United States
CONSUMER PRODUCT SAFETY COMMISSION
Washington, D.C. 20207

VOTE SHEET

Date: OCT - 6 2005

TO : The Commission
Todd A. Stevenson, Secretary

FROM : Page C. Faulk, General Counsel PCF
Lowell F. Martin, Assistant General Counsel L.F.M.
Hyun S. Kim, Attorney HSK

SUBJECT : Petition HP 04-2 Request to Ban Sulfuric Acid Drain Openers for Consumer Use

Attached is a briefing package from the staff concerning a petition submitted by Dr. Michael Fox. The petition requests that the Commission issue a ban on sulfuric acid drain openers or, in the alternative, require packaging of sulfuric acid in single use containers with a maximum sulfuric acid concentration of 84 percent. The staff recommends that the Commission deny the petition.

Please indicate your vote on the following options.

I. Grant Petition HP 04-2 and direct the staff to begin developing a draft advance notice of proposed rulemaking.

Signature ______________________ Date ______________________

II. Deny Petition HP 04-2 and direct the staff to prepare a letter of denial to the petitioners.

Signature ______________________ Date ______________________

CPSA 6(b)(1) CLEARED for PUBLIC
NO MFRS/PRVTLBLRS OR PRODUCTS IDENTIFIED 10/6/05
EXCEPTED BY: PETITION RULEMAKING ADMIN. PRO'G

NOTE: This document has not been reviewed or accepted by the Commission.
Initial [illegible] Date 10/6/05

III. Defer decision on HP 04-2.

Signature

Date

IV. Take other action (please specify):

Signature

Date

Briefing Package for Petition Requesting Ban of Sulfuric Acid Drain Openers for Consumer Use (Petition No. HP 04-2)

For Information Contact:

Kristina Hatlelid, Ph.D., M.P.H.
Directorate for Health Sciences
(301) 504-7254

TABLE OF CONTENTS

Executive Summary

The U.S. Consumer Product Safety Commission (CPSC) received a request from Michael Fox, Ph.D. that the Commission ban sulfuric acid drain openers (SADOs) for use by consumers, or in the alternative, that the Commission require SADOs for consumers be packaged in single-use containers and be limited to a maximum sulfuric acid concentration of 84 percent. This request was docketed under the Federal Hazardous Substances Act (FHSA) as Petition No. HP 04-2 on 1 April 2004, and a Federal Register notice soliciting comments was published 5 May 2004 (69 FR 25069).

Chemical drain opener products containing concentrated sulfuric acid have the potential to cause severe injuries from dermal or ocular exposure, inhalation of fumes, or ingestion. However, the possible chemical substitutes for sulfuric acid-based products, such as hydrochloric acid or formulations containing sodium or potassium hydroxide, especially products containing higher concentrations of hydroxide, also have the potential to cause severe injuries. Injury and exposure data show that each type of chemical drain opener is associated with a risk of injury, with medical outcomes ranging from no effect to major effect. There are also reports of fatalities from exposure to chemical drain opener products, including a child who ingested an alkaline-based product; another case involved exposure to fumes from use of a combination of a sulfuric-acid based product and an alkaline product.

Analysis of some specific incidents shows that exposures to a wide variety of chemical drain opener products occurred through a variety of situations. Prompt treatment (*i.e.*, washing with water and seeking medical attention) can greatly reduce the severity of injury from chemical drain opener exposures, and these cases also showed that in many instances, consumers responded appropriately to exposure, such as by initiating washing or removing the victim from the exposure, and by seeking medical care.

The number of sulfuric acid drain opener exposures, as the percentage of exposures with known product type, is consistent with the estimated market share for these products (*i.e.*, sulfuric acid-based products account for 3-10 percent of chemical drain opener treatments used by consumers, and sulfuric acid drain openers are associated with about 6-10 percent of incidents).

If SADOS were banned, the staff believes that consumers are likely to substitute other chemical drain openers for SADOs. Therefore, any reduction in societal costs associated with all drain openers might be low. This might be especially true if consumers substituted the higher concentration alkaline products for SADOs. Effectively, a ban on SADOs might simply shift much of the societal costs now associated with SADOs to other chemical drain openers.

Thus, the staff believes that the available information does not support a conclusion that a ban of these products is necessary to protect public health and safety. Further, the staff does not believe that restrictions on package size or product formulation would necessarily reduce or eliminate the risk or severity of injury since exposure to a relatively small volume of a sulfuric acid product or to a product containing 84 percent sulfuric acid would likely still require prompt washing and treatment in order to lessen the risk and severity of injury. Therefore, the staff recommends that the Commission deny the petition.

UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, DC 20207

Memorandum

Date: OCT - 6 2005

TO : The Commission
Todd A. Stevenson, Secretary

THROUGH: Page C. Faulk, General Counsel
THROUGH: Patricia Semple, Executive Director

FROM : Jacqueline Elder, Assistant Executive Director, Office of Hazard Identification and Reduction
Kristina M. Hatlelid, Ph.D., M.P.H., Toxicologist, Directorate for Health Sciences

SUBJECT : Petition HP 04-2 Request to Ban Sulfuric Acid Drain Openers for Consumer Use

This briefing package presents the staff's analysis of the petition requesting a ban of sulfuric acid drain openers for consumer use and associated data, and provides a summary of comments received in response to the notice published in the Federal Register (69 FR 25069) and the staff responses to the comments.

Petition HP 04-2

On 26 February 2004, the U.S. Consumer Product Safety Commission (CPSC) received a request from Michael Fox, Ph.D. that the Commission ban sulfuric acid drain openers (SADOs) for use by consumers, or in the alternative, that the Commission require SADOs for consumers be packaged in single-use containers and be limited to a maximum sulfuric acid concentration of 84 percent (Tab A). This request was docketed under the Federal Hazardous Substances Act (FHSA) as Petition No. HP 04-2 on 1 April 2004.

Dr. Fox is a consultant and expert witness in chemical-related accidents. He investigated the case of a child who experienced severe burns to the skin of his upper body and head when a sulfuric acid drain opener product fell from a shelf and spilled on him. He states that SADOs are "unreasonably dangerous," and "can cause horrific injuries and are unsafe when used in a reasonable and foreseeable manner by ordinary consumers." He states that SADOs should only be sold to plumbing professionals and should not be sold to ordinary consumers. Dr. Fox believes that previous Commission decisions not to ban SADOs were based on flawed data and analyses. He states that SADOs are more dangerous than other types of chemical drain opener products, and that no label exists that could "properly instruct and warn the ordinary consumer about the dangers" they present. Dr. Fox also raised the issue that he refers to as the "Kleenex effect," *i.e.*, the phenomenon in which some people may tend to identify products of a certain type by one or more well-known brand names regardless of the actual product brand. In this case, Dr. Fox stated that incidents involving a sulfuric acid-based product might be reported as "Liquid Plumber" [sic], which is similar to the name of a widely available alkaline-based drain opener. He claims that this phenomenon affects the accuracy of the incident reports and data by under-reporting SADO cases and inaccurately suggesting that some injuries were caused by alkaline products when, in fact, they resulted from SADO exposure.

Background

Drain Opener Products

Sulfuric acid drain openers generally contain a high concentration of sulfuric acid (about 84-94 percent), as well as other ingredients such as corrosion inhibitors, and have a pH of 1 or less. Other chemical drain opener types are hydrochloric acid (5 to 30 percent; pH less than 1), and liquid or granular alkaline products. The alkaline drain openers contain sodium or potassium hydroxide *(i.e.*, alkalis) and other ingredients such as sodium hypochlorite. The concentrations of the hydroxides in the granular drain openers range from 60 to 100 percent, whereas the sodium hydroxide or potassium hydroxide in liquid alkaline drain openers can range from about 2 to 40 percent with pH values ranging from about 11.5 to 14. Other drain opener or plumbing system maintenance products are bacterial or enzyme-based formulations. Products may also contain detergents, color dyes, scents, and other non-active ingredients.

The granular products and the lower concentration liquid alkaline are generally available in grocery, drug, convenience, hardware and home center stores. Sulfuric acid drain openers and the higher concentration liquid alkaline products are generally found in hardware stores.

Both alkaline and acid drain openers may react with the drain blockage (*e.g.*, hair and grease) via chemical reactions. SADO manufacturers claim that SADO products will also break down cellulosic materials (*e.g.*, paper) that alkaline products cannot. The heat generated by the drain openers may also liquefy the blockage. As discussed in the previous staff briefing package (Petition HP 95-3, Request to Ban Sulfuric Acid Drain Cleaners, 20 June 1996), the exothermic (*i.e.,* energy releasing) reaction of sulfuric acid with water and other chemicals under some circumstances may be violent and result in spattering or eruption of liquid from the drain.

Current Requirements

The potential for serious injury from products that contain sulfuric acid has been addressed previously by the CPSC through labeling and packaging requirements under the Federal Hazardous Substances Act (FHSA) and the Poison Prevention Packaging Act (PPPA).

Section 2(f)(1)(A) of the FHSA defines a hazardous substance as, among other things, "any substance or mixture of substances which is corrosive," if the substance "may cause substantial illness during or as a proximate result of any customary or reasonably foreseeable handling or use, including reasonably foreseeable ingestion by children." 15 U.S.C. § 1261(f)(1)(A). Hazardous substances containing sulfuric acid are corrosive and are thus subject to the regulations under the FHSA. Further, the Commission found that, for sulfuric acid and other substances originally regulated under the Federal Caustic Poison Act, lack of the designation of "poison" may lessen the general public's understanding of the hazard. Thus, hazardous products containing 10 percent or more of free or chemically unneutralized sulfuric acid require the word "POISON" on the label (16 C.F.R. § 1500.129(b)).

In addition to the word "POISON" and the statement of hazard, which in this case is, "CAUSES SEVERE BURNS," hazardous sulfuric acid drain opener products require labeling for precautionary measures during handling and use (15 U.S.C. § 1261(p)(1)(F)). First aid labeling is also required (15 U.S.C. § 1261(p)(1)(G)).

CPSC also regulates the packaging of some household products containing sulfuric acid under the PPPA. Hazardous household products containing 10 percent or more of sulfuric acid require special child-resistant, senior-friendly packaging. Wet-cell storage batteries are exempted from this requirement (16 C.F.R. § 1700.14(a)(9)).

These requirements may also apply to products containing sodium and/or potassium hydroxide. That is, in addition to the other requirements for labeling under the FHSA, hazardous products containing 10 percent or more of free or chemically unneutralized sodium hydroxide or potassium hydroxide require the word "POISON" (16 C.F.R. § 1500.129(j); 16 C.F.R. § 1500.129(i)); hazardous products in a dry form (*i.e.*, granules, powders, flakes) that contain 10 percent or more sodium and/or potassium hydroxide or in any other form that contain two percent or more require special packaging under the PPPA (16 C.F.R. § 1700.14(a)(5)).

Previous Commission Activity

The Commission has twice considered whether to ban SADOs. In 1977, the Commission received a request from the Hercules Chemical Company, Inc. to ban SADOs for household use. This petition was granted, but after a review of injury and economic information, the Commission was unable to find that a mandatory ban was necessary to address the risks associated with these products. Because of concern about the potential for serious injury from the use of SADOs, the Commission directed the staff to participate with the Association of Chemical Producers, Inc. (ACP) in a voluntary effort to enhance public safety through improved labeling, product design, and consumer education.

In 1994, the Commission received a second request to ban SADOs for consumer use from Roger L. Wabeke, president of Chemical Risk Management (Petition HP 95-3). In 1996, the Commission denied this petition based in part on the existence of the voluntary standard developed by ACP, and because the staff's analysis indicated that the injury rate associated with consumer use of SADOs was similar to that of other drain openers and that the likely result of a ban on SADOs would be a shift to other products without a significant reduction in injury rate.

Voluntary Standards

The ACP, formed in the 1980s after the first request to ban SADOs for consumer use, consisted of six sulfuric acid producers, representing about 70 percent of the market. The ACP established voluntary standards for sulfuric acid drain openers, consisting of three parts: minimum standards, compliance testing, and program review. The minimum standards covered educational materials, labeling, packaging, and formulation requirements. The educational and labeling aspects of the voluntary standard complied with and supplemented the FHSA's labeling requirements for products containing sulfuric acid. Companies that were not ACP members were also encouraged to comply with the voluntary standard.

For nearly two decades, the ACP assessed compliance with the voluntary standards and conducted annual reviews of the performance of the standard. The ACP was disbanded in 2002 purportedly due to costs associated with legal representation of the group, although individual firms may have continued to abide by the standards (Personal communication, J. Whitlock, Amazing Products, 2005). Although the staff is uncertain as to the extent to which former ACP member companies, and sulfuric acid producers in general, follow the voluntary standards established by the ACP, data from 2002 indicates that the staff found two products with minor label problems, which were subsequently corrected by the firms.

Hazard

The staff reviewed the toxicology of sulfuric acid. This information is discussed below and detailed at Tab B. In addition, the staff reviewed other chemicals currently used in drain opener products that could be used in place of sulfuric acid should sulfuric acid-based product become unavailable to consumers.

Dilute[1] sulfuric acid (*i.e.*, concentrations less than 10 percent) is considered a primary irritant, whereas sulfuric acid in concentrated form is a strong corrosive[2]. Strong alkalis are also markedly corrosive. The primary health effects of sulfuric acid are due to its irritating and corrosive nature. Depending on factors such as acid concentration and duration of contact with the skin, dermal exposure to sulfuric acid-containing substances may result in injury ranging from superficial burns to severe full-thickness burns. The heat produced from the reaction of concentrated sulfuric acid with the water in tissue may also play a considerable role in causing injury. Depending upon the route of exposure, sulfuric acid can directly affect the skin, eyes, respiratory tract, and/or gastrointestinal tract. Different body regions are more susceptible to damage than are others. For instance, the eye is more sensitive to injury due to its limited buffering capacity and regenerative capabilities. Mucosal membranes are also especially susceptible to injury, and ingestion of concentrated sulfuric acid can cause injury ranging from superficial ulcerations of the esophagus and stomach to gastric and esophageal perforation. Surgical corrections are often required to correct late complications. The immediate pain that sulfuric acid induces when placed in the mouth likely lessens the amount ingested and subsequent injury, although this may not be the case when an individual deliberately attempts to ingest the substance.

Sulfuric acid mists are a strong irritant of the upper respiratory tract causing irritation of the nose and throat, sneezing, and coughing. Exposure to low air concentrations can produce shallow and rapid breathing as a result of reflex bronchospasm, while severe overexposures can result in potentially fatal spasmodic closure of the larynx, and edema of the larynx and glottis. Respiratory irritation can also progress to tracheobronchial or pulmonary edema (*i.e.*, an accumulation of fluid in the lungs) in some cases.

The staff also reviewed the toxicology of other types of chemical drain openers that might be used as substitutes for sulfuric acid-based products. Concentrated hydrochloric acid causes less desiccation and heat release than sulfuric acid, but hydrochloric acid can cause significant tissue injury, including severe skin burns, esophageal and duodenal necrosis, and permanent eye damage. As with sulfuric acid, inhalation of hydrochloric acid vapors can also cause coughing, pain, inflammation of the upper respiratory tract, and in severe cases, pulmonary edema.

Likewise, products containing sodium hydroxide or potassium hydroxide can cause severe tissue damage. Hydroxides penetrate ocular tissues, quickly damaging the cornea and other structures of the eye. Thus, ocular exposure can result in severe injuries, including

---

[1] The *concentration* of a chemical is a measure of the amount of substance present in a given amount of solution; the *strength* of an acid or alkali is an intrinsic chemical property of the substance. Sulfuric acid and hydrochloric acid are strong acids; sodium hydroxide and potassium hydroxide are strong alkalis. Strong acids and alkalis can be concentrated or dilute depending on the amount of the acid or alkali present in a given volume of solution.

[2] Under the FHSA, a corrosive is defined as a substance that causes visible destruction or irreversible alterations in the tissue at the site of contact (15 U.S.C. § 1261(i)).

permanent impairment of vision, especially if the eye is not immediately washed. Dermal exposure to hydroxide-containing substances also results in injury ranging from diffuse erythema of the skin at the site of contact (*i.e.*, superficial burns), to severe full-thickness burns requiring skin grafting. Ingestion of hydroxide-containing substances causes injury ranging from superficial ulcerations of the esophagus and stomach to gastric and esophageal perforation. As with the acids, the potential for injury from the hydroxide-containing products increases with increasing concentration.

Although the mechanisms of action by which acids and alkalis cause injury are different, strong acids and alkalis can produce significant tissue damage and require caution when using. Drain openers containing sulfuric acid, hydrochloric acid, or sodium or potassium hydroxide can be fatal if swallowed and cause permanent impairment of vision if splashed in the eye. While inhalation exposure would not be expected with the hydroxides, sulfuric acid and hydrochloric acid can form a vapor that can cause symptoms ranging from irritation of the nose and throat to lung edema. Dermal exposure to all of these substances can cause full-thickness injury to the skin that may necessitate skin grafting. Concentrated sulfuric acid can cause dermal injury faster than concentrated sodium hydroxide. However, an hydroxide on the skin may not be perceived as rapidly as sulfuric acid, which could cause a delay in treatment following exposure, increasing the risk or severity of injury. Sodium hydroxide is capable of causing severe burns with deep ulceration due to its ability to continue to penetrate to deeper layers of tissue until it is washed away with copious amounts of water.

Drain openers containing sulfuric acid, hydrochloric acid, sodium hydroxide or potassium hydroxide have the potential to produce severe injuries depending on the concentration of the agent, the pH of the product, the site of exposure, the duration of contact, and the amount of the product involved. The likelihood and severity of injury from exposure to any of these chemicals can be lessened with prompt treatment, which generally consists of removing the victim from the source of exposure, washing the skin and eyes with copious amounts of running water, and obtaining medical treatment, especially in the case of ingestion.

Injury Data Analysis

The staff analyzed the available injury and exposure data for chemical drain openers, and sulfuric acid drain openers in particular. This information is discussed below and detailed at Tab C. It should be noted that the product category, "alkaline," may include products with a wide range of hydroxide concentrations (*e.g.*, 2-40 percent for liquids; 60-100 percent for granular formulations).

Using data from the National Electronic Injury Surveillance System (NEISS), the staff estimated that from 1995 to 2003 inclusive, there were an estimated 16,712 injuries associated with drain openers treated in U.S. hospital emergency rooms. A breakdown of the data from 1995 to 2003 according to type of drain opener is shown in Table 1. Intentional and occupational exposures are excluded.

Table 1: Estimated number of emergency-room treated injuries associated with drain openers by drain opener type, 1995-2003

Drain Opener Type	Estimated Number of Injuries	Coefficient of Variation
Sulfuric acid	768	0.25
Alkaline	5,318	0.12
Mixtures	1,402	0.18
Other*	299	0.40
Unknown	8,925	0.13
Total	**16,712**	**0.09**

*Includes hydrochloric acid, acids of unknown type, and enzyme-based drain openers. C.V. is large.
Source: National Electronic Injury Surveillance System, Directorate for Epidemiology, U.S. Consumer Product Safety Commission.

Most of the cases involved drain openers of unknown composition. Of those injuries where the type of drain opener was known, 10 percent involved sulfuric acid drain openers, and 68 percent involved alkaline drain openers. Injuries involving mixtures included situations in which drain openers were mixed with a wide variety of other substances; the most common substance was bleach. While a few mixtures were of an alkaline substance combined with an acid drain opener, mixtures of alkaline drain openers and SADOs were relatively rare.

The staff also reviewed data compiled by the American Association of Poison Control Centers (AAPCC) from all calls made to participating U.S. poison control centers through the Toxic Exposure Surveillance System (TESS). CPSC staff has access to detailed data concerning children under age 5 years; the staff also has access to summarized reports of aggregated cases involving all ages.

For all ages, 48,700 exposures to chemical drain openers of all types were reported through TESS for 1995 to 2003, inclusive. This total differs from the NEISS injury estimates in part because not all exposures reported to poison control centers are treated in hospital emergency rooms. Approximately 33 percent of the exposures to chemical drain openers reported through TESS were treated in a health care facility (*e.g.*, emergency room, clinic, physician's office). In addition, approximately seven percent of the TESS cases were recorded as intentional exposures.

Nearly 2,000 exposures to chemical drain openers were reported through TESS for children under age 5 years for the three-year period, 2000-2002[3]. Excluding unknowns, approximately six percent of the cases involved a sulfuric acid drain opener product, 82 percent involved an alkaline product, three percent involved a hydrochloric acid-containing product, and about nine percent were coded as involving an enzyme or other product. Fifty-five percent of the SADO exposure cases exhibited clinical effects, compared to 42 percent of the alkaline exposure cases. Seventeen percent of cases involving hydrochloric acid exhibited clinical effects, as did 16 percent of cases in the enzyme/other category. In addition, 25 percent of the sulfuric acid drain opener exposures to children under 5 were confirmed or judged as potentially toxic,

[3] The reporting period for the subset children under age 5 extends to 2002, compared to 2003 for the all ages dataset, because 2002 was the latest year for which CPSC staff had detailed data at the time of the analysis (Tab C).

resulting in moderate or major outcome[4]. Fourteen percent of alkaline exposures, six percent of hydrochloric acid exposures, and two percent of enzyme/other exposures fell in the same categories.

An examination of the data collected through TESS for all ages for 2001 through 2003 shows that approximately nine percent of 16,531 cases involved a sulfuric acid product, and about 70 percent of cases involved an alkaline-based product. These data are consistent with the data collected through NEISS, although in TESS for 2001-2003 only about 13 percent of cases were coded as involving "other" or "unknown" drain opener products compared to more than 55 percent in the NEISS data. This subset of TESS data also shows that approximately 40 percent of sulfuric acid product cases and 31 percent of cases involving alkaline-based products were treated in a health care facility. About 32 percent of exposures involving sulfuric acid-based products and 23 percent of alkaline-based product exposures resulted in moderate outcomes; approximately 1.5 percent of sulfuric acid-based product exposures and 2.5 percent of alkaline-based product exposures resulted in major outcomes. It should be noted that the TESS figures include intentional exposures.

The staff also analyzed available records concerning deaths recorded in CPSC's Death Certificate file and the Injury and Potential Injury Incident file. There are six reports of fatalities from exposure to chemical drain opener products for 1995 through November 8, 2004. The products involved are not known for each case, but one death involved a child who ingested an alkaline-based product and at least one case involved exposure to fumes from use of a combination of a sulfuric-acid based product and an alkaline product.

Overall, these data show that about six to ten percent of injury or exposure cases in which the product type was ascertained involved a sulfuric acid drain opener product; 68 to 82 percent of cases were reported as involving an alkaline-based product. Other cases involved hydrochloric acid and enzyme-based product. Each type of chemical drain opener is associated with a risk of injury. Further, a portion of exposures involving these products resulted in clinical effects or were treated in a health care facility. Cases associated with each type of product included moderate or major medical outcomes; deaths were reported from exposures to acid and alkaline products.

Human Factors Analysis

The staff analyzed product labeling and other Human Factors issues. This information is discussed below and detailed at Tab D.

Consumer Behavior

The staff analyzed a selection of 103 in-depth investigation reports collected by the CPSC. These reports do not constitute a random sample of incidents, and may not be representative of drain opener incidents in general. These reports were used to provide background information and scenario details that may be useful in understanding the types of incidents that occur or to help characterize some of the behavior components of drain opener exposure incidents.

[4] A major effect is defined in TESS as life-threatening or resulting in significant residual disability or disfigurement. Moderate outcomes are defined as having more pronounced, prolonged or systemic symptoms than minor cases; moderate outcomes are cases that were not life-threatening, and the patient had no residual disability or disfigurement, but usually some form of treatment is indicated.

The information in these records indicates that for both adults and children, exposure to chemical drain openers, including sulfuric acid-containing products, occurred in a wide variety of ways. Children, for example, ingested drain openers from the package or another source such as a clogged water fountain or cup, spilled or poured contents onto themselves, and mouthed the cap of a bottle. One child was exposed from mouthing a towel that a maintenance worker had used to clean up a spill from a burst pipe. Children were splashed when products were dropped or fell from a store shelf. One child was splashed when he dropped a toy into a floor drain treated with a chemical drain opener, and another was exposed when he played with a toy that had apparently been contaminated by a nearby spill on the floor. Children were also treated after exposure to fumes.

Reports involving adults showed a similar diversity in incident scenarios, involving normal and foreseeable use and misuse. In this group of cases, most victims were exposed during or immediately after use of the product, *i.e.*, while opening the container, walking with container, while pouring the product, or while using a plunger or removing the drain trap. Adults were also exposed to spills, splashes or leaks from bottles in retail stores or during transport from the store after purchase.

The petitioner described an incident in which a young boy was severely injured when a bottle of sulfuric acid drain opener spilled on him. The petitioner stated that neither the police nor the paramedics knew how to respond to the boy's burns, and that instead of washing his skin with large amounts of water, they dribbled small amounts of saline solution on the skin. In addition, another family member who was exposed was first questioned by police before being treated in a hospital emergency room. Few details about this incident were provided, but it appears that no attempt was made to wash the product from either victim between the time of the spill and the arrival of the medical personnel, and no explanation is given as to why the attending medical personnel responded as they did. The staff believes that this case does not demonstrate a typical response to a chemical exposure in the home. On the contrary, the available information in other investigation documents suggests that many consumers are more capable than the petitioner assumes; in many of the cases described in the incident reports, first aid was given following the exposure; *e.g.* the victim's skin, eyes, or mouth were washed with water and those reporting exposure to fumes left the area and sometimes flushed their eyes. Thus, the staff believes that it is reasonably likely that when an exposure occurs, consumers' responses will be at least somewhat effective.

Labeling Issues

In order to ban sulfuric acid drain openers, the Commission would have to show that the public can be protected only by eliminating the product, and that alternatives to banning, such as improvements in labeling or a voluntary standard, could not adequately reduce the risk of injury.

The petitioner claims that "...the label has to *guarantee* that the consumer will read and understand the label before they use the product and follow all safety instructions to the letter." The petitioner's tone suggests that if the label cannot guarantee full knowledge and complete compliance, the consequences of a sulfuric acid exposure are necessarily dire. The staff believes that no label can guarantee that users will read, understand, and follow product warnings and instructions. However, the available data do not support the inevitability of a serious outcome. The staff's review of a number of case reports suggests that although the consumers involved in the incidents likely failed to comply with one or more of the warnings or instructions on the container, some appropriate action, such as washing and seeking medical treatment, occurred in each case. The staff believes, however, that optimization of warning labels and instructions may increase user compliance, and help reduce the risk of injury.

"Kleenex Syndrome"

The petitioner claims that the injury databases used by the staff have inaccurately recorded some cases as involving a well-known brand of an alkaline-based product rather than accurately identifying that the products contained sulfuric acid, which he refers to as the "Kleenex syndrome." The staff agrees that, in general, there is likely to be some element of non-sampling error in records of incidents involving chemical exposure or injury. Errors may be due to poor recall under conditions of stress, delays between the time of the incident and the time of the report, and other factors. However, the available information does not support the claim that errors in recording the product names or other identifying information are particularly widespread in the data analyzed by the staff. For example, information on cases reported in the TESS database is often collected from the victim's home, near the time the incident occurred. Thus, information about the product is likely to be obtained directly from the product label.

The staff believes that under some circumstances, it may be more likely that incidents involving sulfuric acid are recorded as "unknown drain opener," rather than misidentified as an incident involving a commonly-known alkaline-based product. Because sulfuric acid drain openers are not as widely available as low concentration alkaline products, and because sulfuric acid may be more effective in certain cases, consumers who purchased a SADO product might have sought out a product that is different from the widely-available products. In this case, consumers might not remember the name of the product, but many would be aware that what they purchased is not the same as the brand name products available in grocery stores.

To the extent possible, the staff's analysis of the available data was conducted after the databases were inspected for product-identifying information. Cases with missing or ambiguous information were classified as involving an unknown product. Further, special care was taken by the staff to accurately identify the product type in the portions of the staff's analyses that involved discussions of specific cases. While misclassification may never be eliminated, these steps are likely to reduce the impact of such errors.

Economic Information

The staff evaluated available information on the sulfuric acid drain opener market, alternatives to SADOs, and the impact of a ban. This information is discussed below and detailed at Tab E.

Product Information

SADOs are available in pint, quart, half gallon, and gallon containers, with quarts being the most common size. Sulfuric acid drain openers are widely available in hardware stores; they are much less common in other types of retail stores including grocery, drug, and department stores. Most SADO manufacturers recommend 4 to 8 ounces of the product be used per application for most household drains; a second application is often necessary to remove a clog.

Market Information

Assuming that sales of chemical drain openers are a function of the number of occupied housing units, using data from a 1978 study, nearly 74 million units of chemical drain openers were sold in 2003. Information provided by an industry representative indicates that sulfuric acid drain openers probably account for about 3 to 5 percent of units (or packages) of drain openers sold to retail consumers. Based on the instructions for using the different chemical drain openers, in terms of the number of applications, SADOs could account for up to 10 percent of actual consumer applications.

Societal costs

The CPSC Injury Cost Model (ICM) estimates the societal cost of injuries based upon the age, diagnosis, and gender of the victim, and whether or not the victim was hospitalized as recorded in the cases from the National Electronic Injury Surveillance System (NEISS). The ICM uses the weights assigned to the NEISS cases to estimate the number of injuries that were treated in emergency departments, but it also imputes from these cases estimates of the number of medically-attended injuries treated in settings other than an emergency department. The estimated cost includes the costs of medical treatment, work loss, pain and suffering, and liability insurance and litigation costs. Based on the ICM estimates of drain opener injuries for the years 1995 through 2003, the medically-attended injuries involving all chemical drain openers resulted in about $93 million in societal costs annually.

SADOs were involved in about 10 percent of the NEISS cases where the type of drain opener involved could be determined (the type of drain opener could not be determined in more than half of the NEISS cases). If injuries involving SADOs were more severe than injuries involving other chemical drain openers, the average societal costs associated with injuries involving SADOs would be greater than the costs associated with other chemical drain opener products. As discussed in more detail below, alkaline products with higher hydroxide concentrations may be the most comparable to SADO products. It may not be possible to compare the injuries associated with the different types of chemical drain openers, however, because all hydroxide-based products were grouped together in the injury analysis, and information about the dangers of products containing higher concentrations of hydroxide could be masked by the much larger number of lower concentration hydroxide products.

Substitutes and the Effect of a Ban

Alkaline products are more widely available than SADOs. Unlike SADOs, alkaline products are commonly available in grocery, drug, and convenience stores. Like SADOs, they are also available in hardware stores. However, the alkaline products available in grocery, drug, and convenience stores are generally the liquid alkaline products with the lower hydroxide concentrations, and the granular alkaline formulations. The liquid alkaline products with the higher hydroxide concentrations (*e.g.*, greater than 10 percent) are more likely to be found in hardware or home center stores.

Of the alkaline products, the closest substitutes for SADOs are the ones with the higher hydroxide concentrations. It is likely that many people who choose to purchase SADOs do so because they want a product that they perceive as being stronger than the typical alkaline product sold in grocery, drug, and convenience stores, and are willing to make the effort to go to a hardware store to get such a product. Therefore, if SADOs were no longer available, these consumers might be more likely to buy the alkaline products with the higher hydroxide concentrations that are also available at hardware stores rather than the less concentrated alkaline formulations.

Hydrochloric acid drain openers are also possible substitutes.

Drain cleaners containing enzymes or bacteria work on pipes that are slow draining, but that are not completely clogged. Therefore, they may not be good substitutes for either SADOs or alkaline products that can open completely clogged drains.

Mechanical methods for unclogging drains include the use of plungers and snakes or augers. The primary disadvantages of mechanical devices are that they require higher degrees of skill and physical strength on the part of the consumer than do chemical products. Mechanical methods also may require the consumer to remove things such as cross bars, strainers, mechanical drain plugs, and lift bars from the drain before the device can be used. Therefore, the time it takes the consumer to use a mechanical device may be substantially greater than that needed to use a chemical drain cleaner. For consumers who have the skill, time, and strength, these methods may be effective alternatives to sulfuric acid or other chemical drain cleaners. For other consumers, they may not represent viable alternatives.

Professional plumbing services are the most costly substitute for SADOs. The cost to consumers of using a professional to unclog a drain typically exceeds $100 for even a simple clog. Because of the high price of professional plumbers, many consumers probably try other methods first, such as a chemical drain opener or one of the mechanical devices. In comparison, retail prices for liquid alkaline drain openers range from about $1 to $4 per use; prices for the dry or crystal alkaline products are about $0.20 to $0.65 per use; retail prices for plungers start at under $4; and augers can be purchased for as little as $8. Per application, retail prices for sulfuric acid drain openers are approximately $0.80 to $2.00.

Many clogs are likely to be successfully treated with any of the available acid or alkaline-based drain opener products. However, alkaline products may not be effective for eliminating clogs composed of cellulosic materials, such as paper or cloth. While these types of clogs are probably a small fraction of all clogs, if SADOs were no longer available, consumers who have clogs composed of cellulosic material and who would have ordinarily purchased a SADO would have to rely on other methods for removing the clog. Unless the consumer is able to use the various mechanical devices, the consumer will probably have to call a professional plumber. Therefore, for these types of clogs, a ban would increase the cost of unclogging the drains by a factor of greater than 100 (from about $1 with a SADO to over $100 with a professional plumber).

The effect on societal cost of injuries of banning SADOs for consumer use is less clear. If SADOs were not available, consumers that now use them would likely substitute other chemical drain openers, including the alkaline products with the higher hydroxide concentrations that can also cause severe injuries. The reduction in societal costs that could be expected from a ban on SADOs would be the difference between societal costs for injuries associated with SADOs and the expected societal costs of injuries associated with the substitutes for SADOs.

Effect on Suppliers

SADO manufacturers would be adversely affected by a ban. There are probably fewer than a dozen manufacturers of SADOs for the consumer market. Most have fewer than 500 employees and so would meet the Small Business Administration criteria for being considered a small business. In some cases SADOs account for a large proportion of their sales. Other SADO manufacturers have a more diverse product line. However, a ban on SADOs would likely increase the demand for other drain opening products, including other chemical drain openers, mechanical drain openers, and professional drain cleaning services.

Alternatives to a Ban

As an alternative to banning sulfuric acid drain openers, the petitioner urges the Commission to limit the amount of SADO in containers intended for consumer use to just the amount that would be required for one use and to limit the maximum sulfuric acid concentration of SADOs intended for consumer use to no more than 84 percent.

Effect of Restricting Package Size

According to package directions, a typical application of a sulfuric acid drain opener requires 4 to 8 ounces, or one cup or less. There are some pint-sized packages of SADOs available, but technically these could contain enough product for 2 to 4 applications. Manufacturers could incur some costs in retooling their facilities to accommodate the smaller containers and the cost per unit of the SADOs would increase. It is not known if injuries would be avoided if the product is packaged in smaller containers, because package size might not affect the number of injuries that occur while the product is being used. Further, exposure to the amount of product that might be packaged in a single-use package could still result in severe injury and would still require prompt washing and treatment.

Effect of Restricting Product Formulation

The reaction of a reduced concentration SADO would be expected to produce less heat when added to a clogged drain, or when an exposure occurs. Reducing the heat of the reaction could reduce the risk that the acid will cause the contents of the drain to boil or erupt out of the drain, or it could reduce the tissue damage caused by the heat if it gets in the skin or in the eye. The staff has not investigated whether reducing the concentration of sulfuric acid in the product would significantly reduce the risk or severity of injury.

One manufacturer of a SADO with a sulfuric acid concentration of less than 84 percent claims that their product will "bead up" on dry skin, allegedly allowing the consumer time to wipe or rinse the product off before serious injury results. The producers of another product containing a higher concentration of sulfuric acid (approximately 94 percent) claim that their formulation allows up to five minutes to wash it off the skin. While certain characteristics of product formulations might reduce the risk of injury by allowing more time to get treatment before injury occurs, the staff has not tested these claims, nor has the staff determined whether a specific acid concentration in the product might be responsible for reducing the risk or severity of injury, or if other chemicals in the product formulations provide the risk reduction.

Regardless, a product containing approximately 84 percent sulfuric acid is still considered to be a concentrated sulfuric acid product, and once an exposure has occurred, prompt washing and treatment would likely still be required to reduce the risk and severity of injury. In addition, such a restriction might not result in reducing the risk or severity of injury since some products may already have a sulfuric acid content of 84 percent or less. Therefore, the extent of possible risk reduction from limiting the sulfuric acid concentration to 84 percent is not clear. In addition, the cost of alternative sulfuric acid drain opener formulations may be somewhat higher than the cost of other SADOs because of the costs of other ingredients that are added to some products.

Public Comments

CPSC received four comments in response to the Federal Register notice published 5 May 2004 (69 FR 25069). Comments were provided by two individual companies [Roebic Laboratories, Inc. (CH04-5-1) and The Rooto Corporation (CH04-5-2)] and one group of companies (CH04-5-3; CH04-5-4) that produce sulfuric acid drain opener products. The index of public comments is in Tab F. The comments and the staff's responses to the comments are summarized below and detailed at Tab G. The Commission also received additional comments from the petitioner, Michael Fox, and from the previous petitioner, Roger L. Wabeke, reiterating the points they had made previously.

Comment CH04-5-1: Roebic Laboratories, Inc. opposes the requested ban because the company believes that they have already taken steps to offer safe products. The firm claims that their product formulation is less likely to cause thermal burns to the skin, it is designed to make spills noticeable and easy to clean off, the package uses an "anti-glug" spout to limit the spill, and the manufacturer uses over-packaging to protect against accidental spills during transport.

Staff Response: Although it's conceivable that these steps do provide a benefit, the staff has not tested the ability of these features to reduce the risk or severity of injury to consumers.

Comment CH04-5-2: The Rooto Corporation opposes the ban because they believe that the injury data and economic factors do not support that action. They stated that there has been a large volume of product sales, but few complaints, and that banning the products would greatly increase the cost of cleaning blocked drains and could cause more accidents. They believe that a ban on sulfuric acid products would benefit only plumbers, who would be using the same products currently available to consumers. Finally, they disagree that decreasing the acid concentration in the product would be beneficial.

Staff Response: The staff agrees that the alternatives to sulfuric acid drain openers could be more costly to consumers. However, the staff believes that only a fraction of clogs would only respond to a sulfuric acid drain opener or a method such as provided by a professional plumber (Tab E).

Comment CH04-5-3, CH04-5-4: These comments were submitted by a group of nine sulfuric acid drain opener producers. The firms represented in these comments were formerly members of the consortium of sulfuric acid manufacturers known as the Association of Chemical Producers, Inc. (ACP). These commenters oppose the ban and address some specific claims made by the petitioner related to the volume of sales to consumers versus professionals, product labeling, and data reporting. They also point out that sulfuric acid drain openers are useful for certain kinds of drain blockages and that the alternatives (*e.g.*, professional plumber) would be costly.

Staff Response: Although these commenters refuted many of the claims made in the petition, they provided little data to support their own claims. The staff agrees that sulfuric acid products may work better than other types of chemical drain openers for certain clogs, but we believe that these clogs are only a small fraction of all clogs (Tab E).

Options

The following options are available to the Commission:

Grant the Petition

If the Commission concludes that it is appropriate, the Commission could grant the petition and begin a proceeding to ban sulfuric acid drain openers or require restrictions on package size and product formulation.

Deny the Petition

If the Commission concludes that information is not available or likely to be developed to support the findings required by section 2(q)(1)(B) and 3(i)2 of the FHSA to ban sulfuric acid drain openers or require restrictions on package size or product formulation, the Commission could vote to deny the petition.

Defer Decision on the Petition

If the Commission determines that there is insufficient information to make a decision on the petition and that the staff could obtain such information, the Commission could defer the decision and direct the staff to obtain the additional information.

Conclusions and Recommendation

The available toxicity information indicates that chemical drain opener products containing concentrated sulfuric acid have the potential to cause severe injuries from dermal or ocular exposure, inhalation of fumes, or ingestion. Toxicity data also show that some of the possible chemical substitutes for sulfuric acid-based products, such as hydrochloric acid or the higher concentration hydroxide-based products also have the potential to cause severe injuries. In any case, immediate treatment, including thorough washing with water, will help to limit injuries or the severity of injury.

Analysis of the injury and exposure data shows that each type of chemical drain opener is associated with a risk of injury, and that a portion of exposures involving these products resulted in clinical effects or were treated in a health care facility. Medical outcomes ranged from no effect to major effect. There are also reports of fatalities from exposure to chemical drain opener products. The type of product is not known for each case involving a fatality, but one death involved a child who ingested an alkaline-based product and at least one case involved exposure to fumes from use of a combination of a sulfuric-acid based product and an alkaline product. Although it appears from the TESS figures that exposures involving sulfuric acid-based products tended to be more likely to result in injury or to result in more severe injury, there are limitations in the data that complicate interpretation of and reliance on these results. In particular, because all hydroxide-based products are grouped together in this analysis, information about the dangers of products containing higher concentrations of hydroxide may be masked by the much larger number of lower concentration hydroxide products. It is possible that both sulfuric acid-based products and certain higher concentration hydroxide-based products are associated with a greater likelihood and severity of injury than are associated with other products, such as the commonly available low concentration hydroxide products. In addition, incidents for all ages counted in the TESS figures include intentional exposures that could affect the conclusions drawn from the data.

Analysis of some specific incidents showed that exposures to a wide variety of chemical drain opener products occurred through a variety of situations. These cases also showed that in many instances, consumers were capable of responding appropriately to exposure, such as by initiating washing or removing the victim from the exposure, and by seeking medical care.

The number of sulfuric acid drain opener exposures, as the percentage of exposures with known product type, is consistent with the estimated market share for these products (*i.e.*, sulfuric acid-based products account for 3-10 percent of chemical drain opener treatments used by consumers, and sulfuric acid drain openers are associated with about 6-10 percent of incidents). Sulfuric acid-based products represent a small segment of the market, so that any effort to reduce injuries from exposure to these products would have a relatively small effect on the total number of injuries from exposure to all chemical drain openers. In addition, if sulfuric acid-based products were banned, consumers might substitute other chemical drain openers for SADOs. Thus, the societal costs now associated with sulfuric acid drain openers would not necessarily be significantly reduced or eliminated. This might be especially true if consumers substituted the higher concentration alkaline products for SADOs. Effectively, a ban on SADOs might simply shift much of the societal costs now associated with SADOs to other chemical drain openers.

Substitutes for sulfuric acid drain openers (*e.g.*, chemical, mechanical, plumber) could increase costs to treat some clogs (possibly more than 100-fold, in the case of cellulosic clogs that may not respond to other chemical drain opener products). Changes in packaging or product formulation might be expected to increase manufacturing costs of the product. The possible effect of alternatives to a ban (*i.e.*, package or formulation restriction) in reducing societal costs is unknown.

Although sulfuric acid drain openers have the potential to cause injury, other chemical drain opener products also cause injury. In addition, prompt treatment (*i.e.*, washing with water and seeking medical attention) can greatly reduce the severity of injury from chemical drain opener exposures. The staff believes that a ban on sulfuric acid-containing products would not eliminate the costs now associated with sulfuric acid-based products and might not significantly reduce costs. This might be especially true if consumers substituted the higher concentration alkaline products for SADOs, which would shift much of the societal costs now associated with SADOs to other chemical drain openers. Thus, the staff believes that the available information does not support a conclusion that a ban of these products is necessary to protect public health and safety. Further, the staff does not believe that restrictions on package size or product formulation would necessarily reduce or eliminate the risk or severity of injury since exposure to a relatively small volume of a sulfuric acid product or to a product containing 84 percent sulfuric acid would likely still require prompt washing and treatment in order to lessen the risk and severity of injury. Therefore, the staff recommends that the Commission deny the petition.

TAB A

Chemical Accident Reconstruction Services, Inc.

HP 04-2

February 26, 2004 Via Certified Mail

U.S. Consumer Product Safety Commission
ATTN: Office of the Secretary
Washington, D.C 20207

Re: Petition to Ban Sulfuric Acid Drain Openers

Dear Director:

This is an open letter to the Consumer Product Safety Commission (CPSC), which is also posted on the Internet. The purpose of the letter is to petition the CPSC to ban sulfuric acid drain openers (SADOs) for use by ordinary consumers and to limit SADOs to use by professionals only.

The young boy in the photograph below was the victim of an accident involving a sulfuric acid drain opener (SADO) in 2001.

Young Victim of Sulfuric Acid Drain Opener (SADO) Accident

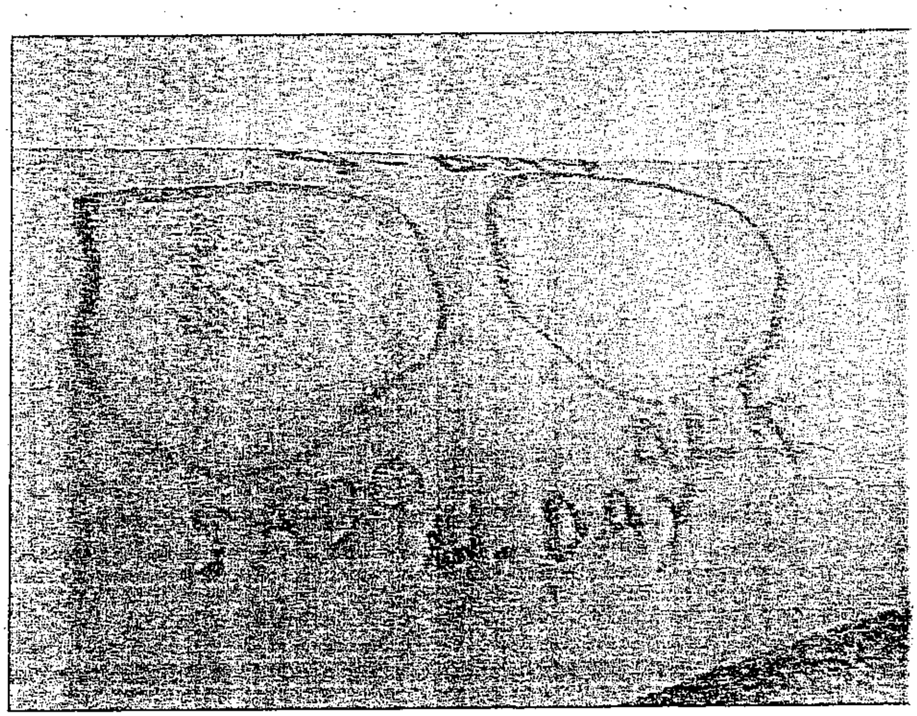
9121 E. Tanque Verde Road #105, Tucson, Arizona 85749
800-MIKE-FOX (645-3369) Fax: 520-749-0861

My involvement in that accident as a chemical expert led me to the CPSC's records concerning the petitions, bans and reviews of SADOs by the CPSC. As a result, I have done a considerable review of the CPSC's files, research and decisions in this regard and have attached a more detailed report of my findings. This cover letter is an Executive Summary.

While I agreed wholeheartedly with the Commission's decision in 1978 to limit the use of SADOs to professional use only and to ban it from use by the ordinary consumer, I was extremely disappointed to see the CPSC reverse itself in 1981 and then to again deny a petition in 1996 to keep SADOs out of the hands of ordinary consumers.

At the same time, I believe I understand the Commission's logic. On the surface, it appeared that the percentage of SADO injuries were about the same as its market share of all drain openers. The Commission never said that SADOs were not dangerous. They simply said that SADOs were no more dangerous than alkali drain openers (ALKDOs).

Before I go any further I would like to ask if anyone at the CPSC ***really*** believes that SADOs are not any more hazardous than ALKDOs. If so, I propose a public demonstration in which I will pour an amount of ALKDO on my forearm while a member of the CPSC pours an equal amount of SADO on their arm. We will then see who heads for the water first and who has the most severe burns. This may seem melodramatic, but I want to get the CPSC's attention on this important matter.



Comparison of SADO vs. ALKDO Skin Contact 4-Day Results
SADO contact time was 25 seconds
ALKDO contact time was 37 seconds

The reason I can make this challenge is that, in addition to seeing the horrific results of SADOs as the young boy pictured above, I have performed the proposed demonstration on my own arm using both ALKDO and SADO simultaneously. I applied the ALKDO first and then the SADO, and then rinsed them both off once the SADO became a serious concern, which was within 25 seconds. The picture above illustrates the results after four days. The SADO produced a burn, which scabbed over, while the ALKDO did not even cause minor skin irritation. There can be little doubt that SADOs are more dangerous than ALKDOs.

The fundamental flaw in the CPSC's logic was to compare the percentage of sales to percentage of injuries. On that basis, it appeared that SADOs were no more hazardous than ALKDOs. In other words, it appeared that the percentage of SADO sales were about the same as the percentage of SADO burns.

However, the hidden flaw in the CPSC's logic was that only 1/3rd of the SADOs were sold to ordinary consumers. The other 2/3rds were sold to professionals. On the other hand, the ALKDOs are primarily consumer products sold in super markets. Since professionals are trained in the use of chemicals (per the OSHA Hazard Communication Standard) they would be far less likely to sustain a chemical injury. In fact, I believe that work-related injuries and intentional injuries were excluded in the CPSC's analysis. When you compare the percent SADO sales to ordinary consumers (3.1%) to the percent SADO injuries (11%) it becomes clear that SADOs are *at least* 3.5X more dangerous than ALKDOs.

One staff member of the CPSC suggested that the frequency of exposure to ALKDOs might be as high as 280 times as much as the frequency of exposure to SADOs. Hence, one would expect that SADOs should contribute less than one percent of all drain opener burns. Yet, SADOs account for at least 11% of all burns. This suggests that SADOs are at least an order of magnitude more dangerous than ALKDOs.

Instead of maintaining the 1978 ban, the Commission decided to allow the SADO industry, referred to as the Associated Chemical Producers or ACP, make voluntary improvements in packaging and labeling. However, if you look at the injury data following the voluntary improvements, as the CPSC's 1996 review did, it becomes clear that things just got worse.

There were 120 injuries due to SADOs in 1980. In 1981 the ACP stepped in with their improved label, yet the injury rate never went below 120 thereafter. It was 170 in 1982 and other years were all above 200. In 1993 there were 680 SADO injuries. This strongly contradicts any notion that the "improved" labeling by the ACP had any measurable effect on injuries.

Another flaw in the CPSC's earlier process is what I call the "Kleenex Syndrome." If you ask someone for a Kleenex, they might hand you a Charmin Bath Tissue. The problem is that "Kleenex" has become a descriptor for an entire product type. Somewhat like a "Xerox copy," even though Xerox is no longer the most common copying machine. Likewise, Liquid Plumber has become a generic term for liquid drain openers. Therefore, the comments section of the National Electronic Injury Surveillance System (NEISS) might say that Liquid Plumber was the cause of a patient's burns when in fact it may have been another type of liquid drain opener. Since Liquid Plumber is an ALKDO, those burns would automatically fall into the ALKDO column and might be missed as SADO burns without further questioning of the patient. Also, the hospital staff might enter Liquid Plumber in the Comments Section even if the patient said it was some type of liquid drain opener. The patient themselves might be confused and say it was Liquid Plumber. The distinction is not immediately obvious to the ordinary consumer.

Concentrated sulfuric acid is so much more hazardous than the typical 10% sodium hydroxide solutions used in ALKDOs that it simply does not make sense that SADOs and ALKDOs present the same danger. There are at least five mechanisms by which SADOs can damage skin (heat, acid, dehydration, oxidation and sink eruptions) while ALKDOs present the single mechanism of alkalinity. Since people are as likely to spill Coke on themselves as they are Pepsi, they are also equally likely to spill a SADO or ALKDO on themselves. Given the ultra hazardous nature of the SADO compared to an ALKDO, it is simply illogical that SADOs would present the same probability of injury as ALKDOs.

My final comment about SADOs mirrors several comments from some CPSC staff members. It is not possible to label a SADO so as to properly instruct and warn the ordinary consumer about the dangers it presents. Once a consumer gets splashed with a SADO he or she must know what to do immediately. Therefore, the label has to *guarantee* that the consumer will read and understand the label before they use the product and follow all safety instructions to the letter. To my knowledge, there is no such label.

Once a consumer is splashed with SADO, there is no time to then read the warnings or first aid instructions on the container. In fact, anyone who works with sulfuric acid on any regular basis is extensively trained *and drilled* on what to do without thinking in an emergency. Without that kind of knowledge and training the ordinary consumer is helpless. For example, in the case involving the young boy pictured above not even the paramedics or police knew how to properly respond to this boys SADO burns. They were dribbling saline solution on him when they should have been washing him with a garden hose full force. Another member of the family who also got sulfuric acid on her skin was not even washed until she got to the hospital emergency room. Instead of washing her with copious amounts of water, the police were questioning her.

There is absolutely no merit to the argument that SADOs are easy to use and offer sufficient economic benefits to offset their risk. It is not a life-saving or otherwise essential chemical product. It is simply a drain opener. There are other, less-risky and less-costly ways to unclog a drain. SADOs can also cause corrosion problems in certain types of plumbing.

As discussed in the attached report, the risks of SADOS greatly exceed their benefits. Furthermore, SADOs are ultra hazardous when compared to other economical means to open drains and can also corrode the plumbing systems themselves.

It is my professional opinion, as someone who has spent his entire professional life in the field of chemistry, chemical safety and chemical accident investigation that:

1) Sulfuric acid drain openers (SADOs) are unreasonably dangerous and should not be sold to ordinary consumers. SADOs can cause horrific injuries and are unsafe when used in a reasonable and foreseeable manner by ordinary consumers. The risk of danger inherent in SADOs greatly outweighs their benefits.

2) SADOs should only be sold to plumbing professionals who have had the benefit of the training required by the OSHA Hazard Communication Standard.

3) If SADOs MUST be sold to ordinary consumers, they should be packaged in one-shot containers, and

4) If SADOs MUST be sold to ordinary consumers in one-shot containers, they should not be greater than 84% in concentration in order to provide a (slight) time-based safety factor and to reduce the thermal component of SA injury.

With greatest respect and sincerity, I hereby request that the CPSC reevaluate and reconsider the use of SADOs by ordinary consumers. I am willing to participate in that reevaluation process at no charge to the CPSC.

Please call (800-MIKE-FOX) if you would like to discuss any of the above or the attached report and my willingness to participate in the review process.

Sincerely,

MICHAEL FOX

Michael Fox, Ph.D.
Founder
Chemical Accident Reconstruction Services, Inc.

REPORT

Sulfuric Acid Drain Openers

by

Michael Fox, Ph.D.
Chemical Accident Reconstruction Services, Inc.
4651 N. Lason Lane
Tucson, Arizona 85749
800-MIKE-FOX (645-3369)

February 2004

Sulfuric Acid Drain Openers
by
Michael Fox, Ph.D.

Prepared February 2004

1.0 Introduction and Background

In 1977 Hercules Chemical Corporation (Hercules) petitioned the Consumer Product Safety Commission (CPSC) to ban sulfuric acid drain openers (SADOs) from consumers and to limit their use to trained professionals.

The CPSC's Technical Advisory Board (TAB) enthusiastically endorsed the petition but before the ban could be implemented an ad hoc group of SADO producers calling themselves the Associated Chemical Producers (ACP) began to lobby the CPSC to oppose the ban. As a result, in 1981 the CPSC decided to reverse itself on the proposed ban, much to the expressed and documented disappointment of Hercules. There was another petition to the CPSC to ban SADOs in 1994 by Roger Wabeke, a consultant in chemical risk management. The CPSC performed another review of injury data and in 1996 again decided against banning SADOs. The photo below is an example of an injury that could have been avoided if the CPSC had banned SADOs from consumer use in 1977 or 1994.

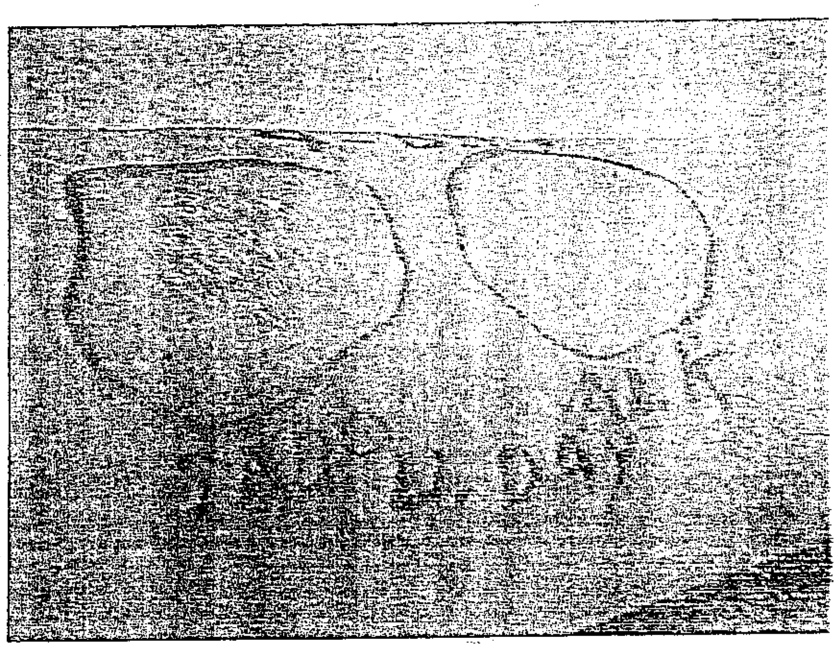
2001 SADO Injury

Because of my involvement as a chemical expert in the accident that led to the young boy's injuries pictured above I have had the opportunity to review the CPSC's actions in 1997, 1981, 1994 and 1996. This report documents my findings and conclusions based on that in-depth review.

2.0 CPSC's 1981 and 1996 Decisions

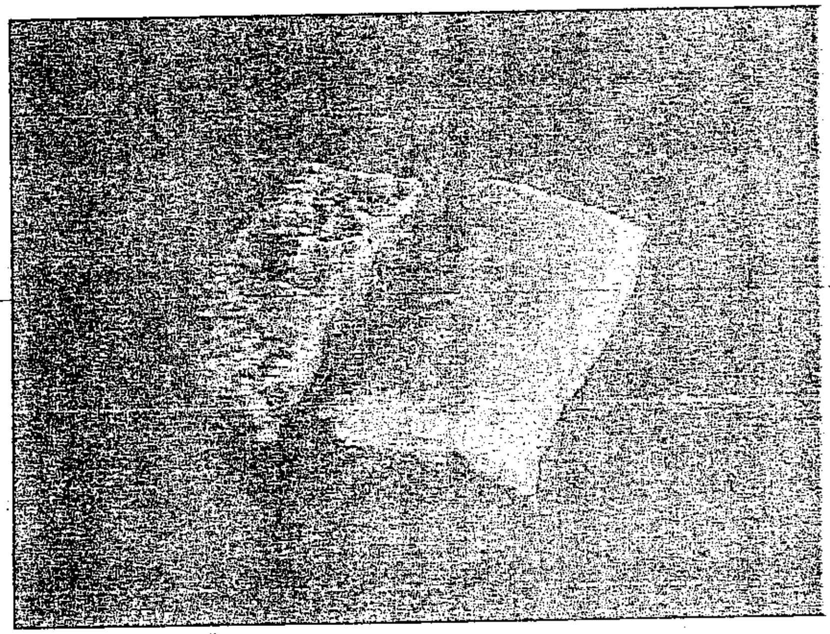
The Commission never said that SADOs were not dangerous. They simply said that SADOs were no more dangerous than alkali drain openers (ALKDOs). For the record, in my petition to the CPSC to ban SADOs I have offered to perform a public demonstration. I will pour an amount of a common ALKDO on my forearm while a member of the CPSC pours an equal amount of a concentrated SADO on their forearm. We will then determine who runs for the water first and whose injuries are the greatest.

The reason I can make this challenge is that, in addition to seeing the horrific results of SADOs as the young boy pictured above, I have performed the proposed demonstration on my own arm using both ALKDO and SADO simultaneously. I applied the ALKDO first and then the SADO, and then rinsed them both off once the SADO became a serious concern, which was with in 25 seconds. The picture above illustrates the results after four days. The SADO produced a second-degree burn (which later scabbed over) while the ALKDO did not even cause minor skin irritation. There can be little doubt that SADOs are more dangerous than ALKDOs.



Comparison of SADO vs. ALKDO Skin Contact 4-Day Results
SADO contact time was 25 seconds
ALKDO contact time was 37 seconds

I have also performed exposure tests on pigskin for longer periods of time. The results are shown below. The sample on the left was exposed to a SADO while the pigskin on the right was exposed to an ALKDO.



SADO vs. ALKDO Tests on Pigskin
Sample on Left was exposed to SADO
Sample on the right was exposed to ALKDO

3.0 CPSC's 1981 and 1996 Actions

Instead of the 1978 ban, the Commission decided to allow the SADO industry make voluntary improvements in packaging and labeling.

Unfortunately, the CPSC never realized that the voluntary industry effort to improve the safety of SADOs was being put into the hands of a single individual who did not understand the first thing about the chemistry of sulfuric acid, did not have any formal chemistry education or training, did not have any label or human factors education or training, did not have any packaging training, and he was not going to hire anyone (even as a temporary consultant) who had the proper education and training to help him and the ad hoc Association of Chemical Producers (ACP) make SADOs safer for consumer use. In other words, I do not believe that the CPSC realized that they were turning over this important public safety mission mostly to one individual who was basically void of any type of education or training relevant to the chemical safety task at hand. More importantly, as a recent 2002 SADO burn injury shows, the ACP has not convinced other SADO manufacturers to comply with the voluntary standards.

4.0 Flaws in CPSC Logic

The fundamental flaw in the CPSC's logic when they reversed their ban was to compare the percentage of sales of SADOs to the percentage of SADO injuries relative to the total sales and injuries of all liquid chemical drain openers (see June 20, 1996 response to Petition HP 95-3). On that basis, it appeared that SADOs were no more hazardous than ALKDOs. In other words, it appeared that the percentage of SADO sales was proportional to the percentage of SADO burns.

However, the CPSC did not pay close attention to their own in-house data to the effect that only 1/3rd of the SADOs were sold to ordinary consumers and 2/3rds were sold to professionals. Since professionals are trained in the use of chemicals (per the OSHA Hazard Communication Standard) they would be far less likely to sustain a chemical injury. Furthermore, the CPSC excluded work-related injuries. When you compare sales to ordinary consumers (3.1%) relative to the SADO injuries (11%) it becomes clear that SADOs are at least 3.5X more dangerous than ALKDOs.

The staff of the CPSC (see Roy Sammarco Memo dated February 24, 1981) suggested that the consumer's exposure to ALKDOs might be as much as 280 times higher than their exposure to SADOs. If true, SADOs should contribute only 0.36% of all drain opener burns. Yet, SADOs account for at least 11% of all burns (which I believe is an under-estimate). These figures suggest that SADOs may be 30X more hazardous than ALKDOs.

5.0 Voluntary Label Improvements

In Table 1 of Tab B (page 5 of the February 1996 report) there were 120 injuries due to SADOs in 1980. In 1981 the ACP stepped in with their improved label and the injury rate never went below 120 since. It was 170 in 1982 and other years were all above 200. In 1993 there were 680 SADO injuries. This strongly contradicts any notion that the "improved" labeling by the ACP had any measurable effect on injuries. The years close to 1980 (81 & 82) showed marked increases thereby ruling out the gradual increase in sales of SADOs over the years to account for the increases in injuries.

Even a manufacturer of SADOs testified that you need to see reduction in injuries to establish that the new warnings are working. Since we do not see a reduction in injuries it can be concluded that the warnings are not working.

6.0 Risks v. Benefits

There is absolutely no merit to the argument that SADOs are easy to use and offer sufficient economic benefits to offset their risk. It is not a life-saving or otherwise essential chemical product. It is simply a drain opener. There are other, less-risky and less-costly ways to unclog a drain.

Assuming the average number of yearly SADO injuries is 354 (per CPSC 1981-1994 data) and the *total* average cost per injury is $100,000, the total annual cost of SADOs calculates out to be $35.4 million. Over a ten-year period that becomes $354 million. Note ACP's own data indicates 391 injuries in 1977 alone.

Had the *consumer* SADO industry shut down in 1980 the economic impact would have been the loss of about 73 jobs (per ACP documents). Admittedly, this did not include all SADO producers, but it could be argued that 2/3rds of the 73 jobs are in the production of SADOs for professional use.

U.S. companies lay off 1,000's of workers almost every day and firms like World Com, Kmart and Enron go bankrupt. These laid-off people eventually find other employment, most likely within a year or less. Hence, the total economic loss to the SADO industry would be on the order of 1/3 x 73 = 24 employee-years. If each employee-year was worth $30,000 that would calculate out to be $0.72 million. That doesn't even come close to the $35.4 million in SADO injuries in one year or $354 million in ten years. This does not consider the toll in terms of the immediate pain and suffering of a SADO victim or the longer-term emotional consequences of disfigurement. Also, it is likely that jobs would be gained in the ALKDO industry. The same logic might be applied to plungers and snakes. More of those might be sold and there would be more jobs in those industries.

It should be further recognized that a *consumer* ban on SADOs would not be a total ban. Companies that once focused on the consumer market could re-direct their efforts at professional plumbers who would be able to continue the use SADOs. The SADO producers might also re-direct their efforts at alkali drain openers for consumer use.

Furthermore, if the CPSC took the position that SADOs should only be sold in one-shot containers, no jobs would be lost from the SADO industry.

The above discussion of risks and benefits does not even include the well-known corrosion and materials problems that SADOs can cause when misused in plumbing systems.

7.0 SADO Crime

Sulfuric acid is a dangerous chemical. In Bangladesh, throwing acid in a woman's face is a growing crime, called the *barbaric crime of the century*." Some Bangladesh men have thrown acid in the face of women after they refuse a marriage proposal. Many women are blinded, lose their hearing, or die.

In Ohio, a man was sentenced 16 years in prison for throwing acid on four people. An article in The Journal of Trauma, March 1998 reported that the University of Louisville Kentucky School of Medicine noticed a substantial number of both accidental *and intentional* burns caused by SADOs. Of 21 reported SADO burn cases, 13 involved the use of SADO as a weapon.

The only reason SADO crime is mentioned in this report is to further show that SADO is a powerful chemical capable of horrific injuries and crimes. It many ways, it is an ultimate personal weapon perhaps more feared than a gun or knife.

8.0 CPSC History of Product Recalls

The CPSC will recall and ban products that present far less danger to society than SADOs. The following is brief list of examples of products that have been recalled:

1. CPSC recalled 136,000 cans of Fire Cap fire and smoke suppressant even though it was not aware of any injuries. The recall was conducted to prevent *possible* injuries.

2. CPSC recalled 190,000 cans of Party Time "Happy String" because it is flammable and one four-year old boy received only 1st and 2nd degree burns on his face and arm. A woman was burned on her ear in another incident.

3. CPSC recalled 912,000 can of "Crazy Ribbon" and "Crazy String" because of flammability. One 11-year old boy suffered serious burns that left permanent scars.

4. CPSC recalled 80,000 Martha Stewart Potpourri Simmering Pots. One consumer received minor burns.

5. CPSC recalled 618,000 Star Wars Light sabers. There were 3 reports of minor burns and one consumer experienced an eye irritation.

6. CPSC recalled 24,000 Martha Stewart Brand Tea Kettles. There were 3 minor burn injuries.

7. CPSC recalled 296,000 cans of aerosol "Smatter" spray foam. One child reportedly suffered a minor bump on the head when a can of Smatter broke apart (exploded) in a hot car.

8. CPSC recalled 200,000 cans of Simonize Quick Gloss because the aerosol container may rupture (explode). No injuries were reported.

9. CPSC recalled 500,000 cans of Shave Gel because the container may corrode and rupture. No injuries were reported.

10. CPSC recalled 124,400 Soap Making Kits. There were 10 reports of children being burned.

11. CPSC recalled 50,000 cans of EASY-OFF because an improperly attached valve assembly can separate from the can. There were 12 reports of burn injuries to skin and eyes. Note that EASY-OFF is chemically similar to alkaline drain openers.

9.0 CPSC History on Product Bans

The following is a very brief list of some products that were ban by the CPSC. Note that item B is the ban of a particular product size.

A. CPSC banned Large Reloadable Shell Fireworks. There were a total of 39 incidents from 1985 thru 1991. During this 6-year period 31 injuries were reported or 5.16 per year. This should be compared to the 3,271 injuries per year from chemical drain openers and the 354 from SADOs.

B. CPSC banned extremely flammable contact adhesives sold in larger than one-half pint containers. Since 1970 there had been 130 injuries including 15 deaths, or 4.81 injuries per year and 0.55 deaths per year. This should be compared to the 3,271 injuries per year from chemical drain openers. **It should also be noted that this was a ban of a SIZE of a product.**

C. CPSC banned toy phonographs because of possible electric shock. No injuries had been reported.

The point of listing some examples of CPSC recalls and bans is to accentuate the inconsistency of the CPSC in performing their duty to protect the consumer from unreasonable risk.

10.0 Kleenex Syndrome

Another flaw in the CPSC's earlier process is what I call the "Kleenex Syndrome." If you ask someone for a Kleenex, they might hand you a Charmin Bath Tissue. The problem is that Kleenex has become a descriptor for an entire product type. Somewhat like a Xerox copy, even though Xerox is no longer the most common copying machine.

Likewise, Liquid Plumber has become more or less a generic term for liquid drain openers. Therefore, the comments section of the NEISS database might say that Liquid Plumber was the cause of a patient's burns when in fact it may have been another type of liquid drain opener.

Since Liquid Plumber is an ALKDO, those burns would automatically fall into the ALKDO column and might be missed as SADO burns without further questioning of the patient. Also, the hospital staff might enter Liquid Plumber in the Comments Section even if the patient said it was some other type of liquid drain opener. Even the patient themselves might be confused and say it was Liquid Plumber as the distinction between SADO and ALKDO is not immediately obvious to the ordinary consumer.

Most would agree that people are as likely to spill Coke on themselves as they are Pepsi. Both Coke and Pepsi come in the same sizes and shapes of containers and are about the same weights and slipperiness. The same is true for SADOs and ALKDOs. Therefore, one would expect people to be about as likely to spill a SADO on themselves as they are an ALKDO.

Concentrated sulfuric acid (93-99%) is so much more hazardous than the typical 10% sodium hydroxide solutions used in ALKDOs that it simply does not make sense that SADOs and ALKDOs present the same danger. There are at least five mechanisms by which SADOs can damage skin (heat, acid, dehydration, oxidation and sink eruptions) while ALKDOs present the single mechanism of alkalinity. In other words, the likelihood of exposure would be expected to be proportional to sales, but the odds of injury are much greater for the SADO.

The bottom line of the Kleenex Syndrome is that the data found in the comments sections of NEISS databases is likely to be falsely weighted toward ALKDOs as the cause of burns. And the Coke-Pepsi analogy tells us that the likelihood of injury from a SADO exposure is far greater than for an ALKDO and any conclusion to the contrary simply doesn't make sense.

11.0 CPSC – Miscellaneous Notes

In 1981, there was no consensus in the CPSC about a course of action re SADOs. The team agreed that the theoretical evidence of the potential of SADOs to cause immediate and severe injury plus the apparent difficulty by consumers in safely following label instructions support the contention that the SADOs should be banned for all but professional use. The Directorate for Compliance and Administrative Litigation said that the hazards of SADOs could be addressed adequately only through a banning action. The Directorate of Health Sciences abstained from making a recommendation, but in 1978 they made a very strong recommendation to ban SADOs. Hence, it does not appear that there was any real consensus within the CPSC about SADOs. The Commission appeared divided, even in the face of ACP lobbying.

Per CPSC, between 1980-1994 there have been 5,070 SADO related injuries. (Total chemical drain opener injuries is 49.070.) 11% involved hospitalization or death. 11% x 5,070 = 557.7. It's difficult to compare the economic hardship of 73 healthy workers who might have to find new jobs to 557.7 who may not ever be able to work again or could be seriously handicapped. Assume that all 73 of the ACP workers would have been out of work one year. The total loss would have been 73 man-years. Some of that loss would have been covered by unemployment benefits. Some by companies finding other positions for the laid-off workers. There might have also been some type of job training program to benefit these 73 workers in the longer term. One would also want to look at employee turnover in the SADO industry before making a firm judgment about the long-term (employee) cost of a ban. Filling bottles with concentrated sulfuric acid would not seem to be a highly sought after career.

If the average injury rate of 354/yr from SADOs were projected from 1994 to 2004, there would have been another 3,540 injuries thru 2004 of which 11% (389) would have involved hospitalization or death. Compare this to 73 (uninjured) industry workers who might have to look for another job.

CPSC had 16 Death Certificates associated with chemical drain openers 1980-1994. At least three were known to be for SADOs. Assume these 3 SADO-injured people each had an average of 25 working years remaining, the lost man-years from the economy workforce was 75. That is greater than the 73 man-years that would have been lost had the ban been enforced. This does not include the deaths or man-years lost since 1994 thru 2004, another 10 years. It does not include lost man-years from injuries without death.

Keep in mind that 2/3 of the SADO sales are to professionals, therefore the loss of all 73 jobs would have been unlikely.

In 1981 the CPSC recommended the following for SADOs:

- Low-Flow containers
- Low-spill containers
- A time-based safety factor
- One-shot containers
- Outer cardboard boxes to allow for more prominent warnings and instructions.

The Ad Hoc ACP report to CPSC in 1981 states:

> "However, if for any reason the Commission determines that additional safety measures should be considered, ACP pledges its full cooperation to assist the Commission in that regard."

- Did ACP use one-shot containers? No.
- Did ACP use containers less likely to tip over? No.
- Did ACP use low-flow containers? No.
- Did ACP use a time-based safety factor? No. (Except One)

To my knowledge, none of the above CPSC recommendations were implemented by the SADO industry.

It is my further opinion that:

a) SADOs present a high degree of risk of harm to a person,
b) The harm to an untrained consumer will most likely be great,
c) The risk is difficult to eliminate by the exercise of reasonable care or labeling given that accidental spillage is a common human occurrence, particularly for the ordinary and untrained consumer who also is not trained how to respond in a timely manner.
d) The use of concentrated sulfuric acid is not a matter of common usage by the ordinary consumer, and
e) Risks greatly exceed benefits.

In many states, the above criteria fit the legal definition of an ultra hazardous activity.

12.0 Summary and Recommendations

It is my professional opinion, as someone who has spent his entire professional life in the field of chemistry, chemical safety and chemical accident investigation that:

1) Sulfuric acid drain openers (SADOs) are unreasonably dangerous and should not be sold to ordinary consumers. SADOs can cause horrific injuries and are unsafe when used in a reasonable and foreseeable manner by ordinary consumers. The risk of danger inherent in SADOs greatly outweighs their benefits.

2) SADOs should only be sold to plumbing professionals who have had the benefit of the training required by the OSHA Hazard Communication Standard.

3) If SADOs MUST be sold to ordinary consumers, they should be packaged in one-shot containers, and

4) If SADOs MUST be sold to ordinary consumers in one-shot containers, they should not be greater than 84% in concentration in order to provide a (slight) time-based safety factor and to reduce the thermal component of SA injury.

TAB B

UNITED STATES
CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, DC 20207

Memorandum

DATE: August 3, 2005

TO : Kristina Hatlelid, Ph.D., M.P.H., Project Manager for the SADO Petition

THROUGH: Mary Ann Danello, Ph.D., Associate Executive Director, Directorate for Health Sciences

Lori E. Saltzman, M.S., Director, Division of Health Sciences

FROM : Patricia M. Brundage, Ph.D., Pharmacologist, Division of Health Sciences

SUBJECT : Toxicity Review of Sulfuric Acid Drain Openers

I. Introduction

The U.S. Consumer Product Safety Commission (CPSC) received a request from Michael Fox (petitioner) to issue a ban on sulfuric acid drain openers (SADOs) for use by consumers, or in the alternative, to require packaging of sulfuric acid in single use containers with a maximum sulfuric acid concentration of 84 percent. This request was docketed as petition number HP04-2. The petitioner asserts that such action is necessary because SADOs present an unreasonable risk of injury to the ordinary consumer.

This memorandum describes the potential health effects from exposure to sulfuric acid, which is found in concentrated form in certain drain openers. The health effects associated with alkalis will also be presented for comparison since they constitute the majority of the drain openers on the market (Franklin, 2005), and they are potential substitutes if sulfuric acid drain openers were no longer available.

There are a number of chemical drain openers on the U.S. market including liquid acid products, liquid alkaline products, and granular alkaline products. Concentrated liquid acid drain openers typically contain between 84 and 94 percent sulfuric acid generally with a pH of one or less. Other acid drain openers contain hydrochloric acid in concentrations of 5 to 30 percent (pH<1). The liquid alkaline and granular alkaline drain openers contain sodium or potassium hydroxide (i.e., alkalis) and other ingredients such as sodium hypochlorite. The concentrations of the hydroxides in the granular drain openers range from 60 to 100 percent, whereas the sodium hydroxide in liquid alkaline drain openers can range from 2 to 40 percent with pH values ranging from about 11.5 to 14. The potassium hydroxide in other liquid alkaline drain openers ranges from 2 to 45 percent. Both alkaline and acid drain openers react with the organic matter and material of the drain blockage via chemical reactions. The heat generated by the drain openers also liquefies the blockage.

Dilute sulfuric acid (i.e., concentrations less than 10 percent) is considered a primary irritant, whereas sulfuric acid in concentrated form is a strong corrosive. Strong alkalis are also markedly corrosive. Both sulfuric acid and alkalis are direct acting toxicants; systemic reactions are the result of local tissue injury. Exposure can occur from skin or eye contact, inhalation of mist/vapor, and ingestion. Both acids and alkalis can cause death from a number of complications including circulatory shock, asphyxia from glottic or laryngeal edema, gastrointestinal tract perforation, intercurrent infection, or stricture formation (Gosselin et al., 1984).

As hydrochloric acid can cause health effects similar to those caused by sulfuric acid, this memo will not specifically address the clinical consequences of hydrochloric acid drain opener exposures, but will address the clinical consequences of all acid drain opener exposures. Similarly, this memo will not deal with the injuries specifically caused by potassium hydroxide, as they are similar to those caused by sodium hydroxide drain openers, but will provide comparative clinical information on alkalis, in general. Though, the focus will be principally on the higher concentration alkalis because they are the closest substitute for sulfuric acid drain openers (Franklin, 2005).

Hazardous household substances containing sulfuric acid are generally subject to the Federal Hazardous Substances Act (FHSA). In addition to other cautionary labeling required by the FHSA for hazardous household substances containing sulfuric acid, sulfuric acid and any preparation containing 10 percent or more of free or chemically unneutralized sulfuric acid requires the word "POISON" in place of a signal word as a result of the Federal Caustic Poisons Act. 16 CFR § 1500.129(b). Such products are generally corrosive meaning that they can cause "visible destruction or irreversible alterations in the tissue at the site of contact." Likewise, hazardous household substances containing free or chemically unneutralized sodium hydroxide in a concentration of 10 percent or more require the word "POISON." 16 CFR § 1500.129(j).

Additionally, the Poison Prevention Packaging Act (PPPA) requires special packaging (i.e., packaging that is child-resistant (CR) and senior-friendly) for hazardous household substances containing 10 percent or more of sulfuric acid, except such substances in wet-cell storage batteries. 16 CFR § 1700.14(a)(9). Likewise, hazardous household substances containing 10 percent or more sodium and/or potassium hydroxide in dry form such as granules, powder, and flakes or 2 percent or more in any other form require CR packaging under the PPPA. 16 CFR § 1700.14(a)(5).

II. Background on Acids and Alkalis

There are some fundamental differences between acids and alkalis. An acid is a compound which releases hydrogen ions (H^+) in an aqueous solution, and an alkali is a compound that produces hydroxide ions (OH^-).

The extent to which a substance dissociates defines the strength of the acid or alkali (Bates, 1999). The strength of an acid or alkali is an intrinsic chemical property and is unrelated to the concentration of the acid or alkali. Strong acids in solution dissociate more or less entirely to release hydrogen ions. Some examples of strong acids include hydrochloric acid (HCl), sulfuric acid (H_2SO_4), perchloric acid ($HClO_4$), and nitric acid (HNO_3). Weak acids will only partially dissociate in water, with only a small fraction of their hydrogens becoming ions. Acetic acid and carbonic acid are considered weak acids. In the same manner, strong alkalis dissociate almost entirely in solution to release hydroxide ions, while weak alkalis only partially dissociate, releasing a smaller fraction of their hydroxide ions. Sodium hydroxide (NaOH) and potassium hydroxide (KOH) are examples of strong alkalis, while sodium bicarbonate and ammonia are examples of weak alkalis. In general, the degree of dissociation correlates with the degree of potential tissue damage.

Concentration is a quantitative measure of the amount of one substance in a given amount of a sample. In this memo, concentration is a measure of the amount of dissolved substance present in a given amount of solution. The concentration can be expressed as a percentage (i.e., the weight of a substance in a given volume) or as a molarity (i.e., the number of molecules in a given volume). A strong acid or alkali can be concentrated or dilute depending on the amount of the acid or alkali present in a given volume of solution.

The logarithmic pH scale[1] measures the concentration of hydrogen ions, ranging from 0 to 14 with the value of pH 7 indicating neutrality (i.e., neither acidic or alkaline). A strong acid, which dissociates almost entirely in water to release hydrogen ions, may have a pH of one or less, even at concentrations below one percent. Above pH 7, the concentration of hydroxide ions is greater than the concentration of hydrogen ions. A strong alkali, which dissociates more or less entirely in a higher pH solution, may have a pH of 13 or more, even at concentrations below one percent.

III. Toxicity

A. Mechanism of Injury

The tissue damage associated with an acid is correlated to the amount of free hydrogen ions that dissociate from that acid. For alkalis, the free hydroxide ions are responsible for the tissue damage. Consequently, the mechanism of injury differs somewhat between acids and alkalis (Donner et al, l98l).

[1] The logarithmic pH scale measures the concentration of hydrogen ions; a decrease in pH by one unit is equivalent to a 10-fold increase in the concentration of hydrogen ions.

Acids primarily cause injury to tissue by a process called coagulation necrosis[2], which is characterized by the formation of a coagulum or eschar (i.e., a crust formed on the surface of a wound) that can serve to limit the penetration of the acid and prevent further damage. Hydrogen ions are highly reactive and readily react with proteins disrupting their structure and ability to function (i.e., denaturation). Concentrated acids also release considerable heat causing thermal damage as they induce dehydration of the cells at the injury site (Sykes et al., 1986). The result is a brittle spongy black mass of carbon. Moreover, the osmotic gradient created by the cellular desiccation causes water to move from the plasma to the injured tissue producing marked edema[3] at the site of injury.

In contrast, alkaline agents cause cellular death as a result of emulsification and disruption of cellular membranes, which leaves the affected areas soft, gelatinous, and friable. This process, referred to as liquefaction necrosis[4], involves fat saponification[5] and solubilization of proteins and collagen. The resulting injury does not limit tissue penetration and can cause deep tissue destruction. Initially, the free hydroxide ions of the alkali bind to fat and protein molecules in the tissue as a result of the high pH and are consumed in the reaction. However, the lower pH of the fluids surrounding the affected tissue can regenerate hydroxide ions. The hydroxide ions can then react with other proteins and fats in a cascading reaction until the hydroxide concentration is diluted with either tissue fluid or externally added fluids to stop the regeneration. Alkaline agents also have a dehydrating action on cellular tissue at the site of the injury (Yano et al., 1993).

B. Factors Influencing Extent of Injury

The severity of the burn is related to a number of factors including the concentration of the agent, the pH of the agent, the site of exposure, the duration of contact, and the amount of the agent involved.

Concentrated acids and alkalis have the capability of causing more severe burns than do less concentrated forms. Extremes of pH also have a greater potential to cause tissue injury (Homan, 1993). The pH of cells must be maintained within a narrow range, otherwise cellular function diminishes or cellular death ensues. The closer the pH value of a substance is to 7.4 (i.e., normal physiological pH), the less damage it causes due to pH disturbance.

The degree of cellular damage is also determined by the type of tissue and area exposed. Skin varies in thickness based on anatomic location. Keratinized epidermal depth varies considerably by body area, from less than

---

[2] Coagulation necrosis is the term given to the structural changes indicative of cell death, in which tissue becomes a dry, opaque mass resulting from the breakdown of proteins.
[3] Edema is the presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body.
[4] Liquefaction necrosis is the death of living tissue which has become softened and liquefied.
[5] Saponification is the breakdown of fatty acids and lipids into soap and glycerol.

1 millimeter (mm) in the thinnest areas (eyelids, genitals) to 5 mm (palms and plantar surfaces), offering varying degrees of thermal protection. Mucosal membranes[6] and the eye are also more vulnerable to injury from acids and alkalis.

Furthermore, the severity of the injury is directly proportional to the length of time the substance remains in contact with the exposed tissue. Quick removal of the substance and dilution with water significantly reduces the extent of injury (Leonard et al., 1982; Mozingo et al., 1988).

C. Classification of Injury

Burns are classified by the depth of injury to the skin. The depth classifications of burn injuries include superficial, superficial partial-thickness, deep partial-thickness, and full-thickness. Superficial burns, characterized by red skin and no blisters, can be very painful but involve only the superficial layer of skin (i.e., epidermis). Superficial partial-thickness burns involve the epidermis and superficial dermis. These erythematous[7] and painful burns are characterized by ruptured weeping blisters and typically heal within 1 to 3 weeks, usually without scarring. Deep partial thickness burns cause injury to the epidermis and deeper dermis; some viable dermis remains. Blister formation, with or without denuding, and pink to mildly pale tissue with intact sensation indicate deeper partial-thickness injury. Full-thickness burns destroy all layers of the skin (i.e., both epidermis and dermis) with damage possibly extending beyond the skin to the nerves, blood vessels, muscle, fat, and bone. Full-thickness injuries, characterized by areas that are leathery and insensitive, will not heal spontaneously.

D. Signs and Symptoms of Exposure

1. Dermal Exposure

a) Acids

Depending on the pH, duration of contact, strength, and concentration, acids can cause a variety of injuries to the skin including erythema, blistering, and penetrating ulcers[8] (i.e., superficial and deep partial-thickness burns). In severe cases of dermal exposure (e.g., delayed washing of substantial exposure), concentrated sulfuric acid is capable of causing full-thickness burns destroying the skin, muscle, fat, as well as the underlying bone (Mozingo et al., 1988). This is a result of tissue desiccation and excessive heat generation.

The necrotic tissue typically separates in two to three weeks, resulting in a well demarcated ulceration that will fill in from below with new tissue growth,

---

[6] Mucosal membrane is the moist tissue lining some organs and body cavities which secretes a mucous.

[7] Erythema is redness due to capillary dilation.

[8] Ulcers are lesions, usually with inflammation, through the skin or mucous membranes which result from loss of tissue.

which is typically very fragile and contains a high density of blood vessels. The later period of healing may be characterized by scar formation and contracture[9].

Significant injuries may be associated with a systemic response caused by a loss of the skin barrier, the release of vasoactive mediators from the wound, and subsequent infection. Metabolic acidosis[10] and hemolysis[11], which can cause renal damage, may also occur (Hummel, 1982; Sigurdsson et al., 1983). Another potential complication associated with severe acid burns is circulatory collapse.

Moderate concentrations of acids are relatively well tolerated on human skin. In a controlled experiment, a 10 percent sulfuric acid solution applied to both intact and abraded human skin for 4 hours was considered non-irritating (Nixon et al., 1975). However, in rats and mice, 1 mL/kg of a 10 percent sulfuric acid solution placed on unoccluded electric razor-shaved skin of the back caused erosion of the skin; the application area was examined one week after application (Sekizawa et al., 1994). In the same study, a 5 percent solution caused erythema and edema, and a 2.5 percent solution had no effect.

b) Alkalis

Alkali substances can cause injury ranging from diffuse erythema of the skin at the site of contact (i.e., superficial burns), to severe full-thickness burns requiring skin grafting. A 4 percent solution of sodium hydroxide applied to the skin of volunteers' arms for 15 to 60 minutes produced damage that progressed to total destruction of all layers of the epidermis (i.e., outer layer of skin) within 60 minutes in several subjects (Nagao et al., 1972).

Another study demonstrated that a 50 percent solution of sodium hydroxide took longer than a 96 percent solution of sulfuric acid to cause tissue injury (Davidson, 1927). Immersion of the hind leg of a rat in 96 percent sulfuric acid for 15 seconds immediately caused edema and redness, while a 50 percent solution of sodium hydroxide produced a similar degree of tissue injury five minutes after a one-minute immersion. Vigorous washing with water of the extremity exposed to the sulfuric acid caused the redness and edema of the extremity to subside within 24 hours. Equivalent treatment of the extremity exposed to sodium hydroxide resulted in moderate edema of the toes and excoriation (i.e., abrasion) of the skin of the foot which healed without deformity.

Concentrated sodium hydroxide applied to human skin caused a delayed sense of irritation compared to the relatively rapid perception of acid (Davidson, 1927). Subjects perceived a 96 percent solution of sulfuric acid after

---

[9] Contracture is a state of permanent stiffness or contraction of the muscles.
[10] Metabolic acidosis is a decrease in the pH in the body fluids because of loss of alkali or accumulation of acids.
[11] Hemolysis is the destruction of red blood cells (i.e., erythrocytes) with the release of their cellular content.

approximately 4 seconds, and a 50 percent sulfuric acid solution after approximately 50 seconds. In comparison, 25 and 50 percent sodium hydroxide solutions were not perceived during the 3 minute test. The 10 and 25 percent solutions of sulfuric acid also produced no stimulation after 3 minutes of contact. Another study demonstrated that a solution of 4 percent sodium hydroxide solution does not cause a sensation of irritation until after several hours (Morris, 1952). This suggests that the sodium hydroxide drain openers, such as those found in grocery, drug, and convenience stores, may not be perceived on the skin before skin damage occurs (Nagao et al., 1972).

2. Ocular Exposure

The eyes are especially susceptible to damage from acids and alkalis due to their limited ability to buffer extremes in pH and their reduced regenerative potential. Agents with a pH level of ≤ 2 or ≥ 11.5 are generally accepted by both U.S. governmental agencies, including the CPSC, the Food and Drug Administration (FDA), the U.S. Environmental Protection Agency (EPA), and international groups, including the European Union (EU) and the committee on the Global Harmonization System (GHS), as the pH levels at which ocular corrosive injury can occur. Splash contact with concentrated strong acids and alkalis can cause severe injuries. Typically, weak or dilute acids cause less damage than weak or dilute alkalis.

a) Acids

Fine sprays of sulfuric acid in the air can cause acute stinging and burning of the eyes in addition to lacrimation, but the rapid dilution of the acid by the tears typically prevents significant injury (Flury and Zernik, 1931). Sulfuric acid vapors may cause a transient punctuate keratoconjunctivitis[12] (Klein, 1928).

Direct exposure to low concentrations typically results in transient injury with full recovery (Grant, 1974). Dilutions of sulfuric acid up to 0.03 M (pH 1.75) applied as a drop to the normal human eye caused an immediate burning sensation persisting for about an hour. However, this dilution (0.03 M) had no influence on vision and caused no residual evident clinical damage.

Weak or dilute acids cause damage as a result of pH alteration, and protein precipitation and denaturation in the corneal epithelium and superficial stroma (Friedenwald et al., 1946). However, this protein coagulation, which causes some turbidity[13] of the cornea that may or may not be reversible depending on the extent of damage, functions as a barrier preventing further penetration and protecting the anterior structures (i.e., iris, ciliary body, and lens). Furthermore, many corneal proteins bind the acid and act as chemical buffers (Dua et al., 2001).

[12] Punctuate keratoconjunctivitis is small areas of inflammation of the cornea and conjunctiva.
[13] Turbidity is the loss of transparency because of sediment or insoluble matter.

Burns caused by weak or dilute acids may cause increased blood flow, edema, and small ecchymoses[14] in the conjunctiva (Grant and Shuhman, 1993). Recovery from such ocular surface burns is dependent on the extent of damage to cornea, limbal cells (corneal stem cells), and conjunctiva tissues, as the limbal cells and conjunctival epithelium are capable of repopulating the corneal epithelium following injury (Dua et al., 2001). The epithelium usually regenerates if there has not been a total loss of both the limbal and conjunctival epithelium, and the outcome is favorable.

Concentrated acids can rapidly penetrate the ocular tissue. Depending on the degree of penetration, concentrated acids can damage the corneal and conjunctival epithelium, stromal nerve endings, endothelium, lens epithelium, and vascular endothelium of the conjunctiva, iris, and ciliary body (Wagoner, 1997). Penetration of the acid through the corneal and conjunctival epithelium causes shortening of collagen fibrils which elevates intraocular pressure. Furthermore, a marked inflammatory response, involving leukocyte infiltration and the release of inflammatory mediators due to the presence of necrotic tissue, can cause corneal and conjunctival ulceration, tissue proliferation, and scarification. The considerable heat produced by concentrated sulfuric acid also causes thermal damage (Wagoner, 1997). In monkeys, concentrated sulfuric acid injury has been shown to result in poor adhesion of regenerated epithelium due to a permanent alteration of the basement membrane and stroma edema (Hirst et al., 1982). Clinical outcomes of severe sulfuric acid eye injury that have an impact on eyesight include glaucoma (Peyresblanques and Le Goff, 1965), cataracts (Homan, 1993; Peyresblanques and Le Goff, 1965), and severe inflammation of the retina (Homan, 1993).

b) Alkalis

Alkalis are damaging to ocular tissue, especially if the eye is not immediately irrigated. A pH of 11.5 was determined to be a general guideline for the prediction of ocular injury (Grant and Kern, 1955; Murphy et al., 1982). Alkalis attack the membrane lipids of the eye, causing liquidation necrosis. Alkalis penetrate the ocular tissues, quickly damaging the corneal stroma, epithelium, and endothelium, as well as the anterior structures of the eye (Paterson and Pfister, 1974; Paterson et al., 1975). Injury may also occur as a result of collengenase (i.e., enzyme that breaks down collagen proteins), which is released from the cells in the cornea when the cornea is damaged (Jarudi and Golden, 1973).

Perforation of the cornea was observed one minute after rabbit eyes were exposed to approximately 0.6 mL of a 4 percent sodium hydroxide solution (Renard et al., 1978). Renard et al. (1978) also found that the same amount of a two percent solution of sodium hydroxide caused reversible destruction of the corneal epithelium and damage to the endothelium that remained unhealed by

[14] Ecchymoses are small spots of ruptured blood vessels in the skin or mucous membrane forming a nonelevated, rounded or irregular, blue or purplish patch.

day 28. Larger amounts of lower concentrations of sodium hydroxide were also shown to cause ocular tissue damage. Twenty drops (approximately one milliliter) of a one percent sodium hydroxide solution (pH 12.7) applied to the eyes of rabbits caused the destruction of both the corneal endothelium and epithelium after a one minute exposure (Bolkova and Cejkova, 1984). When the concentration was reduced to 0.4 percent, the solution caused partial reversible destruction of the corneal epithelium without damaging the endothelium. A 0.2 percent solution caused only a slight decrease in epithelial alkaline and acid phosphatase levels which returned to normal within a week. In another study (Murphy et al., 1982), a one percent solution of sodium hydroxide (pH 13.1) instilled into the eyes of six rabbits caused corneal opacity in three of the six animals. When the concentration was reduced to 0.3 percent (pH 12.8), no corneal opacity was observed.

3. Ingestion

Strong acids and alkalis can severely damage the gastrointestinal tract if ingested. A recent retrospective study that evaluated the late sequelae of corrosive injury to the upper gastrointestinal tract caused by the ingestion of both acids (n=120) and alkalis (n=35) found that both damage the esophagus and stomach with an equal degree of severity (Nagi et al., 2004).

a) Acids

Ingestion of acid can injure the mucous membranes of the mouth, throat, esophagus, stomach, and upper portion of the small intestine (duodenum). The outcomes of acid ingestion are quite variable. Damage to the gastrointestinal tract, established by endoscopy, is graded by means of a commonly used scale (Zargar et al., 1991). Injury to the tissue lining the upper gastrointestinal tract ranges from edema and erythema (grade 1); to superficial localized ulcerations and blisters (grade 2A); to circumferential lesions (grade 2B); to multiple, deep ulcerations and areas of extensive necrosis (grade 3); to perforation (grade 4). Extensive necrosis and perforation are more common following the ingestion of a large volume of acid, or a highly concentrated acid (Christesen, 1995; Zarkovic et al., 1997).

The lethal dose and the time before death are difficult to predict with any useful degree of accuracy (Gosselin et al., 1984). In an adult, a lethal dose of a 95 percent sulfuric acid solution has been estimated to be one ounce (i.e., 2 tablespoons) (Polson and Tattersall, 1959).

Acid ingestion can cause corrosive injury to the mucous membranes of the mouth, throat, and esophagus producing immediate pain and dysphagia[15] (Gosselin et al., 1984). Erythema, edema, and ulcers of the mouth and throat are common. The immediate pain caused when strong acids are taken into the mouth is thought to limit the amount swallowed (Friedman and Lovejoy, 1984).

---

[15] Dysphagia is difficulty swallowing.

Acid ingestion can cause significant esophageal damage (Broor et al., 1989; Muhletaler et al., 1980a; Nagi et al., 2004; Zargar et al., 1989). The esophagus undergoes coagulation necrosis when exposed to 9 percent sulfuric acid for as little as 30 seconds (Ashcraft and Padula, 1974). However, the greatest damage occurs in the stomach following acid ingestion. The pyloric antrum[16] is the most common site of injury (Franken, 1973; Muhletaler et al., 1980b). Initial exposure of the pylorus to acid initiates pylorospasm, prolonging exposure of this area to the acid. Pylorospasm commonly spares the duodenum from damage (Dilawari et al., 1984). Occasionally cases of small intestinal injury are seen, presumably due to the relaxation of the pyloric sphincter (Ritter et al., 1968). Damage to the duodenal wall (e.g., perforation) can cause necrosis of the pancreas, biliary duct, and mesentery (Casetti et al., 1980).

The state of the stomach prior to ingestion can affect the injury caused by acid ingestion (Homan, 1993). If the stomach is empty, acid tends to exert its greatest effect on the lower two-thirds of the gastric mucosa and the antrum (Chaudhary et al., 1996; Subbarao et al., 1988). In a full stomach, an acid is more likely to produce distributed damage (Palmer and Scott, 1949; Steigmann and Dolehide, 1956).

There are a number of acute symptoms associated with the ingestion of an acid. Drooling, and the inability or refusal to swallow are often associated with marked mucosal edema and ulceration of the throat and esophagus. Dyspnea[17] and stridor[18] may develop as a result of injury to the throat and esophagus. Other consequences of acid ingestion include vomiting of mucoid and "coffee ground" material, intense thirst, hematemesis[19], abdominal tenderness, and epigastric pain indicating gastrointestinal hemorrhage and perforation. Acute respiratory distress could occur as a result of posterior pharyngeal edema or the inhalation of acid into the airways (i.e., aspiration) (Wasserman and Ginsburg, 1985). Small amounts of acid which may be aspirated during ingestion or vomiting can cause considerable lung injury. A pneumonia may develop if aspiration of the acid occurs (Gonzales et al., 1954).

Gastric hemorrhage or perforation due to the ingestion of concentrated acid or a large volume of acid may cause a considerable loss of fluid from the plasma and subsequent drop in blood pressure. This, combined with the generalized trauma of acid ingestion, may cause circulatory shock or circulatory collapse (Friedman and Lovejoy, 1984; Gosselin et al., 1984; Homan, 1993). Uncorrected circulatory shock or collapse can cause acute renal failure, and ischemic lesions of the liver and heart. Circulatory shock is most often the cause of immediate death following acid ingestion (Gosselin et al., 1984). Metabolic acidosis and hemolysis are also consequences of acid ingestion.

---

[16] Pyloric antrum is the area of passage between the stomach and the small intestine.
[17] Dyspnea is shortness of breath; difficult or labored breathing.
[18] Stridor is a harsh, high-pitched respiratory sound; often a sign of respiratory obstruction.
[19] Hematemesis is the vomiting of blood.

The initial clinical presentation does not always reliably reflect the severity of the injury in some individuals (Wasserman and Ginsburg, 1985). The ingestion of acid may cause considerable necrosis of the gastrointestinal tract, which will slough off over time. After the sloughing off of the necrotic tissue, there can be considerable blood loss as a direct result of ulceration and/or perforation of the gastrointestinal tract (Ramasamy and Gumaste, 2003). Concentrated acids that induce rapid full-thickness necrosis of the stomach wall will often lead to this delayed perforation, often in the antrum region (Gosselin et al., 1984). Complications of perforation include septic shock, multi-organ failure, and death.

Delayed complications include stricture[20] and fistula[21] formation as scar tissue forms. The most common complication is pyloric stenosis, which is typically evident after several weeks (Boikan and Singer, 1930). Strictures are often associated with deep burns (McAuley et al., 1985; Nagi et al., 2004). Clinical symptoms of antral pyloric stenosis include post prandial epigastric fullness, persistent vomiting, and visible gastric peristalsis (McAuley et al., 1985; Subbarao et al., 1988; Tekant et al., 2001). Other late sequelae of gastric acid injury include intractable pain, achlorhydria[22], protein-losing gastroenteropathy, duodenal atonicity[23], and gastric carcinoma. There is also the risk of the development of esophageal carcinoma (Eaton and Tennekoon, 1972; Ramasamy and Gumaste, 2003), although this etiological relationship is not well established.

b) Alkalis

Ingestion of alkaline substances typically affects the lips, mouth, oropharynx, and esophagus (Friedman and Lovejoy, 1984). Gastric injury has also been reported after the alkali ingestion by humans (Nagi et al., 2004; Zargar et al., 1992). Signs and symptoms of alkali ingestion may include visible burns around the oral cavity, hypersalivation, difficulty swallowing, and epigastric pain (Muhlendahl et al., 1978). Ingestion of alkali solutions with a pH of 11.5 or greater can cause deep penetrating tissue injury of the esophagus and stomach due to the emulsification and disruption of cellular membranes, and may proceed to the development of strictures. Additionally, with the ingestion of alkalis, there is the risk of aspiration into the airway causing laryngeal edema and respiratory distress that may progress to pneumonia or asphyxiation. The development of esophageal carcinoma is another late sequela of alkali ingestion in patients with esophageal stricture formation (Gumaste and Dave, 1992).

---

[20] Stricture is a narrowing or constriction (i.e., stenosis) of a hollow structure, such as the stomach, that develops as scar tissue contracts.
[21] Fistula is an abnormal passage, usually between two internal organs.
[22] Achlorhydria is the absence of hydrochloric acid from maximally stimulated gastric secretions to digest food.
[23] Atonicity is the relaxation, or lack of tone or tension of the small intestine.

In rabbits, a 3.8 percent solution of sodium hydroxide applied to the esophagus for 10 seconds penetrated the mucosa, submucosa, and some fibers of the longitudinal muscle wall; a 10.7 percent solution of sodium hydroxide penetrated the mucosa and submucosa and burned the longitudinal and circular muscle walls; while a 22.5 percent solution perforated the esophagus (Krey, 1952). In another study (Hoffman et al., 1989), which grossly and histologically evaluated the effects of sodium hydroxide applied to sections of dog esophagus for 30 seconds prior to copious irrigation with tap water, a one percent solution made from crystalline drain opener (one percent sodium hydroxide) caused erosion into the muscle layer of the esophagus.

4. Inhalation Exposure

a) Acids

Sulfuric acid mists are a strong irritant of the upper respiratory tract causing irritation of the nose and throat, sneezing, and coughing (Finkel, 1983; Proctor and Hughes, 1978; Sittig, 1985). Acute exposure to low concentrations in the air can also produce reflex shallow and rapid breathing as a result of reflex bronchospasm (Finkel, 1983; Sittig, 1985). Severe overexposures can result in spasmodic closure of the larynx, and edema of the larynx and glottis that can be fatal due to blockage of the airway. Respiratory irritation can also progress to tracheobronchial or pulmonary edema (i.e., an accumulation of fluid in the lungs) in some cases. Sequelae of acute lung injury may include pulmonary fibrosis, residual bronchitis, bronchiectasis[24], and pulmonary emphysema (Finkel, 1983; Proctor and Hughes, 1978).

Exposure to sulfuric acid irritates respiratory tissues by its ability to modify receptor ligands[25] and other biomolecules, which either directly damages the membrane or activates sensory reflexes causing inflammation (Costa, 2001). However, the ammonia that exists in the air and in the lung naturally is typically able to neutralize some of the irritant potential of sulfuric acid.

Due to airway hypersensitivity, asthmatics appear to be slightly more sensitive to the bronchoconstricitive effects of sulfuric acid than are healthy individuals (Koenig et al., 1989). In guinea pigs, considered the best model for asthmatic humans (Amdur, 1989), sulfuric acid causes increased flow resistance due to reflex airway narrowing (i.e., bronchoconstriction) which impedes the flow of air into and out of the lungs. Airway hyperactivity that was observed in guinea pigs two hours after a 1-hour exposure to 200 $\mu g/m^3$ sulfuric acid appeared to be associated with pulmonary inflammation. The degree of the response is related to both acid concentration and particle size (Amdur, 1958; Amdur et al., 1978).

---

[24] Bronchiectasis is a chronic dilatation of the bronchi marked by rancid breath and convulsive coughing, expelling mucous and pus.

[25] Receptor ligands are molecules that bind specifically to molecular structures on the surface of the cell or within the cell.

b) Alkalis

Sodium hydroxide does not readily form a vapor or mist.

E. Treatment

Time is critical in preventing full-thickness burn injury due to both acid and alkaline exposure. After a splash or spill, the rapid removal of garments and irrigation of the exposed area with copious amounts of water will partially remove the agent and decrease the concentration (Curreri et al., 1970). Water dilution decreases the rate of chemical reaction and hygroscopic action of the chemical while restoring the pH level towards normal. The use of a constant flow of water will dissipate the heat of dilution. In general, better results are obtained with immediate and long dilution times (Sykes et al., 1986). There is no clear benefit from neutralizing acid injuries, and it may cause additional tissue damage as a result of the heat released from the exothermic reaction produced by the neutralizing agent. In addition to lavage, appropriate fluid restoration should be initiated.

For ocular exposure, the exposed eye should be held open with fingers and immediately rinsed with copious amounts of water or saline. At the medical facility, additional irrigation may be necessary if the pH of the tears does not return to normal (pH of 7 to 8) and remains abnormal 30 minutes after the discontinuation of irrigation (Brodovsky et al., 2000). For significant alkaline or concentrated acid burns with evident eye injury, irrigation may need to continue for at least two to three hours (Smilkstein and Fraunfelder, 2002).

Mild ocular burns will typically heal without effect if secondary infection is prevented (Grant and Shuhman, 1993). Burns due to strong alkalis and acids may require a longer, more extensive course of treatment. Several treatments used to increase healing in the case of a severe burn include the debridement[26] of the necrotic tissue to minimize ulceration, promoting re-epithelialization (e.g., suppression of collagenases), the prevention of infection, and the prevention of increased intraocular pressure (Brodovsky et al., 2000; Grant and Shuhman, 1993). Conjunctival transplantation, penetrating keratoplasty[27], eyelid reconstruction, and surgical correction of symblepharon[28] may be necessary to repair enduring damage after initial treatment (Grant and Shuhman, 1993).

In the case of acid ingestion, dilution therapy and supportive care are the recommended initial treatments (Friedman and Lovejoy, 1984). Respiratory and hemodynamic stability should be assured. If the individual is able to swallow, is not in respiratory distress or altered mental status, and is not nauseous or vomiting, it is recommended that adults be given milk or water in sips of four to eight ounces (two to four ounces for children) to minimize the risk of vomiting as vomiting increases the risk of aspiration and may cause additional injury to the

[26] Debridement is the removal of dead tissue and foreign matter from a wound.
[27] Penetrating keratoplasty is a transplant of a section of full-thickness cornea.
[28] Symblepharon is scarring and adhesion of the conjunctiva of the eyelid and the eyeball.

esophagus (Klasco, 2005). Induction of emesis and use of charcoal are contraindicated. Charcoal would interfere with an endoscopic evaluation. Intravenous infusion should also be initiated to correct circulatory shock. Esophagastroscopy should be undertaken to assess the extent and severity of damage. Prompt surgical intervention is required in cases of perforation. Treatment classically involved antibiotics to prevent infectious complications and corticosteroids to minimize stricture formation, although there is no good evidence documenting the efficacy of this treatment.

Strictures of the gastrointestinal tract due to the intense collagen deposition associated with healing that cause dysphagia and malnutrition are typically managed by antegrade or retrograde dilation. Other surgical procedures used to correct obstructions arising from corrosive injury include pyloroplasty, or a partial or total gastrectomy[29] (Kaushik et al., 2003).

Treatment for inhalation exposure involves moving the individual from the exposure area to fresh air immediately. If breathing is labored, oxygen or other respiratory support should be administered. The individual should be monitored for signs of respiratory distress as symptoms of pulmonary edema can be delayed up to 48 hours after exposure.

F. Human Exposure Information

1. In-Depth Investigation Reports

Staff reviewed 106 in-depth investigation reports from the In-Depth Investigation database (INDP) from 1995 to 2003. These reports, based on incidents from sources such as NIESS reports, CPSC Hotline phone calls, and Internet complaint reports, do not represent a random sample. Of the 106 reports, ten were associated with sulfuric acid drain openers. The scope of injuries ranged from minor chemical burns; to difficulty breathing; to eye injuries; to the formation of strictures in the esophagus following ingestion. The following are summaries of some of the incidents involving sulfuric acid drain openers.

A 59-year old woman spilled sulfuric acid drain opener on her hands when she lifted the container out of the car by the screw top cap, which came off. Her hand started burning right away. Her boyfriend immediately flushed her hands with cold water and applied baking soda in an attempt to neutralize the acid. There were no chemical burns sustained.

A sulfuric acid drain opener splashed into the eyes of a 28-year old male as he poured about three cups of the drain opener into his bathroom sink. He experienced an immediate burning sensation and jumped into the shower to rinse his eyes. His wife brought him to the emergency department because his eyes continued to burn and he was unable to see despite rinsing his eyes. At the hospital, his eyes were flushed for an additional length of time. He was given

[29] Gastrectomy is the excision of all (total) or part (subtotal or partial) of the stomach.

eye drops, referred to an eye doctor, and released. He was unable to work for one week. There were no permanent effects.

In a similar case, a 25-year old male splashed sulfuric acid drain opener in his eye and on his forearm and forehead while pouring the drain opener from the bottle into a clogged bathtub drain using a funnel. In the process, he dropped the bottle and was splashed with the drain opener in the funnel. The burning began immediately and his forehead started to blister slightly. He began flushing his eye at home right away. After which, he went to a community urgent care center where his eye was flushed for an additional 20 minutes. He also received a saline irrigator and a dye test to check for damage. Before his release, he was given sulfur drops and referred to an ophthalmologist. No mention of treatment for his forearm and forehead was made. There was a full recovery from the injuries.

After using a sulfuric acid drain opener to unclog a sink in his small bathroom that lacked ventilation, a 23-year old male plumber experienced severe difficulty breathing. X-rays at the hospital showed no damage to his lungs and his breathing eventually returned to normal.

In another incident, a 79-year old male was splashed with sulfuric acid drain opener from a cup his 4-year old grandson drank from thinking it contained juice. The boy threw the cup down immediately after ingesting about two tablespoons or less of the drain opener, splashing about a half cup on the grandfather's face, eyes, arms, and head. The boy sustained burns on his left jaw, gums, arm, hand, and left foot. An attempt was made to wash out the boy's mouth with water before calling 911. The boy was transported to the hospital by ambulance; the grandfather was transported separately by car. At the hospital the boy vomited a great deal. He was transferred to a special burn care unit where his stomach was drained. During his two week hospitalization, he had surgery to dilate his esophagus and was fed through a feeding tube. The grandfather's eyes were flushed and his face and arms were treated with an antibiotic cream; he returned to the hospital the day after for additional treatment of a burn on his head that was not detected on his initial visit. A little over a month after the accident, the grandfather had fully recovered from his injuries. However, the 4-year old was still unable to eat whole foods and was using a feeding tube. He was also returning to the hospital weekly for dilation of the esophagus.

2. Published Case Reports

a) Dermal

(1) Acids

In a retrospective review of children and adults admitted to the burn units of two hospitals in Kentucky over a 13-year period ending in 1996, 21 patients (13 children, 8 adults) sustained cutaneous burns from concentrated sulfuric acid drain openers (Bond et al., 1998). A total of eight were accidental, while 13 were assaults. No deaths were reported. The median total body surface area burned was 5 percent (range, 1 to 25 percent). Generally, the burns were small, involving less than 10 percent of the total body surface. Skin grafting was necessary in 14 patients (66 percent). Six of the 14 required multiple grafting sessions.

An accident involving concentrated sulfuric acid spilled from a rooftop drum in Saudi Arabia resulted in seven children (age range, 3 to 7 years) sustaining chemical burns of various depths (Husain et al., 1989). The type of sulfuric acid product spilled was not specified; though it was most likely not a drain opener. No first aid was administered and the contaminated clothes were not removed prior to their arrival at the hospital 30 minutes after the accident. Three had major burns with one sustaining full-thickness burns on approximately 60 percent of the body, which involved both the upper and lower limbs, the right side of the face, the chest wall, genitalia, and abdomen. This 4-year old boy presented with metabolic acidosis, hemolysis, and hypovolomia. During his 166 day hospital stay, he underwent eight autografting procedures and two additional operations to correct contractures after release. A few more corrective procedures for contractures were anticipated. However, he was expected to lead a normal active life.

A 16-month old female, who knocked over a bottle of commercial drain opener containing concentrated sulfuric acid, sustained contact injury to approximately 28 percent of her body surface area including her back, both legs, the right forearm, right flank, and three patches on her face (Dominic et al., 1987). Immediately following the accident, she was partially submerged in a swimming pool for several minutes and washed with a dilute solution of baking soda by her mother. At the burn center, she received intravenous fluid resuscitation. Full-thickness punch biopsies obtained the second day after injury showed epidermal injury to the leg, as well as tissue necrosis into, but not entirely through, the dermis of the patient's back. Skin grafting was needed to treat the full-thickness burns on the face. The greater extent of injury to the face was attributed to the initial partial submersion which diluted the acid on the individual's back and extremities more than on the face. By post burn day 18, the remainder of the wounds had re-epithelialized. Minimal evidence of hypertropic scarring was evident four months after discharge.

(2) Alkalis

In a study looking at domestic chemical burns in Saudi Arabi, 75 percent of the study population (n=44) sustained burns from sodium hydroxide drain openers (Pitkanen and Al-Qattan, 2001). The mean total body surface area affected was four percent (range, 1 to 15 percent). The majority of these incidents (n=33) involving sodium hydroxide openers occurred during use of the openers for clogged toilets, drains, or sinks. Only a small number of the individuals received copious water irrigation prior to arrival at the hospital (n=8).

Of those eight, six patients healed with no grafting, whereas skin grafting was required in all the other patients (n=38).

b) Ocular

(1) Acids

A series of 93 cases over 8.5 years of eye injuries resulting from working with automobile wet-cell batteries containing sulfuric acid (approximately 30 percent) were reviewed (Holekamp, 1977). Two-thirds of the cases (n=69) were relatively minor (i.e., healed without known sequelae within 48 hours). The most common injuries were conjunctival and corneal chemical burns. All severe injuries (n=9), requiring hospitalization or resulting in significant permanent ocular damage, were caused by battery explosions, which caused injury from the force of the explosion as well as the chemical burn from the acid. The severe cases involved damage to the eye lids, anterior chamber of the eye, and retina. Fifteen cases involved acid being spilled or splashed into the eye; injuries and outcomes were not provided.

The eyes of a 21–year old male deliberately splashed with concentrated sulfuric acid were extensively examined (Schultz et al., 1968). The individual sustained chemical burns to his face, anterior trunk, and extremities, involving approximately 50 percent of the body surface. Upon initial presentation, the eyelids of the patient were swollen and denuded. Both corneas were opaque and edematous. During the next three days, the corneas began to clear and his vision improved. Five days after the accident when the patient died from extensive chemical damage to the respiratory tract, histological preparations of the eyes revealed partial necrosis of the conjunctiva, corneal epithelium, and corneal stroma. However, despite the severe external ocular injury, the intraocular structures suffered no damage.

(2) Alkalis

Of 101 patients with severe eye burn (chemical and thermal) who sought treatment at the regional hospital, five patients sustained burns from alkali drain openers (Kuckelkorn et al., 1995). In all five cases, long-term visual acuity was limited to light perception.

c) Ingestion

(1) Acids

A prospective study looked at the injury spectrum and clinical outcome of 16 adult patients (age range, 16 to 60 years) admitted to a hospital in India following the ingestion of common acid cleaning agents used in Indian household, including 10 individuals who ingested sulfuric acid cleaning agents (Dilawari et al., 1984). The amount ingested was known for eight of the individuals and ranged from 15 to 40 mL (approximately 1 to 2.7 tablespoons). The concentration of the sulfuric acid was estimated to be in the range of 26.4 N to 35.4 N (approximately 73 to 98 percent) based on samples obtained from the market. Epigastric pain, vomiting, hematemesis, and oropharyngeal burns were observed in the majority of the patients. The extent and severity of injury to the upper gastrointestinal tract was assessed using fibreoptic endoscopy. Two of the 10 were found to have only moderate injury (i.e., superficial ulcerations) to the esophagus and stomach; neither suffered any complications or required surgical intervention. Of the eight classified as having severe injury (i.e., extensive and deep ulcerations) of the esophagus and stomach, four had major early complications including gastric perforation (2/4), massive hematemesis (1/4), and severe bronchopneumonia (1/4). Two of the four died. Late complications in the six surviving individuals, which included antral and esophageal stricture, were treated with gastric resectioning and stricture dilation. The duodenum was not injured in the majority of the patients (8/10).

In a review of 214 cases of acid or alkali ingestion over a seven year period, 34 were admitted with a possible history of acid ingestion (Hawkins et al., 1980). Fourteen of the 34 ingested sulfuric acid household products. The concentration of the acid was not established. Five sustained burns of the esophagus and stomach penetrating beyond the mucosa as evidenced by deep ulcerations, whitish membrane, edema, and friability; four of the five developed strictures of the esophagus. Spontaneous perforation of the esophagus occurred in one patient and was successfully treated with thoracotomy and drainage of the mediastinum. A 14-month old child suffered from gastric perforation that was corrected with surgical closure; she recovered with stricture of the esophagus, stomach, and duodenum. Another patient who ingested sulfuric acid developed supraglottic edema that was severe enough to necessitate a tracheotomy. None of the eight deaths in the review were attributed to the ingestion of sulfuric acid (4 ingested alkaline; 2 hydrochloric acid; 1 nitric acid; and 1 phosphoric acid). In this case review, alkali agents were most often involved; 117 patients were suspected of ingesting sodium hydroxide.

Erythema of the oropharyngeal mucosa was the only finding initially in a five-year old girl admitted shortly after swallowing a concentrated sulfuric acid solution (Zamir et al., 1985). The type of sulfuric acid product ingested was not cited. Treatment involved intravenous fluids, antacids, antibiotics, and steroids. During the following two months, she developed dysphagia and was treated with repeated dilations. Extensive esophageal stricture, and antral scarring and contraction were noted. A gastroduodenostomy and esophageal replacement by interposition of the right colon were performed. One year following the operation, the child was eating well and was symptom free.

A 37-year old man ingested an unspecified quantity of a 97 percent sulfuric acid drain opener (Litovitz et al., 1998). The man, who was awake and alert one hour post-ingestion, soon became unresponsive. Exploratory laparoscopy revealed massive full-thickness burns, with the full or partial destruction of most of the abdominal structures. Twenty hours after presentation the man died. Necrosis and perforations were noted along the entire length of the gastrointestinal tract. Necrosis was also noted in the lungs, pancreas, and diaphragm.

A 45-year old man was admitted to the emergency room 30 minutes after intentionally ingesting three to four ounces of an unspecified concentrated sulfuric acid product (Litovitz et al., 2002). The individual had perioral burns, was unable to speak, and was in respiratory distress. A rigid abdomen soon developed. Full-thickness burns were noted from the oropharynx to the stomach using esophagastroscopy. Despite resectioning of the esophagus, stomach, and part of the bowel, the individual died after two weeks due to complications, which included sepsis.

A 48-year old man who ingested battery acid (approximately 30 percent sulfuric acid) was admitted to the emergency department with nausea, vomiting, and abdominal pain (Litovitz et al., 2002). He subsequently developed tachypnea and dyspnea requiring intubation. Portions of his stomach and intestines were necrotic and resected. At the time of surgery, the liver was hyperemic, but no organs were necrotic. The individual expired a day later due to cardiac arrest. On post-mortem, necrosis was noted in the esophagus, colon, liver, a portion of the pancreas, the left diaphragm, and abdominal musculature. The trachea and bronchi were also found to be erythematous.

(2) Alkalis

Although no deaths were attributed to the ingestion of sulfuric acid in a published review of 214 cases involving acid and alkali ingestions, there were two deaths associated with the ingestion of concentrated sodium hydroxide drain openers (specific concentrations not noted) (Hawkins et al., 1980). After 22 days in the hospital, one patient with extensive injury to the esophagus, stomach, duodenum, and jejunum died from recurrent massive gastrointestinal hemorrhage. The second patient died as a result of a sudden massive hemorrhage nine days after ingestion; esophageal and gastric necrosis with erosion from the esophagus into a bronchial vein was found after death. Ingestions of sodium hydroxide were responsible for most of the complications in this review.

d) Inhalation

While working alone in a manhole, a 23-year old male exposed to fumes from a 95 percent sulfuric acid mixture expelled from a pipe for approximately 30 minutes was hospitalized for 12 days (Knapp et al., 1991). He was re-hospitalized three days after his release when a pulmonary lung abscess was identified. Four months after this massive, accidental inhalation of sulfuric acid fumes the individual was clinically asymptomatic and back at work.

IV. Summary and Conclusion

The primary health effects of sulfuric acid are due to its irritating and corrosive nature. Depending upon the route of exposure, sulfuric acid can directly affect the skin, eyes, respiratory tract, and/or gastrointestinal tract. The extent of injury caused by a corrosive substance, such as sulfuric acid, is dependent on the concentration of the agent, the pH of the agent, the site of exposure, the duration of contact, and the amount of the agent.

Acids and alkalis can be concentrated or dilute. In general, concentrated acids and alkalis will cause greater tissue damage than more dilute substances regardless of whether or not they are considered a strong acid or alkali.

Concentrated sulfuric acid can cause tissue injury via cellular desiccation and thermal injury, in addition to cellular damage caused by the change in pH. The heat evolved from the reaction of concentrated sulfuric acid and the water in tissue plays a considerable role in the damage caused to tissue. Experimental data included in a U.S. patent demonstrated that a concentration of sulfuric acid between 80.8 and 84.4 percent effectively dissolved a wetted sanitary napkin within 2 minutes while reaching a maximum temperature of 165°F when diluted one to one by volume with tap water (60°F) (Van Vlahakis, 1978). This was in contrast to sulfuric acid concentrations of 85 percent and greater which yielded temperatures of 195°F and higher when diluted one to one with tap water. The author of the patent concludes that concentrations of 84.4 percent will cause less tissue damage based on the reduced maximal temperature of the reaction. Staff is not aware of other studies that demonstrate that the reduced heat generated by sulfuric acid in concentrations of less than 84 percent would lessen tissue injury.

Different body regions are more susceptible to damage than are others. For instance, the eye is more sensitive to injury due to its limited buffering capacity and regenerative capabilities. Mucosal membranes are also especially susceptible to injury.

The ultimate outcome is influenced by whether or not adequate first aid is administered immediately after exposure. This is particularly true in the case of dermal and ocular exposures. Concentrated sulfuric acid is more likely to cause severe dermal injury (e.g., full-thickness burns requiring skin grafting and surgery to correct contracture) when proper first aid is not administered promptly (Dominic et al., 1987; Husain et al., 1989).

Ingestion of concentrated sulfuric acid can cause injury ranging from superficial ulcerations of the esophagus and stomach (Dilawari et al., 1984) to gastric and esophageal perforation (Hawkins et al., 1980). Surgical corrections are often required to correct late complications such as stricture and fistula formation. Fortunately, the immediate pain sulfuric acid induces when placed in the mouth likely lessens the amount ingested and subsequent injury. This however may not be the case when a determined or inebriated individual ingests an acid product. In such cases, larger volumes are often ingested resulting in greater direct injury.

If concentrated sulfuric acid drain openers were no longer available, consumers may be expected to buy drain openers containing hydrochloric acid or sodium hydroxide, especially ones with higher hydroxide concentrations. Therefore, it is important to consider the effects of hydrochloric acid and sodium hydroxide on tissue as well.

There are drain openers currently available that contain between 5 to 30 percent hydrochloric acid. Although concentrated hydrochloric acid is a much less vigorous acid than sulfuric acid causing less desiccation and heat release (Davidson, 1927), hydrochloric acid can still cause significant tissue injury, including severe skin burns, esophageal (Sittig, 1985) and duodenal necrosis (Munoz Munoz et al., 2001; Sittig, 1985), and permanent eye damage (Sittig, 1985). Inhalation of hydrochloric acid vapors can also cause coughing, pain, inflammation upper respiratory tract, and in severe cases, pulmonary edema (Clayton and Clayton, 1994).

Other drain openers contain the strong alkali sodium hydroxide in concentrations ranging from 2 to 40 percent (pH 11.5 to 14). Generally, these products can cause severe tissue damage, especially at higher concentrations. Consequently, substances containing free or chemically unneutralized sodium hydroxide in a concentration of 10 percent or more require the signal word "POISON", as mentioned earlier, in addition to other cautionary labeling required by the FHSA. These same substances in a concentration of 10 percent or more sodium and/or potassium hydroxide in dry form such as granules, powder, and flakes or 2 percent or more in any other form also require CR packaging.

Although the mechanisms of action by which acids and alkalis cause injury are different, strong acids and alkalis can produce significant tissue damage and require caution when using. Drain openers containing sulfuric acid, sodium hydroxide, or hydrochloric acid can be fatal if swallowed and cause permanent impairment of vision if splashed in the eye. However, unlike sodium hydroxide, sulfuric acid and hydrochloric acid can form a mist or vapor which can cause symptoms ranging from irritation of the nose and throat to lung edema. As for dermal injury, all three can cause full-thickness injury that may necessitate skin grafting. Concentrated sulfuric acid can cause dermal injury faster than concentrated sodium hydroxide, although sodium hydroxide is not perceived as rapidly as sulfuric acid on skin, which may delay treatment. Sodium hydroxide is capable of causing severe burns with deep ulceration due to its ability to continue to penetrate to deeper layers of tissue until washed away with copious amounts of water.

None of the acid and alkali drain openers are innocuous substances. In particular, the drain openers containing sulfuric acid or sodium hydroxide both have the potential to produce severe injuries depending on the concentration of the agent, the pH of the agent, the site of exposure, the duration of contact, and the amount of the agent involved. Sulfuric acid drain openers do have the potential to cause dermal injury more rapidly than alkaline drain openers. However, prompt appropriate treatment will considerably lessen the degree of injury.

REFERENCES

Amdur, M. O. (1958). The respiratory response of guinea pigs to sulfuric acid mist. AMA Arch Ind Health *18*, 407-414.

Amdur, M. O. (1989). Health effects of air pollutants: sulfuric acid, the old and the new. Environ Health Perspect *81*, 109-113; discussion 121-102.

Amdur, M. O., Dubriel, M., and Creasia, D. A. (1978). Respiratory response of guinea pigs to low levels of sulfuric acid. Environ Res *15*, 418-423.

Ashcraft, K. W., and Padula, R. T. (1974). The effect of dilute corrosives on the esophagus. Pediatrics *53*, 226-232.

Bates, N. (1999). Acid and alkali injury. Emerg Nurse *7*, 21-26.

Boikan, W. S., and Singer, H. A. (1930). Gastric sequelae of cossosive poisoning. Arch Intern Med *46*, 342-357.

Bolkova, A., and Cejkova, J. (1984). Relationship between various concentrations of NaOH and metabolic effects in experimentally burned rabbit cornea. A biochemical and histochemical study. Graefes Arch Clin Exp Ophthalmol *222*, 86-89.

Bond, S. J., Schnier, G. C., Sundine, M. J., Maniscalco, S. P., and Groff, D. B. (1998). Cutaneous burns caused by sulfuric acid drain cleaner. J Trauma *44*, 523-526.

Brodovsky, S. C., McCarty, C. A., Snibson, G., Loughnan, M., Sullivan, L., Daniell, M., and Taylor, H. R. (2000). Management of alkali burns : an 11-year retrospective review. Ophthalmology *107*, 1829-1835.

Broor, S. L., Kumar, A., Chari, S. T., Singal, A., Misra, S. P., Kumar, N., Sarin, S. K., and Vij, J. C. (1989). Corrosive oesophageal strictures following acid ingestion: clinical profile and results of endoscopic dilatation. J Gastroenterol Hepatol *4*, 55-61.

Casetti, P., Ponzalli, M., Dellarolle, A. C., Duranti, A., Favi, P., and Massimo, C. (1980). [Emergency gastro-duodeno-cephalo-pancreatectomy for gastroduodenal necrosis caused by ingestion of caustics]. Minerva Chir *35*, 409-416.

Chaudhary, A., Puri, A. S., Dhar, P., Reddy, P., Sachdev, A., Lahoti, D., Kumar, N., and Broor, S. L. (1996). Elective surgery for corrosive-induced gastric injury. World J Surg *20*, 703-706; discussion 706.

Christesen, H. B. (1995). Prediction of complications following caustic ingestion in adults. Clin Otolaryngol *20*, 272-278.

Clayton, G. D., and Clayton, F. E., eds. (1994). Patty's Industrial Hygiene and Toxicology, 4th edn (New York, John Wiley & Sons Inc.,).

Costa, D. L. (2001). Air pollution. In Casarett and Doull's Toxicology, C. D. Klassen, ed. (New York, McGraw Hill), pp. 979-1012.

Curreri, P. W., Asch, M. J., and Pruitt, B. A. (1970). The treatment of chemical burns: specialized diagnostic, therapeutic, and prognostic considerations. J Trauma *10*, 634-642.

Davidson, E. C. (1927). The treatment od acid and alkali burns - An experimental study. Ann Surg *85*, 481-489.

Dilawari, J. B., Singh, S., Rao, P. N., and Anand, B. S. (1984). Corrosive acid ingestion in man - a clinical and endoscopic study. Gut *25*, 183-187.

Dominic, W. J., Field, T. O., Jr., and Hansbrough, J. F. (1987). Sulfuric acid burns in a child: histologic examination as an indication of wound depth. J Burn Care Rehabil *8*, 395-397.

Dua, H. S., King, A. J., and Joseph, A. (2001). A new classification of ocular surface burns. Br J Ophthalmol *85*, 1379-1383.

Eaton, H., and Tennekoon, G. E. (1972). Squamous carcinoma of the stomach following corrosive acid burns. Br J Surg *59*, 382-387.

Finkel, A. J. (1983). Hamilton and Hardy's Industrial Toxicology, 4th edn (Boston, MA, John Wright, PSG Inc.).

Flury, F., and Zernik, K. (1931). Schadliche Gase (Berlin, Springer).

Franken, E. A., Jr. (1973). Caustic damage of the gastrointestinal tract: roentgen features. Am J Roentgenol Radium Ther Nucl Med *118*, 77-85.

Franklin, R. (2005). Petition to Ban Sulfuric Acid Drain Openers: Market Information and Economic Considerations (Bethesda, U.S. Consumer Product Safety Commission).

Friedenwald, J. S., Hughes, W. F., and Herrmann, H. (1946). Acid burns to the eye. Arch Ophthalmol *35*, 98-108.

Friedman, E. M., and Lovejoy, F. H., Jr. (1984). The emergency management of caustic ingestions. Emerg Med Clin North Am 2, 77-86.

Gonzales, T. A., Vance, M., Helpern, M., and Umberger, C. J. (1954). Legal Medicine, Pathology and Toxicology, 2nd edn (New York, Appleton-Century-Crofts).

Gosselin, R. E., Smith, R. P., and Hodge, H. C. (1984). Clinical Toxicology of Commercial Products, 5th edn (Baltimore, MD, Williams and Wilkins).

Grant, W. M. (1974). Sulfuric Acid. In Toxicology of the eye (Springfield, IL, Charles C. Thomas), pp. 959-960.

Grant, W. M., and Kern, H. L. (1955). Action of alkalies on the corneal stroma. Arch Ophthalmol *54*, 931-933.

Grant, W. M., and Shuhman, J. S. (1993). Toxicology of the Eye, 4th edn (Springfield, IL, Charles C Thomas).

Gumaste, V. V., and Dave, P. B. (1992). Ingestion of corrosive substances by adults. Am J Gastroenterol *87*, 1-5.

Hawkins, D. B., Demeter, M. J., and Barnett, T. E. (1980). Caustic ingestion: controversies in management. A review of 214 cases. Laryngoscope *90*, 98-109.

Hirst, L. W., Fogle, J. A., Kenyon, K. R., and Stark, W. J. (1982). Corneal epithelial regeneration and adhesion following acid burns in the rhesus monkey. Invest Ophthalmol Vis Sci *23*, 764-773.

Hoffman, R. S., Howland, M. A., Kamerow, H. N., and Goldfrank, L. R. (1989). Comparison of titratable acid/alkaline reserve and pH in potentially caustic household products. J Toxicol Clin Toxicol *27*, 241-246.

Holekamp, T. L. (1977). Ocular injuries from automobile batteries. Trans Sect Ophthalmol Am Acad Ophtalmol Otolaryngol *83*, 805-810.

Homan, C. S. (1993). Acids and Alkalies. In Handbook of Medical Toxicology, P. Viccellio, ed. (Boston, Little, Brown and Company).

Hummel, R. (1982). Clinical Burns Therapy (New York, John Wright).

Husain, M. T., Hasanain, J., and Kumar, P. (1989). Sulphuric acid burns: report of a mass domestic accident. Burns *15*, 389-391.

Jarudi, N. I., and Golden, B. (1973). Ammonia eye injuries. J Iowa Med Soc *63*, 260-263.