UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

OAKLAND DIVISION

VIETNAM VETERANS OF AMERICA, a Non-Profit Corporation; SWORDS TO PLOWSHARES: VETERANS RIGHTS ORGANIZATION, a California Non-Profit Corporation; BRUCE PRICE; FRANKLIN D. ROCHELLE; LARRY MEIROW; ERIC P. MUTH; DAVID C. DUFRANE; WRAY C. FORREST; TIM MICHAEL JOSEPHS; and WILLIAM BLAZINSKI, individually, on behalf of themselves and all others similarly situated,

Plaintiffs,

vs.

CENTRAL INTELLIGENCE AGENCY; DAVID H. PETRAEUS, Director of the Central Intelligence Agency; UNITED STATES DEPARTMENT OF DEFENSE; LEON PANETTA, Secretary of Defense; UNITED STATES DEPARTMENT OF THE ARMY; JOHN MCHUGH, United States Secretary of the Army; UNITED STATES DEPARTMENT OF VETERANS AFFAIRS; and ERIC K. SHINSEKI, UNITED STATES SECRETARY OF VETERANS AFFAIRS,

Defendants.

Case No. CV 09-0037-CW

EXPERT REPORT OF JEFFREY D. LASKIN, PH.D.

I. INTRODUCTION

A. Retention

1. I have been retained by Morrison & Foerster LLP on behalf of its clients, plaintiffs in this matter, Vietnam Veterans of America, Swords to Plowshares: Veterans Rights Organization, Bruce Price, Franklin D. Rochelle, Larry Meirow, Eric P. Muth, David C. Dufrane, Wray C. Forrest, Tim Michael Josephs, and William Blazinski (collectively "Plaintiffs") to serve as a consultant and expert witness in the above captioned action.

2. I expect to testify at trial regarding the matters discussed in this expert report, and in any supplemental reports or declarations that I may prepare for this matter. I may also testify at trial regarding matters related to my opinions addressed by any expert or fact witness testifying on behalf of Plaintiffs or Defendants Central Intelligence Agency; David H. Petraeus, Director of the Central Intelligence Agency; United States Department of Defense; Leon Panetta, Secretary of Defense; United States Department of the Army; John McHugh, United States Secretary of the Army; United States Department of Veterans Affairs; and Eric K. Shinseki, United States Secretary of Veterans Affairs (collectively "Defendants"), including but not limited to any reports, testimony, exhibits, references, or demonstratives presented by Defendants.

3. I reserve the right to supplement or amend this report if additional facts and information that affect my opinions become available. It is my understanding that Plaintiffs have retained other experts and that Defendants may serve expert reports concerning one or more of the issues I address in this report. I reserve the right to testify concerning such other reports or testimony, and to respond to any such report from Defendants' expert(s) and to rebut at trial any opinions expressed in such a report or in such testimony. I also understand that depositions of additional fact witnesses may take place and that Defendants have just recently produced or will be producing additional documents that are still undergoing review. Furthermore, it is my understanding that Defendants have produced, and continue to produce, a substantial quantity of documents and other information in formats that are inaccessible or exceedingly difficult to access or evaluate properly, and that Plaintiffs' counsel is continuing to attempt to convert such information into a usable format. Should Plaintiffs' counsel's efforts be successful, and

information from these sources becomes available to me, I reserve the right to supplement this report to incorporate that information.

4. The headings in this report have been added to create sections for ease of organization. I do not intend these headings to be in any way restrictive of the information contained in the respective sections.

B. Compensation

5. I am being compensated for my work on this matter at my customary rate of $350 per hour, plus expenses. My compensation is not conditioned on my opinions, testimony at deposition or trial, or the outcome of this matter.

II. MY BACKGROUND AND QUALIFICATIONS

6. I received my Bachelor of Arts degree in Chemistry and Biology from New York University in 1973 and my Ph.D. in the field of Pharmacology from the Department of Experimental Therapeutics, Roswell Park Memorial Institute, State University of New York at Buffalo in 1977. From 1977 to 1981, I was a Post-Doctoral Fellow and Staff Associate in the Institute of Cancer Research, College of Physicians and Surgeons of Columbia University.

7. In 1981 I became an assistant professor at the College of Medicine and Dentistry of New Jersey-Rutgers Medical School[1] in the department of Environmental & Community Medicine[2]. In 1987 I became an associate professor and in 1993 a full professor. Since 1995 I have been Chief of the Division of Toxicology. During my academic career I have mentored more than thirty graduate students and post-doctoral fellows who have gone on to prestigious positions in academia and industry.

8. In 1982 I became a member of the graduate faculty for the Joint Graduate Program in Toxicology at Rutgers University. In 2003 I became Deputy Director of the Joint Graduate Program in Toxicology between UMDNJ and Rutgers.

[1] The institution's name has since changed to the University of Medicine and Dentistry of New Jersey Robert Wood Johnson Medical School ("UMDNJ-Robert Wood Johnson Medical School").

[2] The department has since been renamed Environmental & Occupational Medicine.

9. I have been a member of the Environmental and Occupational Health Sciences Institute ("EOHSI") at UMDNJ and Rutgers since 1986 and was made the director of the Division of Toxicology there in 2005.

10. I am the Center Director for the UMDNJ-Rutgers University CounterACT Research Center of Excellence. The purpose of CounterACT is to develop new and improved countermeasures against high priority chemical weapons threats, including sulfur mustard.

11. I am also a founding member of the University Center for Disaster Preparedness and Emergency Response at UMDNJ.

12. I have served on the New Jersey Department of Homeland Security Preparedness College as well as on the Advisory and Executive Committee of the New Jersey Universities Homeland Security Research Consortium.

13. I am a member of the American Association for Cancer Research and the Society of Toxicology.

14. Throughout my career my research has focused on environmental and chemical toxicology. I have lectured and developed courses, as well as published extensively, on the subject of toxicology.

15. Sulfur mustard, also called mustard gas, and closely related derivatives such as half mustard, have been a significant focus of my research. I have both published widely and been invited to give numerous presentations on the subject.

16. I have published more than 150 original research and review articles in peer reviewed journals on a variety of subjects including chemical toxicology and the effects of vesicants like sulfur mustard on various model systems. I have been invited to present my research work at numerous professional meetings both in the United States and internationally. I have also organized numerous meetings on topics related to my work, including the threat posed by terrorists armed with chemical weapons like mustard gas. A current copy of my *curriculum vitae* is attached hereto as Exhibit 1, which includes a complete list of my publications to date.

17. I have not testified as an expert witness or prepared an expert report in any matter in the last four years.

III. BASIS AND SCOPE OF MY OPINIONS

18. I have been asked to provide an overview of the physiological effects on the human body of various chemical compounds, broadly classified as irritants and vesicants, studied in various testing programs conducted by Defendants. These compounds include various irritants in addition to the vesicants sulfur mustard, nitrogen mustard and Lewisite. Moreover, I have been asked to provide my opinion about the potential long-term health problems linked to exposure to such agents. In addition, I have been asked to opine about whether service members exposed to irritants or vesicants like mustard gas and/or Lewisite in the various chemical weapons testing programs operated by Defendants during the last century can reasonably be expected to develop adverse long-term health problems as a result. I may testify about any or all of these topics.

19. In arriving at my opinions, expressed in detail in this report, I have relied on my personal experience as well as various additional resources. I have relied upon the types of information and resources that are normally relied upon by experts in my field, such as articles in peer reviewed journals, treatises and similar scholarly works, and published reports regarding the testing programs at issue. In particular, I have reviewed several studies commissioned by the Department of Veterans Affairs examining the long-term impact on the health of service members experimentally exposed to various chemical agents including irritants or vesicants during their service. Of particular note is the report titled *Veterans at Risk: The Health Effects of Mustard Gas and Lewisite*,[3] published in 1993, and compiled by the Institute of Medicine. I have also reviewed relevant portions of Volumes 2 and 3 of the National Research Council's report *Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents*[4] which focuses primarily on the testing programs conducted at Edgewood Arsenal.

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[3] *Veterans at Risk: The Health Effects of Mustard Gas and Lewisite*, C. Pechura and D. Rall eds., National Academy Press (1993) (hereinafter "*Veterans at Risk*").

[4] *Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents*, Vols. 2 and 3, National Academy Press (1984) (hereinafter "*NRC Report Vol. 2 or 3*"). I have reviewed and relied on the NRC reports for their factual content and descriptions of the various chemical weapons testing programs. My reliance on the factual information contained in those reports does not mean that I agree with the ultimate conclusions reached. I understand that another expert for Plaintiffs will be opining on the validity and design of the analysis presented in those reports, and I express no opinion on that topic.

20. I have also reviewed documents from various other sources which contain contemporaneous reports and accounts of actual tests involving irritants, mustard gas and Lewisite. These documents were helpful to my understanding of the circumstances surrounding the experiments performed in the various testing programs and example test protocols used.

21. These are some of the primary references I have reviewed and relied upon in reaching my opinions; a complete list of documents I have consulted and considered is included as Exhibit 2 to this report. Throughout my report I have cited specific documents, and portions of those documents, to illustrate technical and historical points. These citations are only illustrative, not exhaustive, and I may rely on other specific portions of these documents, as well as any of the references listed in Exhibit 2 to support any of these points. Moreover, to the extent Defendants provide an expert report responding to any of the points addressed in this report, I reserve the right to consider, comment on, or rely on any documents referenced in any such report.

22. I reserve the right to provide further exhibits to be used as a summary of, or as support for, my opinions or testimony, including any testimony by experts or other witnesses at trial.

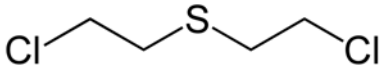
IV. VESICANTS

A. Vesicants as Weapons of War.

23. Sulfur mustard is likely the best known, and most widely studied, vesicant. It is known by a variety of names, including Yperite, S-mustard, Lost, S-Lost, yellow cross, and H or HD, though it is most commonly called mustard gas. (*Veterans at Risk* at 22; *Medical Aspects of Chemical and Biological Warfare – Textbook of Military Medicine, Sidell, F., et al., Office of the Surgeon General (1997) (hereinafter "Military Medicine")*at VET004_001134, 1136-37.) Though its toxic properties were known as early as the 1880s, sulfur mustard was first used as a weapon of war in Belgium, near Ypres, in 1917, where its characteristic odor of mustard or garlic led to its name. (*Veterans at Risk* at 21-22; *see also* Gordon, M. *et al.*, *Chapter 39: Ocular Toxicity of Sulfur Mustard*, Handbook of Toxicology of Chemical Warfare Agents, Elsevier Inc., 575-594 (2009) (hereinafter "*Handbook of Toxicology Chapter 39*") at 575-76; Gerecke, D. *et al.*, *Chapter 41: Dermal Toxicity of Sulfur Mustard*, Handbook of Toxicology of Chemical Warfare

Agents, Elsevier Inc., 611-630 (2009) (hereinafter "*Handbook of Toxicology Chapter 41*") at 611.) By the end of World War I, sulfur mustard was responsible for more than 400,000 casualties. (*Veterans at Risk* at 9.) Unfortunately, the use of sulfur mustard as a chemical weapon did not end with the end of World War I. Over the ensuing decades, sulfur mustard has been used throughout the world on numerous occasions, most recently in the 1980s by Iraq during its war with Iran. (*Veterans at Risk* at 9-10.)

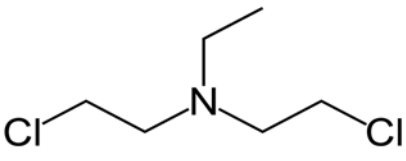
24. The chemical structure of sulfur mustard is shown below:



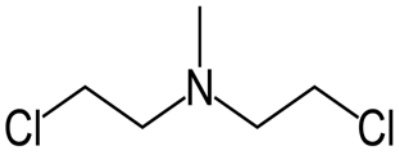
Though called a gas, sulfur mustard is typically liquid at room temperature. (*Veterans at Risk* at 22.) Exposure of the skin, eyes, respiratory tract or gastrointestinal tract to liquid, gaseous, or aerosolized sulfur mustard can cause serious injury, particularly under hot, humid conditions. (*Id.*)

25. One of sulfur mustard's most insidious characteristics is its persistence. At lower temperatures liquid sulfur mustard can remain on contaminated surfaces, such as ground or clothing, for long periods causing an ongoing exposure hazard. (*Id.*; *Military Medicine* at VET004_001138; *NRC Report Vol. 2* at 104-105.) Similarly, sulfur mustard can persist for long periods on the skin and clothing without causing immediate pain or injury; thus soldiers exposed to sulfur mustard may not be aware of the exposure until hours later, when symptoms such as skin reddening, or erythema, first appear. (*See Military Medicine* at VET004_001143, 1151). Despite decades of research, there is still no known antidote for sulfur mustard exposure. (*Id.* at VET004_001136; *Handbook of Toxicology Chapter 39* at 589-590.)

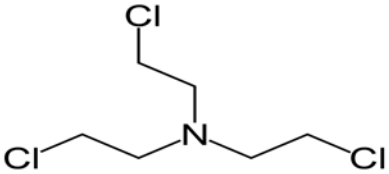
26. The nitrogen mustards, a family of chemically related compounds, were first synthesized in the 1930s. (*Military Medicine* at VET004_001136.) The chemical structures of the three nitrogen mustards, HN1, HN2 and HN3, are shown below:



HN1



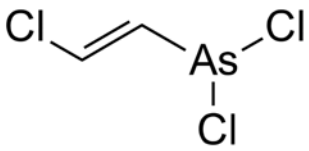
HN2



HN3

The nitrogen mustards are chemically and structurally similar to sulfur mustard, the primary difference being the replacement of the central sulfur atom with nitrogen. The nitrogen mustards have similar vesicant activity to sulfur mustard, though, based on animal testing, they appear to have more pronounced systemic and central nervous system effects. (*Id.* at VET004_001137.) The nitrogen mustards are not, however, suitable for military use, and have never been used on the battlefield. (*Id.*) One of the nitrogen mustards, HN2, was developed as an effective chemotherapy agent for the treatment of cancer, specifically certain neoplasms. (*Id.* at VET004_001136; *Veterans at Risk* at 44-45.)

27. Lewisite is a member of a different class of vesicants called arsenicals, due to the presence of arsenic in the chemical structure, as shown below:



Lewisite was developed in the United States towards the end of World War I, but never saw battlefield use. (*Military Medicine* at VET004_001136, 1156.) Lewisite is more volatile than sulfur mustard, and remains fluid at low temperatures, making it better adapted for use in colder climates. (*Id.* at VET004_001156; *Veterans at Risk* at 25.) Its rapid hydrolysis, however, makes it ill-suited for use in warm, humid conditions, where it can be difficult to maintain biologically active concentrations of the vapor. (*Military Medicine* at VET004_001156)

B. Physiological Effects of Mustard Gas and Lewisite.

28. Mustard gas and Lewisite are broadly classified as vesicants based on their ability to produce vesicles, or blisters, on exposed skin, though they can also cause severe damage to the eyes and respiratory tract as well. (*Veterans at Risk* at 21-22.) At sufficiently high doses these

effects are lethal. (*Id.*) Moreover, mustard is rapidly absorbed and distributed throughout the body, where it can damage all cell types and organs, including the bone marrow, leading to immune suppression. (*Military Medicine* at 1170.) The effects of mustard gas are also cumulative, meaning that even small doses can build up in the body and produce toxic effects.

1. General Mechanisms of Action.

29. The exact biochemical mechanism of action of sulfur and nitrogen mustards is not well understood, but both are known to be potent alkylating agents. (*Military Medicine* at VET004_001139-41; Shakarjian, M. *et al.*, "Mechanism mediating the vesicant actions of sulfur mustard after cutaneous exposure," *Tox. Sci.* 114(1) 5-19 (2010) (hereinafter "Shakarjian 2010").) As such, they can react with a range of macromolecules in the human body, including DNA, RNA, proteins and cellular membrane components. (*Military Medicine* at VET004_001139-41; Shakarjian 2010 at 9-13; *Handbook of Toxicology Chapter 41* at 612-613.) Mustard mediated injury may be initiated by alkylation of DNA,[5] interaction with other macromolecules, or some combination of these processes. (*Military Medicine* at VET004_001139-41; *see also Veterans at Risk* at 71-80; Shakarjian 2010 at 9-13; *Handbook of Toxicology Chapter 39* at 582-585; *Handbook of Toxicology Chapter 41* at 612-616.) Regardless of the precise sequence of events, injuries caused by mustard typically involve a disruption of the epidermal-dermal junction, inflammation, and blister development. (*Military Medicine* at VET004_001139-41; *Veterans at Risk* at 162-164; Shakarjian 2010 at 6, 8-14; *Handbook of Toxicology Chapter 41* at 613-616; *see also Handbook of Toxicology Chapter 39* at 582-585 (explaining these effects in the eye.).)

30. Like mustard, the exact biochemical pathway of Lewisite activity is not well understood. (*Military Medicine* at VET004_001157.) As with other arsenicals, Lewisite can

[5] Alkylation is a type of chemical modification of DNA that typically leads to DNA damage and genetic mutation. For example, chemotherapeutic agents used to fight cancer, such as nitrogen mustard, routinely kill cancer cells by damaging their DNA through alkylation.

inhibit a variety of enzymes, which likely leads to its toxic properties, particularly inhibition of carbohydrate metabolism. (*Id.*; *see also Veterans at Risk* at 71-80, 166-167.)

31. Most information on mechanisms of irritation and toxicity has been obtained using mustard gas and the related vesicant half mustard (2-chloroethyl ethyl sulfide also referred to as CEES). Aqueous physiological solutions of mustard gas are highly reactive, displaying a half-life of only 24 minutes at room temperature, rates of hydrolysis increase with increasing temperature. (Shakarjian 2010 at 9). Under these conditions, sulfur mustard forms a cyclic ethylene sulfonium ion intermediate, a process that creates a reactive electrophile and the release of a hydrogen and chloride ions. In tissues, this electrophile can react not only with nucleophilic sites in DNA, but also RNA, proteins, carbohydrates and other biomolecules. Specific target molecules include sulfhydryls, phosphates, ring nitrogens and carboxyl groups. Because sulfur mustard is bifunctional, it can form a second electrophile, again via a sulfonium ion intermediate. This can result not only in monofunctional adducts with biological molecules in cells and tissues, but also bifunctional adducts. This bifunctional nature also means that sulfur mustard can form inter- as well as intramolecular cross-links. (*Id.*)

32. In the case of DNA, mono- or bifunctional lesions generated by sulfur mustard can initiate repair processes through DNA damage signaling cascades. These include homologous recombination as well as nucleotide excision repair and non-homologous end joining. (Jowsey, P.A., et al., "DNA damage responses in cells exposed to sulphur mustard," *Toxicol Lett.* 209:1-10 (2012).) Sulfur mustard-induced interstrand DNA cross links can also result in potentially toxic DNA strand breaks. Critical for the repair of DNA strand breaks, including those induced by sulfur mustard, is phosphorylation of the histone H2A variant, H2A.X. (Joseph, L.B., et al., "Structural changes in the skin of hairless mice following exposure to sulfur mustard correlate with inflammation and DNA damage," *Exp Mol Pathol.* 91:515-527 (2011).) Phospho-H2A.X recruits DNA damage response proteins important in DNA strand break repair. Each of the DNA damage signaling cascades initiates processes that can promote survival of damaged cells and contribute to alterations in epithelial cell growth and differentiation often associated with the

pathology found in both animal and human tissues following sulfur mustard exposures. (Shakarjian 2010.)

33. An important factor associated with sulfur mustard-induced tissue damage that can mediate irritation reactions as well as aberrant pathology is the production of cytokines, growth factors, and lipid mediators. A number of animal studies have documented that sulfur mustard increases expression of proinflammatory cytokines and growth factors in the skin. For example, in rabbit skin, *in situ* hybridization studies have shown increases in interleukin-1beta, interleukin-8, and monocyte chemoattractant-1. (Tsuruta, J., et al., "The cytokines NAP-1 (IL-8), MCP-1, IL-1 beta, and GRO in rabbit inflammatory skin lesions produced by the chemical irritant sulfur mustard," *Inflammation.* 20:293-318 (1996).) In mouse skin, sulfur mustard has been reported to increase interleukin-1beta, interleukin-6, tumor necrosis factor-alpha and granulocyte monocyte-colony stimulating factor. (Ricketts, K.M., et al., "Inflammatory cytokine response in sulfur mustard-exposed mouse skin," *J Appl Toxicol.* 2000 Dec; 20 Suppl 1:S73-S6 (2000); Sabourin, C.L., et al. "Alterations in inflammatory cytokine gene expression in sulfur mustard-exposed mouse skin," *J Biochem Mol Toxicol.* 14:291-302 (2000).) Generally similar increases in cytokines have been reported in the skin of weanling pigs, an animal model that closely resembles human skin. (Sabourin, C.L., et al. "Cytokine, chemokine, and matrix metalloproteinase response after sulfur mustard injury to weanling pig skin," *J Biochem Mol Toxicol.* 16:263-272 (2002).)

34. It is well recognized that eicosanoids, a class of lipid mediators that include prostaglandins and leukotrienes, are critical for the development of inflammatory reactions. Cycloxygenase-2, the rate limiting enzyme in prostaglandin metabolism, has been reported to be upregulated in mouse skin following sulfur mustard treatment. (Joseph, L.B., *Exp Mol Pathol.* at 91:515-527 (2011).) In a human skin construct model, CEES has also been shown to upregulate cyclooxygenase-2 as well as several additional eicosanoid biosynthetic enzymes including 5-lipoxygenase, microsomal prostaglandin E_2 synthases, leukotriene A_4 hydrolase and leukotriene C_4 synthase. (Black, A.T., et al., "Expression of proliferative and inflammatory markers in a full-thickness human skin equivalent following exposure to the model sulfur mustard vesicant, 2-

chloroethyl ethyl sulfide," *Toxicol Appl Pharmacol.* 249:178-187 (2010).) The importance of prostaglandins in mediating inflammation in the skin following sulfur mustard treatment is supported by work showing that sulfur mustard toxicity is blunted in cyclooxygenase-2 deficient mice and that inhibition of the enzyme reduced sulfur mustard-induced inflammation and dermal necrosis in the mouse ear vesicant model. (Wormser, U., et al., "Reduced sulfur mustard-induced skin toxicity in cyclooxygenase-2 knockout and celecoxib-treated mice," *Toxicol Appl Pharmacol.* 200:40-47 (2004); Young, S.C., et al., "Investigation of anticholinergic and non-steroidal anti-inflammatory prodrugs which reduce chemically induced skin inflammation," *J Appl Toxicol.* 32:135-41 (2012).)

35. The precise injury in tissues leading to basal cell damage and blistering is not well understood. In the skin and cornea, sulfur mustard damages many layers of the tissue. In the skin, damage can be found in the epidermis and dermis. However, it is generally thought that blistering is the result of damage to the dermal-epidermal junction. In animal models, sulfur mustard-induced damage to the basement membrane has been observed in skin as well as cornea. (McNutt, P., et al., "Pathogenesis of acute and delayed corneal lesions after ocular exposure to sulfur mustard vapor," *Cornea.* 0:1-11 (2012); (Shakarjian 2010).) Despite the relatively non-selective chemical reactivity of sulfur mustard, basal keratinocytes overlying the basement membrane of the dermal-epidermal junction in the skin appear most sensitive and blistering involves detachment of these cells from their basement membrane adherence zones. (Shakarjian 2010.) The mechanisms leading to detachment of these cells is multifactorial. Sulfur mustard may directly modify basement membrane proteins and weaken basal cell attachments. For example, sulfur mustard can modify intracellular actin microfilaments and keratin intermediate filaments, both of which are known to be critical in maintaining epithelial connections with the basal lamina. (Shakarjian 2010.) Direct modifications of these proteins *in vitro* have also been demonstrated to block keratinocyte adherence to these matrix proteins, a response blocked by sulfur mustard scavengers. (Zhang, Z., et al., "Assessment of sulfur mustard interaction with basement membrane components," *Cell Biol Toxicol.*, 11:89-101 (1995).) Alternatively, sulfur mustard is known to stimulate target tissues to produce matrix metalloproteinases, a class of

enzymes released by keratinocytes that can directly target and degrade proteins in the basement membrane. (Shakarjian, M.P., et al., "Preferential expression of matrix metalloproteinase-9 in mouse skin after sulfur mustard exposure," *J Appl Toxicol.* 26:239-246 (2006).) In the above studies, a mouse ear vesicant model was used to show that sulfur mustard markedly increases matrix metalloproteinase-9, an enzyme that can degrade collagen IV and other components of the basement membrane.

36. Another important consequence of sulfur mustard-induced DNA alkylation is the activation of poly(ADP-ribose) polymerase (PARP), a family of nuclear signaling enzymes that regulate poly-ADP ribosylation of DNA-binding proteins that are involved in repair processes. Excessive activity of this enzyme in basal cells can deplete cells of NAD+ and adenosine triphosphate resulting in cytotoxicity. This is thought to result in apoptosis or necrosis of basal cells and contribute to the process by which these cells detach from the basement membrane to cause blisters. (Kehe, K., et al., "Molecular toxicology of sulfur mustard-induced cutaneous inflammation and blistering," *Toxicology* 26:12-19 (2009); Shakarjian 2010.)

37. As discussed in more detail below, these agents have various specific physiological effects depending on the dose and exposed tissue. The most detailed information has been reported in humans exposed to sulfur mustard. The skin, respiratory tract and eyes are the most common sites of exposure to sulfur mustard.

2. Dermal Effects

38. One of the hallmarks of human exposure to sulfur mustard is irritation reactions. In the skin, exposures can result in both acute and chronic reactions. Chronic effects have been reported many decades following sulfur mustard exposures. Both acute and chronic effects are dependent on dose and duration of exposure as well as environmental conditions. For instance, higher temperatures and increased humidity exacerbate acute irritation reactions. Grossly, acute irritation, which often arises within several hours of sulfur mustard exposure, includes pruritus, an itching or burning sensation, pain, erythema, swelling, and vesicle and blister formation. (*See* Poursaleh, Z., et al., "Pathogenesis and treatment of skin lesions caused by sulfur mustard," *Cutan Ocul Toxicol.* 1-9 (2011).) Soon after initial exposure, these reactions can lead to dermal

erosions and ulceration. Microscopically, this is associated with changes in growth and differentiation of the epidermis, disruption of the basement membrane and white blood cell infiltration with increased edema in the dermis. (Naraghi, Z.S., et al., "A clinicopathological study on acute cutaneous lesions induced by sulfur mustard gas (Yperite)," *Eur J Dermatol.* 15:140-145(2005).)

39. Skin damage begins almost immediately after exposure to mustard, though noticeable symptoms generally do not appear for 1-24 hours after exposure, typically appearing more rapidly after exposure to liquid sulfur mustard, and may not be complete for several days. (*Veterans at Risk* at 157-158; *Handbook of Toxicology Chapter 41* at 612-616.) The LD_{50}[6] for skin exposure with liquid sulfur mustard is about 700 mg/kg, which corresponds to exposure of about 25% of the surface of the body, though a drop containing as little as 10 µg of sulfur mustard can cause blistering. (*Military Medicine* at VET004_001139, 1170; *see also Veterans at Risk* at 157, Table 3-4.) Vapor exposure is typically reported as the concentration of the agent (mg/m^3) times the duration of exposure, or C_t. (*Military Medicine* at VET004_001139.) Thus, a longer exposure to a low concentration of agent can be just as dangerous as a brief exposure to a very high concentration. (*Id.*) The threshold vapor exposure for skin damage depends on various factors including skin site, temperature, humidity, and sweat, though it generally falls in the range of 50-2,000 $mg \cdot min/m^3$. (*Id.* at VET004_001139, 1143, 1170; *Veterans at Risk* at 158.)

40. Skin blisters caused by sulfur mustard are typically dome-shaped, thin walled and surrounded by erythema. (*Military Medicine* at VET004_001144; *Handbook of Toxicology Chapter 41* at 618-620.) Severe lesions are prone to necrosis and secondary infection. (*Military Medicine* at VET004_001144.) Healing depends on the severity of the injury—erythema may resolve in a matter of days while blisters may take weeks to months to fully heal. (*Military Medicine* at VET004_001146-47.) In either case, exposure routinely leads to changes in skin pigmentation. (*Id.*; *Veterans at Risk* at 159-162.) Irritation reactions can persist, with burning, itching and psoriasis-like skin lesions reported many decades post sulfur mustard exposure,

[6] The LD_{50} is the dose of a given compound that is lethal to 50% of the exposed population.

including severe cases in which patients present with bullous lesions, lichen simplex, prurigo and eczema. (Ghassemi-Broumand, M., et al., "Delayed Ocular, Pulmonary, and Cutaneous Complications of Mustards in Patients in the City of Sardasht, Iran," *Cutan Ocul Toxicol.*, 27:295-305 (2008) (hereinafter "Ghassemi-Broumand 2008"); Rowell, M.,et al., "The chronic effects of sulfur mustard exposure," *Toxicology* 263:9-11 (2009); Namazi, S., et al., "Long-term complications of sulphur mustard poisoning in intoxicated Iranian veterans," J Med Toxicol., 5:191-195 (2009) (hereinafter "Namazi 2009").)

41. Unlike mustard gas, skin exposure to Lewisite is almost immediately painful. (*Military Medicine* at VET004_001156-57; *Veterans at Risk* at 25.) Drops containing as little as 14 μg of Lewisite can cause vesication, while the LD_{50} for skin exposure is about 30 mg/kg. (*Id.* at VET004_001157; *Veterans at Risk* at 164-166.) Erythema appears within minutes of exposure, with blisters developing within a few hours. (*Military Medicine* at VET004_001157; *Veterans at Risk* at 166.) The resulting lesions are typically less severe than with mustard exposure, heal more rapidly, are less prone to secondary infection, and produce changes in skin pigmentation less often. (*Military Medicine* at VET004_001157.)

3. Pulmonary Effects

42. Acute inhalation exposure to sulfur mustard can cause irritation of the respiratory tract, a result of non-specific inflammation of the mucosa and submucosa, which can develop into a condition closely resembling acute respiratory distress syndrome. (Sohrapour, H., "First World Congress on Biological and Chemical Warfare Agents, Belgium," 291-297 (1989); Kehe, K., et al., "Acute effects of sulfur mustard injury- Munich experiences," *Toxicology* 263:3-8 (2009).) Chronic lung irritation, which has been observed many decades following sulfur mustard exposure, can result in changes that compromise lung function including losses of vital capacity and forced expiratory volume and chronic obstructive pulmonary disease. These signs are often associated with respiratory distress and abnormal lung auscultation. (Bijani, K. and Moghadamnia, A.A., "Long term effects of chemical weapons on respiratory tract in Iraq-Iran war victims living in Babol (North of Iran)," *Exotoxicology & Environmental Safety* 53:422-424 (2002); Namazi 2009; *see also* Brown, E. "Pulmonary Effects Following Chronic Exposure to HS

Vapor," at VET123-008141-47 (report noting pulmonary disabilities reported by munitions factory workers chronically exposed to mustard gas during the 1940s).)

43. Mustard-mediated lung damage is dose dependent, beginning with the upper airways and descending into the lower airways as the dose of mustard increases. (*Military Medicine* at VET004_001148; Weinberger, B. *et al.*, "Sulfur mustard-induced pulmonary injury: therapeutic approaches to mitigating toxicity," *Pulmonary Pharm. & Therapeutics* 24:92-99 (2011) (hereinafter "Weinberger 2011") at 92-93.) The C_t for airway injury with gaseous sulfur mustard is 100-500 mg•min/m^3. (*Military Medicine* at VET004_001139, 1170; *see also Veterans at Risk* at Tables 3-4 and 7-1.) Lung tissue is particularly susceptible to toxins compared to other types of tissue, making inhalation an especially damaging form of exposure. The inflammation associated with mustard gas exposure can vary from mild to severe, with necrosis of the airway epithelium common in more serious cases. (*Military Medicine* at VET004_001148; *Veterans at Risk* at 113-118; Weinberger 2011 at 93-95.) Airway injury typically takes several days to fully develop, and in extreme cases can lead to acute inflammation of the upper and lower airways, discharge, inflammatory exudate, pseudomembrane formation, tissue necrosis, and airway obstruction due to sloughing of damaged tissue. (*Military Medicine* at VET004_001148-149; *Veterans at Risk*; 113-118.) Symptoms of mustard gas exposure include coughing, hoarseness, airway inflammation, and toneless voice. (*Military Medicine* at VET004_001148; *Veterans at Risk*; 113-118.) Of those casualties who die from mustard exposure, the majority are due to massive pulmonary damage from inhaling gaseous mustard, often complicated by infection and sepsis. (*Military Medicine* at VET004_001150.)

44. Lewisite produces effects similar to mustard gas, and has an LD_{50} by inhalation of about 1,500 mg•min/m^3 with airway damage occurring at a C_t of about 500 mg•min/m^3. (*Military Medicine* at VET004_001157.) Lewisite vapor causes immediate irritation of the respiratory tract, which typically causes those exposed to seek immediate protection and treatment, thus limiting their exposure and the extent of their injuries. (*Id.* at VET004_001158; *Veterans at Risk* at 117-118.)

4. Ocular Effects

45. While the eye is the organ most sensitive to sulfur mustard, as with skin exposure, there can be a prolonged latency period after exposure before symptoms appear. (*Military Medicine* at VET004_001147; *Handbook of Toxicology Chapter 39* at 575.) The time before symptoms appear varies by both individual sensitivity and the concentration of sulfur mustard, though the latency period is generally shorter than it is for skin exposure. (*Military Medicine* at VET004_001147; *Handbook of Toxicology Chapter 39* at 576 (noting a typical latency of 6-8 hours).)

46. Acute irritation reactions of the eye following sulfur mustard exposure include tearing, conjunctivitis, pain and grittiness under the eyelid, blepharospasm and corneal edema. Corneal damage and ulceration have also been reported. (Kehe, K., and Szinicz, L., "Medical aspects of sulphur poisoning," *Toxicology* 214:198-209 (2005).) Chronic ophthalmic complications reported many decades following sulfur mustard exposure include photophobia, foreign body sensation, burning, itching, lacrimation, redness, blurred vision, pain, and difficulty while reading; hyperemia, conjunctival edema, sub-conjunctival hemorrhage, conjunctival vascular dilation, conjunctival concretion, and altered visual acuity have also been reported as well as alterations in corneal structure and functioning, edema and neovascularization. (Pleyer, U., et al., "Delayed mustard gas keratopathy: clinical findings and confocal microscopy," Am J Ophthalmol. 128: 506-507 (1999); Ghassemi-Broumand 2008; Namazi 2009.)

47. The C_t for eye injury with gaseous sulfur mustard is 10-70 mg•min/m^3. (*Military Medicine* at VET004_001139, 1170; *Handbook of Toxicology Chapter 39* at 579-580; *see also Veterans at Risk* at Tables 3-4 and 8-1.) Mild irritation generally appears after a low C_t exposure. (*Military Medicine* at VET004_001139; *Handbook of Toxicology Chapter 39* at 575-580.) As the C_t increases subjects experience progressively worsening conjunctivitis, blepharospasm, pain, and corneal damage. (*Military Medicine* at VET004_001139; *Handbook of Toxicology Chapter 39* at 575-580.) Subjects also typically experience persistent photophobia that can last for weeks, even with mild exposures. (*Military Medicine* at VET004_001139; *Handbook of Toxicology Chapter 39* at 575-580; *see also Veterans at Risk* at 133-139.)

48. Liquid mustard exposure causes even more severe eye injuries. Symptoms can appear within minutes of a droplet of mustard entering the eye. (*Military Medicine* at VET004_001148.) In extreme cases liquid mustard contamination of the eye can lead to perforation of the cornea, loss of vision, and even loss of the eye. (*Id.*)

49. Laboratory studies with rabbits have shown that liquid mustard is rapidly absorbed and dispersed into the eye, a process taking approximately five minutes. (*Military Medicine* at VET004_001148.) Prompt decontamination of the eyes after exposure to liquid mustard is thus vital to avoid serious local or systemic injury.

50. Lewisite is somewhat less toxic to the eye than mustard gas, producing damage at a C_t of about 150 mg•min/m^3. (*Military Medicine* at VET004_001157.) Moreover, eye injuries from Lewisite tend to be less severe than those caused by sulfur mustard, because the immediate pain and irritation caused by even small exposures to Lewisite triggers rapid blepharospasm, preventing further contamination. (*Id.* at VET004_001158.)

5. Systemic Effects

51. Both sulfur and nitrogen mustards are known cellular poisons with mutagenic effects making them recognized human carcinogens. (*Veterans at Risk* at 4, 22, 71-80.) Due to its limited study in humans, the carcinogenic and mutagenic effects of Lewisite are not known, though it has been found to produce chromosomal abnormalities in mammalian cells, which suggests that similar effects may occur in humans. (*Military Medicine* at VET004_001159; *Veterans at Risk* at 25.)

52. As a potent alkylating agent, DNA is one of mustard's most sensitive targets. (*Military Medicine* at VET004_001177, 1180; *Veterans at Risk* at 82-85; *NRC Report Vol. 2* at 105-106.) Animal testing has shown that pulmonary and dermal, as well as subcutaneous, exposure to sulfur mustard can cause localized cancers, though typically not systemic ones. (*Military Medicine* at VET004_001177-78.) Studies of human exposures, including on the battlefield, chronic exposures in munitions factories, and accidental exposures, indicate that sulfur mustard is a human carcinogen. (*Id.* at VET004_001178.) In studies of chronic exposure of factory workers, a definite link was found between increased incidences of respiratory tract

cancers and prolonged exposure to mustard gas. (*Id.*; *see also Veterans at Risk* at 87-103; *NRC Report Vol. 2* at 107-111; *see also* Doi, M. *et al.,* "Effect of mustard gas exposure on incidence of lung cancer: a longitudinal study," *Am J Epidemiol.* 173:659-666 (2011).)

53. Sulfur mustard has also been found to be teratogenic and gonadotoxic, meaning that it attacks the reproductive organs leading to infertility. (*See* Amirzargar, M.A. *et al.*, "Chronic mustard toxicity on the testis: a historical cohort study two decades after exposure," *Int J Androl* 32:411-416 (2009) (hereinafter "Amirzargar 2009").) Researches found a significant decrease in fertility among men exposed to sulfur mustard during the Iran-Iraq war, though the authors were unable to correlate the incidence of infertility with the extent of mustard exposure. (*Id.* at 413-415.) Moreover, there have been reports of an adverse impact on the health of children born to parents exposed to mustard gas, indicating some as yet poorly understood heritable impact from mustard gas. (*See* Abolghasemi, H. *et al.*, "Childhood physical abnormalities following paternal exposure to sulfur mustard gas in Iran: a case control study," *Conflict and Health* 4:13 (2010).)

C. U.S. Testing of Mustard Gas and Lewisite.

54. The use of sulfur mustard and other chemical weapons during World War I left a lasting impression on military planners. Even before the Japanese attack on Pearl Harbor in 1941, the United States had begun clandestine experiments with mustard gas. (*See Veterans at Risk* at *v*, 10.) This research was conducted by two primary groups, the Committee on Medical Research, which focused on treatments and protective ointments through the National Research Council's Committee on the Treatment of Gas Casualties, and the National Defense Research Committee, which studied protective clothing, such as uniforms and gas masks, through military units such as the Chemical Warfare Service. (*Id.* at *v*, 29-48.)

1. Pre-1950 Testing.

55. In response to the extensive, and devastating, use of chemical weapons during World War I, the U.S. began developing its own chemical weapons capability. (*See Health Effects from Chemical, Biological and Radiological Weapons*, Department of Veterans Affairs (2003) (hereinafter "*Health Effects*") at 3.) By 1942 the U.S. military had determined that animal

testing was insufficient to evaluate the effects of such weapons, and embarked on a program of human experimentation with, among other chemical agents, sulfur mustard and Lewisite. (*Health Effects* at 3; *NRC Report Vol. 2* at 254-257.) The program focused both on improving the U.S. chemical weapons arsenal and the means to protect service members from such weapons. (*Health Effects* at 3.) When World War II finally ended, upwards of 60,000 U.S. service members had participated as human test subjects in these mostly secret experiments. (*Id.*; *Veterans at Risk* at *v.*) Of these, at least 4,000 were exposed to high concentrations of mustard gas or Lewisite in chamber or field tests. (*Health Effects* at 3; *Veterans at Risk* at 1.)

56. Exposure protocols in the program varied widely, but fell into three general categories:

- **Drop or patch tests** – drops of mustard or Lewisite were placed on the skin or clothing to test protective equipment, antidotes, treatments for burns, sensitization, and the effects of physical exertion on the extent and severity of reactions to mustard or Lewisite exposure.[7]
- **Chamber tests** – subjects were placed in gas chambers, with or without protective equipment, and exposed to gaseous mustard or Lewisite. Subjects were typically exposed multiple times until they developed erythema.
- **Field tests** – subjects were sent into contaminated areas, typically to test the effectiveness of protective equipment, though some tests involved unprotected subjects, and to evaluate the persistence and extent of contamination.

(*Health Effects* at 3-4; *Veterans at Risk* at 31-41.)

a. Drop or Patch Tests

57. There appears to have been little or no standardization in the drop and patch type tests.

[7] Mustard drop tests were also a common element of basic training to impress on recruits the dangers of chemical weapons and the need for prompt response if such weapons were used. (*Health Effects* at 3-4; *Veterans at Risk* at 31-41.)

Moreover, the tests often involved different protective ointments or treatments. (*Veterans at Risk* at 35.) Given this high degree of variability in the tests, it is difficult to determine the cumulative dose subjects experienced, although it appears to have always been sufficient to cause a visible response—at minimum erythema but more likely a blister. (*Id.*) Moreover, given that mustard is rapidly absorbed into the system, and accumulates over time, even relatively small drops could present an appreciable systemic exposure.

b. Chamber Tests

58. The chamber tests were similarly poorly controlled. (*Veterans at Risk* at 36.)

They were then examined 24 hours later for signs of erythema, an indication of failure of the protective equipment. (*Veterans at Risk* at 36.) This process was repeated either daily or every second day until the subject developed moderate to intense erythema. (*Id.* at 36-37.) These were called "man-break" tests since the endpoint was the subject showing signs of injury from the vesicant being tested. (*Id.*)

59. Using the development of erythema as an endpoint would, in my opinion, have led to substantial exposure. The development of erythema during these tests was an indication that the protective equipment had failed. However, given the lag between exposure and the development of erythema, many of the subjects who eventually did develop erythema were likely exposed to mustard gas for a prolonged period, since they would have remained in the chamber until the end of an exposure period, unaware that their equipment had already failed and they were effectively unprotected. Moreover, some chamber test subjects, given incomplete or inadequate protective equipment to begin with, experienced dangerously high exposures, and developed severe burns and crusted lesions, particularly to the genitals, that took up to a month to heal. (*Health Effects* at 4; *Veterans at Risk* at 39.)

60. It is difficult to determine with any degree of accuracy the dose of mustard subjects in these chamber tests actually received.[8] At minimum, they received a sufficient cumulative dose to suffer skin damage, since development of erythema was the endpoint of the tests. (*Veterans at Risk* at 52.) This means they received a minimum of 100-300 mg•min/m^3 of mustard, while some likely received much higher doses, in the 1,000-2,000 mg•min/m^3 range.

[8] It is my understanding, based on conversations with Plaintiffs' counsel, that Defendants have compiled a "Mustard Gas Database." While that database includes some information about exposures, it includes little or no dose information. I have not seen any other records that provide useful information about the actual doses received by test subjects. I therefore have relied on secondary indications of dose, such as physical manifestations of exposure.

(*Id.*) Given the hot, humid conditions of the chamber tests, the effective doses were likely significantly higher, since mustard tends to preferentially attack moist skin. (*Id.*)

61. Beyond the direct exposure during the test itself, the requirement that subjects remain in their "equipment," unmasked, for 4-24 hours after exiting the chamber likely exposed them to additional mustard from their contaminated clothing. (*Id.*) Finally, the repetition of such tests, every day or two, further compounded the exposure due to accumulation of the mustard dose.

62. In addition to skin exposure, *Veterans at Risk* describes the likely leakage of mustard through the gas masks used in the tests, indicating that subjects may have received substantial additional exposure to the eyes and respiratory tract even through properly functioning masks. (*Id.* at 53-54.) I have reviewed the calculations reported in *Veterans at Risk* and find them reasonable, though I have not independently verified them.

63. Taken together, these facts indicate that the subjects used in chamber tests likely received substantial cumulative doses of mustard. (*Id.* at 55.)

c. Field Tests

64. Apparently little is known about the protocols for field tests, although the available evidence indicates that over the years of the testing programs more than 1000 service members were involved. (*Veterans at Risk* at 40.)

65. Given the uncertainty in testing conditions (particularly temperature and humidity), and levels and efficacy of protective equipment used, it is difficult to determine the

likely level of exposure suffered by these subjects.

66. Based on the discussion in *Veterans at Risk* and other references I have reviewed, it appears that a very large number of service members experienced significant exposures to sulfur mustard and Lewisite in the pre-1950 military chemical weapons testing programs. These test subjects likely suffered significant acute injuries and, as discussed below, are, in my opinion, at an increased risk of developing a range of disorders traceable to their exposure to mustard gas.

2. Post-1950 Testing and the Edgewood Arsenal Program.

67. The vesicant testing program continued at Edgewood Arsenal after World War II, but the scope of the program was far more limited. Records indicate that at least 147 individuals were exposed to sulfur mustard at Edgewood Arsenal. (*NRC Report Vol. 2* at 124; *Veterans at Risk* at 46-47.) The bulk of the subjects were involved in chamber tests. (*NRC Report Vol. 2* at 124.) As with much of the World War II era testing, chamber tests at Edgewood were designed to test protective clothing and masks. (*NRC Report Vol. 2* a 124; *see also* DTIC Report 462053 "Protection Afforded By Experimental XXCC3-Impregnated Navy Work/Combat Clothing Worn By Men Exposed to Mustard Vapor" at 3, 7-16; "Agent List 1955-1975 Edgewood" VET102_000129 at 219-221.) And, as with the World War II era tests, subjects received repeated exposures until the test equipment failed and they developed erythema, in some cases as many as fourteen sequential exposures. (*NRC Report Vol. 2* at 124-127; DTIC Report 462053 at 15.) While in some instances information about the target concentration of mustard in the chamber is available, since the tests involved testing protective equipment to the point of failure

using development of erythema as a guide, it is difficult to know what total dose these subjects actually experienced. (*Id.* at 124-127.) Given that the protocols seem similar to those described in *Veterans at Risk* for the World War II era testing, I believe it is reasonable to assume that subjects in the Edgewood gas chamber experiments received total doses of mustard gas in the same range estimated for the subjects involved in the World War II era experiments—namely significant mustard exposures.

68. Approximately thirty subjects at Edgewood were also used in sulfur mustard drop tests to test protective ointments and treatments. (*NRC Report Vol. 2* at 124-126.) These subjects sometimes developed erythema and blistering at the site of application. (*Id.*) As noted above, the rapid absorption of mustard through the skin means that these drop tests also likely gave these test subjects a substantial systemic exposure to sulfur mustard. Again, exactly how large of a dose is difficult to know without more precise information about the size of drop used, the concentration of sulfur mustard in the drop, ambient conditions (including temperature and humidity), the solvent, and the condition of the subject's skin, particularly whether they were sweating.

69. There were, apparently, no reports of respiratory or ocular injuries in the 147 men exposed to sulfur mustard during the Edgewood testing. (*NRC Report Vol. 2* at 127.) It is, however, likely that they did experience some level of mustard exposure to the eyes and respiratory system, given that, as noted in *Veterans at Risk*, gas masks are imperfect and will typically allow some level of leakage. Moreover, subjects required to continue wearing contaminated clothing *without* a mask would almost assuredly suffer some mustard exposure to their eyes and respiratory system.

V. IRRITANTS

70. In addition to the large-scale testing of vesicants discussed above, particularly sulfur mustard, Defendants tested a wide range of irritants. (*See NRC Report Vol. 2* at 101-103, 135-184, 203-210, 231-253.)

Human and animal testing has shown that each of these agents causes acute toxicity

and, in some cases, persistent toxicity has been noted. The possibility of long-term health effects caused by a particular agent are dependent on a variety of factors including the acute dose, duration of exposure, ambient environmental conditions (temperature, humidity, etc.), sensitization from prior exposure and inter-individual variability. (*See, e.g., NRC Report Vol. 2* at 101-103, 135-184, 203-210, 231-253.) Unfortunately, the major irritants tested at Edgewood, Adamsite, CN, CS and EA 1778, have not been well studied; there is a lack of clinical data, particularly longitudinal studies of subjects exposed to these compounds. In the case of individuals responding to acute chemical irritant exposure, however, persistent adverse health effects including irritant-induced asthma continue to be noted. (Brooks, S.M., et al., "Reactive airways dysfunction syndrome. Case reports of persistent airways hyperreactivity following high-level irritant exposures" *J Occup Med.* 27(7):473-476. (1985).)

A. Adamsite

71.

Tightness of the chest has been reported. According to the NAS report on Possible Long-Term Health Effects of Short-Term Exposure to Chemicals in studies at Edgewood, "predominant symptoms were related to respiratory tract irritation: burning sensations of the respiratory passages, choking sensations, dysphonia, dyspnea, coughing, and sneezing. Nausea was common. Other, less frequent effects were retching, anorexia, headache, dizziness, lacrimation, salivation, and urinary frequency." (*NRC Report Vol. 2* at 209.)

72. According to the CDC/NIOSH, "Exposure to higher concentrations of Adamsite (DM) can result in more severe, longer-lasting redness, itching, and swelling possibly followed

by blister (vesicle) formation" and in the eye, exposures can lead to necrosis of the corneal epithelium. (CDC, NIOSH, The Emergency Response Safety and Health Database: Vomiting Agent: Adamsite, http://www.cdc.gov/niosh/ershdb/EmergencyResponseCard_29750017.html.) Such injuries can have long-term health consequences.

73. Sittig's Handbook of Toxic and Hazardous Chemicals and Carcinogens states that, "No assessment has been made of possible long-term effects of short-term exposures to Adamsite." However, medical surveillance including pulmonary function tests and radiographic imaging is suggested especially in those who have persistent symptoms of dyspnea, cough or chest discomfort. This emphasizes the possibility that exposure to respiratory irritants, particularly repeated or high dose exposures, can lead to long term respiratory effects.

B. CN

74. CN is known by a number of names, including mace, phenacyl chloride, chloroacetophenone, and phenyl chloromethyl ketone**.**

According to the CDC, NIOSH, exposure to CN in the eyes can cause "inflammation of the cornea (keratitis), inflammation of the conjunctiva (conjunctivitis), chemical burns, loss of the outer layer of the cornea (corneal epithelium), sensitivity to light (photophobia), and blurred vision. Partial eye opacity is possible and may be permanent." (CDC, NIOSH, CHLOROACETOPHENONE (CN): Riot Control/Tear Agent, http://www.cdc.gov/NIOSH/ershdb/EmergencyResponse Card_29750033.html.) In the skin, mild to moderate responses include irritation and pain while severe responses can cause erythema, blistering, and denuded areas. In the lung, severe responses can cause pulmonary edema, bronchospasm and bronchopneumia. Severe reactions to CN as described may have long term adverse health consequences. According to the CDC, "severe exposures, such as those in enclosed spaces, may cause permanent damage to the eyes, including blindness." (*Id.*)

75. According to the *Report Volume 2*, "CN, a moderately toxic irritant, has immediate effects on the eyes, skin, and respiratory tract. CN is a strong skin-sensitizing agent, but is rarely lethal. The Committee found no evidence of lasting ocular or respiratory effects in 99 volunteers exposed experimentally at Edgewood between 1958 and 1972 when subjects were evaluated 2 wk. after cutaneous administration or inhalation of aerosol. Allergic contact dermatitis or hypersensitivity in these volunteers on re-exposure to CN is possible. There has been no systematic study of the possible mutagenic and neoplasm-promoting effects of CN with current scientific methods." (*NRC Report Vol. 2* at xv.) Echoing the concern about the lack of information about potential long-term consequences of exposure to such compounds, Olajob and Salem have noted that "As with other xenobiotics, not enough is known concerning the long-term/chronic effects of riot control agents." (Olajos, E.J. and Salem, H., "Riot control agents: pharmacology, toxicology, biochemistry and chemistry," *J Appl Toxicol.* 21(5):355-391 at 355 (2001).)

C. EA 1778

76. An irritant developed at Edgewood Arsenal, EA 1778 is also known as nonanoyl morpholide and pelargonic morpholide. EA 1778 is a lacrimator that causes coughing, a burning sensation of the nose, throat, and eyes, and in some cases nausea. (*NRC Report Vol. 2* at 231-234.) It has also been reported to cause respiratory tract irritation including rhinorrhea, substernal pain, and dyspnea. (*Id.*)

77. In its report, the NAS stated that EA 1778 appeared to be less toxic than other irritants tested, and did not expect long term health effects from the dosages used at Edgewood. (*Id.* at 233, 253 ("The Committee does not expect long-term health effects in subjects tested with nonanoyl morpholide at the dosages used at Edgewood.").) They noted, however, that as with the other irritants tested, there was no specific toxicological information available regarding the potential for long-term health effects. (*Id.* at 233, 235 ("As with CA, DM and CHT, specific toxicologic data regarding its potential in this regard are not available.")

D. CS

78. By far the irritant most widely tested Edgewood was CS, also called o-chlorobenzylidene malononitrile or tear gas. Service members were subjected to full body exposures in wind tunnel tests, to more limited exposures in mask and smaller wind tunnel tests, to ocular exposures, and skin tests. (*See NRC Report Vol. 2* at 135-166; *see, e.g.*, Edgewood Arsenal Technical Report EATR 4246 The Effects of the Riot Control Agent CS on Visual Acuity; Edgewood Arsenal Technical Report EATR 4252 An Evaluation of the Irritant Potential of CS Aerosols on Human Skin Under Tropical Climatic Conditions; Edgewood Arsenal Technical Report EATR 4301 Toxicity of o-Chlorobenzylidene Malonitrile (CS) in Trioctylphosphate (TOF) Solutions; Edgewood Arsenal Technical Report EATR 4309 Toxicology of Riot Control Agents—CS, CN, and DM at 14-25; Edgewood Arsenal Technical Report EATR 4377 CS in Water: Effects on Human Eyes.) CS vapors are extremely irritating to the eyes and respiratory tract. According to the *NRC Report*, the immediate effects of CS "include a burning, pricking, or peppery sensation in the eyes, nose, mouth, throat, and skin; lacrimation, rhinorrhea, and salivation; blepharospasm and injection of the conjunctivas and margins of the eyelids; photophobia lasting up to 1 h in 10% of subjects; tightness of the chest associated with gripping pain, breathholding, dyspnea, coughing, and sneezing; erythema and occasionally vesiculation of exposed skin; and nausea, vomiting, headache, and apprehension." (*NRC Report Vol. 2* at 148.)

79. As with the other irritants discussed above, studies of long-term effects of CS are lacking. According to Blain (2003) "[t]here is no evidence that a healthy individual will experience long-term health effects from open-air exposures to CS or CR." (Blain, P.G. "Tear gases and irritant incapacitants. 1-chloroacetophenone, 2-chlorobenzylidene malononitrile and dibenz[b,f]-1,4-oxazepine," *Toxicol Rev.* 22(2):103-110 at 104 (2003).) According to the *NRC Report*, "[t]here are no data to suggest that the low dose CS-exposure of 105 subjects at Edgewood would give rise to long-term health effects in the primary target organs, the eyes and respiratory tract" and "[t]here is virtually no evidence that CS poses a mutagenic or carcinogenic hazard." (*NRC Report Vol. 2* at 165-66.) The *NRC Report* also notes that "[r]esults of

experimental studies in microorganisms and short-term experiments in laboratory animals suggest that long-term medical abnormalities in soldiers exposed to CS are unlikely. Acute tissue changes produced in animals and humans seem reversible and not likely to become chronic in the absence of recurrent exposures." (*Id.* xiv.) However, "[f]ollow-up information on the long-term state of health of exposed soldiers is not available, but no reports indicate that Edgewood subjects have experienced any long-term sequelae." (*Id.*)

VI. LONG-TERM HEALTH EFFECTS

A. Vesicants.

80. As early as the 1930s it was known that exposure to mustard gas could produce long-term health effects. (*See Veterans at Risk* at 28-29.) The U.S. military, however, apparently failed to include this research in at least its 1941 training manual on how to treat victims of chemical weapon attacks, and chose not to follow the long-term health of the service members used in its chemical weapons testing programs. (*Id.*)

81. As noted above, I have reviewed a variety of sources that have examined the likely long-term health effects from exposure to mustard and Lewisite. These include summaries of studies of humans exposed to mustard under battlefield conditions, particularly during World War I and the Iran-Iraq war, chronic occupational exposures of workers in munitions factories, and clinical exposures, as well as studies in animal models. Of particular importance in my review was the discussion in *Veterans at Risk* as well as more recent scientific reports of the health and welfare of Iranians exposed to mustard during the Iran-Iraq war.

82. It is my understanding that the *Veterans at Risk* report was commissioned by the Department of Veterans Affairs ("VA") in response to the disclosure that tens of thousands of service members had been used as human test subjects in experiments with mustard gas and Lewisite, a rise in veterans presenting with health problems likely linked to these exposures, and growing public pressure to determine the possible long-term health effects these victims might be suffering as a result of their participation in the various testing programs. (*See Veterans at Risk* at *v-vi*, 1-2, 11.) As described in *Veterans at Risk*, the men and women who conducted that investigation reviewed thousands of pages of documents describing the testing programs as well

as first-hand accounts, where possible, consulted the scientific literature about mustard gas and Lewisite, and examined medical records from victims of the various branches of this testing program. (*Id.* at *vi*, 2-3, 11-12.)

83. Based on their extensive investigation, the authors of *Veterans At Risk* found a causal relationship between exposure to mustard gas or Lewisite and the following conditions:

- Respiratory cancers
  - Nasopharyngeal
  - Laryngeal
  - Lung
- Skin cancer
- Leukemia (typically acute nonlymphocytic type, associated with nitrogen mustard exposure)
- Skin pigmentation disorders
- Chronic skin ulceration and scar formation, including related sexual dysfunction due to scrotal or penile scarring
- Chronic respiratory disorders
  - Chronic bronchitis
  - Chronic laryngitis
  - Emphysema
  - Asthma
  - Chronic obstructive pulmonary disease
- Recurrent corneal ulcerative disease
- Delayed recurrent keratitis of the eye
- Chronic conjunctivitis
- Bone marrow depression and related immunosuppression
- Psychological disorders[9]

[9] I express no opinion about the long-term *psychological* effects of the mustard gas and Lewisite testing programs on the test subjects, except to state that these effects must also be

(Footnote continues on next page.)

(*Veterans at Risk* at 4-5, 214-226; *Military Medicine* at VET004_001177; *see also* Namazi, S. *et al.*, "Long-term complications of sulphur mustard poisoning in intoxicated Iranian veterans," *J Med Tox.* 5(4):191-195 (2009); Rowell, M. *et al.*, "The chronic effects of sulfur mustard exposure," *Toxicology* 263:9-11 (2009).) The authors also found a likely, though not confirmed, link between exposure to sulfur mustard and 1) acute nonlymphocytic leukemia, and 2) reproductive dysfunction due to the mutagenicity, teratogenicity and genotoxicity of mustard agents. (*Veterans at Risk* at 4-5. 214-226.)

84. While I have not reviewed all of the underlying data and documents, I have reviewed *Veterans at Risk* and find the reasoning and conclusions of the report sound and consistent with the other scientific and medical literature I have reviewed. (*See, e.g., Veterans at Risk* at 87-111, 118-130, 139-147,167-178.) It is my opinion, based on the discussion in *Veterans at Risk*, as well as the other references I have reviewed and my extensive experience in this field, that short term exposure to mustard gas or Lewisite can cause any or all of the diseases and disorders listed above, and that they can arise years after the original exposure, particularly the various cancers. Moreover, given the types and extents of exposures discussed above, and the high degree of uncertainty about the actual dose subjects suffered, I agree that there is likely a causal relationship between exposure to mustard gas or Lewisite during the U.S. testing programs and development of one or more of these diseases or disorders, even years or decades later. Indeed, given the relatively conservative estimates of exposure levels reported in *Veterans at Risk* I believe that test subjects in the military mustard gas and Lewisite testing programs suffered substantial exposures that likely will lead to long-term health effects.

85. Evidence from studies of occupational and battlefield exposure to sulfur mustard indicates a causal connection between such exposures and long-term effects such as respiratory and skin cancers. (*See Veterans at Risk* at 97-111.) Given the doses likely experienced by

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(Footnote continued from previous page.)

evaluated to arrive at a comprehensive assessment of the effects of the testing of mustard gas and Lewisite on veterans of these testing programs. I understand that the topic of psychological effects will be addressed by a different expert.

subjects of gas chamber and other tests, as discussed above, it is my opinion that they are likely at increased risk for these types of cancers. Moreover, the evidence from therapeutic uses of nitrogen mustard demonstrates a causal relationship between exposure to nitrogen mustard and an increased risk of at least leukemia. (*Id.* at 102-103.) Again, subjects of the various testing programs exposed to nitrogen mustards are, in my opinion, at increased risk of developing such cancers.

86. Acute and severe exposure to sulfur mustard, leading to erythema and blistering, has been linked with long-term recurrent skin pigmentation abnormalities, chronic skin ulceration and skin cancers. (*Veterans at Risk* at 167-178; *see also* Namazi 2009 at 192-195; Rowell 2009.) Given that development of erythema was the endpoint for most, if not all, chamber tests, and that the injuries could become much more severe (including severe blistering and crusted lesions as discussed above), it is my opinion that subjects involved in chamber tests would be at an increased risk for developing these kinds of disorders. Similarly, subjects involved in drop or patch type tests, where a subject's skin was exposed directly to liquid or gaseous mustard, would also be at an increased risk for the skin disorders discussed above at least at the site of the test. Moreover, given the rapid absorption and systemic distribution of sulfur mustard, these subjects could experience systemic effects as well. The long-term effects of exposure to mustard gas and Lewisite may become manifest many years after the time of initial exposure or exposures.

87. Beyond the increased risk of respiratory cancer, studies of occupational and battlefield exposure to sulfur mustard and/or Lewisite demonstrate that exposure to these compounds increases the risk of victims developing a variety of respiratory disorders, including chronic bronchitis, chronic laryngitis, emphysema, asthma and chronic obstructive pulmonary disease. (*See Veterans at Risk* at 119-130; *see also* Weinberger 2011 at 93; *see also* Namazi 2009 at 192-195; Rowell 2009; Ghanei, M. and Harandi, A.A., "Long term consequences from exposure to sulfur mustard: a review," *Inhalation Tox.* 19:451-456 (2007) (hereinafter "Ghanei 2007").) Moreover, while an acute response to exposure is a strong indication that subjects will later develop respiratory disorders, even relatively modest exposures are associated with later development of respiratory disorders. (*Veterans at Risk* at 119-130.) Given the extent of

exposure subjects used in the human testing programs experienced, particularly during the gas chamber tests, it is my opinion that they are likely at higher risk for these kinds of respiratory disorders, as noted in *Veterans at Risk*.

88. With regard to ocular disorders, severe acute injury from sulfur mustard exposure has been linked to recurrent corneal ulcerative disease, while more mild exposures are linked to at least prolonged conjunctivitis. (*See Veterans at Risk* at 139-147; *Handbook of Toxicology Chapter 39* at 576, 578 (noting the appearance of recurrent, persistent ocular ulcerations after a latency of 10-25 years in severe acute exposures); *see also* Namazi 2009 at 192-195; Rowell 2009 at 10 (noting the emergence of an aggressive form of keratitis 15-20 years after initial exposure).) There seem to have been few reports of ocular involvement during the testing programs— erythema seems to have been the primary endpoint for at least the chamber and drop tests. However, given the prolonged exposure, particularly in the chamber tests, the likely leakage rates of the gas masks used, and the fact that these subjects spent substantial periods of time unmasked while continuing to wear contaminated clothing— I find it likely that subjects were exposed to sufficient levels of sulfur mustard to suffer some eye injury and could be at an increased risk for the disorders mentioned above. Moreover, given the exquisite sensitivity of the eye to sulfur mustard, subjects exposed to sulfur mustard *without* masks or similar protective clothing would, in my opinion, definitely have suffered some acute ocular damage and would likely be at an increased risk for corneal ulcerative disease, prolonged conjunctivitis, and recurrent keratitis of the eye.

89. The authors of *Veterans at Risk* recommended that the VA and the Department of Defense, to the extent possible, identify every test subject exposed to any level of mustard gas or Lewisite, inform them of the exposure and the possible health effects, provide a medical evaluation, and give treatment for any disorders linked to their exposure. (*Veterans at Risk* at 224-226.) I believe that this is an appropriate approach for at least two reasons. First, it will provide medical evaluation and care to a cohort of veterans. Second, it would provide exceedingly valuable information about the health of these test subjects and add to the body of knowledge about the long-term health effects of exposure to mustard gas and Lewisite. The

longitudinal studies currently underway in Iran examining the effects decades after mustard exposure during the Iran-Iraq war are testament to the importance of such monitoring, and have already yielded important insights into the negative long-term health effects of mustard exposure. (*See, e.g.,* Abolghasemi, H. et al., "Childhood physical abnormalities following paternal exposure to sulfur mustard gas in Iran: a case control study," *Conflict and Health* 4:13 (2010); Namazi 2009; Rowell 2009.) While there can be no dispute that exposure to mustard is linked to a variety of long-term health disorders, more data is always helpful in elucidating such effects, and revealing previously unknown ones.

90. I have reviewed several letters sent by the Department of Veterans Affairs to veterans or used internally by the VA, and I believe that they are inaccurate in some respects. (*See* Mustard Gas Outreach Letter at VET001_015113-115; Chem-Bio Outreach Letter at VET001_015129-134; Training Letter 06-04 at VET001_015121-128; Clinician's Letter at VET001_015606-9; Clinician's Letter at DVA012 000269-271.) For example, the letters note the linkage between exposure to mustard gas and the diseases mentioned above, but suggest that only subjects who experienced "full-body" exposure are at risk of these conditions. (*See* VET001_15607-8; VET001_015114; DVA012 000270.) According to the letters, patch or drop tests are not considered "full-body" exposures and thus do not pose a risk of long-term effects. (*See* VET001_015114 ("VA may grant compensation to veterans who have certain diseases associated with **full-body exposure** to mustard agents or Lewisite during military service. This means that the entire body was exposed rather than just one or more locations on the skin, such as in a 'patch test.' Information from DoD shows that while you were exposed to mustard agents or Lewisite, this exposure was not full-body exposure.") (emphasis in original); DVA012 000270.) The letters also suggest that there is no conclusive link between "full-body" exposure and long-term health effects. (*See* DVA012 000270 ("VA may grant compensation to veterans who have certain diseases associated with **full-body exposure** to mustard agents or Lewisite during military service.") (emphasis in original).) In my opinion, such statements are medically and scientifically unsupportable. Rather, in my opinion, there is a consensus in the field that mustard gas and Lewisite cause both adverse short and long-term health effects. And, I can state to a reasonable

degree of scientific certainty, that the kinds of exposures to mustard gas or Lewisite I understand took place during Defendants' testing programs, whether it be "full-body" or some other type of exposure, would cause adverse health effects such as those discussed above. Stated another way, assuming a population of approximately 60,000 test subjects exposed to mustard and Lewisite, it is scientifically unassailable that at least some of those exposed, regardless of whether "full-body" or another type of exposure, will experience long-term health effects.

91. In my opinion the vast majority of the subjects, if not all of them, suffered significant exposure to mustard and/or Lewisite. Given that many of the tests were conducted to the point of failure of the "protective" equipment being tested, I would consider that "full-body" exposure. But even apparently more limited exposures, like skin drop or patch tests, were effectively systemic exposures given the rapid absorption and dissemination of mustard in the human body coupled with its cumulative effects and persistence in the system. Thus, I see no scientific rationale for differentiating between test subjects based on whether they received "full-body" exposure as VA has chosen to define it. All human test subjects exposed to any mustard effectively experienced full-body exposures and are, in my opinion, at an increased risk of long-term health effects resulting from their participation in the military testing programs. And, in my opinion, there is medical value to the subjects and their doctors of knowing the substances, doses, and possible health effects, from a standpoint of prevention and treatment.

92. I have also been asked to review the VA's form Chem-Bio Outreach Letter, which I understand was sent to at least some veterans exposed to mustard gas or Lewisite. (VET001_015129-130). The Chem-Bio Outreach Letter is not particularized to reflect the circumstances of any individual's exposures or those of any discrete group. It does not identify what compound or compounds a veteran receiving it was exposed to, nor does it disclose what sorts of long-term effects a veteran might suffer as a result of their exposure. Rather, it states generally, and in my opinion incorrectly, at least with respect to mustard gas and Lewisite, that there are no known long-term health effects from the chemicals tested. (*See* VET001_015130 ("Scientists know much about many of the agents used in these tests. In order to best serve veterans and their families, VA continues to study the possibility of long-term health effects

associated with in-service exposure to chemical and biological agents. If the medical community identifies such health effects, I assure you that we will share this information with you and other veterans as it becomes available to us.") Yet the long-term health effects of mustard gas in particular are well known and significant. And, in contrast to the Outreach Letter, I understand that the Department of Veterans Affairs' internal Chem-Bio Information Letter to clinicians indicates that there are long-term health effects from mustard gas and Lewisite. (*See* VET001_015608 ("Available evidence and follow-up study in general does not support significant long-term, physical harm among subjects exposed to acutely toxic amounts of these agents other than mustard agents and Lewisite.").)

93. The Fact Sheet accompanying the Chem-Bio Outreach Letter also states that the program "evaluated the effects of low-dose exposures to chemical agents." (VET001_15131.) As noted above, however, for at least the mustard chamber and field tests it was virtually impossible to determine *what* dose subjects actually experienced. I thus find it inaccurate to characterize the doses as "low." Many, arguably most, of the subjects experienced a sufficiently high dose to cause erythema if not worse, which I consider a significant dose of sulfur mustard. In my opinion, the doses were not so inconsequential as to rule out the possibility of adverse health effects.

B. Irritants.

94. As noted above, little research has been done on the long-term health effects of the irritant compounds tested at Edgewood, including Adamsite, CS, CN and EA-1778. The *NRC Report* speculates that long-term health effects are unlikely, though admits that there is no scientific or clinical research to directly support this position. I too have been unable to locate sufficient reliable data demonstrating whether the test subjects exposed to irritants at Edgewood would or would not be more likely to develop long-term health problems than unexposed subjects. In my opinion, however, the subjects of irritant exposures should be contacted and medically evaluated. First, such an outreach program could provide much needed data about the long-term effects of these compounds, the very sort of information that at this time does not exist in the literature. Second, it appears that many of the subjects were exposed to more than one

compound, or underwent multiple exposures, and while it is difficult to know the long-term effects of single exposures, it is possible that subjects who endured exposures to compounds that might interact with one another are at even greater risk of adverse health effects. In my opinion it is vital to contact these test subjects precisely because the likely effects of their exposures cannot be determined based on the information currently available.

95. As I noted previously, the Chem-Bio Outreach Letter states that test subjects were exposed only to low doses of chemicals. But I do not see how that determination could be made. For example, subjects were frequently exposed to aerosols, and while the aerosol concentration could be determined, the actual exposure was unknown since it depended on how much of the compound was inhaled and absorbed by the skin and eyes. In most, if not all, irritant experiments the exposure was at least sufficient to cause acute effects, which I consider a significant exposure. Indeed, individuals involved in the testing program were apparently concerned by the levels of exposures. I reviewed at least one memorandum criticizing the design of some field tests for exposing subjects to unreasonably high concentrations of CS. (*See* Disposition Form re Volunteer Testing at JK01 0000213-214.) I thus question the basis for the statement in the Chem-Bio Outreach Letter that test subjects were exposed only to low doses.

VII. CONCLUSION

96. It is my opinion that subjects in Defendants' chemical weapons testing programs experienced significant, and often dangerous, levels of mustard and Lewisite exposure, as well as significant irritant exposures. The weight of scientific and medical literature demonstrates that such exposures to mustard and Lewisite can have significant health impacts decades after the acute exposure, while the effects of irritant exposures are currently not fully known.

97. It is my opinion that the service members who were subjects in the military testing programs are at an increased risk for the kinds of long-term health effects I discuss above.

Respectfully submitted,

Dated: August 7, 2012

Jeffrey D. Laskin, Ph.D.

Exhibit 1

CURRICULUM VITAE
July 18, 2012

NAME: Jeffrey D. Laskin, Ph.D.

PRESENT TITLE: Professor and Chief
Division of Toxicology

ADDRESS: Department of Environmental & Occupational Medicine
University of Medicine and Dentistry of New Jersey
Robert Wood Johnson Medical School
170 Frelinghuysen Road
Piscataway, NJ 08854

EDUCATION:

B.A. (Chemistry and Biology), June 1973, New York University,
University College of Arts and Science, New York
Ph.D. (Pharmacology), September 1977, Dept. of Experimental Therapeutics, Roswell Park Memorial Institute, State University of New York at Buffalo, Buffalo, New York
Post-doctoral Fellow, Staff Associate, 1977- 1981, Division of Environmental Sciences, Cancer Center/Institute of Cancer Research, College of Physicians and Surgeons of Columbia University, New York, NY

ACADEMIC APPOINTMENTS:

Chief, Division of Toxicology (7/1/95 – present)
Department of Environmental & Occupational Medicine
UMDNJ-Robert Wood Johnson Medical School
Piscataway, New Jersey
Professor, Department of Environmental & Occupational Medicine (7/1/93-present)
UMDNJ-Robert Wood Johnson Medical School
Piscataway, New Jersey
Assistant Professor; 5/1/81-6/30/87
Department of Environmental & Occupational Medicine
UMDNJ-Robert Wood Johnson Medical School
Piscataway, New Jersey
Associate Professor; 7/1/87-6/30/93
Department of Environmental & Occupational Medicine
UMDNJ-Robert Wood Johnson Medical School
Piscataway, New Jersey
Member of the Graduate Faculty, Rutgers, The State University of New Jersey, Graduate Programs in Toxicology, Pharmacology, Biochemistry, Microbiology (1/15/82-present)
Deputy Director, Joint Graduate Program in Toxicology, UMDNJ and Rutgers University

(2003-present)
Member of the Environmental and Occupational Health Sciences Institute (EOHSI) UMDNJ and Rutgers University (1986-present)
Director, Division of Toxicology, EOHSI (2005-present)
Center Director, UMDNJ-Rutgers University CounterACT Research Center of Excellence
Member of the Cancer Institute of New Jersey (1995-present)
Member of the Corporation of the Marine Biological Laboratory, Woods Hole, MA (1997-present)
Founding member, Woods Hole Toxicology Forum, Woods Hole, MA (2007-present)
Founding member and Executive Committee member, University Center for Disaster Preparedness and Emergency Responses (UCDPER)(2007-present)
Advisory Committee, Maria Ferrari Westchester Children's Environmental Health Center, 2009-present.

HONORS

2011 Foundation of UMDNJ Excellence in Research Award, UMDNJ School of Biomedical Sciences

LECTURES AND COURSE DEVELOPMENT

Environmental Medicine, UMDNJ-Robert Wood Johnson Medical School (1981-present)
Environmental Toxicology, UMDNJ-School of Public Heath (2003-present)
Skin and Ocular Toxicology, UMDNJ/Rutgers University Joint Graduate Program in Toxicology, (1986-present)
Mechanisms of Chemical Toxicity- UMDNJ-School of Public Health (2002-present)
Mechanism in Drug Toxicity, Lehigh University Satellite Education Network, Distance Education Program (2006-2008)
Environmental Toxicology, New York Medical College School of Public Health (2008-present)

UMDNJ/RWJMS COMMITTEES

9/1/93-present	Appointments and Promotions, Dept. Env. and Occupational Medicine
7/1/97-6/30/00	Committee of Review
9/1/97-8/30/99	Cancer Institute of New Jersey Instrument Committee
9/1/04-8/31/07	Institutional Core Facility Committee
9/16/03-10/1/10	Research Day Organizing Committee
9/1/96-present	IACUC Animal Care Committee
1/15/08-present	UCDPER Executive Committee

EOHSI COMMITTEES

2003-present:	EOHSI Directors Cabinet
2003-present:	EOHSI Space Committee
2008-present:	EOHSI faculty recruitment Committee
2008-present:	Student affairs

STATE OF NEW JERSEY COMMITTEES

2008-present:	NJ Department of Homeland Security Preparedness College
2008-present:	Advisory Committee; NJ Universities Homeland Security Research Consortium

2008-present: Executive Committee; NJ Universities Homeland Security Research Consortium

MEMBERSHIP IN PROFESSIONAL ORGANIZATIONS

American Association for Cancer Research
Society of Toxicology
Dermatology Specialty Section, Society of Toxicology
Secretary/Treasurer (2000, 2001), elected position, Dermatology Specialty Section, Society of Toxicology

LOCAL PROFESSIONAL ORGANIZATIONS

1994-present: Founder and organizer, New Jersey Skin Club
2008-present: Founder and organizer, Basic & Applied Dermatology Forum
2008-present: Chairman, Program Committee, Basic & Applied Dermatology Forum
2007-present: Organizing committee, New Jersey Skin Workshop

INTERNATIONAL MEETINGS ORGANIZED

Symposium, "Nitric Oxide in Health and Disease", Piscataway, NJ; 6/23/93-6/26/93
Symposium, "Advances in the Biology of the Skin: Pharmacology and Toxicology", Piscataway, NJ; 6/24/96-6/27/96
Symposium, "Advances in the Biology & Treatment of the Skin", Piscataway, NJ 6/23/99-6/25/99
"Fourth International Conference on Nitrosative and Oxidative Stress in Disease", New York, NY (sponsored by the New York Academy of Sciences), 10/28/09-10/30/09

RECENT LOCAL MEETINGS ORGANIZED

NJ Spotlight on Skin Research; Minisymposium, Biomaterials Research Center, Rutgers University. 6/25/07
Basic and Applied Dermatology Research Forum, "Wound Healing and Positive Deviance", EOHSI-Rutgers University/UMDNJ-Robert Wood Johnson Medical School, 11/04/09

RECENT SERVICE ON FEDERAL COMMITTEES

NIH Study Section, XES1 LWJ-B (MM), 10/18/04-10/19/04
NIH National Institute of Arthritis and Musculoskeletal and Skin Diseases Roundtable on Wound Healing, 1/16/07-1/17/07
Department of Defense, DTRA FY08 Joint Science and Technology Office for Chemical and Biological Defense (JSTO-CBD) Scientific Review; Respiratory and Systemic Therapeutics: 4/04/07-4/05/07
NIH, Allergy, Immunology & Transplantation Research Review Committee (AITRC): 3/03/08-3/04/08
Department of Defense, DTRA FY09 Joint Science and Technology Office for Chemical and Biological Defense (JSTO-CBD) Scientific Review: 4/07/08-4/08/08
NIH Study Section. CounterACT review ZNS1 SRB-R (33): 4/22/08-4/23/08
NIH Study Section, NIEHS ONES ZES1 JAB-G-R3: 2/10/09-2/11/09
NIH Study Section, NIEHS ONES ZES1 TN G T1 C: 2/10/09
NASA Advanced Environmental Health/Advanced Food Technology Committee, Houston, TX,

11/8/09-11/7/12
NIH Study Section, NIH ZRG1 IMST-A: 11/12/09-11/13/09

RECENT INVITED PRESENTATIONS (2007-present):

4/26/07, "Laminins and the Extracellular Matrix as Targets for Sulfur Mustard", 1st Annual CounterACT Network Research Symposium, Arlington, VA.
6/25/07, "Oxidative Stress and Skin Toxicity", NJ Spotlight on Skin Research; Minisymposium, Biomaterials Research Center, Rutgers University.
10/12/07, "Sulfur Mustard Countermeasures: NATO conference on Defense against the Effects of Chemical Hazards, Edinburgh, Scotland.
11/14/07, "Mechanism of Cutaneous Inflammation", Dermal Clinical Evaluation Society, Glenpointe Marriott, Teaneck, NJ
2/21/08, "The UMDNJ/Rutgers University CounterACT Research Center of Excellence", Robert Wood Johnson Medical School Executive Council, New Brunswick, NJ
4/16/08, "Oxidative Stress in Chemical-induced Skin Injury", 2nd Annual CounterACT Network Research Symposium, Washington, DC.
4/25/08, "Treatments for Sulfur Mustard Poisoning", Department of Chemistry, Lehigh University, Bethlehem, MA
8/03/08, "Risks from Exposure to Sulfur Mustard", Woods Hole Toxicology Forum, Woods Hole Oceanographic Institute, Woods Hole, MA
9/18/08, "Perspectives in Homeland Security Research", Department of Environmental Health, New York Medical College School of Public Health
10/24/08, "The UMDNJ/Rutgers University CounterACT Research Center of Excellence", Biomedical Advanced Research and Development Authority (BARDA), 2009 HHS Public Health Emergency Medical Countermeasures (PHEMC) Enterprise Stakeholders Workshop, Marriott, Arlington, VA
11/5/08, "Oxidative Stress in Sulfur Mustard Toxicity"; Lovelace Respiratory Research Institute, Sulfur Mustard Symposium, Albuquerque, NM
12/18/08, "Thioredoxin reductase as a target for sulfur mustard", Biomaterials Center, Rutgers University
4/15/09, "Antioxidants as Countermeasures to Sulfur Mustard", 3rd Annual CounterACT Network Symposium, Washington, DC
6/8/09, "Drug Development under the FDA Animal Efficacy Rule", Johnson and Johnson Pharmaceutical Research Institute, Raritan, NJ.
7/28/09; "Mechanisms of inflammation", Javelin Pharmaceuticals, Cambridge, MA.
7/29/09; "Redox cycling of 2- and 4-hydroxyestrogen catechol metabolites in breast epithelial cell lines", 2009 Gordon Research Conference on Hormones & Cancer, Holderness School, NH
8/7/09; "Mechanisms mediating chemical redox cycling", Woods Hole Oceanographic Institute, Woods Hole, MA
8/8/09; "US efforts to combat chemical terrorism", Woods Hole Educational Forum, Woods Hole, MA
11/30/09; "Oxidative stress induced by chemical alkylating agents", New York Academy of Sciences symposium on Oxidative and Nitrosative Stress, New York, NY.
5/2/10; "Mechanism of action of sulfur mustard and related alkylating agents, Department of Chemistry, Lehigh University.
3/8/11; Control of Stem Cell Differentiation in the Lung. Society of Toxicology, Washington, DC.
4/29/11; The Threat of Chemical Terrorism, Department of Environmental Medicine, New York University, Sterling Forest, NY
6/22/11; Mechanisms of Action of Chemical Threat Agents, 5st Annual CounterACT Network Research Symposium, Washington, DC
6/26-28/11: Overview of the UMDNJ-Rutgers University CounterACT Research Center of Excellence, 6th Annual CounterACT Network Research Symposium, San Francisco, CA

MENTORED GRADUATE STUDENTS	***Current position***
Linda Piccinini (Microbiology, Ph.D., 1985)	Medical Scientist at Biogen Idec
Christopher Molloy (Toxicology, Ph.D., 1986)	Rutgers University, Ernest Mario School of Pharmacy (Professor II & Dean)
Edmund Lee (Toxicology, MD/Ph.D., 1987)	Rockefeller University (Chief of Dermatology)
Edward Yurkow (Toxicology, Ph.D., 1988)	Johnson & Johnson (Principal Scientist)
Fred Mermelstein (Toxicology, Ph.D., 1990)	Javelin Pharmaceuticals (President & CEO)
Adrienne Garcia-Welsh (Nutrition, Ph.D., 1993)	Consultant in Nutrition Sciences
Diane E. Heck (Toxicology, Ph.D., 1994)	New York Medical College (Professor & Chair)
Anthea Dokidis (Biochemistry, Ph.D., 1995)	Affymetrix, Inc. (Staff Scientist)
Carol Faaland (Biochemistry, Ph.D., 1997)	Consultant in Toxicology
John Mitchell (Toxicology, Ph.D., 1997)	Hurley Consulting (Consultant in Toxicology)
Yang Jin (Toxicology, MD/Ph.D.,1997)	Brigham and Women's Hospital, Harvard University (Assistant Professor)
George DeGeorge (Pharmacology, Ph.D., 1999)	M & B Laboratories (President and CEO)
Blase Billack (Toxicology, Ph.D., 2001)	St. Johns University (Associate Professor)
Anna Vetrano (Biochemistry, Ph.D., 2002)	UMDNJ-Robert W Johnson Medical School (Assistant Professor)
Valescia John (Toxicology, M.S., 2003)	Johnson & Johnson (Scientist I)
Adrienne Black (Toxicology, Ph.D., 2007)	Rutgers University (Research Teaching Spec)
Karma Fussell (Toxicology, Ph.D., 2007-2011)	Res. Scientist, BASF, Germany
Ruijin Zheng (Toxicology, 2005-present)	
Irene Wolman (Toxicology, 2008-present)	
Ronald Udasin (Toxicology, 2009-present)	

POST-DOCTORAL FELLOWS	***Current position***
Tanveer Abidi, Ph.D. (1987-1989)	Kean University (Assistant Professor)
Leslie Helyer, Ph.D. (1990-1993)	Consultant in Nutrition Science
Chitra Punjabi, Ph.D. (1991-1993)	Consultant in Toxicology
Randy Shuler, Ph.D. (1991-1994)	Environmental Resources Management (Senior Toxicologist)
Runa Sur, Ph.D. (2000-2001)	Johnson and Johnson (Staff Scientist)
Rupa Mukhopadhyay, Ph.D. (2003-2005)	Johnson and Johnson (Staff Scientist)
Anna Vetrano, Ph.D. (2003-2005)	UMDNJ-Robert W Johnson Medical School (Assistant Professor)
Joshua Gray, Ph.D. (2003-2006)	US Coast Guard Academy (Assistant Professor)
Adrienne Black, Ph.D. (2009-2011)	Nanotox (Staff Scientist)
Vladimir Mishin, Ph.D. (2005-present)	
Yun Wang, Ph.D. (2006-present)	
Shaojun Yang, Ph.D. (2007-present)	
Yi-Hua Jan, Ph.D. (2007-present)	
Jamie Bernard, Ph.D. (2011-present)	

RESEARCH ASSISTANT PROFESSORS

Diane Heck, Ph.D. (1996-2004)	New York Medical College (Professor & Chair)
Thomas Mariano, Ph.D. (1997-2003)	UMDNJ-CABM (Research faculty)
Michael Shakarjian, Ph.D. (2005-2008)	New York Medical College (Assistant Professor)

Joshua Gray, Ph.D. (2006-2008) US Coast Guard Academy (Assistant Professor)

STUDENT COMMITTEES:

Active member of Rutgers University and University of Medicine and Dentistry of New Jersey graduate programs in Toxicology, Molecular Biosciences, Biochemistry, Cell and Molecular Biology, and Nutrition. Involved in teaching and serving on various program and student committees including qualifying examination committees and doctoral defense committees.

PATENTS

US patent #5,216,176 (June 1, 1993) "7-Alkoxycoumarins, dihydropsoralens and benzodipyranones as photoactivated therapeutics"
US patent #5,356,929 (October 18, 1994) "Reduced and quaternized psoralens as photoactivated therapeutics"
US patent #5,473,083 (December 5, 1995) "Reduced and quaternized psoralens as photoactivated therapeutics"
US patent #5,695,761 (December 9, 1997) "Suppression of nitric oxide production by osteopontin"
US patent #6,177,424 (January 23, 2001) "4'-substituted-4',5'-dihydropsoralens and therapeutic Uses thereof"
US patent #6,255,324 (July 3, 2001) "Amino- and mercurio-substituted 4',5'-dihydropsoralens and therapeutical uses thereof"
US patent #7,015,022 (March 21, 2006) "Mammalian catalase-dependent oxidation processes and methods for stimulating oxidative activities"
US patent #7,105,511 (September 12, 2006) "Fluorescent fused-ring triazoles that inhibit cell proliferation and uses thereof"
US Patent #7,150,967 (December 19, 2006) "Fluorescent tags for amino acid and nucleic acid analysis"
US Patent #7,598,238 (October 6, 2009) "Fluorescent fused-ring triazoles that inhibit cell proliferation and uses thereof"
US Patent #8,071,642 (December 6, 2011) "Dimethyl amino ethyl ether psoralens and methods for their production and use"
US patent pending (publication number 20030225148) "Biological methods of use of 4-amino-3-mercapto-triazoles"
US patent pending (publication number 20040225000) "Methods of producing 4-amino-3-mercapto-triazoles"
US patent pending (submitted by UMDNJ November 3, 2009) "Unique dual action therapeutics"
US Provision patent (submitted by UMDNJ September 15, 2009) "Pharmacologically active vanilloid carbamates"

PUBLICATIONS

JOURNAL ARTICLES

1. Laskin JD, Evans RM, Slocum HD, Burke D, Hakala MT. (1979). Basis for natural variations in sensitivity to 5-fluorouracil in mouse and human cells in culture. Cancer Research, 39, 383-390.

2. Mufson RA, Laskin JD, Fisher PB, Weinstein IB. (1979). Melittin shares certain cellular effects

with phorbol ester tumor promoters. Nature, 290, 72-74.

3. Laskin JD, Mufson RA, Weinstein IB, Engelhardt DL. (1980). Identification of a distinct phase during melanogenesis that is sensitive to extracellular pH and ionic strength. J. Cellular Physiology, 103, 467-474.

4. Laskin DL, Laskin JD, Weinstein IB, Carchman RA. (1980). Modulation of phagocytosis by tumor promoters and epidermal growth factor in normal and transformed macrophages. Cancer Research, 40, 1028-1035.

5. Evans RM, Laskin JD, Hakala MT. (1980). Assessment of growth limiting events caused by 5-fluorouracil in mouse and human cells. Cancer Research, 40, 4113-4122.

6. Laskin DL, Laskin JD, Weinstein IB, Carchman RA. (1981). Induction of chemotaxis in mouse peritoneal macrophages by phorbol ester tumor promoters. Cancer Research, 41, 1023-1028.

7. Laskin JD, Mufson RA, Piccinini L, Engelhardt DL, Weinstein IB. (1981). Effect of tumor promoters on newly synthesized proteins in mouse epidermis. Cell, 25, 441-450.

8. Evans RM, Laskin JD, Hakala MT. (1981). Effect of excess folates and deoxyinosine on the activity and site of action of 5-fluorouracil. Cancer Research, 41, 3288-3295.

9. Pietropaolo C, Laskin, JD, Weinstein IB. (1981). Effect of tumor promoters on sarc gene expression in normal and transformed chick embryo fibroblasts. Cancer Research, 41, 1565-1571.

10. Matthew E, Laskin JD, Zimmerman EA, Weinstein IB, Hsu KD, Engelhardt DL. (1981). Benzodiazepines have high affinity binding sites and induce melanogenesis in B16/C3 melanoma cells. Proeedings of the National Academy Sciences, USA, 78, 3935-3939.

11. Laskin DL, Laskin JD, Weinstein IB, Carchman RA. (1981) Enhancement of macrophage-induced cellular cytotoxicity by phorbol ester tumor promoters, Cancer Research 41, 4523-4528.

12. Laskin JD, Piccinini L, Engelhardt DL, Weinstein IB. (1982). Control of melanin synthesis and secretion in B16/C3 cells. Journal Cellular Physiology, 113, 481-486.

13. Laskin JD, Piccinini L, Engelhardt DL, Weinstein IB. (1983). Specific protein production during melanogenesis in B16/C3 melanoma cells. Journal Cellular Physiology, 114, 114-121.

14. Molloy CJ, Gallo MA, Laskin JD. (1984). Effect of the chemical irritants anthralin and benzoyl peroxide and mouse skin epithelial cell protein production. Journal Soceity Cosmetic Chemists, 35, 197-205.

15. Hsu L, Natyzak D, Laskin JD. (1984). Effect of the tumor promoter 12-O-tetradecanoyl phorbol-13-acetate on neurite outgrowth from sensory ganglia. Cancer Research, 44, 4607-4614.

16. Laskin JD, Lee E, Yurkow E, Laskin D, Gallo MA. (1985). A possible mechanism of psoralen toxicity not involving direct interaction with DNA. Proceedings National Academy

Sciences, USA, 82, 6158-6162.

17. Molloy CJ, Laskin JD. (1985). Alterations in mouse epidermal keratin production induced by dietary vitamin A deficiency. Annals New York Academy Sciences, 455, 739-740.

18. Laskin JD, Lee E, Laskin D, Gallo MA. (1986). Psoralens potentiate UVA light induced inhibition of epidermal growth factor binding. Proceedings National Academy Sciences, USA, 83, 8211-8215.

19. Laskin JD, Piccinini L. (1986). Tyrosinase isozyme heterogeneity in differentiating B16/C3 melanoma. Journal Biological Chemistry, 261, 16626-16635.

20. Yurkow EJ, Laskin JD. (1987). Characterization of a photoalkylated psoralen receptor in HeLa cells, Journal Biological Chemistry, 262, 8439-8442.

21. Molloy CJ, Laskin JD. (1987). Specific alterations in keratin biosynthesis in mouse epidermis in vivo and in explant culture following a single exposure to the tumor promoter 12-O-tetradecanoylphorbol-13-acetate, Cancer Research, 47, 4674-4680.

22. Molloy CJ, Gallo MA, Laskin JD. (1987). Alterations in the expression of specific epidermal keratin markers in the hairless mouse by the topical application of the tumor promoters 2,3,7,8-tetrachlorodibenzo-p-dioxin and the phorbol ester 12-O-tetradecanoylphorbol-13-acetate, Carcinogenesis, 8, 1193-1199.

23. Laskin DL, Gardner CR, Laskin JD. (1987). Induction of chemotaxis in mouse peritoneal macrophages by activators of protein kinase C. Journal Leukocyte Biology, 41, 474-480.

24. Laskin DL, Sirak AA, Pilaro AM, Laskin JD. (1988). Functional and biochemical properties of rat Kupffer cells and peritoneal macrophages. Journal Leukocyte Biology 44, 71-78.

25. Molloy CJ, Laskin JD. (1988). Effect of retinoid deficiency on keratin expression in mouse bladder, Experimental. Molecular Pathology, 49, 128-140.

26. Molloy CJ, Laskin JD. (1988). Keratin polypeptide expression in mouse epidermis and cultured epidermal cells Differentiation 37, 86-97.

27. Laskin DL, Robertson FM, Pilaro AM, Laskin JD. (1988). Activation of liver macrophages following phenobarbital treatment of rats, Hepatology, 8, 1051-1055.

28. Geller HM, Adinolfi A, Laskin JD, Freed WD. (1988). Implantation of catecholamine-secreting cell lines into the rat and mouse brain. Progress Brain Research, 78, 643-646

29. Freed WJ, Adinolfi AM, Laskin JD, Geller HM. (1989). Transplantation of B16/C3 melanoma cells into the brains of rats and mice. Brain Research, 485, 349-362.

30. Mermelstein FM, Abidi TF, Laskin JD. (1989). Inhibition of epidermal growth factor receptor tyrosine kinase activity in A431 human epidermoid cells following psoralen/ultraviolet light treatment. Molecular Pharmacology, 36, 848-855.

31. Abidi TF, Faaland CA, Scala D, Rhein L, Laskin JD. (1989). Ethoxylated alcohol sulfate

(Neodol-12) and other surfactants in the assay of protein kinase C. Biochemistry Biophysica Acta, 992, 362-368.

32. Yurkow E, Laskin JD. (1989). Purification of tyrosinase to apparent homogeneity based on its resistance to SDS-proteinase K digestion. Archiv. Biochemistry Biophysics, 275, 122-129.

33. Laskin DL, Beavis AJ, Sirak AS, O'Connell SM, Laskin JD. (1990). Differentiation of U-937 histiocytic lymphoma cells towards mature neutrophilic granulocytes by dibutyryl cyclic adenosine-3',5'-monophosphate, Cancer Research, 50, 20-25.

34. Robertson FM, Huang MT, Conney A, Laskin JD, O'Brien T. (1990). Identification of epidermal cell subpopulations with increased ornithine decarboxylase activity following treatment of mouse epidermis with 12-O-tetradecanoyl-phorbol-13-acetate. Cancer Research, 50, 4741-4746.

35. Robertson FM, Beavis AJ, Oberyszyn TM, O'Connell SM, Dokidos A, Laskin DL, Laskin JD, Reiners JJ. (1990). Production of hydrogen peroxide by murine epidermal keratinocytes following treatment with the tumor promoter 12-O-tetradecanoylphorbol-13-acetate. Cancer Research, 50, 6062-6067.

36. Sirak AA, Laskin JD, Robertson FM, Laskin DL. (1990). Failure of F-MET-LEU-PHE to induce chemotaxis in differentiated promyelocytic (HL 60) leukemia cells, Journal Leukocyte Biology, 48, 333-342.

37. Robertson FM, Gilmour SK, Beavis AJ, Conney AH, Huang M-T, Laskin JD, Hietala OA, O'Brien TG. (1990). Flow cytometric detection of ornithine decarboxylase activity in epidermal cell subpopulations. Cytometry, 11, 832-836.

38. Huang M-T, Lysz T, Ferraro T, Abidi, TF, Laskin JD, Conney AH. (1990). Inhibitory effects of curcumin on in vitro lipoxygenase and cyclooxygenase activities in mouse epidermis. Cancer Research, 51, 813-819.

39. Jetter MM, Heindel ND, Laskin JD. (1990). Novel synthesis of dihydroxanthyletin and dihydrosesselin derivatives. Journal Heterocyclic Chemistry, 27, 995-997.

40. Yurkow E, Laskin JD. (1991). Mechanism of action of the psoralens: Isobologram analysis reveals that ultraviolet light potentiation of psoralen action is not additive but synergistic. Cancer Chemotherapy Pharmacology, 27, 315-319.

41. Conney AH, Lysz T, Ferraro T, Abidi TF, Manchand PS, Laskin JD, Huang MT. (1991) Inhibitory effect of curcumin and some related dietary compounds on tumor promotion and arachidonic acid metabolism in mouse skin. Advances Enzyme Regulation, 31, 385-396.

42. Laskin JD, Lee E. (1991). Psoralen binding and inhibition of epidermal growth factor binding by psoralens/ultraviolet light (PUVA) in human epithelial cells. Biochemical Pharmacology, 44, 125-132.

43. Laskin DL, Sirak AA, Laskin JD. (1991). Distinct patterns of chemotactic-peptide induced calcium mobilization in differentiated myeloid leukemia cells and peripheral blood

neutrophils. Journal Leukocyte Biology, 49, 369-379.

44. Laskin JD, Dokidis A, Sirak A, Laskin DL. (1991). Distinct patterns of proteoglycan biosynthesis in human granulocytes, monocytes and differentiated myeloid leukemia cells, Leukemia Research, 15, 515-523.

45. Laskin DL, Sirak AA, Laskin JD. (1991). Differentiation of HL 60 myeloid leukemia cells is associated with a transient block in the G2 phase of the cell cycle, Cell Proliferation, 24, 341-355.

46. Laskin JD, Dokidis A, Gardner CR, Laskin DL. (1991). Changes in sulfated proteoglycan production following activation of rat liver macrophages, Hepatology, 14, 306-312.

47. Heindel ND, Van Dongen, JM, Sachais, BS, Phillips JH, Gallo MA, Laskin JD. (1991). Syntheses of psoralen analogues and evaluation of their inhibition of epidermal growth factor binding. Journal Pharmaceutical Sciences, 80, 686-689.

48. Faaland CA, Mermelstein FH, Hayashi J, Laskin JD. (1991). Rapid uptake of tyrphostin into A431 human epidermoid cells is followed by a delayed inhibition of epidermal growth factor (EGF)-stimulated EGF receptor tyrosine kinase activity. Molecular Cellular Biology, 11, 2697-2703.

49. Laskin JD, Dokidis A, Laskin DL. (1991). Characterization of sulfated proteoglycan synthesis in mouse skin following a single topical application of the tumor promoter 12-O-tetradecanoylphorbol-13-acetate. Cancer Biochemistry Biophysics, 12, 69-73.

50. Heck DE, Bisaccia E, Armus S, Laskin JD. (1991). Production of hydrogen peroxide by cutaneous T cell lymphoma following photopheresis with psoralens and ultraviolet light. Cancer Chemotherapy Pharmacology, 28, 344-350.

51. Georgiadis C, Laskin JD. (1991). Differential inhibition of epidermal growth factor binding by the tumor promoter 12-O-tetradecanoylphorbol-13-acetate and the photoactivated psoralens in cells overexpressing protein kinase C. Biochemical Pharmacology, 42, 2003-2009.

52. Huang M-T, Ho CT, Wang ZY, Ferraro T, Finnegan-Olive T, Lou Y-R, Mitchell JM, Laskin JD, Newmark H, Yang CS, Conney AH. (1992). Inhibitory effect of topical application of a green tea polyphenol fraction on tumor initiation and promotion in mouse skin, Carcinogenesis, 13, 947-954.

53. Shenkman RL, Laskin JD, Yalowich JC, LaVoie EJ. (1992). Structure activity relationships of mammalian topoisomerase II inhibitors related to ellipticine, Medicinal Chemistry Research, 2, 165-172.

54. Molloy CJ, Laskin JD. (1992). Altered expression of a mouse epidermal cytoskeletal protein is a sensitive marker for proliferation induced by tumor promoters. Carcinogenesis 13, 963-968.

55. Heck D, Laskin DL, Gardner CA, Laskin JD (1992). Epidermal growth factor suppresses nitric oxide and hydrogen peroxide in keratinocytes. Potential role of nitric oxide in the

regulation of wound healing. Journal Biological Chemistry, 267, 21277-21280.

56. Punjabi C, Laskin, DL, Heck D, Laskin JD. (1992). Nitric oxide production by bone marrow cells: inverse correlation with cellular proliferation, Journal Immunology, 149, 2179-2184.

57. Gardner CR, Laskin JD, Laskin DL. (1993). Platelet activating factor induced calcium mobilization in hepatic macrophages and endothelial cells, Journal Leukocyte Biology, 53, 190-196.

58. Pendino K, Gardner CA, Laskin JD, Laskin DL. (1993). Induction of functionally active platelet-activating receptors in rat alveolar macrophages, Journal Biological Chemistry, 268, 1-4.

59. Laskin JD, Mermelstein FM, Heindel ND, Ron Y. (1993). Selective inactivation of lymphocytes after psoralen/ultraviolet light (PUVA) treatment without affecting systemic immune responses. Journal Leukocyte Biology 54, 138-144.

60. Hwang S-m, Lopez CA, Heck DE, Gardner, CR, Laskin DL, Laskin JD, Denhardt DT. (1993). Osteopontin inhibits induction of nitric oxide synthase gene expression by inflammatory mediators in mouse kidney epithelial cells. Journal Biological Chemistry, 269, 711-715.

61. Hwang S-m, Wilson, PD, Laskin JD, Denhardt DT. (1994). Age and development related changes in osteopontin and nitric oxide synthase mRNA levels in human kidney proximal tubule epithelial cells: contrasting responses to hypoxia and reoxygenation. Journal Cellular Physiology, 160, 61-68.

62. Punjabi CJ, Laskin JD, Huang S-m, MacEachern L, Laskin DL. (1994). Enhanced production of nitric oxide by bone marrow cells and increased sensitivity to macrophage colony stimulating factor (CSF) and granulocyte-macrophage CSF after benzene treatment of mice. Blood, 83, 3255-3263.

63. Punjabi CJ, Laskin JD, Pendino KJ, Goller NL, Durham SK, Laskin DL. (1994). Production of nitric oxide by rat type II pneumocytes: increased expression of inducible nitric oxide synthase following inhalation of a pulmonary irritant. American Journal Respiratory Cell and Molecular Biology, 11: 165-172.

64. Garcia-Welsh A, Laskin DL, Shuler RL, Laskin JD. (1994). Cellular depletion of p56lck during thymocyte apoptosis, Journal Leukocyte Biology, 56, 528-532.

65. Garcia-Welsh AL, Laskin DL, Hwang S-m, Denhardt DT, Laskin JD. (1994). Production of nitric oxide by differentiated LSTRA cells is associated with expression of macrophage inducible nitric oxide synthase, Journal Leukocyte Biology, 56, 488-494.

66. Garcia-Welsh A, Laskin DL, Molloy CJ, Laskin JD. (1994). Alterations in expression of p56lck during myeloid differentiation of LSTRA cells. Cell Growth and Differentiation. 5, 1215-1223.

67. Helyar L, Bundschuh D, Laskin JD, Laskin DL. (1994). Induction of hepatic Ito cell nitric oxide production after acute endotoxemia. Hepatology 20, 1509-1515.

68. Heck DE, Laskin JD, Zigman S, Troll W. (1994). N^G-monomethyl-l-arginine inhibits arbacia fertilization and differentiation. Biological Bulletin, 187, 248-249.

69. Laskin DL, Heck DE, Gardner CR, Feder LS, Laskin JD. (1994). Distinct patterns of nitric oxide production in hepatic macrophages and endothelial cells following acute endotoxemia. Journal Leukocyte Biology. 56, 751-758.

70. Wizemanm TM, Gardner CR, Laskin JD, Quinones S, Durham SK, Goller NL, Ohnishi ST, Laskin DL. (1994). Production of nitric oxide and peroxynitrite in the lung following acute acute exposure of rats to endotoxin. Journal Leukocyte Biology. 56, 759-768.

71. Laskin DL, Pendino KJ, Punjabi CJ, Rodriguez del Valle M, Laskin JD. (1994). Pulmonary and hepatic effects of inhaled ozone in rats. Environmental Health Perspectives, 102, S10, 61-64.

72. Pendino KJ, Shuler RL, Laskin JD, Laskin DL. (1994). Enhanced production of interleukin-1, tumor necrosis factor-a and fibronectin by rat lung phagocytes following inhalation of a pulmonary irritant. American Journal Respiratory Cell and Molecular Biology 11, 279-286.

73 Troll W, Sueoka N, Sueoka E, Laskin JD, Heck DE. (1995). Inhibitors of protein phosphatases (okadaic acid and tautomycin) block sea urchin development. Biological Bulletin, 189:201.

74 Shuler RL, Laskin DL, Gardner CR, Feder LS, Laskin JD. (1995). Lymphocyte mediated nitric oxide production by rat endothelial cells. Journal Leukocyte Biology, 57, 116-121.

75. Gardner CR, Laskin JD, Laskin DL. (1995). Distinct biochemical responses of hepatic macrophages and endothelial cells to platelet activating factor during endotoxemia. Journal Leukocyte Biology 57, 269-274.

76. Laskin JD, Rao NR, Punjabi CJ, Laskin DL, Snyder R. (1995). Distinct actions of benzene and its metabolites on nitric oxide production by bone marrow leukocytes. Journal Leukocyte Biology, 57, 422-426.

77. Faaland CA, Laskin JD, Thomas TJ. (1995). Inhibition of epidermal growth factor-stimulated EGF receptor tyrosine kinase activity in A431 epidermoid carcinoma cells by polyamines. Cell Growth and Differentiation 6, 115-121.

78. Pendino KJ, Meidof TM, Heck DE, Laskin JD, Laskin DL. (1995). Inhibition of macrophages with gadolinium chloride abrogates ozone-induced pulmonary injury and inflammatory mediator production. American Journal Respiratory Cell and Molecular Biology, 13, 125-132.

79. Laskin DL, Rodriquez del Valle M, Heck DE, Hwang S, Ohnishi ST, Durham SK, Laskin JD. (1995). Hepatic nitric oxide production following acute endotoxemia in rats is mediated by increased inducible nitric oxide synthase gene expression. Hepatology 22, 223-234.

80. Stauber KL, Laskin JD, Yurkow EJ, Thomas PE, Laskin DL, Conney AH. (1995). Flow cytometry reveals subpopulations of murine epidermal cells that are refractory to induction of cytochrome P-450-1A1 by ß-napthoflavone. Journal Pharmacology Experimental Therapeutics, 273, 967-976.

81. Laskin JD, Heck DE, Laskin DL. (1996). Nitric oxide production in the lung and liver following inhalation of the pulmonary irritant ozone. Advances Experimental Medicine Biology, 387,141-146

82. Yang J, Heck DE, DeGeorge G, Tian Y, Laskin JD. (1996). 5-Fluorouracil suppresses nitric oxide production in colon carinoma cells, Cancer Research, 56,1978-1982.

83. Laskin DL, Heck DE, Punjabi CJ, Laskin JD. (1996). Role of nitric oxide in hematosuppression and benzene-induced toxicity. Environmental Health Perspectives, 104,1283-1297

84. Pendino KJ, Gardner CR, Shuler RL, Laskin JD, Durham SK, Barton DS, Ohnishi S, Ohnishi T, Laskin DL. (1996). Inhibition of ozone-induced nitric oxide synthase expression in the lung by endotoxin. American Journal Respiratory Cell Molecular Biology, 14, 516-525

85. DeGeorge GL, Heck DE, Laskin JD. (1997). Arginine metabolism in keratinocytes and macrophages during nitric oxide production: multiple modes of action of nitric oxide synthase inhibitors. Biochemical Pharmacology, 54, 103-112.

86. Gardner CR, Heck DE, Yang CS, Thomas P, Laskin JD, Laskin DL. (1998). Role of nitric oxide in acetaminophen induced hepatotoxicity in the rat. Hepatology, 26, 748-754.

87. Weinberger B, Fakhrzadeh L, Heck DE, Laskin JD, Gardner CR, Laskin DL. (1998). Inhaled nitric oxide primes lung macrophages to produce reactive oxygen and reactive nitrogen intermediates. American Journal Respiratory Critical Care Medicine, 58, 931-938.

88. Laskin DL, Sunil V, Guo Y, Heck DE, Laskin JD. (1998). Increased nitric oxide synthase in the lung after ozone inhalation is associated with activation of NF-κB. Environmental Health Perspectives, 106,1175-1178

89. Jabin I, Heindel ND, Rapp RD, Laskin JD. (2000). Synthetic approaches to 3-substituted-5'-(N-pyridiniummethyl)-4',5'-dihydropsoralen. Journal Heterocyclic Chemistry, 36,1-9.

90. Heck DE, Louis L, Gallo MA, Laskin JD. (2000). Modulation of the development of plutei by nitric oxide in the sea urchin Arbacia punctulata. Biological Bulletin, 199, 195-197.

91. Laskin DL, Heck DE, Punjabi CJ, Laskin JD. (2000). Nitric oxide as a mediator of benzene-induced hematosuppression and toxicity. Journal Toxicology Environmental Health A, 61, 413-417.

92. Weinberger B, Laskin DL, Heck DE, Laskin JD. (2001). The toxicology of inhaled nitric oxide. Toxicological Sciences 59, 5-16.

93. Billack B, Heck DE, Porterfield DM, Malchow RP, Smith PJ, Gardner CR, Laskin DL, Laskin JD, (2001). Minimal amidine structure for inhibition of nitric oxide biosynthesis. Biochemical Pharmacology, 61, 1581-1586

94. Whittemore M, Heindel N, Guillon C, McNeel T, Rapp R, Mariano T, Heck DE, Laskin JD, (2001). Synthetic approaches to 4,8-dimethyl-4´-(N-pyridiniummethyl)-4´,5´-dihydropsoralens and their activity against PAM 212 keratinocytes. Heterocycles, 55, 1081-1093.

95. Whittemore MS, Heindel ND, Jabin I, Guillon C, McNeel TE, Rapp RD, Heck DE, Laskin JD, (2001). Synthetic approaches to 4,8-dimethyl-5'-(N-pyridiniummethyl)-4',5'-dihydropsoralens and 4,8-dimethyl-5'-(N-aminomethyl)-4',5'-dihydropsoralens, Journal Heterocyclic Chemistry, 38, 909-916.

96. Porterfield DM, Laskin JD, Malchow RP, Billack B, Smith PJS, Heck DE, (2001). Proteins and lipids define the diffusional field of nitric oxide. American Journal Physiology, 281, L904-912.

97. Cox, RL, Mariano TM, Heck DE, Laskin JD, Stegeman JJ, (2001). Nitric oxide synthase sequences in the marine fish *Stenotomus chrysops* and the sea urchin *Arbacia punctulata*, and phylogenetic analysis of nitric oxide synthase calmodulin-binding domains. Comparative Biochemistry Physiology B, 130, 479-491.

98. Heck DE, Roy A, Laskin JD (2001). Nucleic acid microarray technology for toxicology: promise and practicalities. Advances Experimental Medicine Biology, 500, 709-714.

99. Laskin DL, Fakhrzadeh L, Laskin JD (2001). Nitric oxide and peroxynitrite in ozone-induced lung injury. Advances Experimental Medicine Biology, 500, 183-190.

100. Weinberger B, Laskin DL, Mariano TM, Sunil VR, Heck DE, Gardner CR, Laskin JD. (2001). Mechanisms underlying reduced responsiveness of neonatal neutrophils to distinct chemoattractants. Journal Leukocyte Biology, 70, 969-976.

101. Mariano TM, Vetrano AM, Gentile SL, Heck DE, Whittemore MS, Guillon CD, Jabin I, Rapp RD, Heindel ND, Laskin JD. (2002). Cell-impermeant pyridinium derivatives of psoralens as inhibitors of keratinocyte growth. Biochemical Pharmacology, 63, 31-39.

102. Ahmad N, Laskin JD, Laskin DL. (2002). Regulation of cyclooxygenase-2 expression in hepatic macrophages by nitric oxide during acute endotoxemia. Journal Leukocyte Biology, 71, 1005-1011.

103. Billack B, Heck DE, Mariano TM, Gardner CR, Sur R, Laskin DL, Laskin JD. (2002). Induction of cyclooxygenase-2 by heat shock protein 60 in macrophages and endothelial cells. American Journal Physiology Cellular Physiology, 283, 1267-1277.

104. Fakhrzadeh L, Laskin JD, Laskin DL. (2002). Deficiency in inducible nitric oxide synthase (NOSII) protects mice from ozone induced lung injury. American Journal Respiratory Cell Molecular Biology, 26, 413-419.

105. Gardner CR, Laskin JD, Dambach D, Sacco M, Durham SK, Bruno, MK, Cohen S, Gordon MK, Gerecke DR, Zhou P, Laskin, DL. (2002). Reduced hepatotoxicity of acetaminophen in mice lacking inducible nitric oxide synthase. Role of TNFα and IL-10. Toxicolology Applied Pharmacology, 184, 27-36.

106. Laskin DL, Fakhrzadeh L, Heck DE, Gerecke D, Laskin JD. (2002). Upregulation of phosphoinositide 3-kinase and protein kinase B in alveolar macrophages following ozone inhalation. Role of NF-κB and STAT-1 in ozone-induced nitric oxide production and toxicity. Molecular Cellular Biochemistry, 234, 235:91-98.

107. Laskin JD, Heck DE, Laskin DL (2002). The ribotoxic stress response as a potential mechanism for MAP kinase activation in xenobiotic toxicity. Toxicological Sciences, 69, 289-291.

108. Mariano TM, Vetrano AM, Gentile SL, Heck DE, Whittemore MS, Guillon CD, Jabin I, Rapp RD, Heindel ND, Laskin JD. (2002). Cell-impermeant pyridinium derivatives of psoralens as inhibitors of keratinocyte growth. Biochemical Pharmacology, 63, 31-39.

109. Martey CA, Vetrano AM, Whittemore MS, Mariano TM, Gentile SL, Heck DE, Laskin DL, Heindel ND, Laskin JD. (2002). Mechanisms of growth inhibition in keratinocytes by mercurio-substituted 4',5'-dihydropsoralens. Biochemical Pharmacology 63, 2001-2009.

110. Sunil V, Connor A, Laskin JD, Laskin DL (2002). Activation of type II alveolar epithelial cells during acute endotoxemia. American Journal Physiology Lung Cell Molecular Physiology, 282, L872-L880.

111. Sunil V, Connor AJ, Lavnikova N, Gardner CR, Laskin JD, Laskin DL. (2002). Acute endotoxemia prolongs the survival of rat lung neutrophils in response to 12-O-tetra-decanoylphorbol 13-acetate. Journal Cellular Physiology, 190, 382-389.

112. Sunil V, Connor A, Laskin JD, Laskin DL (2002). Activation of adherent vascular neutrophils in the lung during acute endotoxemia. Respiratory Research, 3, 21-31.

113. Sur R, Heck DE, Mariano TM, Jin Y, Murphy WJ, Laskin JD. (2002). UVB light suppresses nitric oxide production by murine keratinocytes and macrophages. Biochemical Pharmacology, 64, 1469-1481.

114. Weinberger B, Laskin DL, Heck DE, Laskin JD. (2002). Oxygen toxicity in premature infants. Toxicology Applied Pharmacology, 181, 60-67.

115. Xu X, Mariano, TM, Laskin JD, Weisel CP. (2002). Percutaneous absorption of trihalomethanes, haloacetic acids, and haloketones. Toxicology Applied Pharmacology, 184:19-26.

116. Heck DE, Vetrano AM, Mariano TM, Laskin JD. (2003). UVB light stimulates production of reactive oxygen species: unexpected role for catalase. Journal Biological Chemistry, 278, 22432-22436.

117. Chiu H, Gardner CR, Dambach DM, Brittingham JA, Durham SK, Laskin JD, Laskin DL. (2003). Role of p55 tumor necrosis factor receptor 1 in acetaminophen- induced antioxidant defense. American Journal Physiology Gastrointestinal Liver Physiology, 285, G959-966.

118. Heck DE, Laskin JD. (2003). Ryanodine sensitive calcium flux regulates motility of arbacia punctulata sperm. Biological Bulletin, 205, 185-186.

119. Gardner CR, Laskin JD, Dambach D, Durham, SK, Bruno MK, Gordon MK, Zhou P, Laskin DL. (2003). Exaggerated hepatotoxicity of acetaminophen in mice lacking tumor necrosis factor-1 receptor. Potential role of inflammatory mediators. Toxicology Applied Pharmacology, 192,119-130.

120. Chiu H, Gardner CR, Dambach DM, Durham SK, Brittingham JA, Laskin JD, Laskin DL. (2003). Role of tumor necrosis factor receptor 1 (p55) in hepatocyte proliferation during acetaminophen-induced toxicity in mice. Toxicology Applied Pharmacology, 193, 218-227.

121. Fakhrzadeh L, Laskin JD, Gardner CR, Laskin DL. (2004). Superoxide dismutase overexpressing mice are resistant to ozone-induced tissue injury and increases in nitric oxide and TNF-α. American Journal Respiratory Cell Molecular Biology, 30, 280-287.

122. Vetrano AM, Heck DE, Mariano TM, Mishin V, Laskin DL, Laskin JD. (2005). Characterization of the oxidase activity in mammalian catalase. Journal Biological Chemistry, 280, 35372-35381.

123. Weinberger B, Hanna N, Laskin JD, Heck DE, Gardner CR, Gerecke DR, Laskin DL. (2005). Mechanisms mediating the biologic activity of synthetic proline, glycine, and hydroxyproline polypeptides in human neutrophils. Mediators Inflammation, 1, 31-38.

124. Hanna N, Vasquez P, Pham P, Heck DE, Laskin JD, Laskin DL, Weinberger B. (2005). Mechanisms underlying reduced apoptosis in neonatal neutrophils. Pediatric Research, 57, 56-62.

125. Fakhrzadeh L, Laskin JD, Laskin DL. (2004). Ozone-induced production of nitric oxide and TNF-alpha and tissue injury are dependent on NF-κB p50. American Journal Physiology Lung Cell Molecular Physiology, 287, L279-L285.

126. Martey CA, Vetrano AM, Whittemore MS, Mariano TM, Heck DE, Laskin DL, Heindel ND, Laskin JD. (2005). Inhibition of interferon-gamma signaling by a mercurio-substituted dihydropsoralen in murine keratinocytes. Biochemical Pharmacology, 70,1726-1734.

127. Dambach DM, Durham SK, Laskin JD, Laskin DL. (2006). Distinct roles of NF-κB p50 in the regulation of acetaminophen-induced inflammatory mediator production and hepatotoxicity. Toxicology Applied Pharmacology, 211, 157-165.

128. Gray JP, Mishin V, Smith PJS, Hong JY,Thiruchelvam M, Cory-Slechta DA, Heck DE, Laskin DL, Laskin JD. (2007). Paraquat increases cyanide-insensitive respiration in murine lung epithelial cells by activating a cytoplasmic NAD(P)H:paraquat oxidoreductase. Journal Biological Chemistry, 282, 7939-7949.

129. Weinberger B, Vetrano AM, Syed K, Murthy S, Hanna N, Laskin JD, Laskin DL. (2007). Influence of labor on neonatal neutrophil apoptosis, and inflammatory activity. Pediatric Research, 61, 572-577.

130. Hofmann PA, Israel M, Koseki Y, Laskin J, Gray J, Janik A, Sweatman TW, Lothstein L. (2007). N-Benzyladriamycin-14-valerate (AD 198): A non-cardiotoxic anthracycline that is cardioprotective through PKC-{epsilon} activation. Journal Pharmacology Experimental Therapeutics, 323, 658-664.

131. Sunil VR, Laumbach RJ, Patel KJ, Turpin BJ, Lim HJ, Kipen HM, Laskin JD, Laskin DL. (2007). Pulmonary effects of inhaled limonene ozone reaction products in elderly rats. Toxicology Applied Pharmacology, 222, 211-220.

132. Chen LC, Gordon RE, Laskin JD, Laskin DL. (2007). Role of TLR-4 in liver macrophage and endothelial cell responsiveness during acute endotoxemia. Experimental Molecular Pathology, 83, 311-326.

133 Sunil VR, Patel KJ, Nilsen-Hamilton M, Heck DE, Laskin JD, Laskin DL. (2007). Acute endotoxemia is associated with upregulation of lipocalin 24p3/Lcn2 in lung and liver. Exp Mol Pathol. 83, 177-187.

134 Black AT, Gray JP, Shakarjian MP, Laskin DL, Heck DE, Laskin JD. (2008). Increased oxidative stress and antioxidant expression in mouse keratinocytes following exposure to paraquat. Toxicology Applied Pharmacology, 231, 384-392.

135 Black AT, Gray JP, Shakarjian MP, Mishin V, Laskin DL, Heck DE, Laskin JD. (2008). UVB light

upregulates prostaglandin synthases and prostaglandin receptors in mouse keratinocytes. Toxicology Applied Pharmacology, 232, 14-24.

136 Weinberger B, Quizon C, Vetrano AM, Archer F, Laskin JD, Laskin DL. (2008). Mechanisms mediating reduced responsiveness of neonatal neutrophils to lipoxin A4. Pediatric Research, 64, 393-398.

137 Chen LC, Laskin JD, Gordon MK, Laskin DL (2008) Regulation of TREM expression in hepatic macrophages and endothelial cells during acute endotoxemia. Experimental Molecular Pathology, 84,145-155.

138 Fakhrzadeh L, Laskin JD, Laskin DL (2008) Regulation of caveolin-1 expression, nitric oxide production and tissue injury by tumor necrosis factor-alpha following ozone inhalation. Toxicology Applied Pharmacology, 227, 380-389.

139 Wang Y, Gray JP, Mishin V, Heck DE, Laskin DL, Laskin JD (2008) Role of cytochrome P450 reductase in nitrofurantoin-induced redox cycling and cytotoxicity. Free Radical Biology Medicine, 44, 1169-1179.

140. Black, AT, Gray JP, Shakarjian MP, Laskin DL, Heck DE, Laskin JD. (2008). Distinct effects of ultraviolet B light on antioxidant expression in undifferentiated and differentiated mouse keratinocytes. Carcinogenesis, 229, 219-225.

141. Sunil VR, Patel KJ, Mainelis G, Turpin BJ, Ridgely S, Laumbach RJ, Kipen HM, Nazarenko Y, Veleeparambil M, Gow AJ, Laskin JD, Laskin DL. (2009). Pulmonary effects of inhaled diesel exhaust in aged mice. Toxicology Applied Pharmacology, 241, 283-293.

142. Mishin V, Gray JP, Heck DE, Laskin DL, Laskin JD. (2010). Application of the Amplex red/horseradish peroxidase assay to measure hydrogen peroxide generation by recombinant microsomal enzymes. Free Radic Biol Med. 48(11):1485-1491.

143. Wang Y, Gray JP, Mishin V, Heck DE, Laskin DL, Laskin JD. (2010) Distinct roles of cytochrome P450 reductase in mitomycin c redox cycling and cytotoxicity, Molecular Cancer Therapeutics, 9:1852-1863.

144. Black AT, Joseph LB, Casillas RP, Heck DE, Sinko PJ, Gerecke DR, Laskin DL, Laskin JD. (2010) Role of MAP kinases in regulating expression of antioxidants and inflammatory mediators in mouse keratinocytes following exposure to the half mustard vesicant, 2-chloroethyl ethyl sulfide. Toxicol Appl Pharmacol. 245(3):352-360

145. Vetrano AM, Laskin DL, Archer F, Syed K, Gray JP, Laskin JD, Nwebube, N., Weinberger B. (2010) Inflammatory effects of phthalates in neonatal neutrophils: role of PPAR-γ. Pediatric Res., 68(2):134-139.

146. Gardner CR, Gray JP, Joseph LB, Cervelli J, Bremer N, Kim Y, Mishin V, Laskin JD, Laskin DL (2010) Potential role of caveolin-1 in acetaminophen-induced hepatotoxicity. Toxicol Appl Pharmacol. 245(1):36-46.

147. Black AT, Hayden PJ, Casillas RP, Heck DE, Gerecke DR, Sinko PJ, Laskin DL, Laskin JD (2010). Expression of proliferative and inflammatory markers in a full-thickness human skin equivalent following exposure to the model sulfur mustard vesicant, 2-chloroethyl ethyl sulfide, Toxicol Appl Pharmacol., 249(2):178-187.

148. Jan YH, Heck DE, Gray JP, Zheng H, Casillas RP, Laskin DL, Laskin JD. (2010) Selective targeting of selenocysteine in thioredoxin reductase by the half mustard 2-chloroethyl ethyl sulfide in lung epithelial cells. Chem Res Toxicol 23:1045-1053.

149. Young S, Fabio K, Guillon C, Mohanta P, Halton TA, Heck DE, Flowers RA 2nd, Laskin JD, Heindel ND. (2010). Peripheral site acetylcholinesterase inhibitors targeting both inflammation and cholinergic dysfunction. Bioorg Med Chem Lett. 20(9):2987-2990.

150. Malaviya R, Sunil VR, Cervelli J, Anderson DR, Holmes WW, Conti ML, Gordon RE, Laskin JD, Laskin DL (2010), Inflammatory effects of inhaled sulfur mustard in rat lungs. Toxicol Appl Pharmacol., 248(2):89-99.

151. Gray JP, Mishin V, Heck DE, Laskin DL, Laskin JD (2010) Inhibition of NADPH cytochrome P450 reductase by the model sulfur mustard vesicant 2-chloroethyl ethyl sulfide is associated with increased production of reactive oxygen species, Toxicol Appl Pharmacol., 247: 76-82.

152. Laskin DL, Chen L, Hankey PA, Laskin JD (2010) Role of STK in mouse liver macrophage and endothelial cell responsiveness during acute endotoxemia, J Leukoc Biol. 2010 May 7. [Epub ahead of print]

153. Fussell KC, Udasin RG, Gray JP, Mishin V, Smith PJ, Heck DE, Laskin JD. (2011) Redox cycling and increased oxygen utilization contribute to diquat-induced oxidative stress and cytotoxicity in Chinese hamster ovary cells overexpressing NADPH-cytochrome P450 reductase. Free Radic Biol Med. 50(7):874-882.

154. Young SC, Fabio KM, Huang MT, Saxena J, Harman MP, Guillon CD, Vetrano AM, Heck DE, Flowers RA, 2nd, Heindel ND, Laskin JD. (2011) Investigation of anticholinergic and non-steroidal anti-inflammatory prodrugs which reduce chemically induced skin inflammation. J Appl Toxicol. 2011 Feb 11. doi: 10.1002/jat.1645. [Epub ahead of print], PMID: 21319177

155. Sunil VR, Patel-Vayas K, Shen J, Gow AJ, Laskin JD, Laskin DL. (2011) Role of TNFR1 in lung injury and altered lung function induced by the model sulfur mustard vesicant, 2-chloroethyl ethyl sulfide. Toxicol Appl Pharmacol. 250(3):245-255. PMID: 21070800

156. Joseph LB, Gerecke DR, Heck DE, Black AT, Sinko PJ, Cervelli JA, Casillas RP, Babin MC, Laskin DL, Laskin JD. (2011) Structural changes in the skin of hairless mice following exposure to sulfur mustard correlate with inflammation and DNA damage. Exp Mol Pathol. 91(2):515-527.

157. Fussell KC, Udasin RG, Smith PJ, Gallo MA, Laskin JD. (2011) Catechol metabolites of endogenous estrogens induce redox cycling and generate reactive oxygen species in breast epithelial cells. Carcinogenesis. 32(8):1285-1293.

158. Dragomir AC, Laskin JD, Laskin DL. (2011) Macrophage activation by factors released from acetaminophen-injured hepatocytes: potential role of HMGB1. Toxicol Appl Pharmacol. 253(3):170-177.

159. Black AT, Hayden PJ, Casillas RP, Heck DE, Gerecke DR, Sinko PJ, Laskin DL, Laskin JD. Regulation of Hsp27 and Hsp70 expression in human and mouse skin construct models by caveolae following exposure to the model sulfur mustard vesicant, 2-chloroethyl ethyl sulfide. Toxicol Appl Pharmacol. 253(2):112-120.

160. Trabaris M, Laskin JD, Weisel CP. (2011) Percutaneous absorption of haloacetonitriles and chloral hydrate and simulated human exposures. J Appl Toxicol. 2011 Mar 1. doi: 10.1002/jat.1657. [Epub ahead of print]

161. Young SC, Fabio KM, Huang MT, Saxena J, Harman MP, Guillon CD, Vetrano AM, Heck DE, Flowers RA 2nd, Heindel ND, Laskin JD. (2011) Investigation of anticholinergic and non-steroidal anti-inflammatory prodrugs which reduce chemically induced skin inflammation. J Appl Toxicol. 2011 Feb 11. doi: 10.1002/jat.1645. [Epub ahead of print]

162. Black AT, Gordon MK, Heck DE, Gallo MA, Laskin DL, Laskin JD. (2011) UVB light regulates expression of antioxidants and inflammatory mediators in human corneal epithelial cells. Biochem Pharmacol. 81(7):873-880.

163. Sunil VR, Patel-Vayas K, Shen J, Gow AJ, Laskin JD, Laskin DL. (2011) Role of TNFR1 in lung injury and altered lung function induced by the model sulfur mustard vesicant, 2-chloroethyl ethyl sulfide. Toxicol Appl Pharmacol. 250(3):245-255.

164. Sunil VR, Patel KJ, Shen J, Reimer D, Gow AJ, Laskin JD, Laskin DL. (2011) Functional and inflammatory alterations in the lung following exposure of rats to nitrogen mustard. Toxicol Appl Pharmacol. 250(1):10-18.

165. Gardner CR, Hankey P, Mishin V, Francis M, Yu S, Laskin JD, Laskin DL. (2012) Regulation of alternative macrophage activation in the liver following acetaminophen intoxication by stem cell-derived tyrosine kinase. Toxicol Appl Pharmacol. 262(2):139-148.

166. Trabaris M, Laskin JD, Weisel CP. (2012) Effects of temperature, surfactants and skin location on the dermal penetration of haloacetonitriles and chloral hydrate. J Expo Sci Environ Epidemiol. 22(4):393-397.

167. Dragomir AC, Sun R, Mishin V, Hall LB, Laskin JD, Laskin DL (2012) Role of galectin-3 in acetaminophen-induced hepatotoxicity and inflammatory mediator production. Toxicol Sci. 127(2):609-619.

168. Sunil VR, Shen J, Patel-Vayas K, Gow AJ, Laskin JD, Laskin DL.(2012) Role of reactive nitrogen species generated via inducible nitric oxide synthase in vesicant-induced lung injury, inflammation and altered lung functioning. Toxicol Appl Pharmacol. 261(1):22-30.

169. Connor AJ, Laskin JD, Laskin DL. (2012) Ozone-induced lung injury and sterile inflammation. Role of toll-like receptor 4. Exp Mol Pathol. 92(2):229-235.

170. Gardner CR, Mishin V, Laskin JD, Laskin DL. (2012) Exacerbation of acetaminophen hepatotoxicity by the anthelmentic drug fenbendazole. Toxicol Sci. 125(2):607-612.

171. Sunil VR, Patel-Vayas K, Shen J, Laskin JD, Laskin DL. (2012) Classical and alternative macrophage activation in the lung following ozone-induced oxidative stress. Toxicol Appl Pharmacol. 2012 Jun 19. [Epub ahead of print]

BOOK CHAPTERS, REVIEWS AND OTHER PUBLICATIONS:

1. Weinstein IB, Mufson RA, Lee LS, Laskin JD, Horowitz A, Ivanovic V. (1980). Membrane and

other biochemical effects of the phorbol esters and their relevance to tumor promotion. In "Carcinogenesis: Fundamental Mechanisms and Environmental Effects", R. Reidel Publishing, New York.

2. Matthew E, Engelhardt DL, Laskin JD, Zimmerman EA (1980) Melanotropic effects of benzodiazepines: correlation with high-affinity receptors. Trans Am Neurol Assoc. 105:38-39.

3. Weinstein IB, Horowitz AD, Mufson RA, Fisher PB, Ivanovic V, Laskin J, Greenebaum E. (1981). Biochemical effects of the phorbol ester tumor promoters and their implications with respect to polycyclic aromatic hydrocarbon carcinogenesis. In "Polycyclic Hydrocarbons and Cancer" (H. Gelboin and P.O.P. Ts'O, eds.), Academic Press, New York.

4. Pietropaolo C, Laskin JD, Weinstein IB. (1982). Effect of tumor promoters on sarc gene expression in normal and transformed chick embryo fibroblasts 1982 Year Book of Cancer (R.L. Clark, Cumley, R.W. and Hickey, R.C., eds.), Year Book Medical Publishers, Inc., Chicago.

5. Hargraves RW, Geller HM, Laskin J, Patel-Vaidya U, Adinolfi AM, Freed WJ (1987) Transplantation of PC12 pheochromocytoma and B-16/C melanoma cells to the rat brain. Annals of the New York Academy of Sciences 495, pp. 715-717

6. Laskin JD, Laskin D. (1988). Role of psoralen receptors in cell growth regulation, in: Psoralen DNA Photobiology, F. Gasparro, ed., CRC Press, Boca Raton, FL, pp. 135-158.

7. Laskin JD. (1989). Keratinization and tumor promotion. J. Amer. Coll. Toxicology 8, 245-250.

8. Laskin JD, Dokidis A, Gardner CR, Laskin DL. (1989). Role of sulfated proteoglycans in Kupffer cell function. In: Cells of the Hepatic Sinusoids. Knook D and Wisse E, eds., Netherlands: Kupffer Cell Foundation, 83-84.

9. Laskin JD, (1989). Role of psoralen receptors in cell growth regulation. J. Amer. Coll. Toxicology, 8, 797.

10. Laskin JD (1989) Mechanisms of psoralen action in the skin. International J. Toxicology, 8: 797-800.

11. Laskin JD, Dokidas A, Gardner CR, Robertson FM, Laskin DL. (1989). Role of sulfated proteoglycans in Kupffer cell function. In: Cells of the Hepatic Sinusoid. Vol. 2. D. Knook and E. Wisse, Eds., Netherlands:Kupffer Cell Foundation, 83-84.

12. Gardner CR, Laskin JD, Faaland CA, Laskin DL. (1991). Role of protein kinases in liver macrophage function. In: Cells of the Hepatic Sinusoid, Vol. 3. E.Wisse, D.L. Knook, R.S. McCuskey, Eds., Netherlands: Kupffer Cell Foundation, 115-117.

13. Gardner CR, Heck DE, Feder LS, Mc Closkey TW, Laskin JD, Laskin DL. (1992). Differential regulation of reactive nitrogen and reactive oxygen intermediate production by hepatic macrophages and endothelial cells. In: The Molecular Basis of Oxidative Damage by Leukocytes. A. Jesaitis and E. Dratz, Eds., Florida, CRC Press, 267-272.

14. Huang MT, Robertson FM, Lysz T, Ferraro T, Wang ZY, Georgiadis CA, Laskin JD, Conney AH (1992) Inhibitory Effects of Curcumin on Carcinogenesis in Mouse Epidermis Phenolic Compounds in Food and Their Effects on Health II, Chapter 27, pp 338-349, ACS Symposium

Series, Volume 507.

15. Laskin JD, Heck DE, Laskin DL, Mitchell JM, Huang MT, Zhi Yuan Wang ZY, Yang CS, Ho CT, Conney AH (1992) Inhibitory Effect of a Green Tea Polyphenol Fraction on 12-O Tetradecanoylphorbol-13-acetate-Induced Hydrogen Peroxide Formation in Mouse Epidermis Phenolic Compounds in Food and Their Effects on Health II, Chapter 24, pp 308-314, ACS Symposium Series, Volume 507

16. Laskin JD (1992). Mechanisms of chemical photosensitization: Ability of photoactivated psoralens to interfere with epidermal growth factor-induced signal transduction pathways. Photochem. Photobiol., 55, 845.

17. Gardner CR, Laskin JD, Laskin DL. (1993). Platelet activating factor (PAF) induced activation of hepatic macrophages and endothelial cells: Role of intracellular calcium. Cells of the Hepatic Sinusoid, Vol. 4, E. Wisse, D.L. Knook, and R.S. McCuskey, eds., Kupffer Cell Foundation, Leiden. pp. 60-62.

18. Laskin DL, Heck DE, Feder LS, Gardner CR, Laskin JD. (1993). Regulation of nitric oxide production by hepatic macrophages and endothelial cells. Cells of the Hepatic Sinusoid, Vol. 4, D.L. Knook, and E. Wisse, eds., Kupffer Cell Foundation, Leiden. pp. 26-28.

19. Helyar L, Laskin JD, Thomas PE, Bundschuh DS, Laskin DL (1993). Modulation of cytochrome P450 expression in liver macrophages and endothelial cells. Cells of the Hepatic Sinusoid, Vol. 4, D.L. Knook, and E. Wisse, eds., Kupffer Cell Foundation, Leiden. pp. 394-396.

20. Helyar L, Bundschuh DS, Laskin JD, Laskin DL (1993). Hepatic fat storing cells produce nitric oxide and hydrogen peroxide in response to bacterially-derived lipopolysaccharide (LPS). Cells of the Hepatic Sinusoid, Vol. 4, D.L. Knook, and E. Wisse, eds., Kupffer Cell Foundation, Leiden. pp. 67-69.

21. Rodriguez del Valle M, Hwang SM, Heck DE, Laskin JD, Laskin DL. (1994) Role of nitric oxide in hepatic injury following acute endotoxemia. Ann N Y Acad Sci. 730:329-331.

22. Laskin JD, Heck DE, Laskin DL. (1994) Multifunctional role of nitric oxide in inflammation. Trends in Endocrinology and Metabolism, 5: 377-382.

23. Laskin JD, Heck DE, Xenobiotic-induced Skin Toxicity, in: Schook, L. and Laskin, D.L. (eds.). Xenobiotic-induced Inflammation. Academic Press, 1994.

24. Laskin JD, Laskin DL. Flow Cytometry. In: *In Vitro Toxicity Indicators*. C.A. Tyson and J.M. Frazier, Eds., Academic Press, N.Y., 431-437, 1994

25. Laskin DL, Heck DE, Gardner CR, Durham SK, Goller NL, Ohnishi ST, Laskin JD. 1995. Enhanced production of nitric oxide by hepatic macrophages (MP), endothelial cells (EC) and fat storing cells (FSC) following acute endotoxemia. Cells of the Hepatic Sinusoid, Vol. 5, E. Wisse, D.L. Knook, and K. Wake, eds., Kupffer Cell Foundation, Leiden. pp. 53-55.

26. Shuler RL, Laskin JD. Natural carcinogens and anticarcinogens, in: Environmental Medicine (eds, S., Brooks, Gochfeld, M., Herzstein, J., Schenker, M., and Jackson, R.), Mosby, St Louis, 1995, pp. 512-514.

27. Laskin DL, Laskin JD (1996) Macrophages, Inflammatory Mediators, and Lung Injury. Methods

10(1):61-70

28. Gardner CR, Durham SK, Barton D, Heck DE, Laskin JD, Laskin DL. (1997) Role of nitric oxide in hepatotoxicity. Cells of the Hepatic Sinusoid, Vol. 6, E. Wisse, D.L. Knook, and C. Balabaud, eds., Kupffer Cell Foundation, Leiden. pp. 268-271.

29. Laskin DL, Laskin JD. (1997) Phagocytes. In: Comprehensive Toxicology, Vol. 5, Toxicology of the Immune System. D.A. Lawrence, ed., Pergamon, N.Y., pp. 97-112.

30. Gardner CR, Heck DE, Chiu H, Laskin JD, Durham SK, Laskin DL. 1999. Decreased hepatotoxicity of acetaminophen in mice lacking inducible nitric oxide synthase. Cells of the Hepatic Sinusoid, Vol. 7, E. Wisse, D.L. Knook, R. DeZanger, and R. Fraser, eds., Kupffer Cell Foundation, Leiden. pp. 104-105.

31. Laskin JD, Mariano TM (1999). Nitric oxide as a cellular mediator of apoptosis. in: Laskin, J.D. and Laskin, D.L. (eds.). Cell and Molecular Biology of Nitric Oxide. Marcel Dekker, Inc., NY, pp. 269-292.

32. Weinberger B, Heck DE, Laskin DL, Laskin JD. (1999) Nitric oxide in the lung: therapeutics and cellular mechanisms of action, Pharmacol. Therapeutics, 84:401-411.

33. Laskin DL, Fakhrzadeh L, Laskin JD (2001). Nitric oxide and peroxynitrite in ozone-induced lung injury, In: Biological Reactive Intermediates VI, Chemical and Biological Mechanisms of Susceptibility to and Prevention of Environmental Diseases, P. Dansette, R. Snyder, M. Delaforge, G. Gibson, H. Greim, D. Jollow, T. Monks, and I. Sipes, eds., New York: Plenum Press, 183-190.

34. Laskin DL, Weinberger B, Laskin JD. (2001) Functional heterogeneity in liver and lung macrophages. J. Leukoc. Biol. 70:163-170

35. Laskin JD, Heck DE, Gardner CR, Laskin DL. (2001). Pro-oxidant and antioxidant functions of nitric oxide in liver toxicity. Antioxidants and Redox Signaling, 3:261-271.

36. Laskin DL, Laskin JD. (2001) Role of macrophages and inflammatory mediators in tissue injury, Toxicology, 160: 111-118.

37. Kagan VE, Laskin JD. (2001) Direct and indirect antioxidant effects of nitric oxide: Radically unsettled issues. Antioxidants and Redox Signaling 3(2):173-175

38. Weinberger B, Hanna N, Gropper CA., Heck DE, Laskin DL, Laskin JD. (2003) Transdermal xenobiotics in newborn skin. J. Toxicol. Cutaneous Ocular Toxicol. 22:51-67.

39. Laskin DL, Gardner CR, Gerecke DR, Laskin JD. (2003). Ozone-induced lung injury: role of macrophages and inflammatory mediators. In: Reactive Oxygen/Nitrogen Species: Lung Injury and Disease. V. Vallyathan, X. Shi, and V. Castranova, eds., New York: Marcel Dekker. pp. 289-316

40. Laskin DL, Fakhrzadeh L, Gerecke D, Heck DE, Laskin JD. (2003) Macrophages and inflammatory mediators in chemically-induced toxicity. Journal UOEH 25: 191-195.

41. Heck DE, Gerecke D, Vetrano A, Laskin JD (2004) Solar ultraviolet radiation as a trigger for cell signal transduction. Toxicol. Appl. Pharmacol., 195(3):288-297.

42. Heck DE, Kagan VE, Shvedova AA, Laskin JD (2005) An epigrammatic (abridged) recounting of the myriad tales of astonishing deeds and dire consequences pertaining to nitric oxide and reactive oxygen species in mitochondria with an ancillary missive concerning the origins of apoptosis. Toxicology. 208(2):259-271.

43. Gerecke DR, Sabourin CLK, Chang YC, Laskin JD, Gordon MK, Casillas, RP (2008) Gene expression profiles of vesicant-induced skin injury; NATO conference on "Defense Against the Effects of Chemical Hazards', Edinburgh, Scotland.

44. Shakarjian MP, Heck DE, Gray JP, Sinko PJ, Gordon MK, Casillas RP, Heindel ND, Gerecke DR, Laskin DL, Laskin JD (2010) Mechanisms mediating the vesicant actions of sulfur mustard after cutaneous exposure; Toxicol Sci. 114(1):5-19

.

45. Laskin JD, Heck DE, Laskin DL (2010) Nitric oxide pathways in toxic responses, General and Applied Toxicology, third edition. B. Ballantyne, T. Marrs, T. Syversen, eds. UK: Wiley-Blackwell, Chapter 17.

46. Laskin DL, Laskin JD (2010) Inflammation and Cancer, Holland-Frei's- Cancer Medicine, 8th edition, W.K. Hong, R.C. Bast, W.N. Hait, D.W. Kufe, R.E. Pollock, R.R. Weichselbaum, J.F. Holland, E. Frei, eds., New York: PMPH Publishing House, chapter 19, pp. 270-278.

47. Laskin DL, Gardner CR, Laskin JD, (2010) "Phagocytes", in: Comprehensive Toxicology, Volume 5, Toxicology of the Immune System. C. McQueen, ed., Elsevier, in press.

48. Laskin JD, Black AT, Jan YH, Sinko PJ, Heindel ND, Sunil V, Heck DE, Laskin DE, (2010) Oxidants and antioxidants in sulfur mustard-induced injury (2010) Ann N Y Acad Sci. 1203:92-100.

49. Heck DE, Shakarjian M, Kim HD, Laskin JD, Vetrano AM. (2010) Mechanisms of oxidant generation by catalase. Ann N Y Acad Sci. 1203:120-125.

49. Weinberger B, Laskin JD, Sunil VR, Sinko PJ, Heck DE, Laskin DL. (2011) Sulfur mustard-induced pulmonary injury: Therapeutic approaches to mitigating toxicity. Pulm Pharmacol Ther. 24(1):92-99.

50. Laskin DL, Sunil VR, Gardner CR, Laskin JD. (2011) Macrophages and tissue injury: agents of defense or destruction? Annu Rev Pharmacol Toxicol. 51:267-288.

BOOKS:

1. Laskin, J.D. and Laskin, D.L. (eds.). Cell and Molecular Biology of Nitric Oxide. Marcel Dekker, Inc., NY, 1999

Exhibit 2

Abolghasemi, H., Radfar, M., Rambod, M., Salehi, P., Ghofrani, H., Soroush, M., Falahaty, F., Tavakolifar, Y., Sadaghianifar, A., Khademolhosseini, S., Kavehmanesh, Z., Joffres, M., Burkle, F., Mills, E. (2010). Childhood physical abnormalities following paternal exposure to sulfur mustard gas in Iran: a case control study. Conflict and Health 4:13. (PLTF 014900-05).

Amirzargar, M.A., Yavangi, M., Rahnavardi, M., Jafaris, M., Mohseni, M. (2009). Chronic mustard toxicity on the testis: a historical cohort study two decades after exposure. Int J Androl. 32:411-416. (PLTF 014815-21).

Asuku, M.E., Milner, S.M., Gerold, K.B. (2011). Beyond tears: the potential hazards of the o-chlorobenzylidene-malononitrile (CS) gas under scrutiny. J Spec Oper Med. 11(4):28-30.

Ballantyne, B., Swanston, D.W. (1978). The comparative acute mammalian toxicity of 1-chloroacetophenone (CN) and 2-chlorobenzylidene malononitrile (CS). Arch Toxicol 40:75-95.

Ballantyne, B. (1979). Evaluation of ophthalmic hazards from an aerosol generator of 2-chlorobenzylidene malononitrile (CS) Mil Med. 144(10):691-694.

Bijani, K., Moghadamnia, A.A., (2002). Long term effects of chemical weapons on respiratory tract in Iraq-Iran war victims living in Babol (North of Iran). Exotoxicology & Environmental Safety 53:422-424.

Black, A.T., Hayden, P.J., Casillas, R.P., Heck, D.E., Gerecke, D.R., Sinko, P.J., Laskin, D.L., Laskin, J.D. (2010). Expression of proliferative and inflammatory markers in a full-thickness human skin equivalent following exposure to the model sulfur mustard vesicant, 2-chloroethyl ethyl sulfide. Toxicol Appl Pharmacol. 249:178-187.

Blain, P.G. (2003). Tear gases and irritant incapacitants. 1-chloroacetophenone, 2-chlorobenzylidene malononitrile and dibenz[b,f]-1,4-oxazepine. Toxicol Rev. 22(2):103-110.

Brooks, S.M., Weiss, M.A., Bernstein, I.L. (1985a) Reactive airways dysfunction syndrome (RADS). Persistent asthma syndrome after high level irritant exposures. Chest. 88(3):376-384.

Brooks, S.M., Weiss, M.A., Bernstein, I.L. (1985b). Reactive airways dysfunction syndrome. Case reports of persistent airways hyperreactivity following high-level irritant exposures. J Occup Med. 27(7):473-476.

Brown, M. (2009). Military Chemical Warfare Agent Human Subjects Testing: Part 1—History of Six-Decades of Military Experiments With Chemical Warfare Agents. 174(10):1041-1048(8).

Brown, M. (2009). Military Chemical Warfare Agent Human Subjects Testing: Part 2--Long-Term Health Effects among Participants of U.S. Military Chemical Warfare Agent Testing. Mil Med. 174(10):1049-1054.

CDC, NIOSH, CHLOROACETOPHENONE (CN): Riot Control/Tear Agent, http://www.cdc.gov/NIOSH/ershdb/EmergencyResponseCard_29750033.html

CDC, NIOSH, Documentation for Immediately Dangerous To Life or Health Concentrations (IDLHs), alpha-Chloroacetophenone, http://www.cdc.gov/niosh/idlh/532274.html

CDC, NIOSH, The Emergency Response Safety and Health Database: Vomiting Agent: Adamsite http://www.cdc.gov/niosh/ershdb/EmergencyResponseCard_29750017.html

Chart of Chemical Dosage Ranges – Chem-Bio Database.

Cleanup of Chemical Explosives and Munitions. Location, Identification and Environmental Remediation, 2nd edition, Elsevier. (2012).

Croddy, E., Wirtz, J., Larsen, J., eds. (2005). Weapons of Mass Destruction: An Encyclopedia of Worldwide Policy, Volume 2.

Doi, M., Hattori, N., Yokoyama, A., Onari, Y., Kanehara, M., Masuda, K., Tonda, T., Ohtaki, M., Kohno, N. (2011). Effect of mustard gas exposure on incidence of lung cancer: a longitudinal study. Am J Epidemiol. 173:659-666. (PLTF 014914-24).

Department of Veterans Affairs. Mustard Gas Outreach Letter. (VET001_015113-15). (**Deposition Exhibit 767)**

Department of Veterans Affairs. Outreach Letter Spreadsheet. (DVA006 104420-839).

Department of Veterans Affairs. Mustard Gas Training Letter 06-04. (2006). (VET001_015121-28). (**Deposition Exhibit 296)**

Department of Veterans Affairs. Chem-Bio Outreach Letter. (VET001_015129-34). (**Deposition Exhibit 296)**

Department of Veterans Affairs. Under Secretary for Health Information Letter (Clinician's Letter). (2006). (VET001_015606-9). (**Deposition Exhibit 275)**

Department of Veterans Affairs. Under Secretary for Health Information Letter (Clinician's Letter). (DVA012 000269-71). (**Deposition Exhibit 766)**

Disposition Form re Volunteer Testing (1969). (JK01 0000213-14).

Email from Mark A. Brown to Kenneth Craig Hyams and others, June 29, 2006, Re: EDMS 352753 – Edgewood Arsenal Notification Letter – Expedite. (DVA052 000113-14).

)

Gerecke, D., Gray, J., Shakarijian, M., Casillas, R. (2009). Chapter 41: Dermal Toxicity of Sulfur Mustard. Handbook of Toxicology of Chemical Warfare Agents, Elsevier Inc., 611-630 (PLTF 014842-61).

Ghanei, M., Harandi, A.A. (2007). Long term consequences from exposure to sulfur mustard: a review. Inhalation Tox. 19:451-456. (PLTF 014787-93).

Ghassemi-Broumand, M., Aslani, J., Emadi, S. (2008). Delayed Ocular, Pulmonary, and Cutaneous Complications of Mustards in Patients in the City of Sardasht, Iran. Cutan Ocul Toxicol. 27:295-305.

Gordon, M., Enzenauer, R.W., Babin, M.C., (2009). Chapter 39: Ocular Toxicity of Sulfur Mustard. Handbook of Toxicology of Chemical Warfare Agents, Elsevier Inc., 575-594. (PLTF 014822-41).

Health Effects from Chemical, Biological and Radiological Weapons. Department of Veterans Affairs. (2003). 1-84. (VET001_009384-9479). (**Deposition Exhibit 333)**

Hellreich, A., Mershon, M., Weimer, J., Kyso, K., Bottiglieri, N. (1969). Edgewood Arsenal Technical Report EATR 4252 An Evaluation of the Irritant Potential of CS Aerosols on Human Skin Under Tropical Climatic Conditions. 1-31.

Horton, D.K., Burgess, P., Rossiter, S., Kaye, W.E. (2005). Secondary contamination of emergency department personnel from o-chlorobenzylidene malononitrile exposure. Ann Emerg Med. 45(6):655-658.

Joseph, L.B., Gerecke, D.R., Heck, D.E., Black, A.T., Sinko, P.J., Cervelli, J.A., Casillas, R.P., Babin , M.C., Laskin, D.L., Laskin, J.D. (2011). Structural changes in the skin of hairless mice following exposure to sulfur mustard correlate with inflammation and DNA damage. Exp Mol Pathol. 91:515-527.

Jowsey P.A., Williams F.M., Blain P.G. DNA damage responses in cells exposed to sulphur mustard. (2012). Toxicol Lett. 209:1-10.

Kehe, K., Szinicz, L. (2005). Medical aspects of sulphur poisoning. Toxicology 214:198-209.

Kehe, K., Balszuweit, F., Steinritz, D., Thiermann, H. (2009). Molecular toxicology of sulfur mustard-induced cutaneous inflammation and blistering. Toxicology, 26:12-19.

Kehe, K., Thiermann, H., Balszuweit, F., Eyer, F., Steinritz, D., Zilker, T. (2009). Acute effects of sulfur mustard injury- Munich experiences, Toxicology 263:3-8.

Ketchum, James S. Chemical Warfare, Secrets Almost Forgotten (2006). Appendix. (JK24 0028935-355). (**Deposition Exhibit 27)**

McNamara, B.P., Owens, E.J., Weimer, J.T., Ballard, T.A., Vocci, F.J. (1969). Edgewood Arsenal Technical Report EATR 4309 Toxicology of Riot Control Agents—CS, CN, and DM. 1-82.

McNutt, P., Hamilton, T., Nelson, M., Adkins, A., Swartz, A., Lawrence, R., Milhorn, D. (2012). Pathogenesis of acute and delayed corneal lesions after ocular exposure to sulfur mustard vapor. Cornea, 0:1-11.

Namazi, S., Niknahad, H., Razmkhah, H. (2009) Long-term complications of sulphur mustard poisoning in intoxicated Iranian veterans. J Med Toxicol. 5:191-195. (PLTF 014895-99).

Naraghi, Z.S., Mansouri, P., Mortazavi, M., (2005). A clinicopathological study on acute cutaneous lesions induced by sulfur mustard gas (Yperite). Eur J Dermatol. 15:140-145.

Oberst, F., Musselman, N., Graf, C., Trapp, G., Dawson, P. (1965). DTIC Report 462053, Protection Afforded By Experimental XXCC3-Impregnated Navy Work/Combat Clothing Worn By Men Exposed to Mustard Vapor. 1-25.

Olajos, E.J, Salem, H. (2001). Riot control agents: pharmacology, toxicology, biochemistry and chemistry. J Appl Toxicol. 21(5):355-391.

Owens, E.J., Punte, C.L. (1963). Human respiratory and ocular irritation studies utilizing o-chlorobenzylidene malononitrile aerosols. Am Ind Hyg Assoc J. 24:262-264.

Pechura, C., Rall, D., eds. (1993). Veterans at Risk: The Health Effects of Mustard Gas and Lewisite, National Academy Press. (PLTF 002954-3399).

Pleyer, U., Sherif, Z., Baatz, H., Hartmann, C. (1999). Delayed mustard gas keratopathy: clinical findings and confocal microscopy. Am J Ophthalmol. 128:506-507.

Pohanish, R.P. Sittig's Handbook of Toxic and Hazardous Chemicals and Carcinogens, Elsevier, 6th Edition, 2012, Adamsite:72-75.

Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents, Volume 1, National Academy Press. (1982). (PLTF 014140-430)

Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents, Volume 2, National Academy Press. (1984). (PLTF 014431-778)

Possible Long-Term Health Effects of Short-Term Exposure to Chemical Agents, Volume 3: Final Report: Current Health Status of Test Subjects, National Academy Press. (1985). (VET013-004999-5104)

Poursaleh, Z., Ghanei, M., Babamahmoodi, F., Izadi, M., Harandi, A.A., Emadi, S.E., Taghavi, N.O., Sayad-Nouri, S.S., Emadi, S.N. (2011). Pathogenesis and treatment of skin lesions caused by sulfur mustard. Cutan Ocul Toxicol. 1-9.

Punte, C.L., Ballard, T., Weimer, J.T. (1962). Inhalation studies with chloracetophenone, diphenylaminochloroarsine and pelargonic morpholide-I. Animal exposures. Am Ind Hyg Assoc J. 23:194-198.

Punte, C.L., Gutentag, P.J., Owens, E.J., Gongwer, L.E. (1962). Inhalation studies with chloracetophenone, diphenylaminochloroarsine and pelargonic morpholide-II. Human exposures. Am Ind Hyg Assoc J. 23:199-202.

Punte, C.L., Owens, E.J., Gutent, P.J. (1963). Exposures to orthochlorobenzylidene malononitrile. Controlled human exposures. Arch Environ Health. 6:366-374.

Punte, C.L., Weimer, J.T., Ballard, T.A., Wilding, J.L. (1962). Toxicologic studies on o-chlorobenzylidene malononitrile. Toxicol Appl Pharmacol. 4:656-662.

Rengstorff, R. (1968). Edgewood Arsenal Technical Report EATR 4246 The Effects of the Riot Control Agent CS on Visual Acuity. 1-13.

Rengstorff, R., Mershon, M. (1969). Edgewood Arsenal Technical Report EATR 4377 CS in Water: Effects on Human Eyes. 1-15.

Review of the Army Non-stockpile Chemical Materiel Disposal Program, Chemical Agent Identification Set, National Research Council, National Academies Press. (1999).

Rice, L.M., Grogan, C.H., Armbrecht, B.H., Reid E.E. (1954). Pungents. Fatty Acid Amides. J Am Chem Soc. 76:3730-3731.

Ricketts, K.M., Santai, C.T., France, J.A., Graziosi, A.M., Doyel, T.D., Gazaway, M.Y., Casillas, R.P. (2000). Inflammatory cytokine response in sulfur mustard-exposed mouse skin. J Appl Toxicol.; 20 Suppl 1:S73-S6.

Rowell, M., Kehe, K., Balszuweit, F., Thierman, H. (2009). The chronic effects of sulfur mustard exposure. Toxicology. 263:9-11. (PLTF 014862-65).

Sabourin, C.L., Petrali, J.P., Casillas, R.P. (2000). Alterations in inflammatory cytokine gene expression in sulfur mustard-exposed mouse skin. J Biochem Mol Toxicol. 14:291-302.

Sabourin, C.L., Danne, M.M., Buxton, K.L., Casillas, R.P., Schlager, J.J. (2002). Cytokine, chemokine, and matrix metalloproteinase response after sulfur mustard injury to weanling pig skin. J Biochem Mol Toxicol. 16:263-272.

Shakarjian, M., Heck, D., Gray, J., Sinko, P., Gordon, M., Casillas, R., Heindel, N., Gerecke, D., Laskin, D., Laskin, J. (2010). Mechanism mediating the vesicant actions of sulfur mustard after cutaneous exposure, Tox. Sci. 114(1) 5-19. (PLTF 014880-94).

Shakarjian, M.P., Bhatt, P., Gordon, M.K., Chang, Y.C., Casbohm, S.L., Rudge, T.L., Kiser, R.C., Sabourin, C.L., Casillas, R.P., Ohman-Strickland, P., Riley, D.J., Gerecke, D.R. (2006). Preferential expression of matrix metalloproteinase-9 in mouse skin after sulfur mustard exposure. J Appl Toxicol. 26:239-246.

Sidell, F.R., Urbanetti, J.S., Smith, W.J., Hurst, C.G. Chapter 7: Vesicants. Medical Aspects of Chemical and Biological Warfare – Textbook of Military Medicine. Office of the Surgeon General, 1997. (VET004_001133-67).

Sidell, F.R., Hurst, C.G. Medical Aspects of Chemical and Biological Warfare – Textbook of Military Medicine. Chapter 8: Longterm Health Effects of Nerve Agents and Mustard. Office of the Surgeon General, 1997. (VET004_001168-87).

Sohrabour, H. (1989) First World Congress on Biological and Chemical Warfare Agents, Belgium. 291-297.

Tsuruta, J., Sugisaki, K., Dannenberg, A.M., Yoshimura, T., Yasuharu, A., Mounts, P. (1996). The cytokines NAP-1 (IL-8), MCP-1, IL-1 beta, and GRO in rabbit inflammatory skin lesions produced by the chemical irritant sulfur mustard. Inflammation. 20:293-318.

Weimer, J.T., Owens, E.J., Ballard, T.A., Ferrell, J.F., Everts, J.S., Averill, H.P., McNamara, B.P. (1969). Edgewood Arsenal Technical Report EATR 4301 Toxicity of o-Chlorobenzylidene Malonitrile (CS) in Trioctylphosphate (TOF) Solutions. 1-50.

Weinberger, B., Laskin, J., Sunil, V., Sinko, P., Heck, D., Laskin, D. (2011). Sulfur mustard-induced pulmonary injury: therapeutic approaches to mitigating toxicity. Pulmonary Pharm. & Therapeutics 24:92-99. (PLTF 014906-13).

Witten, B. (1969). The Search for Toxic Chemical Agents. U.S. Army Chemical Research Laboratory, Edgewood Arsenal, Md. Technical Report, EATR 4210. [Confidential Report]

Wormser, U., Langenbach, R., Peddada, S., Sintov, A., Brodsky, B., Nyska, A. (2004). Reduced sulfur mustard-induced skin toxicity in cyclooxygenase-2 knockout and celecoxib-treated mice. Toxicol Appl Pharmacol. 200:40-47.

Young, S.C., Fabio, K.M., Huang, M.T., Saxena, J., Harman, M.P., Guillon, C.D., Vetrano, A.M., Heck, D.E., Flowers, R.A. II, Heindel, N.D., Laskin, J.D. (2012). Investigation of anticholinergic and non-steroidal anti-inflammatory prodrugs which reduce chemically induced skin inflammation. J Appl Toxicol. 32:135-41.

Zhang, Z., Peters, B.P., Monteiro-Riviere, N.A., Assessment of sulfur mustard interaction with basement membrane components. (1995). Cell Biol. Toxicol., 11, 89-101.