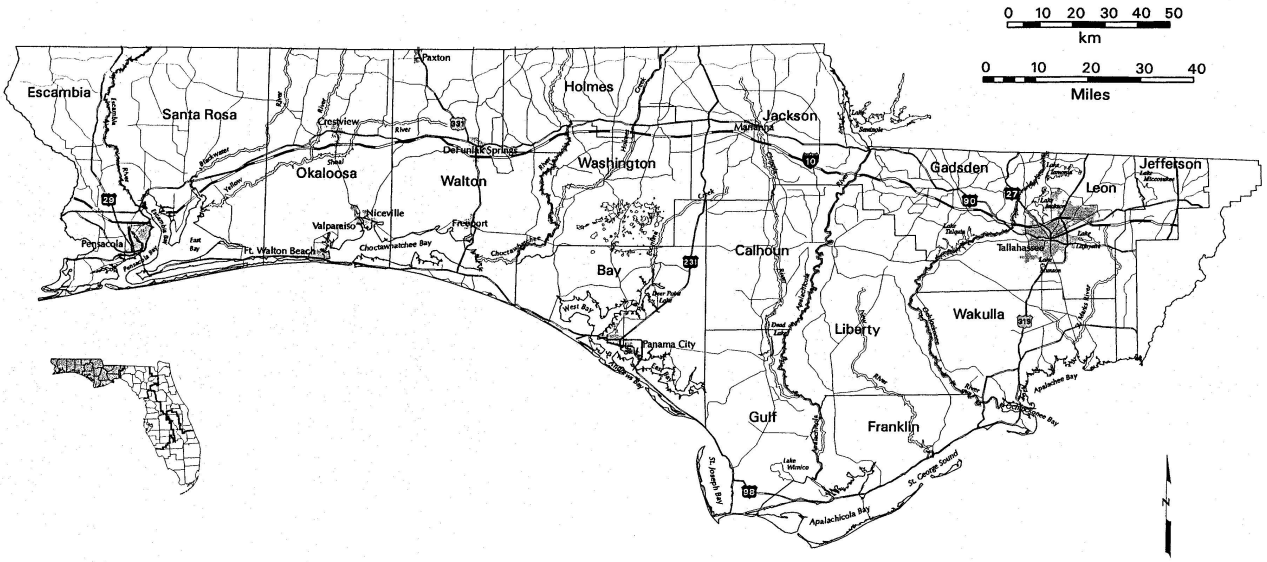
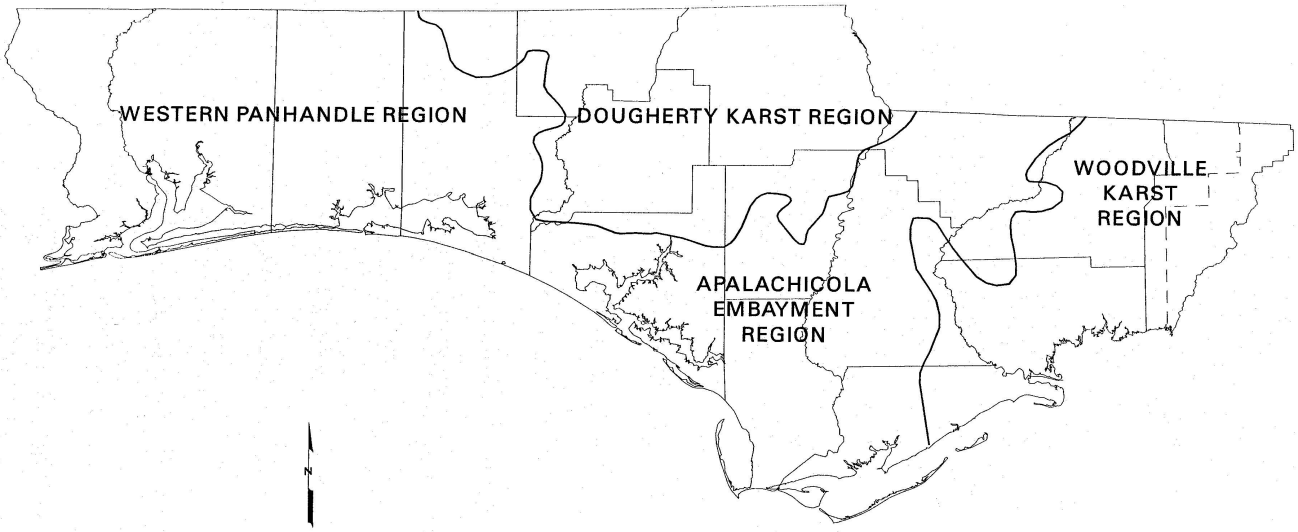


Figure A-1
Northwest Florida Water Management District

Topography and drainage

935. The topography and resultant physiography of northwest Florida are the products of stream and sea wave activity over the past 15 million years.

936. The major physiographic features include the Northern Highlands, the Marianna Lowlands (Dougherty Karst Plain),



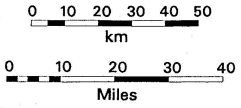


Figure A-3

Ground Water Regions in
the Northwest Florida Water Management District

and the Coastal Lowlands (Figure A-2).

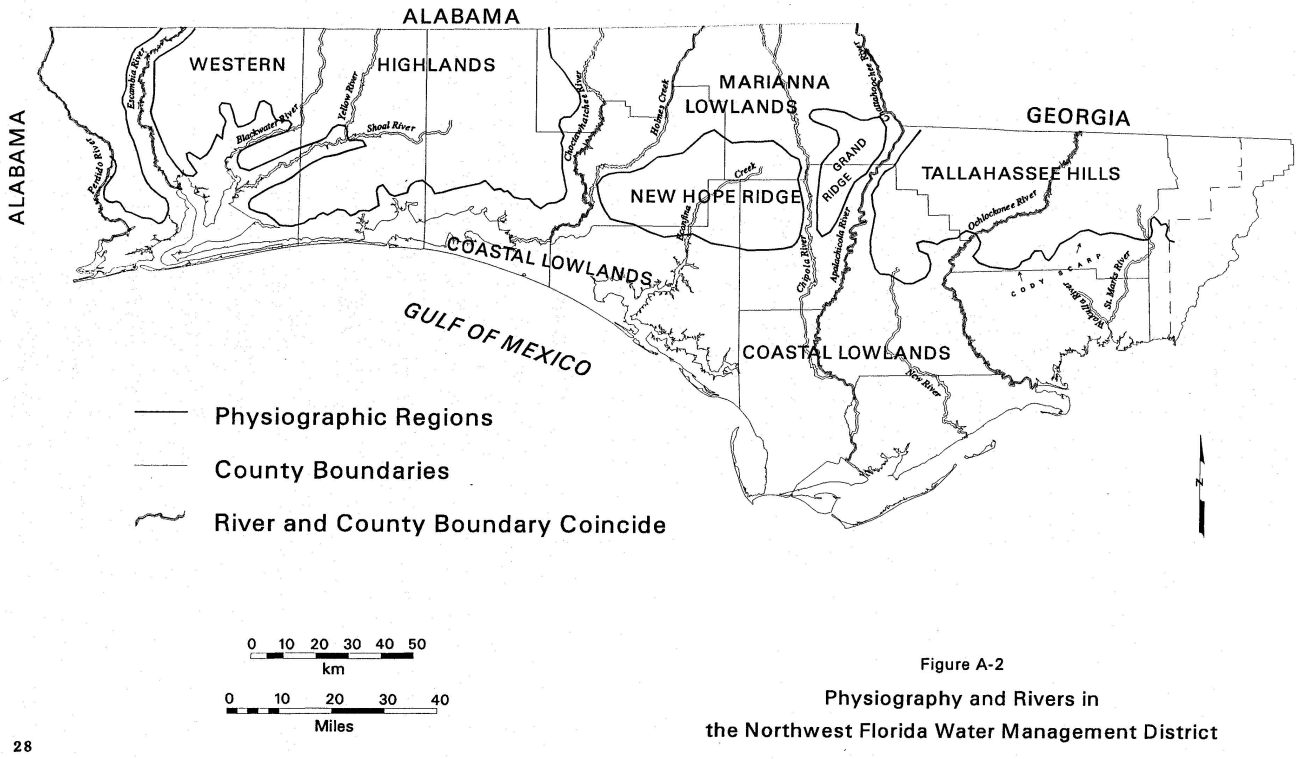


Figure A-2
Physiography and Rivers in
the Northwest Florida Water Management District

937. The Northern Highlands extend across the northern part of the panhandle and north into Alabama and Georgia.

938. Significant landforms within the Northern Highlands include the Tallahassee Hills, Grand Ridge, New Hope Ridge, and the Western Highlands.

939. The Western Highlands and the Tallahassee Hills are separated by the physiographic province referred to as the Marianna Lowlands.

940. Grand Ridge and New Hope Ridge are remnant highland features bordering the southern edge of the Marianna Lowlands.

941. The southern limit of the Northern Highlands is marked by a regionally-extensive outfacing scarp referred to as the Cody Scarp.

942. Elevations in the highlands area range from 50 to 345 feet above sea level.

943. The Cody Scarp represents the northern extent of a Pleistocene sea level transgression that removed Miocene and Pliocene sediments to expose the underlying carbonates of the St. Marks Formation and Suwannee Limestone. This area of exposed carbonates is referred to as the Woodville Karst Plain and represents a distinct physiographic sub-region of the larger Woodville Karst Region.

944. The Marianna Lowlands is actually the southern extent of the Dougherty Karst Plain (Figure A-3), which extends into southeast Alabama and southwest Georgia.

945. The Marianna Lowlands is a product of stream erosion and ground water dissolution activity. The highlands that formerly existed in this area have been reduced, primarily by the major rivers and streams: the Chattahoochee-Apalachicola, Chipola and Choctawhatchee rivers, and Holmes Creek (Figure A-2).

946. The karst plain is well drained and contains many visible sinkholes, as well as paleosinks which have no surface expression.

947. Many areas lack well-defined surface drainage patterns due to the capture of runoff by the subsurface through internal drainage.

948. Elevations within the karst plain range from near sea level to 245 feet above sea level.

949. The Coastal Lowlands lie south of the Northern Highlands and are adjacent to the coastline. Elevations are low, ranging from sea level to about 100 feet above sea level. The land in many areas is poorly drained due to a flat topography and associated high water table.

950. Landforms present within this province include barrier islands, lagoons, estuaries, sand-dune ridges, and relict spits and bars, all of which are the result of marine processes.

Regional Hydrostratigraphy

951. Each system is a collection of lithologic beds that share certain hydrogeologic characteristics.

952. Systems are defined by their ability to conduct or retard the flow of water and, thus, are not constrained by lithologic or stratigraphic boundaries. In general, boundaries between systems separate lithologically distinct units. In some cases, due to variations in a lithologic unit's ability to conduct water, a system boundary may occur within a stratigraphic unit.

953. In descending order from land surface, the four systems are: the Surficial Aquifer System which includes the Sand-and-Gravel Aquifer; the Intermediate System; the Floridan Aquifer System; and the Sub-Floridan System.

954. Due to their lithologic characteristics, the Surficial Aquifer System and Floridan Aquifer System have properties that allow for the storing and transmitting of ground water; however, these aquifer systems are vastly different in that, due to variations in composition and thickness, each has different water-yielding properties.

955. The Intermediate and Sub-Floridan systems function as groups of sediments that retard the vertical movement of ground water. The Intermediate System limits the exchange of water between the Surficial Aquifer System and the Floridan Aquifer System.

956. The Sub-Floridan System forms the base of the Floridan Aquifer System ground water flow regime.

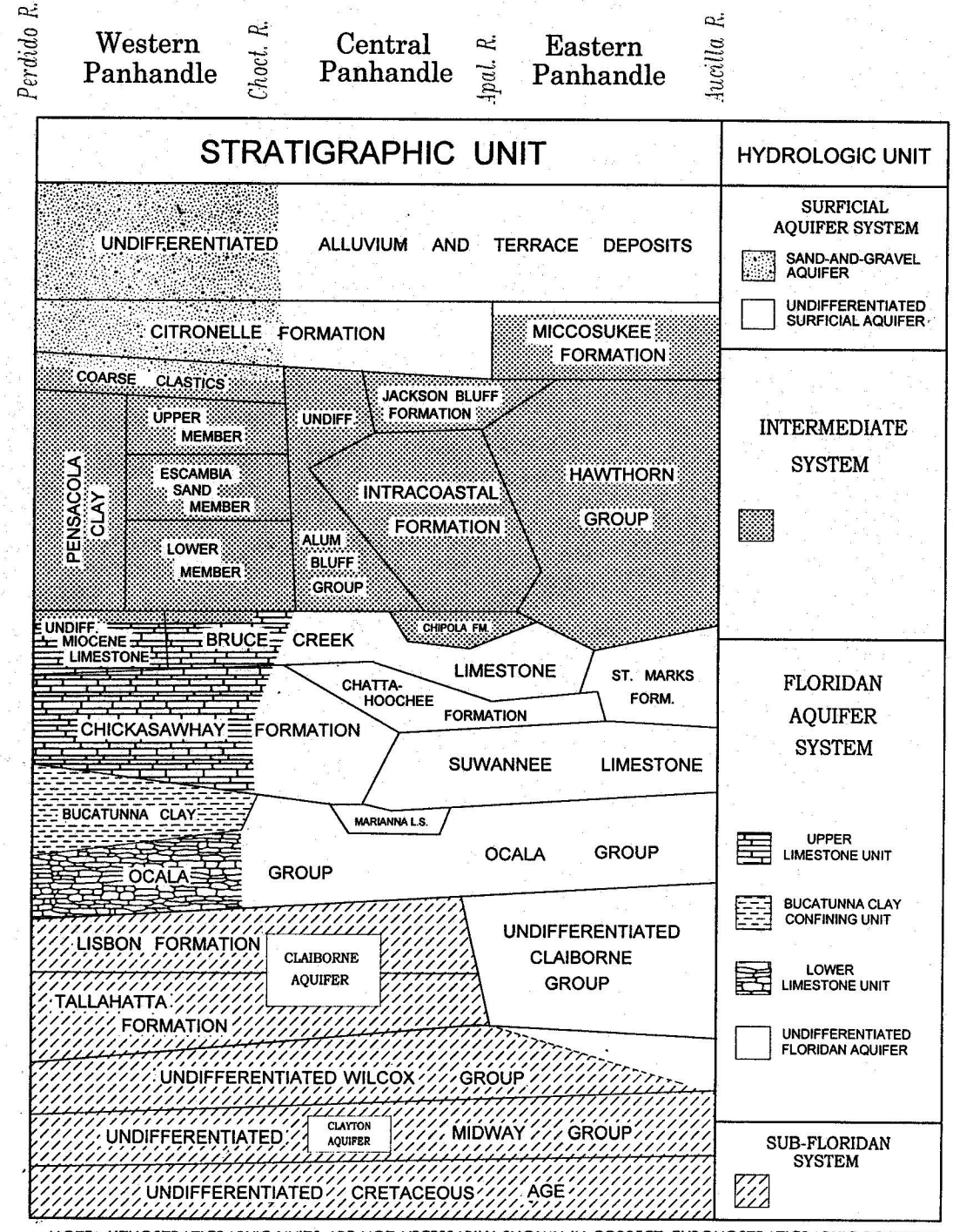
957. The following figure correlates regional stratigraphy to the hydrogeologic systems and shows the variability of the hydrogeologic framework across the panhandle.

958. The lithostratigraphic units in the figure are not necessarily shown in correct chronostratigraphic position.

959. Surficial Aquifer System replaces terms such as water-table aquifer, non-artesian aquifer, shallow aquifer, and sand aquifer. Figure 1 correlates the Surficial Aquifer System to the stratigraphy within northwest Florida.

960. In the panhandle, the Surficial Aquifer System is comprised of the uppermost permeable zones of the Miccosukee Formation, undifferentiated terrace and alluvial deposits, the Citronelle Formation, and those portions of the undifferentiated coarse elastics sufficiently permeable to be included in the basal Sand-and-Gravel Aquifer.

NORTHWEST FLORIDA WATER MANAGEMENT DISTRICT



NOTE : LITHOSTRATIGRAPHIC UNITS ARE NOT NECESSARILY SHOWN IN CORRECT CHRONOSTRATIGRAPHIC POSITION.

Figure 1. Correlation of Hydrogeologic Systems to Stratigraphy in the Panhandle Region.

961. Figure A-5 shows the occurrence area for the Sand-and-Gravel Aquifer and by inference the undifferentiated Surficial Aquifer System.

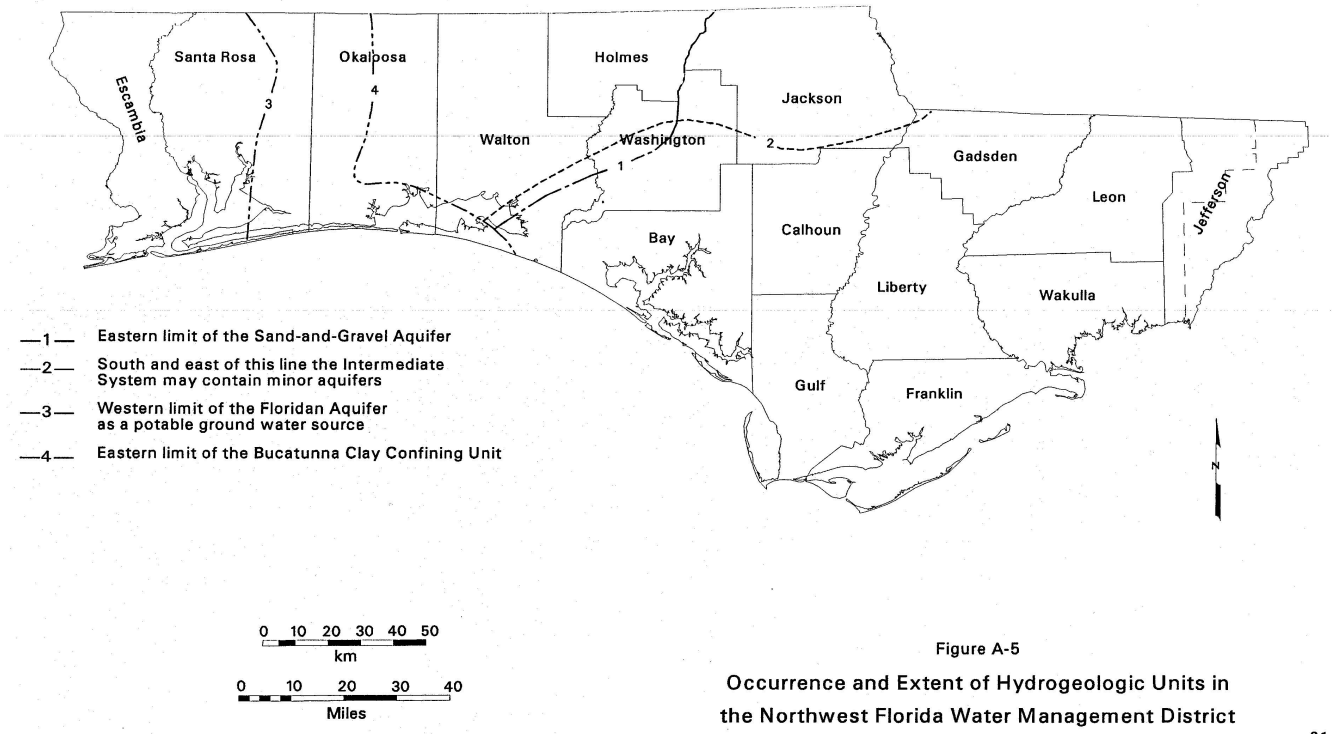


Figure A-5

Occurrence and Extent of Hydrogeologic Units in the Northwest Florida Water Management District

962. The greater the percentage of continuously occurring clay beds and the thicker the clayey sequence, the more effective the Intermediate System is in retarding vertical movement. Where the system is thin or less clayey, or where the beds are breached by higher permeability sediments, the Intermediate System is "leaky" and vertical movement of water to the underlying Floridan Aquifer System is more effective.

963. In portions of northwest Florida, the Intermediate System contains minor aquifers. These aquifers are sandwiched between clayey sediments. Due to vertical hydraulic conductivity contrasts, discrete hydrostatic heads define each zone.

964. The Sub-Floridan System, although primarily a confining sequence, does contain aquifers of regional significance. In the

north-central portion of the panhandle, the aquifers that occur in this system are the southern extents of more prolific aquifers recognized as the Claiborne Aquifer and the Clayton Aquifer in southeast Alabama and southwest Georgia.

Floridan Aquifer System

965. The Floridan Aquifer System consists of a persistent carbonate sequence that includes a variety of geologic formations ranging in age from middle Miocene to Paleocene.

966. Within northwest Florida, the formations display lateral and vertical variations in lithologic characteristics due to changes that occurred in the depositional environments. This variability results in wide permeability contrasts within the aquifer.

967. In general, the Suwannee and Ocala limestones have the highest permeabilities, regardless of geographic area. As wells rarely tap the sediments underlying the Ocala Limestone, the permeability characteristics of these sediments are poorly understood. The younger portions of the system range from low to high permeability.

968. Near the middle portion of the panhandle, middle Eocene to Paleocene rocks change from relatively more elastic in the west to relatively more carbonate to the east. The significance of this change is that the sediments in the west function as a confining unit and are part of the Sub-Floridan System (Figure 1).

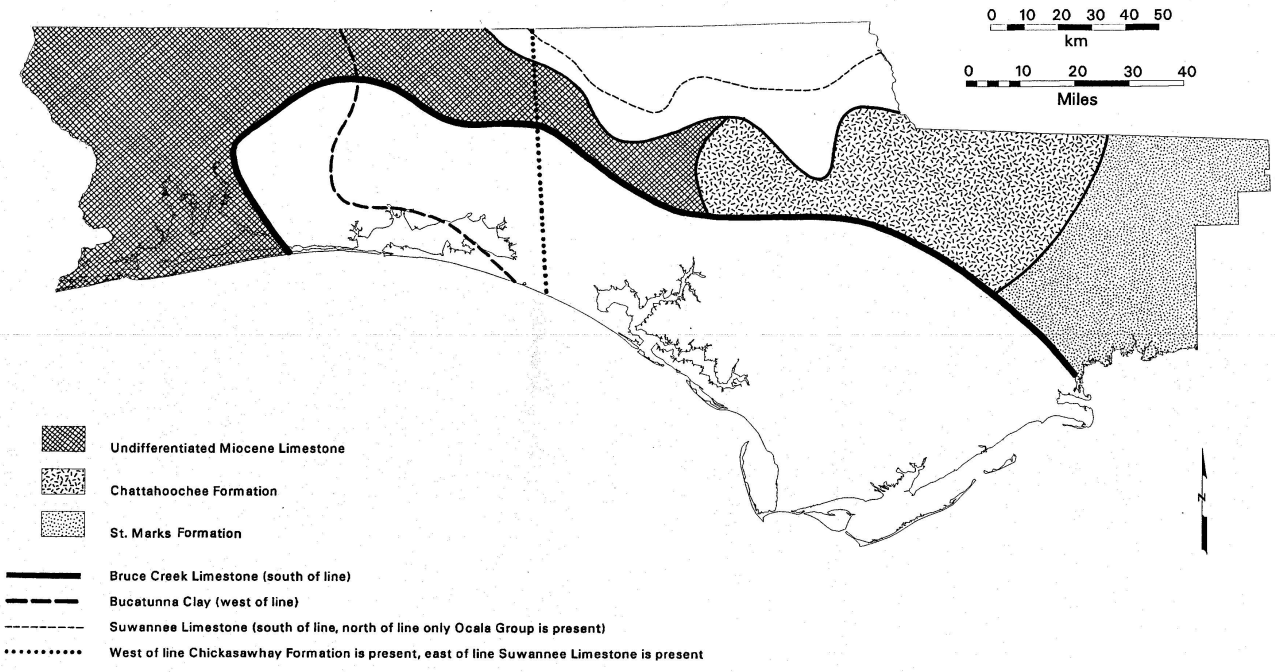


Figure A-7
Areal Extent of Stratigraphic Units
Composing the Floridan Aquifer System

REFERENCES : SCHMIDT AND COE, 1978 ; SCHMIDT, 1984

969. To the east, contemporaneous deposits are carbonates and are hydraulically connected to the overlying younger carbonates. Thus, the thickness of the Floridan Aquifer System increases eastward across the panhandle (Figure 1).

970. In the westernmost portion of the panhandle, the Floridan Aquifer System is split vertically by a regional confining unit. The two parts are referred to as the upper limestone of the Floridan Aquifer System, which includes all or part of the Chickasawhay Formation, the Bruce Creek Limestone, and an undifferentiated Miocene limestone; and the lower limestone of the Floridan Aquifer System, which includes the Ocala Limestone (Figure 1). The confining unit separating the two portions is referred to as the Bucatunna Clay Confining Unit. Where the unit pinches out to the east, the Floridan Aquifer System becomes one vertically undifferentiated unit. The

Bucatunna Clay Confining Unit thickens to the southwest and thins to the north and east.

971. In the westernmost panhandle, the hydrostratigraphy is further complicated by the fact that the Bruce Creek Limestone and the undifferentiated Miocene limestone can contain moderate amounts of clay. Where these clayey intervals are contiguous with the Pensacola Clay lithostratigraphic unit, they are most appropriately included in the Intermediate System.

972. The Floridan Aquifer System is present throughout northwest Florida. However, in the extreme western portion of the panhandle, the conditions are such that the aquifer is not used as a ground water source.

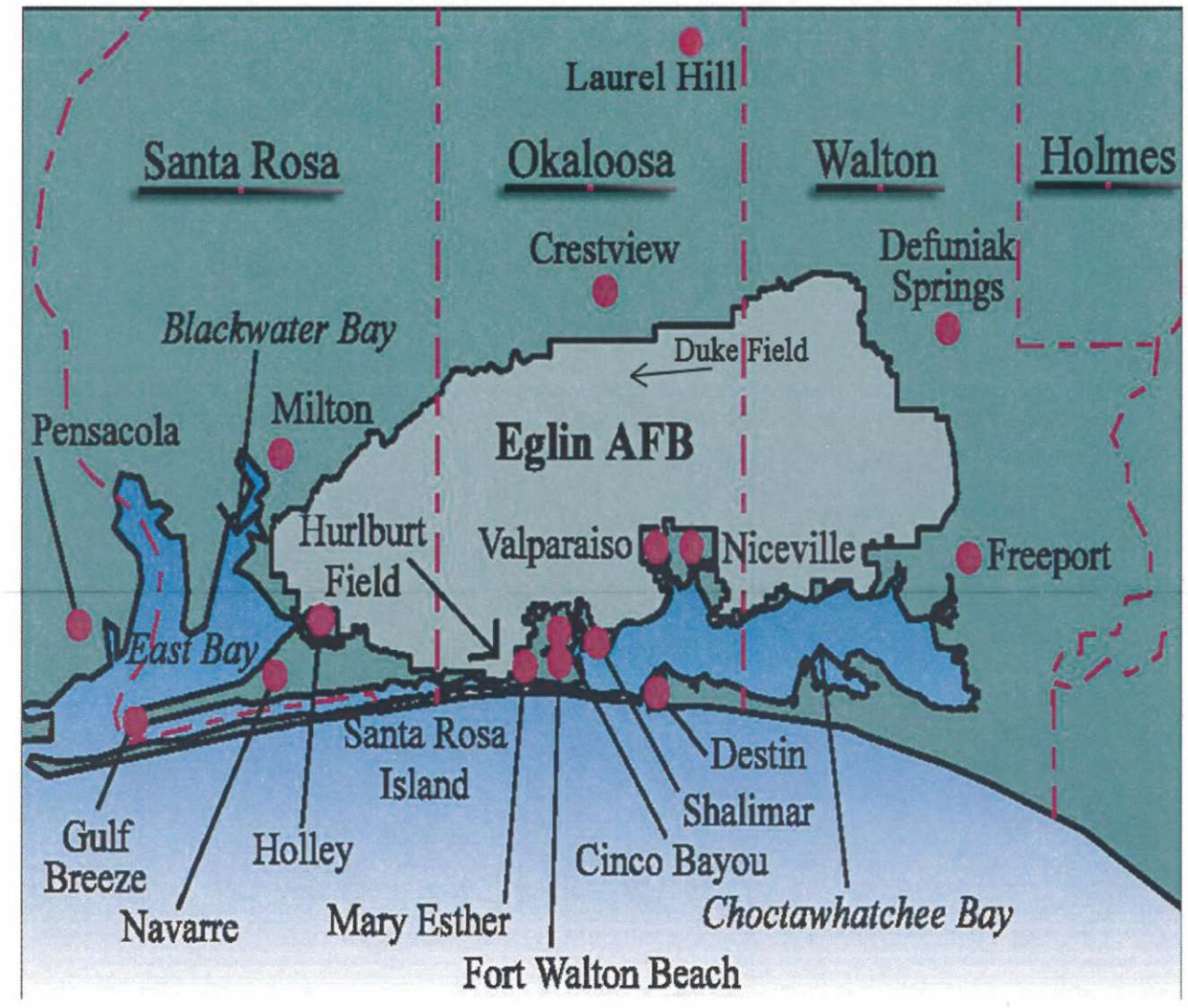
973. In Santa Rosa and Escambia counties mineralization steadily increases in a southwesterly direction in both the upper and lower Floridan Aquifer System. In these areas, the Sand-and-Gravel Aquifer is the primary source for ground water. In most of southern Okaloosa and Walton counties, the lower Floridan is highly mineralized and is not potable. In these counties, the upper limestone of the Floridan Aquifer System is the primary source for ground water, and supplemental sources are derived from the Sand-and-Gravel Aquifer.

974. For much of northwest Florida, the Floridan Aquifer System is the deepest active flow system. In the northcentral portion of the panhandle, freshwater aquifers also exist within the Sub-Floridan System. The base of the Floridan Aquifer System forms a gradational contact with fine-grained elastic sediments of much lower permeability. The term Floridan Aquifer System replaces terms such as principal artesian formations, principal aquifer, principal artesian aquifer, Floridan aquifer, and Tertiary limestone aquifer system.

EGLIN AIR FORCE BASE: MILITARY HISTORY

975. Eglin AFB was established in 1935 as the Valparaiso Bombing and Gunnery Base. It is named in honor of Lieutenant Colonel Frederick I. Eglin (1891–1937), who was killed in a crash of his Northrop A-17 pursuit aircraft on a flight from Langley to Maxwell Field, Alabama.

976. Eglin is also one of the few military air bases in the U.S. to have scheduled passenger airline service. The Destin–Fort Walton Beach Airport (VPS) is co-located on the base property.



Location map: The region

977. Eglin Air Force Base evolved from the 1933 creation of the Valparaiso Airport, when an arrowhead-shaped parcel of 137 acres (0.55 km^2) was cleared for use as an airdrome.

978. In 1931, personnel of the Air Corps Tactical School, newly relocated to Maxwell Field, Alabama, sought a location for a bombing and gunnery range. They saw the potential of the sparsely populated forested areas surrounding Valparaiso and the vast expanse of the adjacent Gulf of Mexico.

979. From October 1941 to October 1945, an AAF Fixed Gunnery School operated at the base supervised by the 75th Flying Training Wing.

980. At its peak during World War II, the base employed more than 1,000 officers, 10,000 enlisted personnel and 4,000 civilians.

981. After the war, Eglin became a pioneer in developing the techniques for missile launching and handling; and the development of drone or pilotless aircraft beginning with the Republic-Ford JB-2 Loon, an American copy of the V-1. The 1st Experimental Guided Missiles Group was activated at Eglin Field, Florida, on 6 February 1946, operating out of Auxiliary Field 3.

982. By March 1950, the 550th Guided Missiles Wing, comprising the 1st and 2nd Guided Missile Squadrons, had replaced the 1st Experimental Guided Missiles Group. The 2nd Guided Missile Squadron, SSM, had 62 pilots manning 14 B-17s, three B-29s, and four F-80 Shooting Stars, yellow-tailed drone aircraft used in the role of testing guided missiles.

983. In December 1955, the Air Munitions Development Laboratory was reassigned from the Wright Air Development Center at Wright-Patterson AFB, Ohio, to the Air Force Armament Center at Eglin by Headquarters Air Research and Development Command. The responsibility for development of

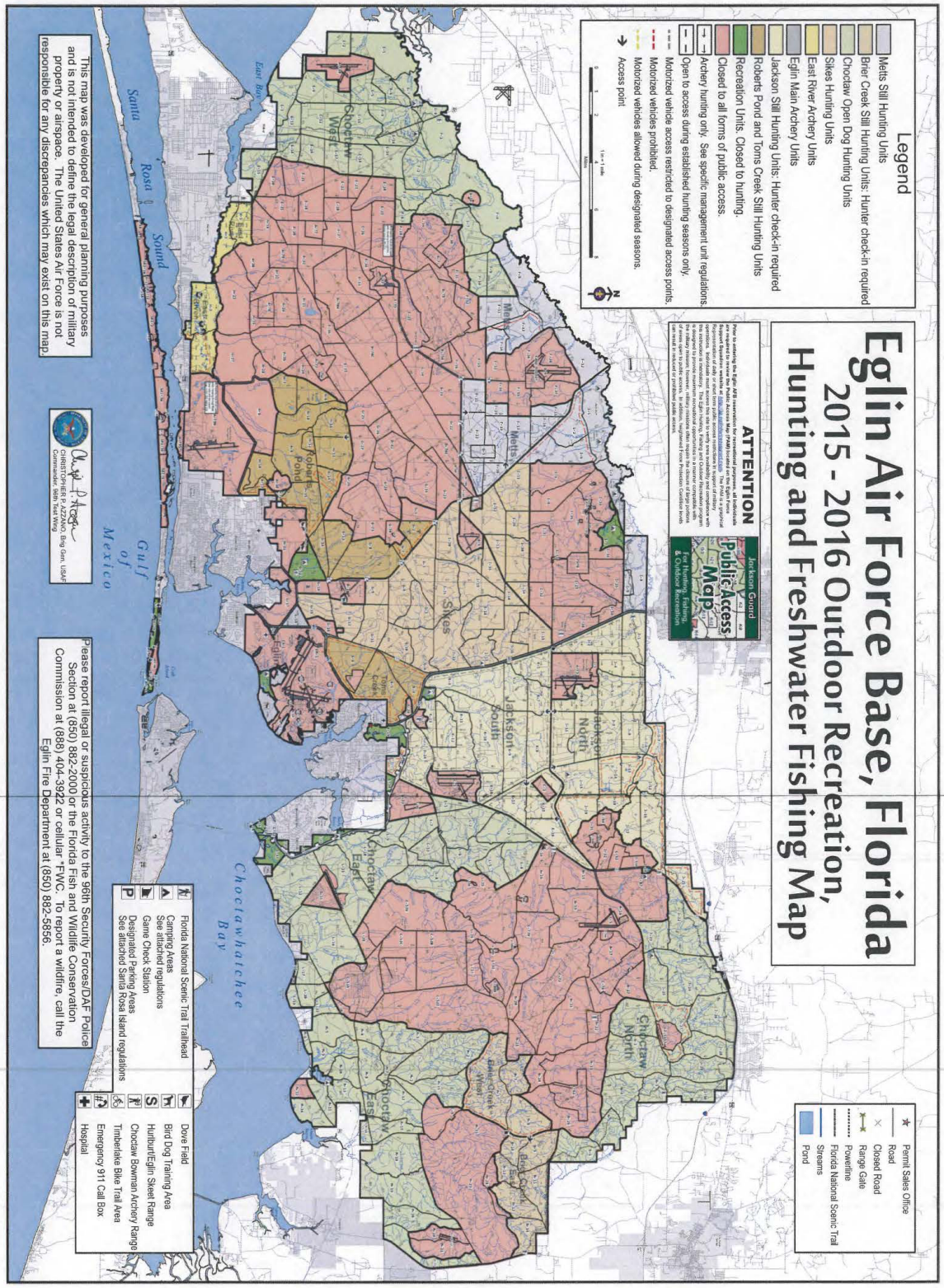
guns, bombs, rockets, fuses, guided missile warheads and other related equipment in the armament field was transferred from the Dayton, Ohio facility at this time. Work on nuclear weapons was not included in this mission.

984. The USAF Special Air Warfare Center was activated 27 April 1962, with the 1st Combat Applications Group (CAG) organized as a combat systems development and test agency under the SAWC. The 1st CAG concentrated on testing and evaluation of primarily short-term projects which might improve Air Force counter-insurgency (COIN) operations. The Special Air Warfare Center, located at Hurlburt Field, undertook to develop tactical air doctrine while training crews for special air warfare in places like Southeast Asia. By mid-1963, SAW groups were in Vietnam and Panama.

985. The USAF Tactical Air Warfare Center was activated on 1 November 1963. It would be re-designated as the USAF Air Warfare Center on 1 October 1991.

986. On 1 August 1968, the Air Proving Ground Center was redesignated the Armament Development and Test Center to centralize responsibility for research, development, test and evaluation, and initial acquisition of non-nuclear munitions for the Air Force.

Eglin Air Force Base Public Access Map



Eglin Air Force Base: The herbicide testing program

987. From 1961-1971,The Air Development Test Center, Eglin Air Force Base (AFB), on the Florida "Panhandle," developed, tested, and calibrated the aerial spray systems used in support of Operation RANCH HAND and the US Army Chemical Corps in Vietnam.

988. Before 1970 very little was known about the toxicity or environmental persistence of the TCDD contaminant, in the 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) containing herbicides, including Agent Orange, which also contained 2,4-dichlorophenoxyacetic acid (2,4-D).

989. The test programs at Eglin AFB involving 2,4,5-T herbicides were conducted under guidelines provided by US Army Chemical Corps and US Department of Agriculture with the recognition that the herbicide was "essentially non-toxic to humans and other mammals."

990. In late 1961, a testing installation, including supporting laboratories and test arrays, was established at Eglin AFB to develop and test aerial spray equipment for disseminating the pesticides (herbicides and insecticides) used in the Vietnam War.

991. Direct aerial applications were restricted to an area approximately 2.6 km2 within Test Area C–52A, one of many test ranges on the approximately 2,000 km2 Eglin Reservation.

992. In 1961,Test Area C–52A covered an area of approximately 8 km2 and was a grassy plain densely covered by Switchgrass, Panicum virgatum, Woolly Panicum, Panicum lanuginosum, and Broomsedge, Andropogon virginicus, surrounded by a forest stand that was dominated by three climax species, Sand Pine, Pinus clausa, Longleaf Pine, Pinus palustris, and Turkey Oak, *Quercus laevis*

993. Twenty major test and evaluation projects of aerial spray equipment were conducted on four fully instrumented test grids, each uniquely arrayed to match the needs of fixed-wing, helicopter, or jet aircraft. Each of the grids was established within the boundary of Test Area C–52A of the Eglin Reservation.

994. The tests, conducted under climatic and environmental conditions similar to those in Vietnam, included the use of the military herbicides (Agents) Orange, Purple, White, and Blue.

995. Although not identical to those of Southeast Asia, the climatic and environmental parameters of the Eglin AFB Reservation were sufficiently similar so that the operational conditions of the aircraft, spray equipment, and behavior of the herbicides were reasonably realistic.

996. An array of test grids was developed where the aircraft and equipment could be monitored and evaluated using the actual herbicides that were deployed for use in Vietnam.

997. The goal was not to test the effectiveness of the herbicides, but rather the effectiveness of the aircraft and spray equipment in disseminating a concentration of herbicide at the "minimum biologically effective ground deposition level."

998. Test arrays were constructed and sampling systems developed to assess the characteristics of dissemination aerially delivered liquid and particulate materials from various aerial-spray-equipment systems.

999. Each of the 196 permanent sampling stations, arranged on 122-m centers to form the 2.6-km2 grid, employed glass plates and Kromekote cards for physical collection of test droplets of material.

1000. From June 1962 through June 1970, the Eglin tests girds received approximately 75,000 liters of Agent Orange; 61,200

liters of Agent Purple, 15,800 liters of Agent White, 16,600 liters of Agent Blue herbicides; and 815 liters of malathion insecticide.

1001. During that period, 222,530 liters (approximately 58,786.207 gallons) of herbicides were sprayed at a test grid on the base.

1002. Approximately 75,000 kg of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 76,000 kg of 2,4-dichlorophenoxyacetic acid (2,4-D) were aerially disseminated on an area of less than 3 km during the period 1962-1970.

1003. These herbicides were estimated to contain at least 3.1 kilograms (~6.834 pounds) of 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin (TCDD), present as a contaminant.

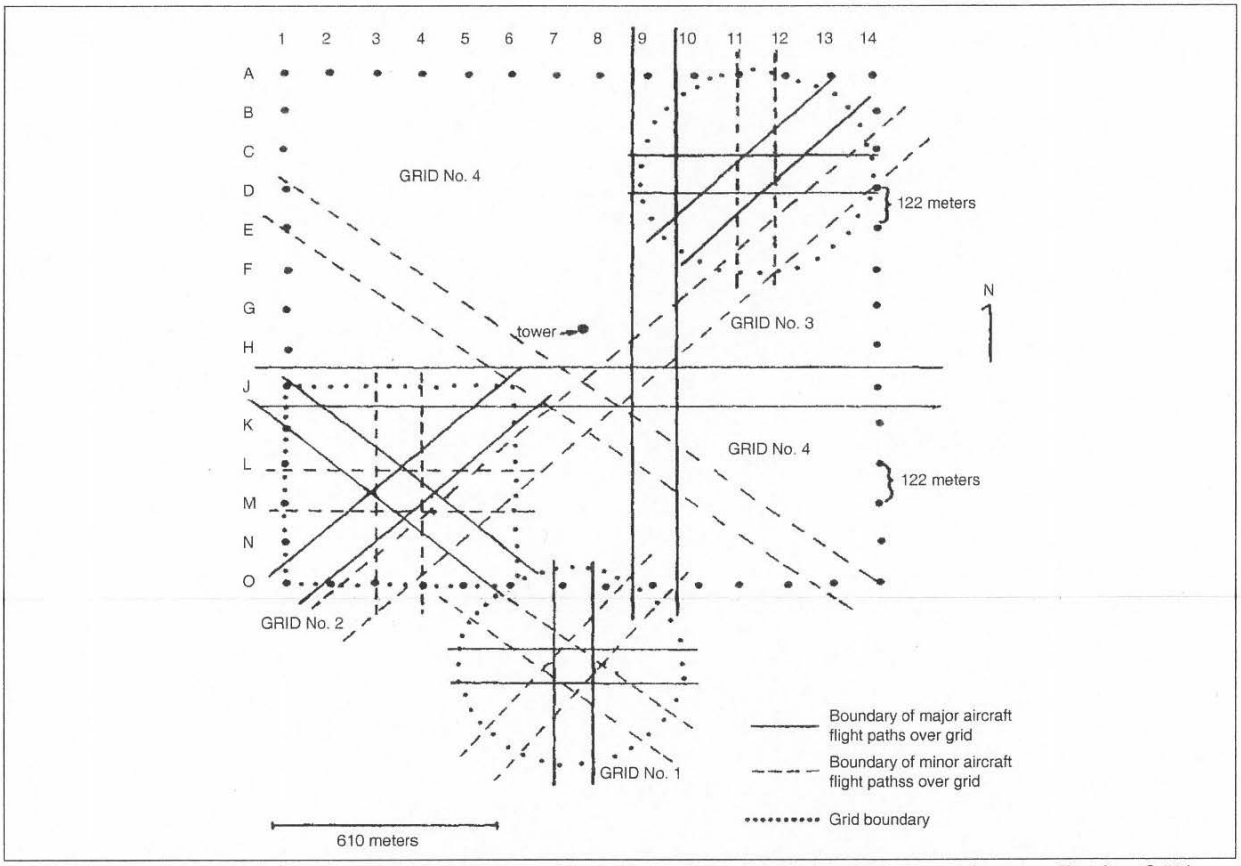


Fig. 2: Location of major flight paths over the individual test grids used for testing and evaluating aerial spray equipment on Test Area C-52A

1004. The last spray equipment test using Agent Orange was in December 1969, while the last test with Agent White was in early 1970.

1005. Despite records of the number of missions and the quantity of herbicide per mission, no attempt has yet been made to determine the exact quantity of herbicides deposited.

1006. Agent Orange consisted of equal volumes of concentrated n-butyl esters of 2,4- di-chloro-phenoxy-acetic acid (2,4-D) and 2,4,5-T herbicides.

1007. Agent Purple consisted of both the n-butyl ester and iso-butyl ester of 2,4,5-T (30% and 20 %, respectively), and the n-butyl ester of
2,4-D.

1008. Agent White consisted of the 1:4 mixture of the tri-isopropanolamine salt of picloram (4- amino-3,5,6-trichloro-picolinic acid) and 2,4-D.

1009. Agent White was commercially available as Tordon 101® manufactured by The Dow Chemical Company.

1010. Agent Blue contained cacodylic acid, as the free acid, and the sodium salt of cacodylic acid, sodium cacodylate.

1011. Agent Blue was commercially available as Phytar 560G® manufactured by Ansul Chemical Company for the government.

Herbicide locations at Eglin Air Force Base

1012. In 1992, the Air Force identified eleven significant known and suspected Herbicide Orange locations at Eglin AFB: C–52A Test Grid (SS-25), Mullet Creek Drum Disposal Site (DP-09), C–52A Aerial Overspray Area (AOC-24), Hardstand 7 (SS-26), Receiver Landfill (LF-08), Upper Memorial Lake (LF-51), 3 sites at Lower Memorial Lake (AOC-81), Field No. 2 Drum

Disposal (DP-11), and Field No. 2 Helicopter Loading Area (AOC-55).

Test and evaluation projects on test area C–52A

1013. Located about a mile and a half northeast of the community of Choctaw Beach in Walton County, Site C–52A is a one square-mile plot carved out of the massive Eglin test ranges.

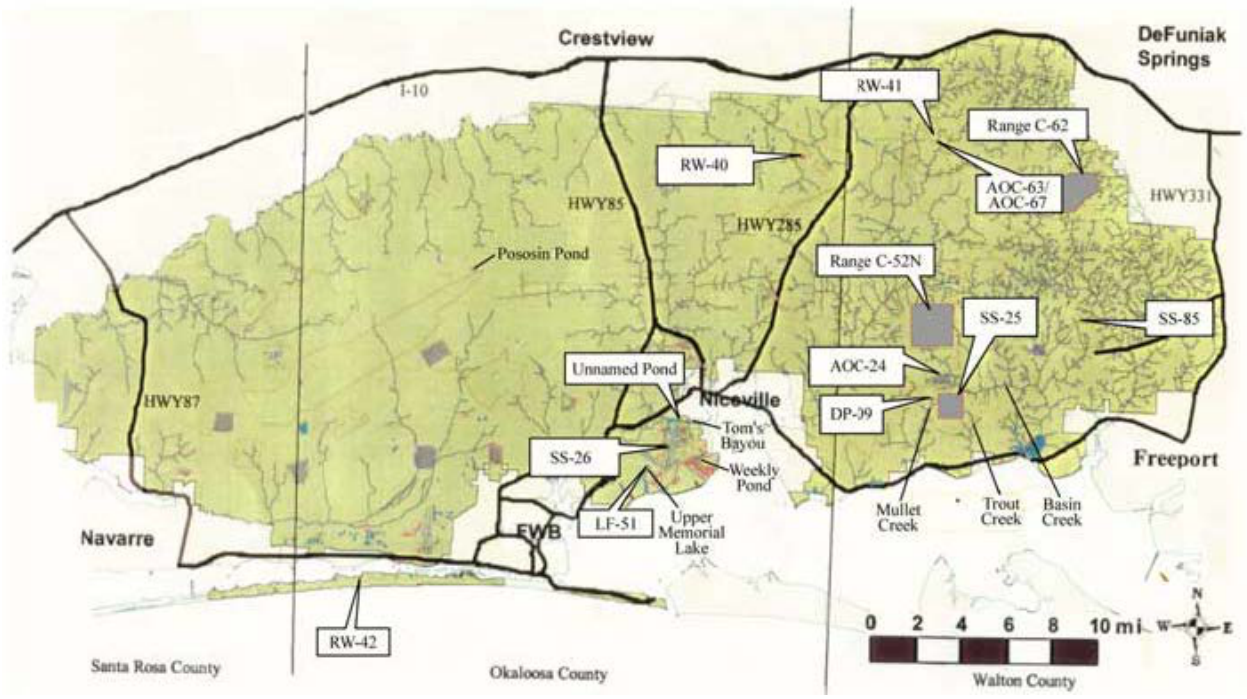
1014. The location is off-limits to the public.

1015. Together, the C–52A Herbicide Test Grid and the Mullet Creek Drum Disposal Site are known as the Herbicide Exposure Unit. The sites are located in a rural area in the southeastern section of the Eglin Reservation, about 3 miles north of Choctawhatchee Bay and 8 miles east of Niceville, Florida.

1016. The C–52A Herbicide Test Grid is located about 10,930 feet from the nearest base boundary.

1017. The Eglin Reservation with sites identified

Eglin Reservation



1018. The Mullet Creek Drum Disposal Site is located about a ½ mile west of the C–52A Herbicide Test Grid about 10,230 feet from the southern base boundary.

1019. The C–52A Aerial Overspray Area is in the vicinity of the Herbicide Exposure Unit. When climatic conditions were not appropriate for aerial spraying at the Herbicide Exposure Unit, aircraft would spray the already-loaded herbicides in this area. TCDD and arsenic in the soil are the primary contaminants.

1020. Upper Memorial Lake is located on Eglin Main Base south of the east-west runway. A site north of the lake was identified as a former burial area used to dispose of herbicide drums and TCDD has been detected in the subsurface soil.

1021. The test grids were uniquely arrayed to match the needs of fixed-wing, helicopter, or high-performance jet aircraft.

1022. According to Plaintiff Donald Cutts of Niceville, "The grid was marked off, and every 500 feet there was what we called a 'sniffer station'. In between each sniffer station was a pole with a six-inch square paper card on top of it where the spray would collect. We'd go down the rows and collect the sample cards in a box and take them back to Eglin after every mission. We didn't wear gloves or masks or nothing."

1023. According to Plaintiff Terrell Gatlin of Dorcas, "During the testing, would set up 'mobile communication units' – basically pick-up trucks topped with a flashing amber light.

1024. "We would park one on the east end of the grid with the light burning, and one on the west end. The aircraft would come across from east to west, and when the pilot reached the amber light on the east side, he turned on his spray. When he reached the light on the other side, he turned it off."

1025. From 1962 through 1964, Grid 1 was used for the calibration and refinements of the UC-123B/MC-1 Defoliant Spray System, the A-lH/FIDAL Helicopter Defoliant Spray System (the AGRINAUTICS System), and the H-34/HIDAL Helicopter Defoliant Spray System.

1026. These systems involved the dissemination of Agent Purple and were the initial defoliation systems deployed in Vietnam.

1027. From 1964 through 1966, modifications of the UC-123B/MC-1 and the newly developed NA45Y-1 Internal Defoliant Dispenser were evaluated for the dissemination of Agent Orange.

1028. From late 1966 through 1968, testing and evaluation were conducted on the jet-modified aircraft (UC-123K) with the NA45Y-1 Internal Defoliant Dispenser.

1029. In 1969 through 1970, additional modifications were evaluated in the UC-123K system.

1030. In addition, first evaluation of both herbicide (Agent Blue) and insecticide dissemination by high performance jet aircraft (F-105 and F-4) with the PAU spray system was undertaken.

Hardstand 7

1031. Site SS-26, Hardstand 7 (previously known as ERP Site T3), is located to the west of the north-south runway on the Eglin Main Base, Okaloosa County, Florida. It is east-northeast of Building 914, the Explosive Ordnance Disposal (EOD) facility (Figure 26-1). Map coordinates are Latitude 30°29'19" N. and Longitude 86°31'10"W

1032. The Hardstand 7 site was used for storage of herbicide drums and transfer of herbicides to aircraft for dissemination on the Herbicide Test Grid at C–52A. Several investigations have been conducted since 1970 to characterize the soil, water quality, and biota in the vicinity of Hardstand 7. In 1985, the entire area around Hardstand Pond and Beaver Pond was fenced and signs were posted banning trespassing and fishing, as recommended in a 1984 report.

Field No. 2 Drum Disposal Area

1033. Site DP-11, previously known as ERP Site D17, is located on Range C-3 (Auxiliary Field No. 2) within the Eglin Reservation (Figure 11-1), approximately 5 miles northeast of Niceville, Florida, on Highway 285, in Okaloosa County, Florida. The Drum Disposal Area is 500 feet west of Highway 285 at the southern end of a field, along a steephead, above an unnamed pond. Map coordinates are Latitude 30°3'14" N. and Longitude 86°26'33" W.

1034. Reportedly, empty fuel drums, partially full solvent drums, and herbicide drums were disposed of along the crest of the steephead and then covered with soil. Drums were found along the steephead in the 1960s and 1970s and subsequently disposed of.

DEFENDANTS DUTY OF CARE AS GOVERNMENT CONTRACTORS

1035. Upon information and belief at all the times relevant to the claims of the Plaintiffs, the United States Air Force in general, and those units of the United States Air Force responsible for operations at Eglin Air Force Base, in particular, were, and they still are concerned about the conduct and individual responsibility of civilian contractors with respect to the health and safety of their employees and other civilian employees of the United States government.

1036. Upon information and belief at all the times relevant to the claims of the Plaintiffs, the United States Air Force in general, and those units of the United States Air Force responsible for operations at Eglin Air Force Base, in particular, were, and they still are intent upon maintaining a productive work environment in which management and employees comply with standards of conduct and responsibilities essential to the effective functioning of the Air Force and accomplishment of its national security mission.

1037. Upon information and belief at all the times relevant to the claims of the Plaintiffs, the United States Air Force in general, and those units of the United States Air Force responsible for operations at Eglin Air Force Base, in particular, did, and they still do, among other requirements, insist that all contractors comply with safety and health standards set for the job environment.

1038. The Defendants were each government contractors, and their management executives, employees, agents and subcontractors who provided materials, goods and services, directly or indirectly, to the United States Department of Defense during the war in Southeast Asia owed the Plaintiffs a duty to take reasonable care to protect them from the dangers and hazards

associated with and resulting from unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1039. The Defendants were each government contractors, and their management executives, employees, agents and subcontractors who provided materials, goods and services, directly or indirectly, to the United States Department of Defense during the war in Southeast Asia knew, or with the exercise of reasonable care and concern for the safety and health of the Plaintiffs should have known, that they had not taken reasonable care to protect the Plaintiffs from the dangers and hazards associated with and resulting from unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site-C52A.

1040. The Defendants owed a non-delegable duty of care to their employees and contractors involved with the herbicide testing program at Eglin Air Force Base each of them was a foreseeable and probable victim of physiological injury, systematic disease, genetic damage and death as a result of their unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1041. That the Defendants had a non-delegable duty to advise the Plaintiffs and their families they had been exposed to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A and warn the Plaintiffs and their families that they were in danger of physiological injury, systematic disease, genetic damage and death as a result of that exposure.

1042. The Defendants were each government contractors, and their management executives, employees, agents and subcontractors

who provided materials, goods and services, directly or indirectly, to the United States Department of Defense during the war in Southeast Asia did comply with safety and health standards set for the job environment by the United States Air Force in general, and those units of the United States Air Force responsible for operations at Eglin Air Force Base, in particular.

THERE IS NO GOVERNMENT CONTRACTOR IMMUNITY

1043. Government contractors are only entitled to immunity by virtue of that relationship if they can establish by a fair preponderance of substantial credible evidence that the United States approved reasonably precise specifications; the herbicides supplied by the Defendant manufacturers conformed to those specifications; and the herbicide manufacturers warned the United States about the dangers in the use of the herbicides that were known to the supplier but not to the United States.

1044. By reason of the unique and specialized knowledge of the defendant herbicide manufacturers, the contamination of the phenoxy herbicides sold to the government with 2,3,7,8-tetrachlor-dibenzo-p-dioxin was not merely a negligent manufacturing defect; it was a conscious, intentional, and discretionary decision by the management and marketing officials of the defendant herbicide manufacturers to use particular time- or cost-saving procedures that increase the probability of contamination with the highly toxic TCDD, and as such became a design defect.

1045. That the overriding specification provided to the manufacturers of the herbicides to be used as chemical defoliants during the war in Southeast Asia were that they must be safe and "non-toxic to humans and animals."

1046. The herbicides supplied by the Defendant herbicide manufacturers were highly toxic to human beings.

1047. The Defendant herbicide manufacturers had actual knowledge about the toxicity of their products that was unknown to the United States at the time the products were used during the war in Southeast Asia.

1048. That at no time during the war in Southeast Asia did the Defendant herbicide manufacturers ever warn any individual in the United States government with decision-making authority concerning the deployment of herbicides is chemical defoliants during the war in Southeast Asia that the herbicides as they were supplied were toxic to human beings.

1049. The Defendant herbicide manufacturers had actual knowledge that the levels of the dioxin contaminant in the phenoxy herbicides supplied to the government for use as chemical defoliants varied.

1050. The Defendant herbicide manufacturers had actual knowledge that the government would indiscriminately mix the chemical defoliants without regard to the manufacturing source.

1051. There is no substantial credible evidence that the Defendant herbicide manufacturers ever shared their knowledge of dangers of which they were aware with the government.

1052. There is no substantial credible evidence that any decision makers in the government had greater knowledge about the contamination of the phenoxy herbicides with 2,3,7,8-tetrachloro-dibenzo-p-dioxin and about the toxicity of TCDD than the Defendant herbicide manufacturers or even that they had equivalent or substantial knowledge about the dangers of phenoxy herbicides in its planned use for chemical defoliation in Vietnam.

1053. That at no time during the war in Southeast Asia did the Defendant herbicide manufacturers ever warn any individual in the United States government with decision-making authority concerning the deployment of herbicides is chemical defoliants during the war in Southeast Asia about the actual and varying levels of TCDD in each batch of the herbicides they supplied and that the TCDD contaminant was highly toxic to human beings.

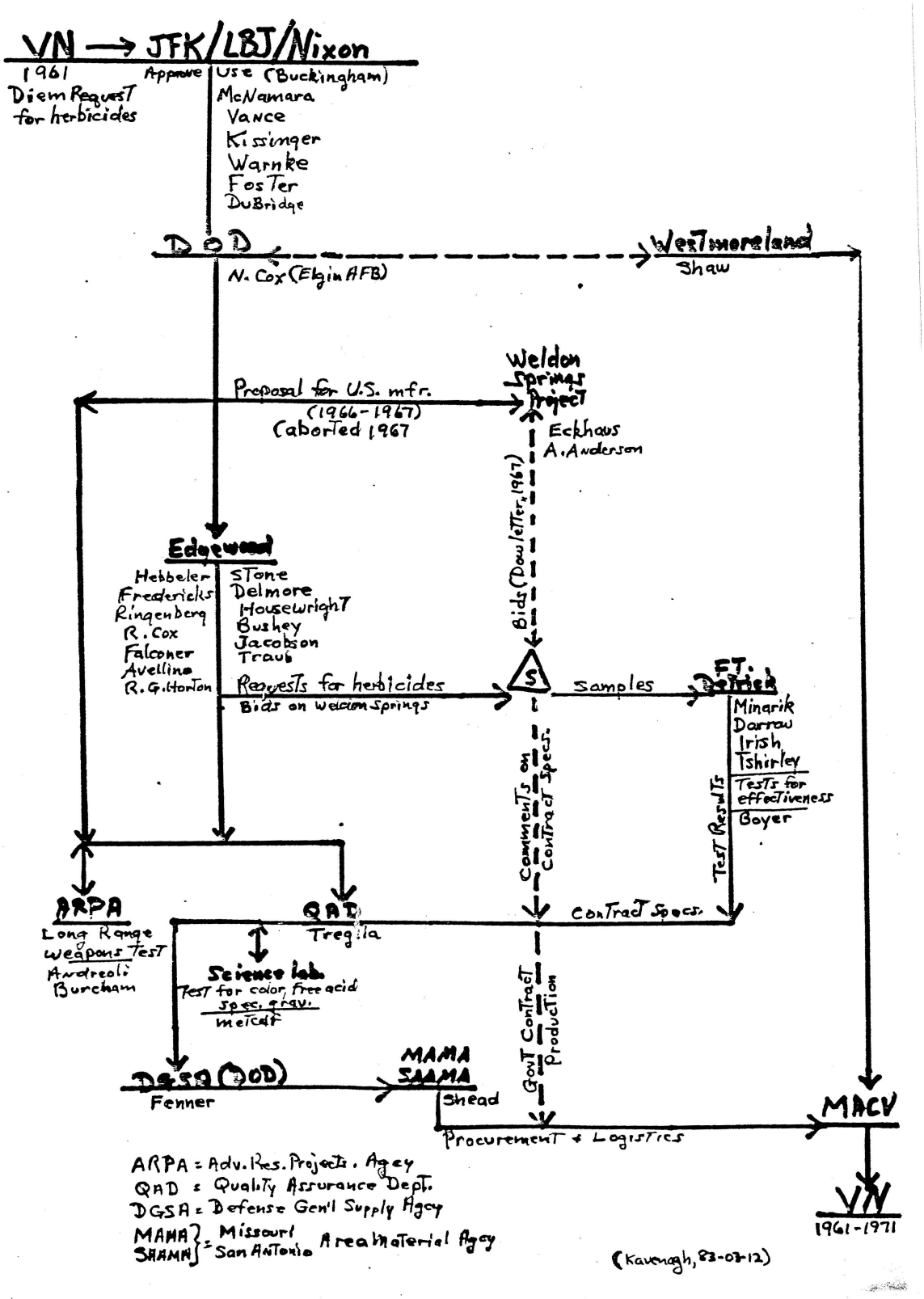
1054. In 1959, Dr. Friedrich Hoffman, a chemical warfare specialist and chief of the Agents Research Branch at Edgewood Arsenal, Md., according to a document still partly classified, reported that he learned in Europe "startling information" about the toxicity of a compound he identified as dioxin. He said because it has caused "several deaths of workers in the plant," Dr. Hoffman's secret report concluded "that dioxin should not be used for chemical warfare because it is too deadly."

THE WEB OF RELATIONSHIPS AMONG GOVERNMENT EMPLOYEES

1055. The depositions taken during the original Agent Orange litigation, MDL–381, from witnesses with actual knowledge of the facts clearly establish the entire web of knowledge about the dioxin contamination of the phenoxy herbicides deployed as chemical defoliants during the war in Southeast Asia.

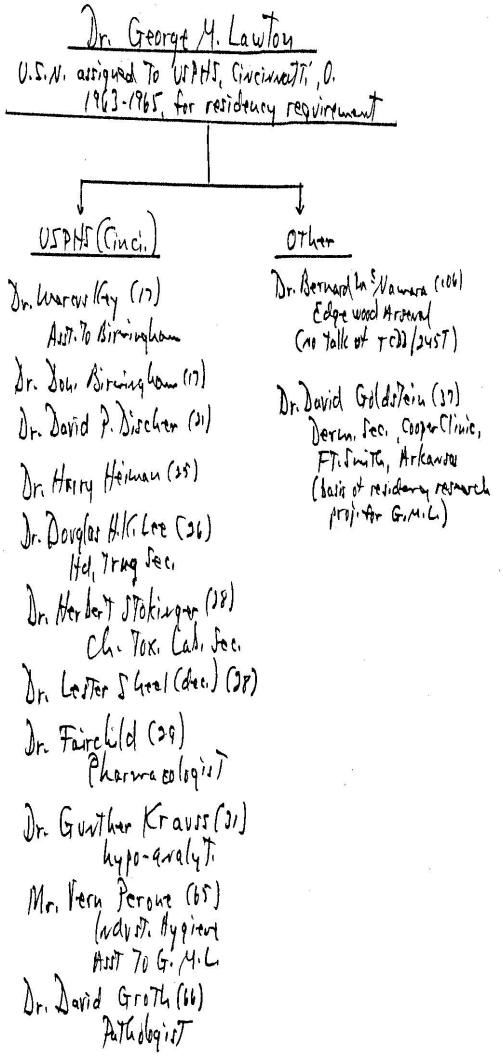
1056. The following are fair and accurate copies of the analysis of the relationships among all those with knowledge of the government herbicide program and the chemicals involved which were submitted by attorneys, W. Keith Kavenagh and Victor John Yannacone, jr. on behalf of the American combat veterans of the War in Southeast Asia in opposition to the motions for summary judgment made by the corporate defendant war contractors in MDL 381. The basis for the motions was the claim of government contractor immunity.

1057. There is no question that any information concerning the dioxin contamination of the herbicides used as chemical defoliants during the War in Southeast Asia was highly compartmentalized, often classified SECRET, and circulated among a small group of individuals who never presented the information to the decision makers responsible for the deployment of chemical defoliants during the War in Southeast Asia.

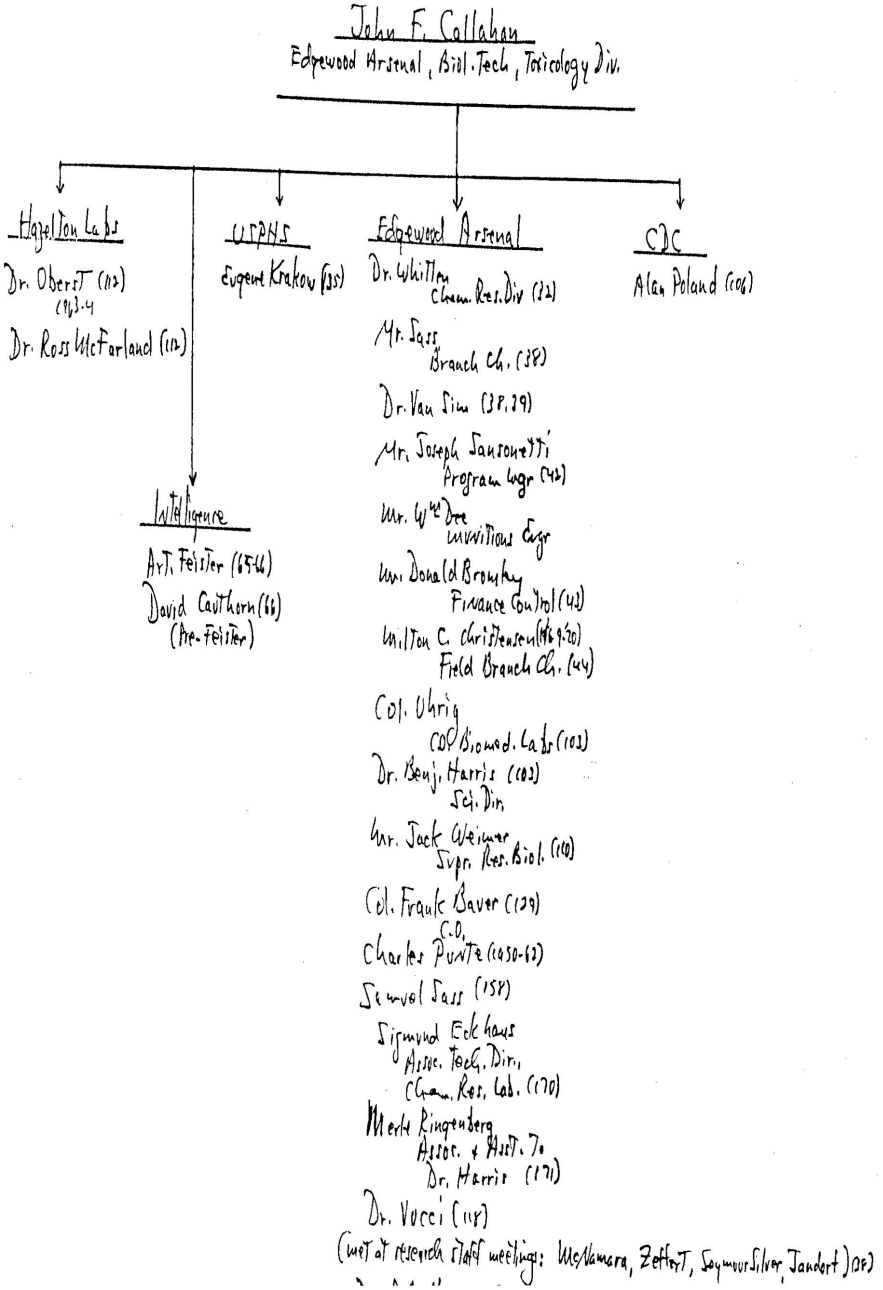


The web of relationships among government employees

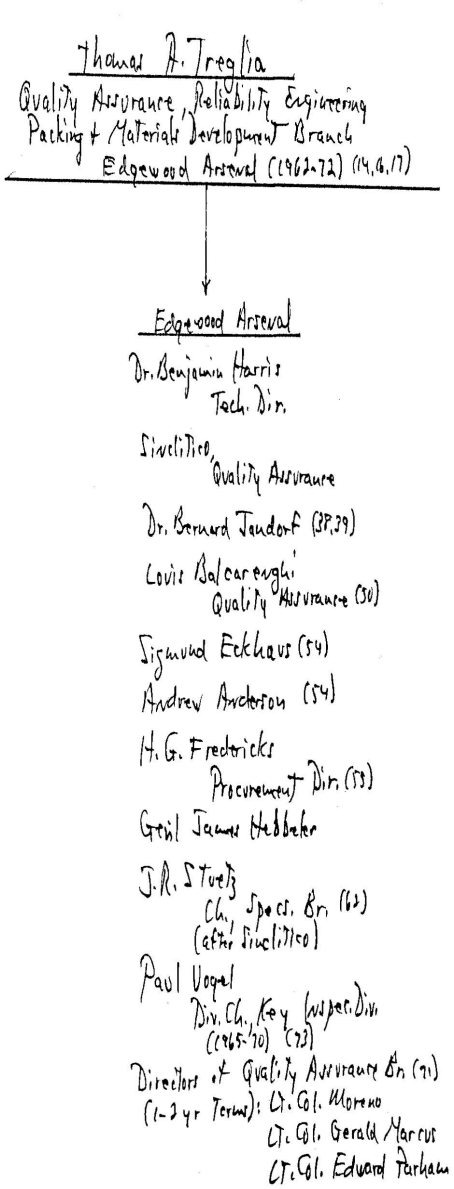
1058. Dr. George Lawton, USN, USPHS, Cincinnati, OH.



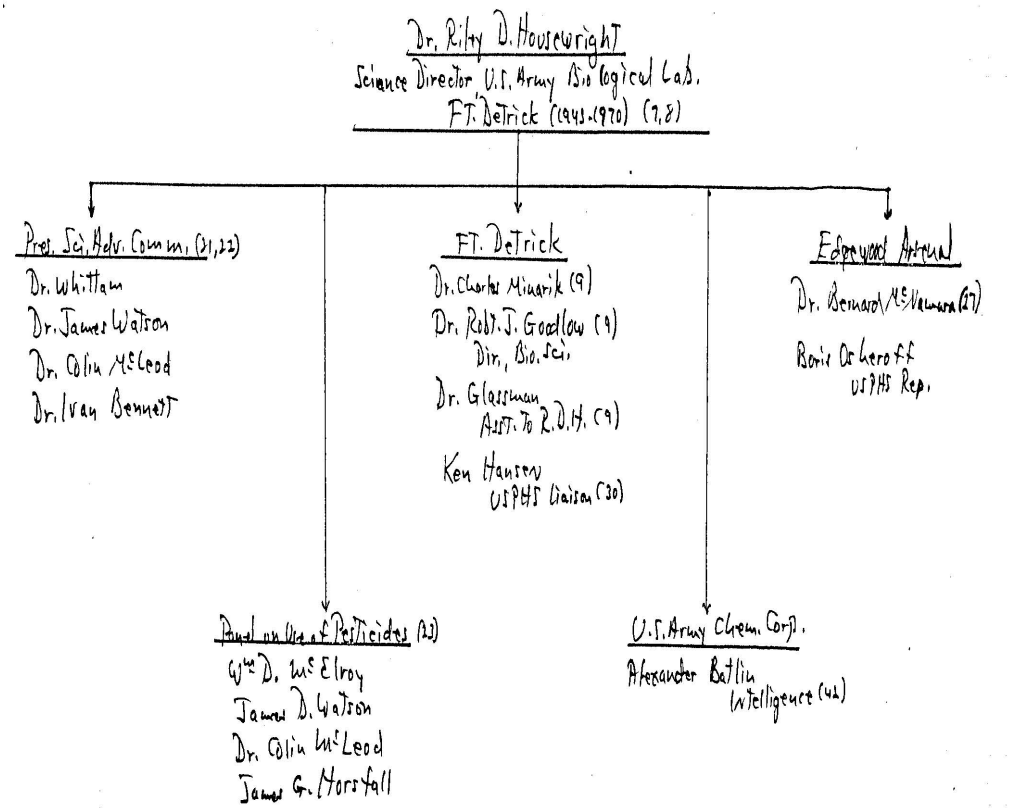
1059. John F. Callahan, Edgewood.



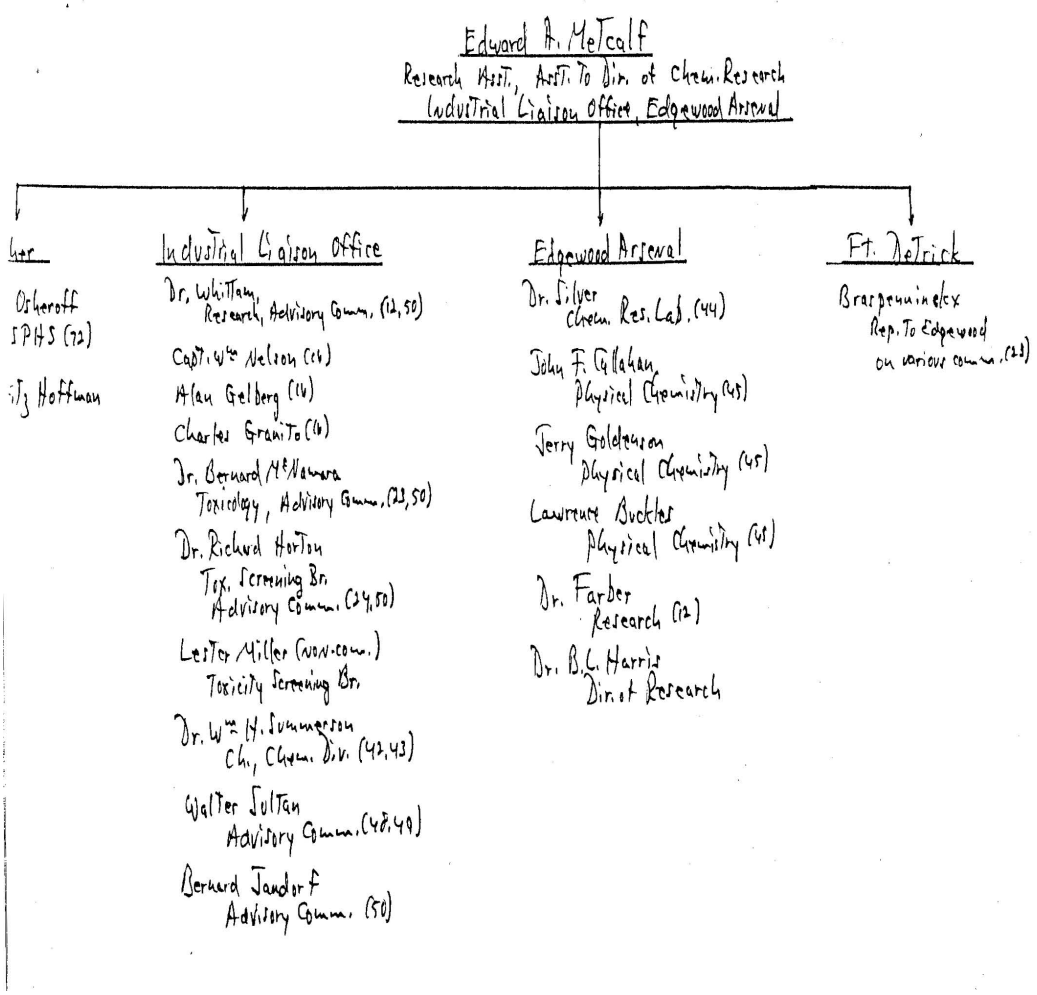
1060. Thomas A. Treglia, Edgewood.



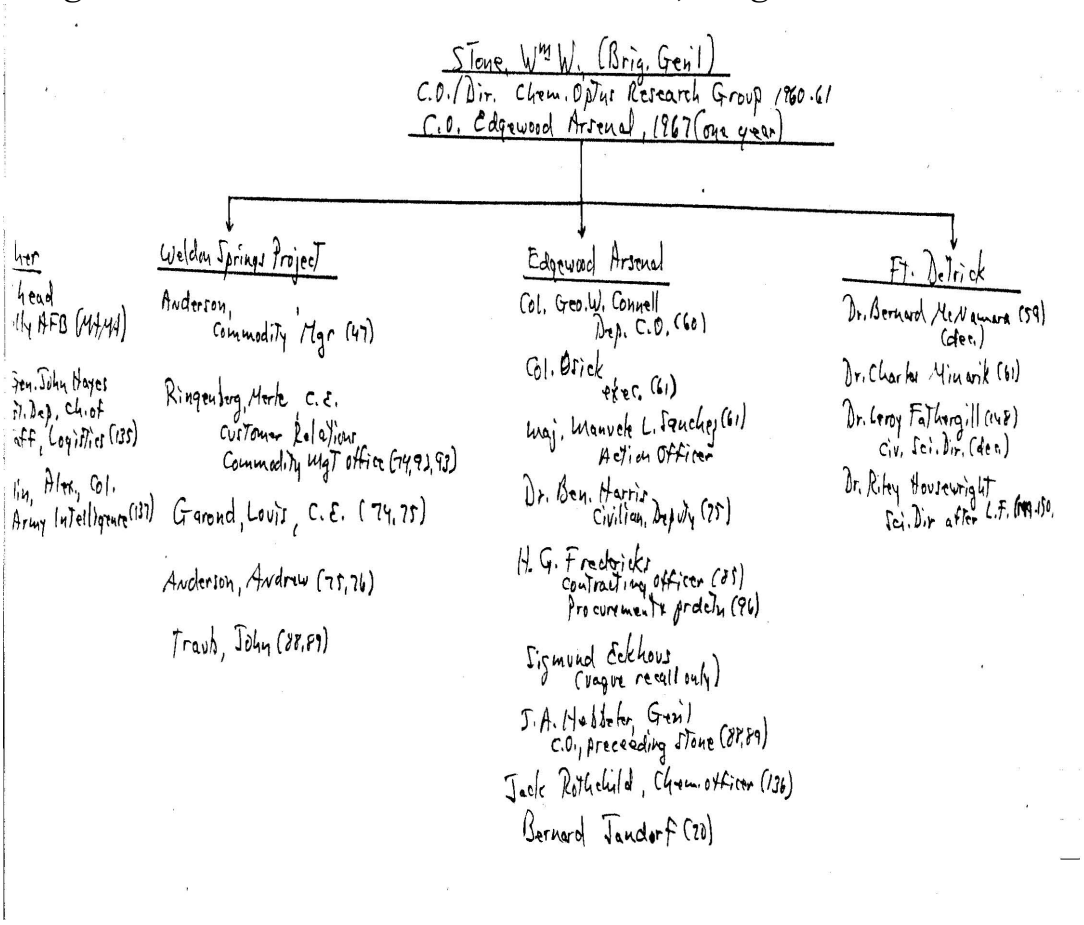
1061. Dr. Riley D. Housewright, Fort Detrick.



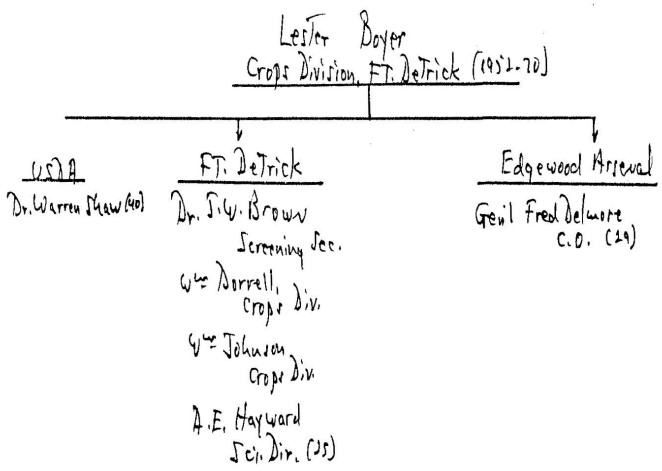
1062. Edward A. Metcalf, Edgewood.



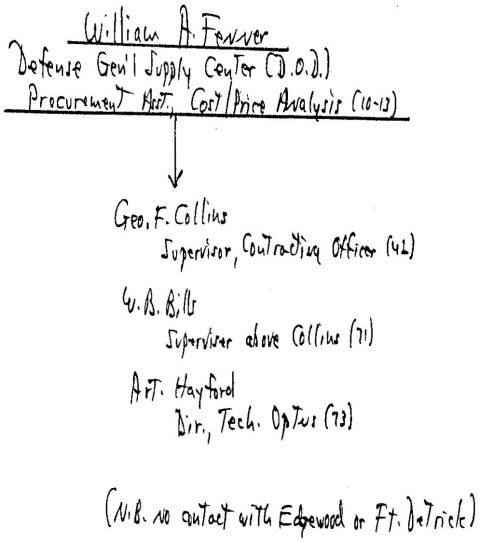
1063. Brigadier General William W. Stone, Edgewood.



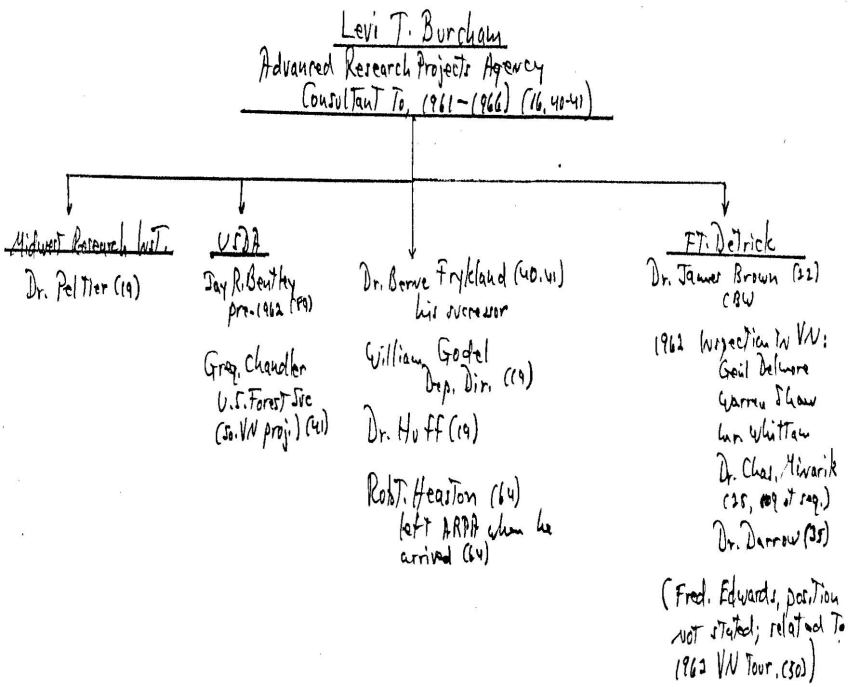
1064. Lester Boyer, Fort Detrick.

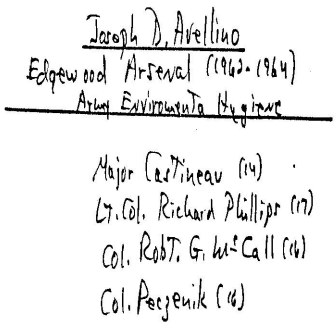


1065. William A. Fenner, Defense General Supply Center.



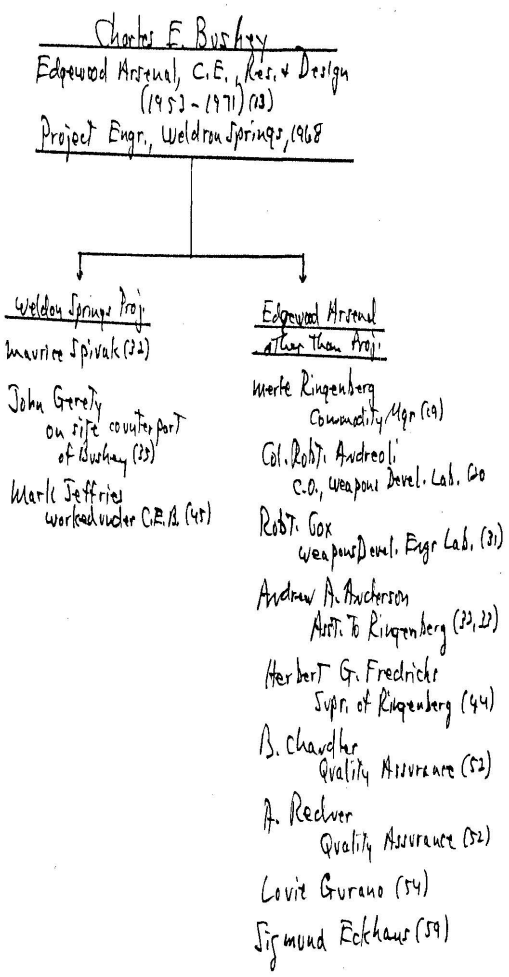
1066. Levi T. Burcham, ARPA and Joseph D. Avellino, Edgewood.





[N.B. vague recall only; remember no other names; Names suggested were those common to other days.]

1067. Charles E. Bushey, Weldon Springs.



The limited actual knowledge of the government

1068. During World War II, the military discovered the herbicidal properties of 2,4,5-T and conducted extensive testing of various possible herbicides.

1069. This research was conducted under the supervision of the Crops Division of the Army Chemical Corps at Camp Detrick, Maryland.

1070. In 1949, Dr. Donald Birmingham of the Public Health Service visited Nitro, West Virginia, where there had been an explosion at Monsanto's 2,4,5-T plant. The report of Dr. Birmingham's colleague, Dr. Louis Schwartz, indicted a

connection between chloracne and the chemicals produced in the plant.

1071. Dr. Birmingham of the U.S. Public Health Service published an article, "New Causes of Occupational Dermatoses, 20 *Industrial Health* 489, 490 (1959), stating that in the manufacture of 2,4,5-T, intermediate hydrocarbons of the chlorine group had caused chloracne in more than 200 chemical workers at a manufacturing plant which we now know was the Monsanto facility at Nitro, West Virginia.

1072. In the early 1950s, C.H. Boehringer Sohn Company of Germany had serious cases of chloracne among workers engaged in the production of trichlorophenol (TCP), a precursor chemical used *inter alia* to manufacture 2,4,5-T. By 1955, the Boehringer company was forced to halt TCP production at two plants.

1073. D. K. H. Schulz, a skin specialist, investigated the Boehringer chloracne problem and in 1957 together with Professor J. Kimmig, reported his findings in an article entitled as translated from the German, "Chlorinated Aromatic Cyclic Ethers as the Cause of Chloracne but published only in German in 44 *Die Naturwissenshaftern* 337 (1957). In this article, the authors stated that they were able to isolate dioxin, which they believed to be the contaminant in TCP that was causing the health problems.

1074. There is no substantial credible evidence that anyone in the government read the Kimmig and Schulz article at the time it was published, and at that time there were no general index systems where a high-level decision-making government official might have routinely come across the article much less related it to the use of phenoxy herbicides during the War in Southeast Asia

1075. Dr. Friedrich W. Hoffman went to Germany in 1959 to search among his former Nazi chemical-biological warfare colleagues for potential chemical warfare agents. In the 5 October 1959 "Hoffmann Trip Report," Dr. Hoffman reported that he had received "startling information" regarding the toxicity of a dioxin compound which was a contaminant of the wood preservative Perna.

1076. In the "Hoffmann Trip Report," Dr. Hoffman described the deaths of several workers in a plant that produced wood preservatives containing trace amounts of dioxin. In addition, he reported that the compound could cause severe, indeed fatal, liver damage. Dr. Hoffman apparently referenced the Kimmig and Schultz paper in a note to the "Hoffmann Trip Report."

1077. At least 10 copies of the Hoffmann report all classified "SECRET" were distributed at the Army Chemical Corps Warfare Laboratories at Edgewood Arsenal, but there is no substantial credible evidence that any government decision maker with management authority over the deployment of phenoxy herbicides during the War in Southeast Asia were aware of the connection between dioxin and TCP or the use of dioxin contaminated TCP in the manufacture of 2,4,5-T.

1078. Dr. Bernard Jandorf, chief of the Army Chemical Research Laboratory, testified that people at Edgewood knew about the toxicity of dioxin since the late 1950s.

1079. Dr. Richard Horton, a toxicologist, testified that he knew dioxin was toxic in 1959, as did Dr. Thomas Simmons, who worked in the Agents Research Branch. Walter Sultan, a pharmacologist in the Toxicity Screening Branch, testified that he had read the Hoffmann report.

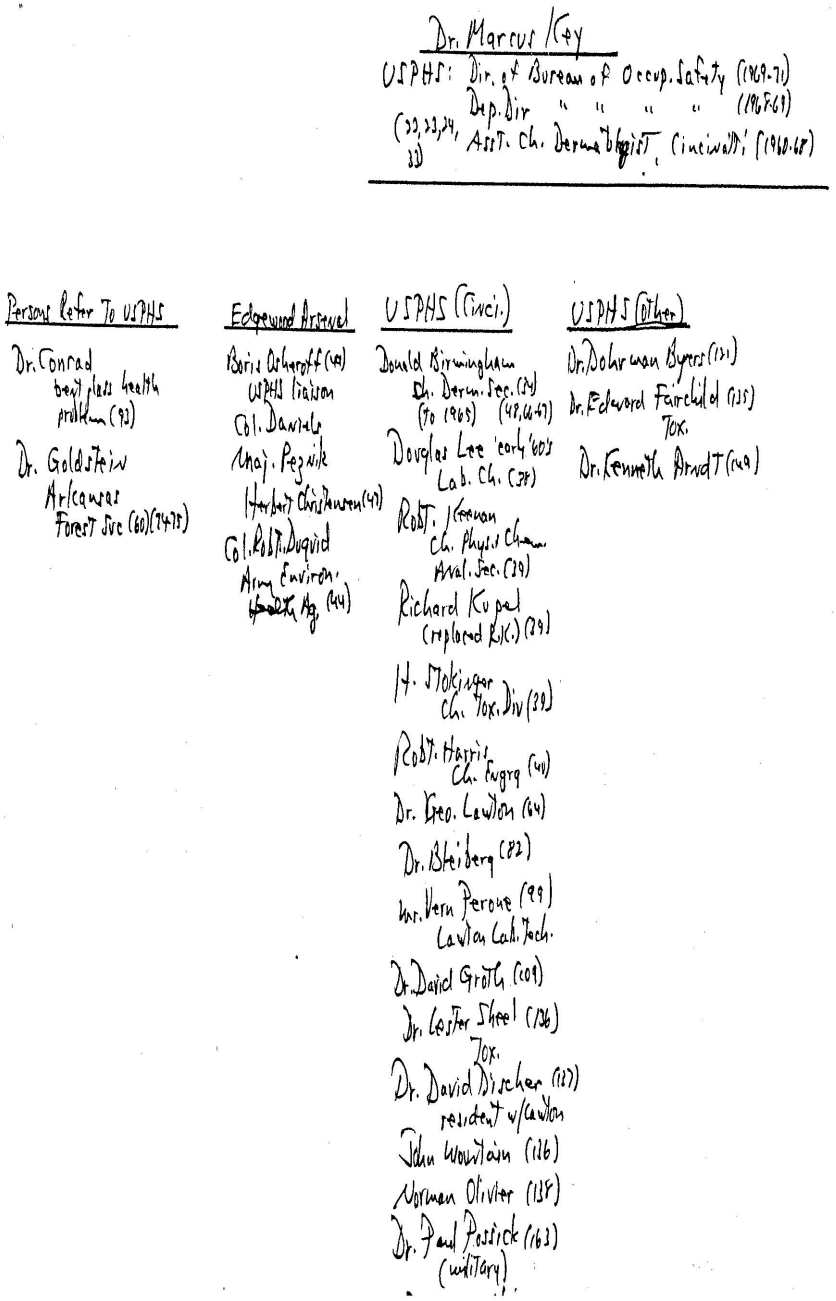
1080. Dr. Marcus Key of the Public Health Service testified that he had learned of the association between hydrocarbons and

chloracne and other diseases at the Harvard School of Public Health in 1953.

1081. Dr. Key of the Public Health Service testified at his deposition that in 1963 he placed a sample of 2,4,5-T herbicide on his forearm to see if it would induce chloracne. He did this three times a week for three weeks and developed chloracne on his forearm. He also testified that he had read Kimmig & Schulz and learned of dioxin from that article. When questioned concerning a June 1964 article by Dr. Jacob Bleiberg, "Industrially Acquired Porphyria," 89 *Archives of Dermatology* 793 (1964), which discussed chloracne and porphyria in workers engaged in 2,4,5-T production, Key stated that he had reviewed the article at the time it was written.

1082. The following is a fair and accurate representation of those with whom Dr. Marcus Key, of the U.S. Public Health Service in Cincinnati, OH had direct contact and/or association with during the period dioxin contaminated herbicides were deployed as chemical defoliants during the War in Southeast

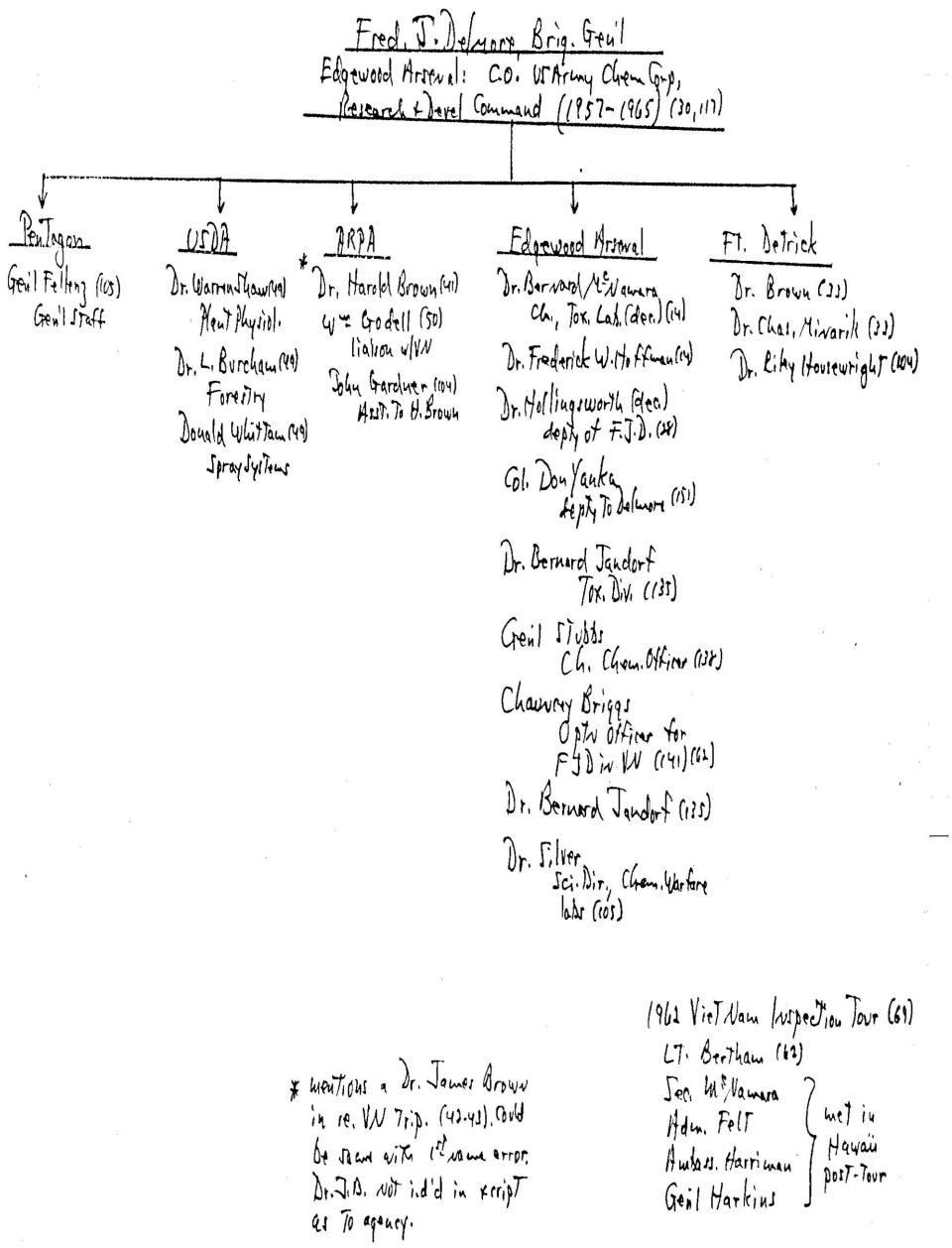
Asia.



1083. In the early 1960s, Dr. Bernard McNamara, Chief of the Toxicology Division at Edgewood, performed a toxicity study at Edgewood Arsenal on Agent Purple, a predecessor of Agent Orange of the toxicity of Agent Purple, another defoliant containing 2,4,5-T that was used by the military. This testing was conducted at the request of General F. J. Delmore,

Commanding General, U.S. Army Chemical Corps, Research & Development Committee and indicated inconclusively that there was some toxicity. However, there is no substantial credible evidence that the toxicity studies performed by Dr. McNamara would ever have revealed the toxicity attributable to the dioxin contaminant, nor is there any substantial credible evidence that Dr. McNamara was aware of the extent of the dioxin contamination of the phenoxy herbicide component of Agent Purple.

1084. The following is a fair and accurate representation of those with whom Brigadier General Fred J. Delmore of Edgewood had direct contact and/or association with during the period dioxin contaminated herbicides were deployed as chemical defoliants during the War in Southeast Asia. .



1085. On April 26, 1963, the Army conducted a meeting at its Edgewood Maryland Arsenal "to evaluate the toxicity of a[n herbicide] mixture known as 'Purple'" to reaching a conclusion "about dose levels and hazards to health of men and domestic animals from 2, 4-D and 2,4,5-T based on the medical literature and unpublished data of various research laboratories."

1086. Those in attendance included officials from various branches of the military and various other government agencies, and representatives from manufacturers Dow Chemical and AmChem Products.

1087. Although there were a number of incidents involving 2,4-D and, 2,4,5-T about which the Defendant herbicide manufacturers were fully aware, the minutes from the 1963 meeting at Edgewood Arsenal contain references to a lack of workplace incidents.

1088. The minutes of the April 26, 1963 Meeting record that an AmChem representative related experiences of "industrial firms making continuous field applications over very large areas" and noted "skin sensitization was the maximum effect produced" in "probably one out of a thousand persons."

1089. The unpublished data relied upon by the Army was provided by the manufacturers, particularly The Dow Chemical Company and at the conclusion of the meeting, the participants adopted "acute toxicity" figures for Agent Purple based upon that unpublished data after concluding from that unpublished data related to 2, 4-D and 2,4,5-T in the context of the way these herbicides had been used for defoliation in military situations in Southeast Asia, that no health hazard is or was involved to men or domestic animals from the amounts or manner these materials were used.

1090. In 1963, The Institute for Defense Analysis wrote a report for the Advanced Research Project Agency, an agency within the Department of Defense. This report stated that herbicides were safe when used commercially, but that there could be increased hazards in military use because greater concentrations might be applied by less experienced personnel under the pressures inherent in battlefield use. The report

noted the connection between chloracne and skin and respiratory irritations and their association with herbicides.

1091. It has been alleged that on May 9, 1963, the President's Scientific Advisory Committee (PSAC) was briefed on the "Possible Health Hazard of Phenoxyacetates As Related to Defoliation Operations in Vietnam." However, there is no substantial credible evidence that such a briefing, if it ever occurred, provided the Committee with information about the contamination of phenoxy herbicides with 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin and how toxic that TCDD contaminant was to human beings. Rather, the domestic safety record of herbicides containing these two chemicals, including the manufacturers' alleged reports to the Department of Agriculture regarding the absence of ill effects from the herbicides on their workers, were presented.

1092. The information which was supplied to the PSAC may have resulted in the "Bionetics Study," a lengthy government-sponsored investigation by Diane Courtney, PhD of the effects of more than 100 pesticides including the herbicide 2,4,5-T.

1093. In July 1966, the director of the National Academy of Sciences wrote to the chief of the Bureau of Medicine and Surgery for the navy advising him of the connection between 2,4,5-T and porphyria and chloracne, neither of which are as life threatening as a Viet Cong/NVA ambush.

1094. In August 1966, the National Academy of Sciences, in response to a request for information, wrote to the Army Surgeon General telling him that 2,4,5-T was toxic and that chloracne was associated with it.

1095. In 1965, the National Cancer Institute contracted with Bionetics Research Laboratory in Maryland to investigate the possible teratogenic effects of a number of pesticides and

herbicides. Among the herbicides tested on mice and rats were 2,4-D and 2,4,5-T.

1096. In 1967, a Rand report commissioned by the Advanced Research Project Agency (ARPA) of the Department of Defense described "actual experience" of health hazards associated with the use of defoliants in Vietnam.

1097. The study, *Evaluation of Carcinogenic, Teratogenic, and Mutagenic Activities of Selected Pesticides and Industrial Chemicals*, the study was published in 1969 and provided the first published indication of the teratogenicity and fetotoxicity of 2,4,5-T. The principal investigator, K. Diane Courtney, concluded that 2,4,5-T was teratogenic, causing malformations and stillbirths in mice when administered in high doses, and that 2,4-D was potentially harmful.

1098. At the request of The Dow Chemical Company, Dr. Courtney repeated the tests with a sample of 2,4,5-tri-chloro-phenoxy-acetic acid (2,4,5-T) supplied by Dow with a very low level of TCDD contamination compared with the original samples which had been, upon information and belief, provided by Diamond Shamrock and were heavily contaminated with dioxin. Dr. Courtney concluded that the cause of toxicity was the contaminant TCDD and that 2,4,5-T itself was not teratogenic.

1099. It was this research that ultimately triggered cessation of the defoliation campaign according to Dr. R.A. Darrow of Fort Detrick.

1100. There is no substantial credible evidence that the information about dioxin or its contamination available to any of the government employees at Edgewood, Camp Detrick/Fort Detrick, the U.S. Public Health Service (USPS), including Dr. Birmingham, Dr. Schwartz, Dr. Hoffman, Dr. Jandorf, Dr. Horton, Dr. Simmons, Mr. Sultan, Dr. Key, General Delmore,

Dr. Stokinger, Colonel Shade, Dr. MacDonald, and Dr. Hornig, about the dioxin contamination of the phenoxy herbicides deployed as chemical defoliants during the War in Southeast Asia or information about the extraordinary systemic toxicity of TCDD were ever transmitted or conveyed to any government decision maker with management authority over the deployment of phenoxy herbicides during the War in Southeast Asia or that such decision makers were personally aware of the connection between dioxin and TCP or the use of dioxin contaminated TCP in the manufacture of 2,4,5-T.

The first limited knowledge by the Department of Defense

1101. In 1970, Dow officials met with military officials to share their concerns over dioxin-contaminated Agent Orange. It followed this up with a letter to the Secretary of Defense, Melvin R. Laird, which said in part: "There is abundant evidence that 2,3,7,8-tertachlorodibenzo-p-dioxin occurring as an impurity in 2,4,5-T is highly toxic." It urged the Secretary to set specifications to ensure that products with no more than one part per million of dioxin be used.

1102. Dr. Robert Darrow, a military official who had met with Dow executives several months earlier in the Pentagon to receive the same information, recalled later in a deposition that he was "surprised" to hear Dow's warning. "In other words," he said, "this represented information that we were receiving for the first time in this respect on the dioxin situation."

DEFENDANTS' ACTUAL KNOWLEDGE

The special secret knowledge of The Dow Chemical Company

1103. The Dow was aware, but the United States government officials involved in the chemical defoliation program for the war in Southeast Asia were *not* aware that in the 1930s the 2,4,5-trichlorophenol which Dow advertised, marketed,

promoted, and sold as the wood preservative, Dowicide, was a source of chloracne.

1104. It is ironic that in 1937, M. G. Butler of Dow published a paper which stated that the chlorophenols should not be used until the mechanism by which they caused chloracne was understood. The Dow plant was closed temporarily. By 1956, Dow learned from secret communications with C.H. Boeringer Sohn in Germany that the toxic contaminant in 2,4,5-trichlorophenol was TCDD.

1105. In 1941, V.K. Rowe and others at the Dow Biochemical Research Laboratory developed a rabbit skin test who did for compounds reported to have caused acneform dermatitis.

1106. In 1943, J.F. Lontz, of the E .I. Dupont Company, patented the use of a monocarboxylic acid as a plant growth hormone. The chemical industry soon recognized that plant growth hormones could be utilized as herbicide. In 1945 and 1946, Mr. Jones with American Chemical Paint Company obtained two patents for the use of halogenated phenoxy monocarboxylic acids.

1107. In 1946, the American Chemical Paint Company accused Dow of infringing the Jones' patent for Methods and Compositions for Killing Weeds.

1108. Dow filed suit against American Chemical Paint Company to have the Jones' patent declared invalid, *American Chemical Paint Co.* v. *The Dow Chemical Company*, 161 F.2d 956 (6th Cir. 1947).

1109. Since 1948 and continuing through the war in Southeast Asia, Dow manufactured a number of commercial herbicides consisting of mixtures of 2,4,5-T and 2,4-D. Dow also patented a number of those commercial mixtures and trademarked and obtained trademark protection for their brand names.

1110. In the manufacture of their phenoxy herbicides, Dow developed internal specifications for their manufacture, "accepted quality control procedures," and "quality requirements," all of which were treated as proprietary trade secrets and never publicly disclosed.

1111. Although Dow has never disclosed and withheld as proprietary and confidential the specific manufacturing processes it used from 1948 through 1970, it is generally known that Dow prepared the sodium salt of 2,4,5-T through a process known as caustic hydrolysis involving, in a mixture subject to heat and pressure.

1112. Dow does not deny knowledge of the 1949 explosion at the Monsanto plant in Nitro, West Virginia, in 1949, and the resulting cases of chloracne.

1113. Three patents issued to Dow on September 19, 1950 for new esters of 2,4-D. W.R. Mullison of Dow was awarded a patent for four new esters of 2,4-D in 1950.

1114. On July 31, 1950, Dow scientists were issued U.S. Patent Number 2,562,855 for new esters of 2,4,5-T.

1115. In the 1950s, C.H. Boehringer Sohn Company had chloracne problems wrote to Dow for help.

1116. Between 1952 in 1954 serious cases of chloracne had appeared at the Boeringer plants in Ingelheim and Hamburg, eventually causing both plants.

1117. To test for toxicity in their manufacturing facilities, rabbits were placed in each of the buildings with the doors and windows closed. All the rabbits died within five days and the necropsies showed pronounced liver changes and liver necrosis.

1118. In 1955, Dow replied by sending a data sheet describing the hazards due to toxicity and the precautions Dow was taking to prevent them.

1119. Dow does not deny knowledge of the outbreak of chloracne among workers at the Diamond Alkali, later Diamond Shamrock, plant in 1956 at Newark, New Jersey.

1120. Dr. K. H. Schulz of the University Skin Clinic in Hamburg-Eppendorf found that the "chloracne inciters" were present in the phenol and the 2,4,5-trichlorophenolate solution. Dr. Schulz further determined that the anisole fraction was the carrier of the chloracnegenic substance which he finally identified as 2,3,7,8-tetrachloro-dibenzo-p-dioxin.

1121. As a result of their identification of TCDD as the chloracnegenic agent in 2,4,5-trichlorophenol, Boehringer initiated certain process changes to prevent the formation of TCDD.

1122. On February 11, 1957 Boehringer provided Dow with information about the process changes necessary to prevent the formation of TCDD and the methods it had developed to prepare TCP so as to avoid "chloracne exciters."

1123. In April, 1957, Dr. Schulz published his findings in the German journal, *Die Naturwissenshaften*, (44:337–338). Dr. Schulz observed "damages to liver functioning" and "high general toxicity" of the compound.

1124. There is no substantial credible evidence that any government decision maker with management authority over the deployment of phenoxy herbicides during the War in Southeast Asia was aware of this paper or that this paper had been translated into English at any time prior to the conclusion of the war in Southeast Asia.

1125. During the Agent Orange litigation, MDL–381, Dow produced copies of the Schulz paper as well as an internal English translation, neither of which were ever provided to any United States government official involved in the chemical defoliation program during the war in Southeast Asia.

1126. Dow developed a "rabbit ear" test, which was non-specific, but was able to determine if a chloracnegen was present.

1127. Dow used this test until 1964

1128. Prior to becoming a "Government Contractor," Dow had invented , patented and sold commercial herbicides containing a 50/50 mixture of 2,4-D and 2,4,5-T.

1129. At some point prior to 1962, Dow moved production from a batch process to a continuous process.

1130. In July, 1963, Dow shut down building 199 in Midland, Michigan where 2,4,5-trichlorophenol was manufactured in order to install new equipment in order to increase its capacity to hydrolyze tetrachlorobenzene to 2,4,5-trichlorophenol. Dow was "pushing the existing reaction to its limit" by increasing the temperature.

1131. Dow had actual knowledge prior to 1964 that, depending upon the manufacturing process chosen, 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin could contaminate 2,4,5-trichlorophenol, an intermediate in the manufacture of 2,4,5-T.

1132. In February, 1964, workers in Building 199 at Dow's plant in Midland, Michigan where 2,4,5-trichlorophenol was being manufactured, more than 40 workers engaged in the manufacture of TCP showed clinical manifestations of chloracne.

1133. After shutting down the plant, Dow investigated the incident and found that there was a high concentration of chloracnegen

in the waste stream from the plant and that this chloracnegen was dioxin.

1134. In August, 1964, Dow used vapor phase chromatography to detect TCDD and its waste oils. The waste oils were fractionated and the TCDD isolated and identified. Dow then proceeded to synthesize TCDD in the laboratory.

1135. Dow developed a method of using gas chromatography to detect dioxin in TCP and in 2,4,5-T at concentration levels as low as 1 part per million (ppm).

1136. Dow researchers were satisfied that there was no chloracnegenic response if the dioxin level was at or below 1 ppm, but they never published their findings in the open peer reviewed scientific literature.

1137. In November, 1964 and later in April and June, 1965 Dow scientists produced a confidential memorandum with only limited circulation internally on a need to know basis within the company describing an analytical method for "The Determination of 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin in 2,4,5-trichlorophenoxyacetic acid by Gas-Liquid Chromatography." The paper described an analytical method for the detection of TCDD in the end product, the herbicide 2,4,5-T, not the intermediate 2,4,5-trichlorophenol.

1138. The Dow Chemical Company knew as early as the middle 1960's about evidence that exposure to dioxin might cause people to become seriously ill and even die, but the company withheld its concern from the Government and continued to sell herbicides contaminated by dioxin to the Army.

1139. The Kimmig & Schulz article was referenced in an internal memo written by a Dow official in 1964.

1140. On May 4, 1965, Dow established a specification requiring a gas-liquid chromatographic analysis of all 2,4,5-T leaving the

plant, but never advised any United States government official involved in the chemical defoliation program during the war in Southeast Asia.

1141. In March 1965, Dow called a meeting attended by Hercules, Diamond Alkali, and Hooker Chemical Corporation to discuss health hazards involved in the production of TCP and 2,4,5-T.

1142. No one from the government was invited to the meeting,

1143. At the time of that meeting, Dow knew that the dioxin problems arose during the manufacturing process and that any dioxin produced at that stage would carry forward into the delivered product.

1144. At the meeting, Dow explained that precautions were necessary to prevent health hazards and stated that it had examined herbicides sold by some other companies and found some to contain "surprising high levels" of dioxin.

1145. In a memo to the file after the March 1965 meeting, Dr. Edward Chandler of Diamond Alkali indicated that Dow thought that repeated exposure to 1 ppm could be dangerous.

1146. In June 1965, V. K. Rowe of the Dow Biochemical Research Laboratory wrote to Ross Mulholland of Dow Chemical of Canada and described the chloracne problems Dow had experienced and stated that Dow did not want any of its customers to develop acne.

1147. The letter also indicates a fear of government intervention into and control of the entire herbicide industry and further that Dow wanted to get the problem under control without governmental regulation. Mulholland was cautioned not to transmit this information to anyone else.

1148. Dow supplied Agent Orange to the military pursuant to seven contracts.

1149. Deliveries were made from September 1965 to December 1968.

1150. Dow admits that it knew prior to this that chloracne was an industrial health hazard present in the production of certain chlorinated hydrocarbons.

1151. In 1967, as the government considered whether to manufacture phenoxy herbicides itself through a contractor, Defendant The Dow Chemical Company in an internal document entitled "Plan 'Orange' Production, which was never shared with any government decision maker involved in the chemical defoliation program stated that "[a] serious potential health hazard to production workers is involved in the production of 2,4,5-T" and noted that its "knowhow regarding elimination of the hazard" could be made available to the government, but it never was.

1152. On February 29, 1967 A. P. Beutel, vice president of Dow, wrote a letter to Brigadier General J. A. Hebbeler concerning the government's plan for producing Agent Orange in a government plant and mentioned "certain health problems" inherent in the manufacturing process.

1153. On April 20, 1967 Beutel wrote to H. G. Fredricks, Deputy Director of Procurement and Production, concerning the same subject, government production of Agent Orange, and mentioning a "serious potential health hazard" to workers.

1154. In August 1967 Beutel and two other Dow representatives told two officials from the office of the Secretary of Defense that caution should be exercised in producing 2,4,5-T.

1155. On September 26, 1967 Beutel wrote to Andrew Anderson of Edgewood indicating that Dow would not bid on the government project to manufacture Agent Orange because of, among other reasons, the chloracne problem.

1156. In March 1970, Dow briefed representatives of the military on the presence of dioxin as an impurity in TCP and 2,4,5-T.

1157. On April 15, 1970, Julius E. Johnson, Vice-President and Director of Research for Dow, gave a statement before a subcommittee of the United States Senate Committee on Commerce stating "Since 1950 we have been keenly aware of the possibility of a highly toxic impurity being formed in 2,4,5-trichlorophenol as a side reaction under conditions of elevated processing temperatures… we also knew that if the impurity was present in the 2,4,5- trichloro–phenol it could be carried forward to the end product, 2,4,5-T.

1158. In June 1970, after temporary suspension of Agent Orange use, Dow wrote to Secretary of Defense Melvin Laird recommending "strongly" that the government set appropriate specifications and controls to ensure that no 2,4,5-T be used if it contained more than 1 ppm dioxin. Dow specifically urged that standard for any 2,4,5-T used as a component of Agent Orange if it was to be used as a defoliant in Vietnam.

1159. Documents suggest that Dow and other chemical companies shielded from the Government their information that one of the herbicides, Agent Orange, contained dangerous levels of dioxin.

1160. Dow, whose research on herbicides has long been relied on by the Government, has maintained that, aside from a skin disease known as chloracne, the company did not know of any harm to humans from dioxin.

1161. Yet in 1965, at a time when the Government was purchasing millions of pounds of Agent Orange, Dow's toxicology director wrote in an internal report that dioxin could be "exceptionally toxic" to humans, and the company's medical director warned, "Fatalities have been reported in the literature."

1162. Phillip Schneider, Dow's manager of media relations, said in July 1983 during the Agent Orange litigation (MDL 381) that "we are not aware of any human fatalities related to 2,3,7,8-

TCDD," the most potent form of dioxin. He said that the references to fatalities in the documents must have been to other substances and that Dow's experiences with its own workers had shown no long-term health effects from chloracne.

T.H. Agriculture and Nutrition Co

1163. T.H. supplied the government with Agent Orange pursuant to contracts dated June 28, 1967, March 1, 1968, and May 20, 1968.

1164. T.H. had manufactured 2,4,5-T for commercial sale as an herbicide prior to 1967. It did not make TCP but rather purchased it from other chemical companies.

1165. According to, T.H., in December, 1964, Dr. David Groth of the Public Health Service wrote to T.H. requesting samples of 2,4,5-T stating that 2,4,5-T was associated with chloracne and that it was suspected that dioxin was the culprit. Groth stated that he was attempting to develop a method to isolate the contaminant.

1166. In December 1964, Lindley S. DeAtley, a Vice President of Research and Development for T.H., wrote to Dr. R. C. Dosser, Laboratory Director at Dow, telling him of Groth's letter.

1167. Dosser replied in a telephone call that there might be some methods of production which led to a toxic compound.

1168. By January 1965, DeAtley had concluded that T.H. should conduct some tests of its own.

1169. On February 19, 1965, DeAtley and D. W. Fuhlage, T.H.'s Supervisor for Process Development, visited Dow's Midland plant.

1170. The minutes of the meeting indicate that DeAtley and Fuhlhage learned that cases of chloracne had recently been more severe, and that Dow had its workers changing clothing and showering at mid-shift.

1171. T.H. admits that it had cases of chloracne among its workers.

1172. In June 1967, when T.H. negotiated its first contract, it did tell the government that there was a chloracne problem in the manufacturing process and that this was factored into the price.

1173. Nor did T.H. ever tell the government that its chloracne manufacturing problem was probably caused by dioxin, or that the contaminant very likely carried over into the delivered Agent Orange.

1174. T.H. knew that gas chromatography could determine dioxin level, but never told the military about it even after it began to supply Agent Orange to the government.

1175. Eventually, samples of T.H. Agent Orange tested at Gulfport, Mississippi, indicate a dioxin level that went as high as 4.1 ppm.

Uniroyal, Inc.

1176. Uniroyal sold Agent Orange to the government pursuant to three contracts between October 6, 1966 and March 1, 1968.

1177. The product sold was not manufactured by Uniroyal, but rather, was supplied to Uniroyal by a Canadian subsidiary, now named Uniroyal, Ltd. (Ltd.).

1178. Evidence of Uniroyal's knowledge of chloracne problems is found in a memo from Ltd. to Uniroyal dated June 11, 1962, which states that five employees at the Clover Bar plant in Canada had symptoms of chloracne.

1179. The memo of T. H. Evans of Ltd. dated August 10, 1965 was found in the files of Walter Harris of Uniroyal and contains Evans' description of his visit to Dow's Midland plant in 1965 and the discussions he had with Dow scientists concerning chloracne problems and dioxin.

1180. Uniroyal knew much more about the possible hazards of Agent Orange use when it entered into its first contract with the government in 1966.

Thompson Chemical Corporation

1181. Originally, Thompson declined to bid for Agent Orange contracts. Acting under appropriate statutory authority, The Defense Production Act of 1950, Sept. 8, 1950, ch. 932, 64 Stat. 798, amended, June 30, 1966, 80 Stat. 235, however, the government required Thompson to supply Agent Orange pursuant to two contracts dated April 19, 1967 and May 24, 1968.

1182. Thompson supplied 333,685 gallons between September 1967 and January 1969. As reflected in test results, the dioxin content of its product ranged from .1 to .3 ppm.

1183. Soon after it began to manufacture Agent Orange, Thompson experienced an incident that caused a few of its employees to develop what was believed to be chloracne.

1184. The principal evidence of Thompson's knowledge of this health hazard is found in two internal memos of Dow Chemical Company dated February 1967.

1185. The first states that M. S. Buckley, Technical Director of Thompson, requested information from Dow to assist him in dealing with a “severe chloracne problem” among some of Thompson's employees.

1186. The second memo, written by V. K. Rowe, indicates that he did not give Buckley a very detailed description of what caused the problem because, in his words, “it was quickly apparent that Mr. Buckley had little understanding of the toxicological aspects of his problem. Had he asked for methods, etc., I would have agreed to send them to him.”

1187. There is no substantial credible evidence that Thompson disclosed this production accident to information about dioxin or its contamination available to any government decision maker with management authority over the deployment of phenoxy herbicides during the War in Southeast Asia.

1188. The principal manufacturers of dioxin contaminated phenoxy herbicides and the cacodylates engaged in a conspiracy' to mislead the Government about the contamination of the phenoxy herbicides with dioxin and the dangers of dioxin.

1189. Even as late as 1982, the defendant war contractors in the Agent Orange litigation (MDL 381) attempted to conceal and withhold documents revealing their unique knowledge about the contamination of the phenoxy herbicides they manufactured with TCDD and the inherent toxicity of TCDD.

1190. As far back as the 1930's, Dow workers contracted skin disorders from exposure to what now appears to have been dioxin, and company scientists had some evidence that exposure could lead to serious illness and death.

1191. Over the years, a pattern of chloracne outbreaks related to dioxin appeared among workers in widely scattered chemical plants.

1192. Dow and other companies shared with one another anxiety over these illnesses and other findings about dioxin, but the companies withheld their concern and their scientific information from the Government.

1193. When Dow in 1970 finally warned some individuals in the Defense Department about "possible" dangers associated with the use of Agent Orange, military officials declared that they were then hearing about the problem for the first time.

1194. Dow was not the first company to make Agent Orange for the defoliation and crop destruction campaign that ran from 1962

to 1970. But, according to a 1975 Air Force history of herbicide use in Southeast Asia, Dow became the largest of nine contractors, providing, at a price of $7 a gallon, nearly one-third of the total 12.8 million gallons supplied to the Government.

Knowledge of adverse health effects, "May be fatal"

1195. In a Dow company report called "Chloracne Dow Experience," a section headed "Chloracne in Humans" states: "Usually not disabling but may be fatal." A chronology notes that in 1955 "severe cases including some suspected fatalities" occurred in Germany.

1196. Dow's medical director, Dr. B.B. Holder, cited the dangers of dioxin in his review of a major outbreak of chloracne at Dow's Midland, Michigan plant in 1964, afflicting 50 workers, which coincided with stepping up of production of trichlorophenol, a precursor of Agent Orange, to meet growing war needs. "The clinical picture of the disease is one that primarily affects the skin," The stimulation of certain skin glands produces multiple blackheads along with bacterial cellulitis, he said.

1197. "In extreme exposures to certain chlorinated compounds, a general organ toxicity can result. This is primarily demonstrated in the liver, hematopoietic and nervous systems." … "Many patients demonstrated continuation of the skin lesions several years after complete removal from the exposure."

1198. After the skin manifestations, Dr. Holder went on, may come the onset of "psychopathological and other systemic findings. Their occurrence is seen only in extreme exposures and has a direct dose relationship. Fatalities have been reported in the literature. There is no specific treatment for this disease."

1199. In the 1930's, Dow introduced a line of chlorophenol wood preservatives. Dow had hardly begun marketing the chemical

when reports of severe chloracne associated with the chlorophenols began reaching the medical literature. One case in 1936 reportedly involved 300 to 400 Mississippi lumber workers stricken with ulceration, severe pimpling or thickening of the skin, urinary disturbances or leg cramps.

1200. The following years brought other outbreaks. In 1949, 228 workers at a Monsanto 2,4,5-T plant in Nitro, West Virginia, developed chloracne as the result of an industrial accident. Other symptoms, included "severe pains in skeletal muscles, shortness of breath, intolerance to cold, palpable and tender liver, loss of sensation in the extremities, demyelination of peripheral nerves, fatigue, nervousness, irritability, insomnia, loss of libido and vertigo."

1201. Chloracne problems continued to plague the Nitro plant for the next 20 years. In 1968, the medical director of Monsanto, Dr. R. Emmet Kelly, wrote in a brief memo on the situation in Nitro: "I don't want to be cynical, but are there any employees in the Department who don't haveThe Diamond Shamrock knowledge

1202. In 1963, Dr. Richard W. McBurney visited a plant of the Diamond Alkali Company, later Diamond Shamrock, in the *Ironbound* section of Newark, N.J. and wrote that he saw similarities with a disease known as *Porphyria Cutanea Tarda*: "This disease is a disease of the blood forming elements of the body in which the hemoglobin of the red blood cells is broken down and essentially the spleen, liver and kidneys are affected to a greater or lesser extent, depending upon the ingestion of such a chlorinated benzene."

1203. Dr. Roger Brodkin, director of the division of dermatology at the University of Medicine and Dentistry of New Jersey, said that he and his late associate, Dr. Jacob Bleiberg, had treated about 50 of the former workers at the Diamond Alkali plant

since 1962 and that he was still treating about 10 of them. The ailments included chloracne, which is a severe form of acne, blackening of the skin, unwanted hair growth, and porphyria.

1204. A former public health official, Dr. Donald J. Birmingham, then a dermatologist with the United States Public Health Service, visited the plant with Dr. Brodkin in 1964. The visit came after Dr. Brodkin and Dr. Bleiberg brought the workers' problems to Dr. Birmingham's attention as well as to the attention of other health officials.

1205. Dr. Birmingham said that New Jersey health officials, who also visited the plant, knew of the complaints, but he did not have any regulatory authority to enforce any action against the company.

1206. Without naming the Diamond plant, Dr. Bleiberg wrote up his findings anecdotally in the *Archives of Dermatology* in June 1964.

1207. Charles Yokum, a spokesman for Diamond Shamrock, said the company had known of the skin disorders since the 1950s and had arranged for Dr. Bleiberg to look for the cause.

1208. There had been two explosions at the Diamond Alkali plant in Newark, one on Feb. 20, 1960, in which one worker was killed, and the other sometime in 1955.

Efforts to conceal evidence of manufacturers' knowledge

1209. Diamond Shamrock Corporation confirmed that employees were ordered on May 16, 1983 in a memo signed by C.E. Stewart, president of the Diamond Shamrock chemical unit which said in part: "All files and records not needed in the performance of one's job or required by law to be kept are to be discarded by Friday, May 20, 1983. Files and records more than two years old are presumably not needed. This applies to every Diamond Shamrock location (plants, research facilities,

offices.)" The memo concluded: "Compliance is mandatory. Overtime and temporary help are being authorized to complete this assignment."

1210. On June 2, a little more than two weeks after the records destruction memo, the New Jersey Department of Environmental Protection announced the first results of dioxin tests taken on April 15 on the grounds of the former Diamond Alkali plant in Newark.

1211. The 1964 outbreak of chloracne at Dow's Midland plant stirred considerable concern, and word that the Government was asking questions prompted a series of meetings to develop a strategy for avoiding Government regulation.

1212. On March 19, 1965, Dow invited representatives of Monsanto, Diamond Alkali, the Hooker Chemical Company and the Hercules Powder Company to Midland to discuss "problems of health" associated with findings of "highly toxic impurities" in 2,4,5-trichlorophenol and related materials.

1213. At the March 24 meeting, Dow discussed its recent chloracne outbreak and its 25 years' experience testing its chemicals on rabbits' ears. The meeting, according to a Diamond Alkali representative, E.L. Chandler, "was obviously designed to help us solve this problem before outsiders confuse the issue and cause us no end of grief."

The 1965 Dow meeting memo

1214. The following pages are a true and accurate copy of that memorandum prepared by Ed Chandler, the Diamond Alkali representative at that meeting.

Exhibit 41

INTER-OFFICE CORRESPONDENCE

DATE 3/25/65

TO: JOHN COOK, JR.

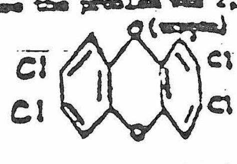
FROM: E. L. CHANDLER

SUBJECT: CHLORACNE - DOW MEETING

cc: F. R. Kennedy - Mgr., Newark plant
J. O. King
K. F. Wilkerson

On March 24, Mike Kennedy and I met with two people from Hooker Chemical Co., two from Hercules, and with the Dow group to discuss the toxicological impurities associated with 2,4,5 trichlorophenol and related materials.

Dr. Rowe of Dow Chemical opened the meeting by stating that they had operated for 25 years without trouble; but, in the last year, they had 60 to 70 cases of chloracne. Ten of these were moderate, and five to ten were extremely severe. Their approach was a qualitative one at first. They wanted to find the causative material, learn how to identify it, and try to avoid continual trouble with the unknown. They tested various materials from tar fractions and from, as they put it, "gunk", etc. They found that there are a number of suspect materials, probably 26 or 27; but the major "bad actor" that they identified and which seemed to consistently cause the problem was 2,3,7,8-tetrachlorodibenzo p-dioxin (symmetrical).



→ 2,3,7,8-TETRACHLORODIBENZ p-Dioxin (Symmetrical)

This incidentally was previously listed as a suspect material by Cy Perkins of our company. A similar material is the unsymmetrical 1,3,7,8-TCDD, also sometimes listed as 2,3,7,9-TCDD.

The Dow people used the white glove approach and found this contaminant on tool handles, benches, instruments, and other facilities. In test animals, they could consistently cause the symptoms to appear. Dr. Holder of Dow, one of their medical doctors, had excellent color slides of the various patients. The difficulty starts with multiple blackheads resulting in closed cystic structures which make the patient look like he needs to wash his face. The disease develops slowly, not appearing until six weeks to two months after mild exposure but appearing in five to seven days with very heavy exposure.

One bench chemist has been under treatment for two years and his face is starting to show signs of clearing. Dr. Holder says that he believes this man's problems will be solved in another six months. Dr. Sadek, who does their microscopic work in connection with their animal laboratory, showed photo micrographs of the cysts as they formed in the ears of rabbits. The cysts correlated with those found on the faces of the men.

DS 1000344

DS 0000343

D. JOHNSON

FEB 6 '83

EXHIBIT 4

Ex Chandler-7

Chlorocene - Dow Meeting (ELC) -2- March 25, 1965

Basically, there is a carotinoid deposit in the hair follicles and oil ducts in the face. These eventually go from the blackhead stage to form a closed, heavy acne deposit. The chemical cannot be found in the facial tissues or in the acne, but the problem still persists after exposure. The best description of the acute stage is that the facial tissues resemble the exaggerated surface texture of an orange, rather glazed and marbly with the enclosed hard acne deposits.

A secondary symptom, which does not correlate directly with the amount of facial dermatitis, is a fatigue reaction where the analogue is completely listless, tired out, and nearly incoordinated. A complete biopsy of liver, kidney, etc., showed no degeneration of major organs. A complete clinical examination of the patients showed no measurable effect on heart, blood pressure, respiration rate, blood sugar, etc. The fatigued patients seemed to be helped by heavy doses of vitamins, perhaps related in some way to the carotene metabolism of the body (involving vitamin A, etc.)

An oral dose of 17 milligrams immediately killed the test animals. The Dow people did not know this dosage to obtain an LD50 but decided that, when they ran future tests, this compound, it should not be in the present. They found that, after exposure to the material, washing within 15 minutes did not help a great deal, but did slow down the speed at which the symptoms appeared. Washing after one hour was of absolutely no help whatsoever in removing total dermatitis or speed of appearance of the reaction. Mechanical scrubbing with detergent does not remove this material. Extremely hard scrubbing can accomplish the task or the use of solvents, such as 1,1,1-trichloroethane.

Dow has developed a new analytical method in which they have confidence in their sensitivity to 1 ppm. They can only state in levels below this that some may be present below the 1 ppm. They stated that they have not used micro-colorimetric methods, and the electron capture tests that they ran made only a very slight improvement in sensitivity with this compound. Their analytical chemist stated that the electron cell saturates because of the presence of other materials in high concentration compared to the dioxin.

This material has some storage properties. It has a fairly high vapor pressure but nevertheless is quite persistent as a contaminant. It can be separated from benzene by boiling if it is not carried down to dryness. The Dow people are extremely careful in all of their work with this compound. They use PVC throwaway gloves, and all samples are burned in a special furnace which operates at 800°F. These samples are sealed before going to the burner. They use discovery methods on rabbits for qualitative screening only.

The Dow people state that they intend to set a limit of zero with sensitivity of plus or minus 1 ppm on this material. They have analyzed materials from other companies, including our company, and have found amounts as high as 10 ppm in 2,4,5-T acid and 20 to 30 ppm in phenates.

They have made a single application to the ears of test rabbits and found that 20 ppm will not give folliculitis. Forty ppm does give a slight effect, and 100 ppm is severe. They have made repeat applications of from 10 to 100 ppb, and 25 of these treatments do not cause a response; however, 1000 ppb (1 ppm) gives a slight response with nine applications and a severe reaction with 11 applications.

They conclude, therefore, that 1 ppm with repeat exposure can create a real problem.

Dow's people outlined a method for extracting and running samples on 2,4,5-T; 2,4,5-T7; and phenols. It involves a chloroform extraction, followed by

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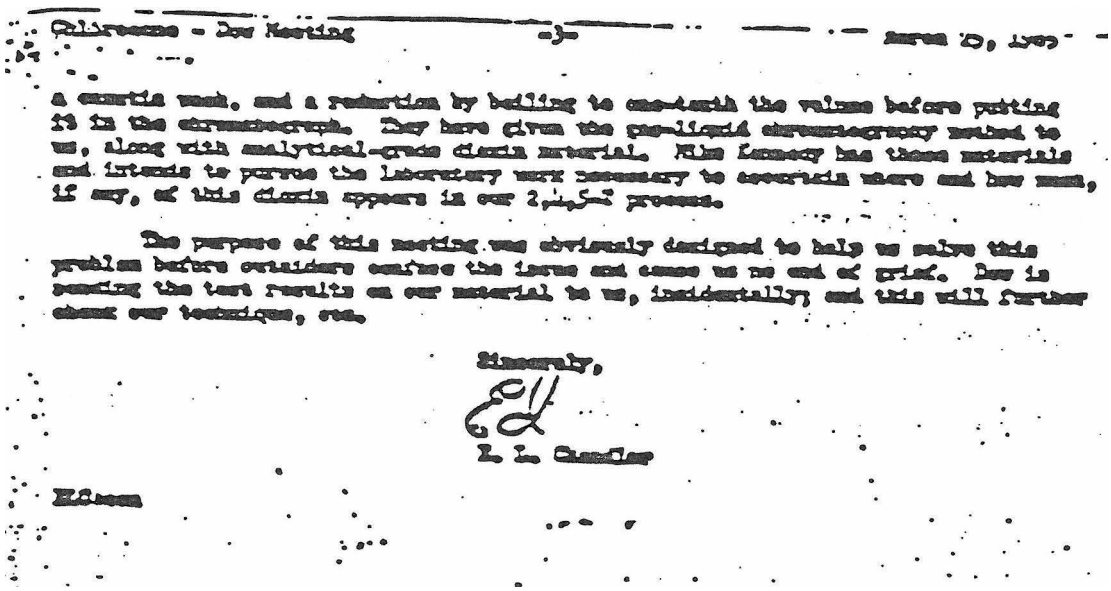


Chloroacne - Dow Meeting -2- June 25, 1965

a caustic wash, and a reduction by boiling to one-tenth the volume before putting it in the chromatograph. They have given the gas-liquid chromatography method to us, along with analytical-grade dioxin material. Mike Kennedy has these materials and intends to pursue the laboratory work necessary to ascertain where and how much, if any, of this dioxin appears in our 2,4,5-T process.

The purpose of this meeting was obviously designed to help us solve this problem before outsiders confuse the issue and cause us no end of grief. Dow is running the test results on our material to us, incidentally, and this will further check our technique, etc.

Sincerely,

E. L. Chandler

ELChandler

1215. Shortly afterward, on June 24, 1965, Dr. Verald K. Rowe, toxicology director for Dow, wrote to Ross Mulholland, bioproducts manager of Dow Chemical of Canada. The letter noted the dangers of dioxin and warned of consequences to Dow, stating, "As you well know, 'we had a serious situation in our operating plants because of contamination of 2,4,5-trichlorophenol with impurities, the most active of which is 2,3,7,8-tetrachlorodibenzodioxin. The material is exceptionally toxic; it has a tremendous potential for producing chloracne and systemic injury.

1216. "One of the things we want to avoid is the occurrence of any acne in consumers. I am particularly concerned here with consumers who are using the material on a daily, repeated basis such as custom operators may use it.

1217. "If this should occur, the whole 2,4,5-T industry will be hard hit and I would expect restrictive legislation, either barring the material or putting very rigid controls upon it. This is the main reason why we are so concerned that we clean up our own house from within, rather than having someone from without do it for us."

1218. The letter concluded: "I trust that you will be very judicious in your use of this information. It could be quite embarrassing if it were misinterpreted or misused."

1219. While exchanging information with each other information about the contamination of phenoxy herbicides with 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin (TCDD) and the dangers associated with exposure to TCDD, in the product, all of the manufacturers conspired to withheld key data from the Government and they succeeded.

1220. On April 22, 1963, a Dow official, C.L. Lynn, director of registration for the Bioproducts Department, wrote to Brigadier General Fred J. Delmore of the United States Army Munitions Command, Chemical-Biological-Radiological Agency, at the Army Chemical Center in Maryland stating that

> "We have been manufacturing 2,4-D and 2,4,5-T for over ten years. To the best of our knowledge, none of the workmen in these factories have shown any ill effects as a result of working with these chemicals."

The Hercules contribution to the conspiracy

1221. Hercules Inc. became aware that the Government's testing of its Agent Orange mixture was less accurate than its own. But the company, withheld its method because, a Hercules researcher noted, "It is possible that their method may be giving beneficially higher analysis, particularly on 2,4-D, than our own method."

1222. The following pages are a true and accurate copy of the confidential documents involving Dr. John P. Frawley of Hercules with respect to this matter.

H0066

Exhibit 386

HERCULES POWDER COMPANY

CC: MR. H. E. WILDER
MR. J. M. EAGAN - SYN. - WILM.
MR. C. L. DUNN - SYN. - WILM.
TECH. FILE

Jacksonville, Arkansas
March 30, 1965

CONFIDENTIAL

TO: DR. JOHN P. FRAWLEY - MEDICAL - WILM.

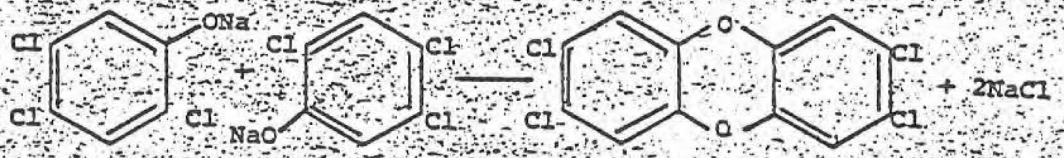
FROM: [redacted] SIDWELL

2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN
COMMENTS ON ITS PROBABLE MODE OF FORMATION

The report of your meeting with representatives of the Dow Chemical Company on the toxicity of trichlorophenol impurities is very interesting and, of course, will be held in confidence.

As outlined to you by telephone at an earlier date, it is our belief that the temperature and concentration conditions which we use in the production of sodium trichlorophenate are such that very little, if any, of the compound 2,3,7,8-tetrachlorodibenzo-p-dioxin is produced. We believe that if higher concentrations and, accordingly, higher temperatures are employed in the production of sodium trichlorophenate, a reaction similar to the following might take place:

Two molecules of 2,4,5-trichlorophenol might react together with the elimination of sodium chloride in such a manner as to produce the compound in question. This is illustrated below:



2 NaTCP → 2,3,7,8-tetrachlorodibenzo-p-dioxin

It is our opinion that such a reaction would be favored by high concentrations of sodium trichlorophenate and high temperature.

H01332

12653-12-59 400M

HERCULES POWDER COMPANY

It may have been a fortunate circumstance that our production procedure was limited in pressure by the design characteristics of the original vessel purchased by Reasor-Hill Corporation, which automatically forced us to operate at lower temperatures and lower concentrations. In any case, the reaction of caustic with symetrical tetrachlorobenzene in methanol is definitely exothermic and, since methanol and dimethyl ether produced as a side product have high vapor pressures, we are limited to a working pressure of approximately 325 pounds psig. Our peak temperature during the reaction occasionally reaches 175°C, and our maximum pressure rarely exceeds 320 pounds psig.

Experiments carried out in a small (1 liter) Parr pressure bomb a number of years ago indicated to us that if the concentration of alcohol is too low, or if the concentration of caustic is too high, excessive temperatures and pressures are reached, even sufficient to cause pyrolytic charring of the charge. Under such conditions, it is possible to use up all the caustic and actually generate hydrogen chloride, developing pressures of above 900 psig.

AES:bg

A. E. Lidwell

Exhibit 39

June 10, 1965

Mr. V. K. Rowe
Biochemical Research Laboratory
1701 Building
The Dow Chemical Company
Midland, Michigan 48640

Dear V. K.:

Thank you for your letter of May 13 regarding analysis of 2, 3, 7, 8-tetrachloro-p-dioxin in 2,4,5-T acid. I have not replied before now because I have not had much to report other than continued frustration of our analytical people.

As you probably know, two of our analysts visited Mr. Gill in Midland and reported a pleasant and satisfactory visit. In brief, the major difficulty was associated with a difference in the standard adjustment of the flame impinger in relation to the emission coil of two different models of the gas chromatographs. It appears this is a critical point for this determination which no one ever anticipated, including GC manufacturers. This problem could have gone on for years. I think we are finally underway.

Thanks again for your help.

Sincerely,

John P. Frawley, Ph.D.
Chief Toxicologist

JPF/esw

cc: Mr. E. P. Wheeler

HO 1364
EXHIBIT

Defendants independently chose their manufacturing processes

1223. The Defendants, jointly and severally, individually and collectively, exercised discretion in choosing the process and process parameters they would use in manufacturing phenoxy herbicides for use as chemical defoliants during the war in Southeast Asia. The choice of manufacturing process and the operating conditions under which the reactions would be conducted irrevocably determined the levels of TCDD which would contaminate the final product delivered to the United States military for testing at Eglin Air Force Base prior to use as a chemical defoliant during the war in Southeast Asia.

1224. The Defendants, jointly and severally, individually and collectively, knew that the greater the extent of TCDD contamination the greater the danger of physiological injury, illness, disease, and death to those exposed.

1225. There is no substantial credible evidence that any United States government official involved in the chemical defoliation program during the war in Southeast Asia had knowledge of equivalent to, or even any knowledge at all, about the contamination of the phenoxy herbicides manufactured, marketed, promoted, and sold by the Defendants with TCDD.

1226. There is no substantial credible evidence that any United States government official involved in the chemical defoliation program during the war in Southeast Asia imposed any requirements upon the Defendants with respect to their manufacturing processes.

1227. In 1964, however, according to B.B. Holder, Medical Director of Dow, "It was well understood by all involved [referring to Dow management] that we had to be extremely careful in process changes to prevent the formation of excessive chlorinated dioxins and similar toxic compounds."

1228. No United States government official involved in the chemical defoliation program during the war in Southeast Asia knew that the phenoxy herbicides being tested at Eglin Air Force Base were contaminated with TCDD and how toxic TCDD was.

1229. There is no substantial credible evidence that any United States government official involved in the chemical defoliation program during the war in Southeast Asia was aware that different temperatures, pressures, alcohols, and phenolate water content resulted in different levels of TCDD contamination, otherwise the lengthy history of government procurement practices since World War I clearly indicate that they would have specified, required, and insisted upon the safest method.

1230. An internal memorandum produced by Defendant Dow during MDL–381, stated: "Chlorophenol and derivatives producers are Dow, Monsanto, Hercules, Diamond and Hooker. Dow and Hooker isolate and distill the trichlorophenol prior to sales and use, and analyses of samples of their sales products show no exciter present. It is suspected that Hercules also distills the trichlorophenol as samples of their 2,4,5-T acid shows no exciter. Diamond and Monsanto do not isolate the trichlorophenol to purify it but make all derivatives from the sodium salt. Analysis of Monsanto's 2,4,5-T acid shows 3–8 ppm exciter, and analyses of Diamond's sodiumtrichlorophenate shows 8–24 ppm exciter."

1231. The different manufacturing processes chosen by the individual Defendants resulted in different levels of TCDD contamination and Dow certainly, and each of the other manufacturers probably, were aware of that fact. There is no substantial credible evidence that any United States government official involved in the chemical defoliation program during the war in Southeast Asia was.

GOVERNMENT RELIED UPON THE MANUFACTURERS

1232. That at all the times relevant to the claims of the Plaintiffs, all of the government decision-makers involved in the deployment of chemical defoliants during the war in Southeast Asia relied upon representations by the Defendant manufacturers as to health and safety, toxicity, effectiveness, mode of application, methods of application, and amounts of application in making determinations concerning the procurement and use of such chemical defoliants.

1233. That at all the times relevant to the claims of the Plaintiffs, the Defendant manufacturers were fully aware that the government decision-makers involved in the deployment of chemical defoliants during the war in Southeast Asia intended to rely upon and did, in fact, rely upon representations by the Defendant manufacturers as to health and safety, toxicity, effectiveness, mode of application, methods of application, and amounts of application in making determinations concerning the procurement and use of such chemical defoliants.

1234. That the Defendant manufacturers had a non-delegable duty to inform and warn the government decision-makers involved in the deployment of chemical defoliants during the war in Southeast Asia of all the Defendant manufacturers knew as manufacturers, about the health and safety, toxicity, effectiveness, mode of application, methods of application, and amounts of application in making determinations concerning the procurement and use of such chemical defoliants.

1235. That the Defendant manufacturers failed to inform and warn the government decision-makers involved in the deployment of chemical defoliants during the war in Southeast Asia of all the Defendant manufacturers knew as manufacturers, about the health and safety, toxicity, effectiveness, mode of application, methods of application, and amounts of application in making

determinations concerning the procurement and use of such chemical defoliants.

1236. The Defendant herbicide manufacturers had actual knowledge of a danger that might have negatively influenced the military conclusion that "operational use" of Agent Orange posed "no health hazard to men or domestic animals," and its presumably related decision to continue to purchase Agent Orange as it was then being produced by the defendants, had it been shared with the government decision maker involved in the chemical defoliation program.

1237. There is no substantial credible evidence that the Defendant herbicide manufacturers delivered to the government precisely what the government requested, phenoxy herbicides which posed no health to men or domestic animals from the amounts or manner these materials were used as chemical defoliants in Vietnam.

1238. There is no substantial credible evidence that the decision by the government to purchase phenoxy herbicides from the Defendant herbicide manufacturers was a fully informed decision.

THE CAUSES OF ACTION

1239. In addition to general, special, and exemplary damages, Plaintiffs seek declaratory judgment and equitable relief and bring this action on a number of causes rooted in equity and sounding in tort.

Failure to warn

1240. That during the time that the Plaintiffs were exposed to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A, each of them and all their families relied upon statements from

representatives of the Defendants about the safety of the materials, particularly the phenoxy herbicides, to which they were exposed at Eglin Air Force Base.

1241. That during the decades since the Plaintiffs worked at Eglin Air Force Base, none of the Plaintiffs, nor any of their family members have ever been advised by the Defendants that they were at risk of physiological injury, systematic disease, genetic damage and death as a result of their unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1242. That during the decades since the Plaintiffs worked at Eglin Air Force Base, none of the Plaintiffs, nor any of their family members have ever been warned by the Defendants that they were at risk of physiological injury, systematic disease, genetic damage and death as a result of their unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1243. That the failure to warn the Plaintiffs that they were at risk of physiological injury, systematic disease, genetic damage and death as a result of their unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A contributed to and was a cause of the physiological injury, systematic disease, genetic damage and death of the Plaintiffs.

Strict liability; defective products

1244. That the dioxin contaminated phenoxy herbicides and the cacodylates manufactured, marketed, promoted, and sold by the Defendants, jointly and severally, individually and collectively, were defective products because the risk of harm to the Plaintiffs from exposure to dioxin contaminated phenoxy

herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A was foreseeable.

1245. That the dioxin contaminated phenoxy herbicides and the cacodylates manufactured, marketed, promoted, and sold by the Defendants, jointly and severally, individually and collectively, were defective products because the risks of harm from the product could have been reduced or avoided by providing reasonable instructions or warnings, and the failure to provide those instructions or warnings made the dioxin contaminated phenoxy herbicides and the cacodylates unreasonably dangerous and the cause of physiological injury, systematic disease, genetic damage and death of the Plaintiffs.

Fraud and deceit

1246. That the Defendants, jointly and severally, individually and collectively, made false statements of fact about the safety of the herbicide testing program at Eglin Air Force Base.

1247. That the Defendants, jointly and severally, individually and collectively, made false statements of fact about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base.

1248. That the Defendants, jointly and severally, individually and collectively, knew at time they made the false statements about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base that such statements were false.

1249. That the Defendants, jointly and severally, individually and collectively, made the false statements about the safety of the herbicide testing program at Eglin Air Force Base and about

the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base knowing that the Plaintiffs would act in reliance upon those false statements.

1250. That the Plaintiffs did act to their detriment relying upon the false statements made by the Defendants, jointly and severally, individually and collectively, about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base.

1251. That as a result of relying upon the false statements made by the Defendants, jointly and severally, individually and collectively, about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base, the Plaintiffs suffered physiological injury, systematic disease, genetic damage and death.

Intentional misrepresentation

1252. That the Defendants, jointly and severally, individually and collectively, knowingly and intentionally made false statements about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base.

1253. That the Defendants, jointly and severally, individually and collectively, actually knew that their representations about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing

program at Eglin Air Force Base were false when they were made.

1254. That the Defendants, jointly and severally, individually and collectively, intentionally made the false statements about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base intending that the Plaintiffs would act in reliance upon those false statements**.**

1255. That the Plaintiffs relied to their detriment upon the false statements intentionally made by the Defendants, jointly and severally, individually and collectively, about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base.

1256. That the false statements intentionally made by the Defendants, jointly and severally, individually and collectively, about the safety of the herbicide testing program at Eglin Air Force Base and about the safety of the dioxin contaminated phenoxy herbicides and the cacodylates used in the herbicide testing program at Eglin Air Force Base upon which the Plaintiffs relied to their detriment caused the Plaintiffs to suffer physiological injury, systematic disease, genetic damage and death.

Negligence and negligent undertaking

1257. That the Defendants, jointly and severally, individually and collectively, negligently and carelessly failed to establish safeguards with respect to the handling and application of dioxin contaminated protected the Plaintiffs from exposure to

dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1258. That the Defendants, jointly and severally, individually and collectively, negligently and carelessly failed to implement industry-standard "best practices" with respect to the handling of the inherently dangerous dioxin contaminated phenoxy herbicides and the cacodylates so as to protect the Plaintiffs from exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1259. That the Defendants, jointly and severally, individually and collectively, negligently and carelessly failed to comply with and implement generally accepted principles of industrial hygiene with respect to the handling of inherently dangerous materials such as dioxin contaminated phenoxy herbicides and the cacodylates so as to protect the Plaintiffs from exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1260. That during the time that the Plaintiffs were exposed to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A, and during the decades since, each of them and all their families relied upon statements from representatives of the Defendants about the safety of the materials, particularly the phenoxy herbicides, to which they were exposed at Eglin Air Force Base.

1261. That the Plaintiffs and their families relied to their detriment upon the public assurances of the Defendants, jointly and severally, individually and collectively, about the safety of the materials, particularly the phenoxy herbicides, to which they were exposed at Eglin Air Force Base.

1262. That the Defendants, jointly and severally, individually and collectively, undertook to provide materiel, goods, and services to the government of the United States for good and valuable consideration assumed a duty to not put the Plaintiffs at an undue risk of harm and to take any and all actions necessary and appropriate for the protection of the Plaintiffs.

1263. That the failure of the Defendants, jointly and severally, individually and collectively, to exercise reasonable care in the manufacture and use of dioxin contaminated phenoxy herbicides and the cacodylates to protect the Plaintiffs increased the risk of physiological injury, systematic disease, genetic damage and death for the Plaintiffs.

1264. That the Plaintiffs relied upon then undertaking of the Defendants, jointly and severally, individually and collectively, and have suffered physiological injury, systematic disease, genetic damage and death as a result of their reliance upon the undertaking of the Defendants.

1265. That the manufacture and use of dioxin contaminated phenoxy herbicides and the cacodylates by the Defendants, jointly and severally, individually and collectively, created a generalized and foreseeable risk of physiological injury, systematic disease, genetic damage and death to the Plaintiffs.

1266. That by reason of their undertaking to provide materiel, goods, and services to the government of the United States for good and valuable consideration, the Defendants, jointly and severally, individually and collectively, assumed a duty to lessen and mitigate the risk of physiological injury, systematic disease, genetic damage and death to the Plaintiffs.

1267. That by reason of their undertaking to provide materiel, goods, and services to the government of the United States for good and valuable consideration, the Defendants, jointly and severally, individually and collectively, assumed a duty to not

put the Plaintiffs at an undue risk of harm and to take sufficient and appropriate precautions to protect the Plaintiffs from physiological injury, systematic disease, genetic damage and death as a result of unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

Reckless disregard for worker safety and health

1268. According to the Plaintiffs, while the planes were flying directly overhead, none of the workers wore any protective gear.

1269. According to Plaintiff Terrell Gatlin, "Our only directions were to get in the truck and roll the windows up. But most of the time you couldn't roll them up because it didn't have air conditioning, and it would be 100 degrees outside."

1270. According to Plaintiff Terrell Gatlin, "We were told as soon as the test was over to go and wash the vehicle. There was never any mention of washing ourselves."

1271. According to Plaintiff Terrell Gatlin, the herbicide that rained down from the sky turned the trucks a bright pink. "Even after we washed them, it would take weeks before the sun would bleach them white again,"

1272. The planes often came in as low as 50 feet above the ground, resulting in considerably greater exposure for the range technicians than for the American combat veterans.

1273. According to the Plaintiffs, the spray missions lasted about an hour. Usually there would be 15 to 25 people on the site, performing a variety of tasks, including filming the missions.

1274. According to Plaintiff Tommy Brown, a photographer, on one occasion, he was told to shut down his camera immediately. "They called me on the radio and said, 'Don't ask no questions,' By the time we closed the camera, this stuff hit us like mud

before we could get down from the top of the tower where we were filming. We got the full impact."

1275. That the Defendants, jointly and severally, individually and collectively, failed to use reasonable care and take reasonable precautions to safeguard the health, safety, welfare and lives of the Plaintiffs and protect them from physiological injury, systematic disease, genetic damage and death as a result of their unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1276. The gross, flagrant, and egregious character of the conduct of the Defendants, jointly and severally, individually and collectively, with respect to their manufacture and use of dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base was evidence of such reckless disregard of human life and the personal safety and health of those exposed without proper protection to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A and want of care for those so exposed as to establish a presumption of a conscious indifference to the consequences of such exposure upon the Plaintiffs, and such wantonness, recklessness, and grossly careless disregard of the health, safety, and welfare of the Plaintiffs, and such reckless indifference to their rights as to be an intentional violation of those rights.

Civil conspiracy

1277. That the Defendants, jointly and severally, individually and collectively, conspired to allow the physiological injury, systematic disease, genetic damage and death of the Plaintiffs by withholding and concealing information about the hazards and risks associated with unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1278. That the Defendants, jointly and severally, individually and collectively, conspired to allow the physiological injury, systematic disease, genetic damage and death of the Plaintiffs by misrepresenting the hazards and risks associated with unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates at Eglin Air Force Base designated ranges, including site C–52A.

1279. That by reason of the conspiracy of the Defendants, jointly and severally, individually and collectively, the Plaintiffs suffered physiological injury, systematic disease, genetic damage and death.

THE CONSPIRACY OF SILENCE

1280. By 1965, Dow had entered into a conspiracy of silence which made it impossible for any United States government official involved in the chemical defoliation program during the war in Southeast Asia to reach parity of knowledge about the contamination of phenoxy herbicides with TCDD or about the extraordinary toxicity of TCDD.

1281. In 1963, made an affirmative misrepresentation to the United States military, "[W]e have been manufacturing 2,4,-D and 2,4,5-T for over 10 years. To the best of our knowledge, none of the workman in these factories have shown any ill effects as a result of working with these chemicals." At the time Dow made that misrepresentation, its own industrial experience and its knowledge of the Boehringer experience, established it was untrue.

1282. In December of 1964, the conspiracy of silence was nearly exposed by Dr. David Groth of the United States Public Health Service who requested samples of 2,4,5-T to analyze for a suspected contaminant.

1283. When Dow learned of Dr. Groth's intentions, a number of meetings were arranged among the Defendant manufacturers, conversations held, and memos to file written.

1284. Unfortunately, Dr. Groth was unable to test the samples due to a lack of funds to purchase gas chromatography equipment.

1285. Dow sought to advise the other manufacturers of 2,4,5-T that their products were contaminated. Dr. R.C. Dosser of Dow pointed out to Mr. E .T. Upton of T.H. Agriculture and Nutrition Company in a telephone conversation on or before January 12, 1965: "[t]hat if a compound found in 2,4,5-T acid or its esters, proved to be damaging that this might lead to a flurry of successful claims by users of the herbicide who allegedly had been injured by it.

1286. On February 18, 1965, Dow held an internal "Exciter" Problem Meeting, the minutes of which state:

1287. "Otis introduced the meeting by indicating that recent information indicates "Exciter" components may be present in Dow 2,4,5-trichlorophenol. Bioproducts is concerned about 2,4,5-T acid, esters, formulations, silvex, ronnel, and Erbon.

1288. This meeting is to review status of our knowledge of this subject, potential hazards, possible effect on Dow image, legal implications, and need for possible quarantine. These basic decisions are to be made without consideration of economic impact."

1289. As a result of that meeting Dow determined to meet with the other producers of 2,4,5-T.

1290. Prior to March 1, 1965, V.K. Rowe of Dow contacted the Medical Director, Emett Kelly, M.D. and Assistant Medical Director at Monsanto and the Chief Toxicologist at Hercules, John P. Frawley, concerning dioxin contamination.

1291. On March 19, 1965, V. K. Rowe of Dow wrote identical letters to Emett Kelley, M.D., Dr. J. Wilkenfeld and Mr. Raymond Verhoeze of Hooker Chemical Corporation, Mr. Francis Kennedy, Plant Manager and Dr. Ed Chandler, Technical Services, Diamond Alkali Company and Dr. John P. Frawley of Hercules

1292. "inviting each of you to come to Midland to discuss the toxicological problems caused by the presence of certain highly toxic impurities in certain samples of 2,4,5-trichlorophenol and related materials. *** Our discussions will deal only with toxicological and analytical aspects of the problem. We will not discuss manufacturing know how, sales, or anything else not dealing with the problems of health."

1293. The meeting occurred on March 24, 1965 and "V.K. Rowe recapped the Dow situation in terms of the problem and the initial studies by Toxicology and Environmental Research Laboratory regarding the in-plant situation. He expanded this in general terms to the study of end products, ours and other peoples." A number of the attendees at the March 25, 1965 meeting drafted memos to file.

1294. A memo to file from E .L. Chandler of Diamond Alkali Company notes among other things:

> "The Dow people state that they intend to set a limit of zero with sensitivity of plus or minus 1 ppm on this material. They have analyzed materials from other companies, including our company, and have found amounts as high as 10 ppm in 2,4,5-T acid (end product) and 20 to 30 ppm in phenates.
>
> "They have made a single application to the ears of test rabbits and have found that 20 ppm will not give folliculitis. Forty ppm does give a slight effect, and 100 ppm is severe. They have made repeated applications of from 10 to 100 ppb, and 25 of these treatments do not cause a response; however, 1000 ppb (1 ppm) gives a slight response with nine applications and a severe reaction with 11 applications.

"They conclude, therefore, that 1 ppm with repeat exposure can create a real problem."

1295. Dow believed in 1965 that 1 ppm with repeated exposures presented a significant health hazard, yet this information was never presented to any United States government official involved in the chemical defoliation program during the war in Southeast Asia.

1296. C.L. Dunn of Hercules Powder Company also prepared a memo to file. In that memo Mr. Dunn notes,

"Rowe expressed concern that carry-through of toxic materials into final formulated herbicide products may occur unless precautions are taken." ***

"Evidence that the chloracnegens may be systemic." ***

"Hooker and Diamond people were quite aware of chloracne. One of the Hooker people said that some cases are believed to relate to exposures occurring 20 years before." ***

[The detection limits for Dow's gas chromatography are:] "2,4,5-TCP — 1 ppm." [and] "2,4,5-T acid — 1 ppm." ***

"Competitor products — Dow has examined all manufacturer's 2,4,5-T products. Some have "surprisingly high" amounts of chloracnegens. This defined as 10 ppm. in 2,4,5-T acid, and up to 30 ppm. in 2,4,5-trichloro-phenate."

1297. Similarly, J. Wilkenfeld of Hooker Chemical Company recorded the information he had received at Dow on March 24, 1965 as:

"Work with rabbits with washing after exposure, sometimes as soon as 15 minutes after application, did not stop the development of the chloracne and single, oral dose toxicity tests (not (?) m 50) resulted in the death of some rabbits at 17 micrograms per kilo. Peripheral liver cell necrosis occurred in some rabbits where the washing was done. ***

"The current in plant working limit is "no response" with less than one ppm by chromatography considered safe. When they

were having difficulty, chloracne causers were in the order of 20–30 ppm."

1298. No United States government official involved in the chemical defoliation program during the war in Southeast Asia was invited to attend the meeting, nor is there any substantial credible evidence that any of the information presented by Dow at the meeting or any report of the discussions among the representatives of the other Defendants at the meeting ever reached any government official or any member of the United States military responsible for the chemical foliation program, and the information certainly never reached the Plaintiffs who were being exposed without protection during their work at Eglin Air Force Base.

1299. On June 24, 1965, V.K. Rowe of Dow's Biochemical Research Laboratory wrote a letter to Ross Mulholland, Manager, Bioproducts, Dow Chemical of Canada. In that letter, Mr. Rowe stated:

"As you well know, we had a serious situation in our operating plants because of contamination of 2,4,5-trichlorophenol with impurities, the most active of which is 2,3,7,8-tetrachlorodibenzodioxin. This material is exceptionally toxic; it has tremendous potential for producing chloracne and systemic injury. If it is present in the trichlorophenol, it will be carried through into the T acid and into the esters and hence into formulations which are to be sold to the public. One of the things which we want to avoid is the occurrence of any acne in consumers. I am particularly concerned here with persons who are using the material on a daily, repeated basis such as custom operators may use it. If this should occur, the whole 2,4,5-T industry will be hard hit and I would expect restrictive legislation, either barring the material or putting very ridged [*sic*] controls upon it. This is the main reason why we are so concerned that we clean up our own house from within, rather than having someone from without do it for us. In this way, we can approach the problem in an orderly manner. If the producers and handlers of this

material will cooperate, there is no reason why we cannot get this problem under strict control and thereby hopefully avoid restrictive legislation; in other words, let us practice good citizenship. ***

"We are not in any way attempting to hide our problem under a heap of sand, but we certainly do not want to have any situations arise which will cause the regulatory agencies to become restrictive. Our primary objective is to avoid this.

"I trust that you will be very judicious in your use of this information. It could be quite embarrassing if it were misinterpreted [*sic*] or misused."

1300. As an example of "practicing good citizenship" while furthering the conspiracy of silence, V.K. Rowe wrote as a postscript, "under no circumstances may this letter be reproduced, shown, or sent to anyone outside of Dow."

1301. In a memo to file recording a conversation on July 9, 1965, Mr. Earl Farnham telephoned J.P. Frawley of Hercules Powder Company at the behest of Dr. Donald Baldwin, Vice-President of Dow. Mr. Farnham sought to inquire: "[H]ow serious I considered the chloracne problem in relation to the consumer use of 2,4,5-T." When Mr. Frawley suggested that the matter should be discussed with Hercules management, rather than him, Mr. Farnham stated, "that on the advice of their toxicologist, Dow has gone to great expense to alter their manufacturing conditions in order to produce 2,4,5-T acid which has less than one ppm acnegen." Mr. Farnham further stated:

"[T]hat Dow was extremely frightened that this situation might explode. They are aware that their competitors are marketing 2,4,5-T which contains "alarming amounts" of acnegen and if the government learns of this the whole industry will suffer. They are particularly fearful of a congressional investigation and excessive restrictive legislation on manufacture of pesticides which might result.

1302. Mr. Frawley of Hercules indicated that Hercules shared Mr. Farnham's fear but that Hercules was unaware of his allegation that the competitors' products were hazardous.

1303. Mr. Frawley's memo to file of July 12, 1965 is marked confidential.

1304. Dow never advised the military about the contamination of 2,4,5-T with dioxin and the associated health hazards until March 6, 1970 when representatives of Dow provided a briefing to representatives of the United States military finally admitting that TCDD was an impurity in 2,4,5-TCP and 2,4,5-T and at that briefing, for the first time, Dow representatives presented the following information on 2,4,5-T:

> "Since 1950 it has been known that 2,3,7,8-tetrachlorodibenzo-p-dioxin is an impurity associated with 2,4,5-trichlorophenol (TP). The latter material is used in the manufacture of 2,4,5T. Dioxin is a highly toxic material… In 1964, Dow closed their plant which manufactured 2,4,5T due to widespread acne among plant workers. Dioxin and other impurities w re found in the 2,4,5T. The plant was cleaned and the manufacturing process was changed to eliminate the dioxin and other impurities. The Dow 2,4,5T now contains less than 0.5 ppm of dioxin."

1305. Dr. Robert Darrow attended the meeting on March 6, 1970 at the Pentagon with Dr. Charles Minarik as representatives of the Crops Division at Fort Detrick.

1306. During examination by defendant's counsel, Dr. Darrow, one of the two experts in the United States military about herbicides clearly established that they were never aware that the phenoxy herbicides, particularly 2,4,5-T were contaminated with TCDD, dioxin, and that he was highly toxic.

Q. Can you recall a meeting which took place on or about March 11, 1970, at which various representatives of Dow met with Gen. William Sloan [sic] and various others concerning Agent Orange?

A. I was at a meeting with Dow's representatives but I don't recall the date of it. The name General Sloan doesn't register with me. I'm not sure.

Q. Do you recall what if anything you said at that meeting.
A. I just listened. I didn't say anything.

Q. Do you recall what if anything Dr. Minarik said at that meeting?
A. The presentation was given by the Dow people. That was it. They presented information.

Q. Anyone at the meeting, Dr. Darrow, did they say in words or in substance to the Dow representatives why didn't you tell us about this before?
A. I think the feeling was there but I'm not sure it was said. We were surprised when we got the information at this time.

Q. By "we" who are you referring to?
A. Dr. Minarik and myself.

Q. Do you recall what if anything was said in that regard by anyone at the meeting?
A. No specific comments, no. In other words, this represented information that we were receiving for the first time in this respect on the dioxin situation.

Q. Where was that meeting held? Do you recall that?
A. It was in the Pentagon. It was in Washington, D.C. , in the Pentagon.

PLAINTIFFS HAVE TIMELY FILED THIS ACTION

1307.

Defendants actions preclude constructive notice to the Plaintiffs

1341. At the time the physiological injury sustained by each plaintiff as a result of their unprotected exposure to dioxin contaminated phenoxy herbicides and the cacodylates during testing of chemical defoliation agents at Eglin Air Force Base became clinically manifest, because of the conscious and deliberate actions of the Defendants, there was no information available to any of the individual Plaintiffs about the association between their exposure to dioxin contaminated

phenoxy herbicides and the cacodylates at Eglin Air Force Base and their disease and in some cases death years later.

1342. To this day, the Defendants, jointly and severally, individually and collectively, continue a conscious and deliberate campaign to deny the toxicity of the dioxin contaminated phenoxy herbicides and the cacodylates they developed as chemical defoliants for use during the war in Southeast Asia.

1343. Even though the Congress of the United States has promulgated legislation and the Veterans Administration of the United States has reluctantly accepted the fair preponderance of credible scientific evidence which clearly recognizes an association between exposure to dioxin contaminated phenoxy herbicides in a wide panoply of disease states associated with compromise of the immune system and the biochemical detoxification processes associated with a number of liver enzymes, the Defendants, jointly and severally, individually and collectively, still rigorously deny the evidence and claim there are no illness or disease processes associated with their dioxin contaminated phenoxy herbicides and that the organic arsenic goals are toxic only when ingested in large doses.

1344. During the period of public concern over dioxin raised during the original Agent Orange litigation from 1979 through its settlement in the mid-1980s, the Plaintiffs in this action believed that they were healthy and that there was no cause for alarm over their exposure to the dioxin contaminated phenoxy herbicides and the cacodylates they had tested at Eglin Air Force Base.

1345. There is no substantial credible evidence indicating that at any time since the contamination of phenoxy herbicides with 2,3,7,8-tetrachlor-dibenzo-p-dioxin became a concern of The

Dow Chemical Company in 1965, at no time did any of the Defendants make any effort to warn the medical profession, much less the general public, about the dangers of exposure to TCDD.