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September 13, 1989

VISTA

Mr. Robert Hardeman
Whitaker Oil Company
1557 Marietta Road, N.W.
Martech Station
Atlanta, Georgia 30377

Dear Robert:

Below are responses to questions and issues you raised in your letter of August 16. In general, I believe you should emphasize the lower degree of health risk concerns and lower volatility as it relates to environmental release potential, i.e. VOC content, and not dwell on the disposal conditions.

The information below is relatively general but I believe responsive to your questions and needs. I can try to provide further details in those areas where you see a need for more specific information.

TOXICITY

You make a statement in your letter that LPA is nontoxic and noncarcinogenic. It is important for you to know that this type of statement in advertising literature could be challenged, especially when made in such absolute terms. In comparison to RMS, LPA is relatively nontoxic and presents far less exposure potential under normal use conditions. Also you should be aware that LPA has never been tested for carcinogenic potential. Our determination that no carcinogenic labeling is required is based on the absence of aromatic compounds, and comparison of the physical and chemical properties of LPA to other oils that are known to be or suspected to be carcinogenic. The attached LPA Hazard Determination details this determination.

With regard to the acute toxicity of Regular Mineral Spirits, there is some confusion with the terminology when reviewing the scientific literature. It is difficult to determine if the literature reports are dealing specifically with the product or products you are attempting to displace.

However, I have enclosed what I believe to be appropriate pages from a NIOSH criteria document on refined petroleum solvents and the ACGIH TLV documentation for Stoddard Solvent. The animal and human

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toxicity discussed clearly indicates potential toxic effects from the inhalation of the solvent under normal use conditions. Effects noted include significant skin irritation, central nervous system effects, and blood system effects, such as anemia.

As to the carcinogenicity of RMS, there is data in the literature, much of it based on API studies, indicating a skin carcinogenic potential for sweetened, but not hydrotreated, kerosine middle distillate fractions. RMS may fall in this category in some cases.

IARC Monograph 33 is a document entitled Mineral Oils: Lubricant Base Oils and Derived Products. In the monograph this broad category of oils is broken down into different classes based on physical properties and production parameters. These classes are then categorized as to their carcinogenic potential. The practical implication of this document is that the OSHA Hazard Communication Standard requires any IARC identified carcinogen to be labeled as a carcinogen.

Based on the physical properties and process used to manufacture LPAs, we believe that these solvents are not covered by IARC Monograph 33. This determination also allows us to conclude the Proposition 65 Mineral Oil Listing does not apply. We have written confirmation from California on that interpretation.

The applicability of IARC Monograph 33 to mineral spirits is difficult based on the terminology differences and my knowledge of the processes used to manufacture these oils.

EXPOSURE LIMITS - As discussed, there is no specific TLV or OSHA exposure limit applicable to LPA solvents. The NIOSH criteria document discussed above does recommend a TLV of 350 mg/m³ for low aromatic kerosine type oils. This limit is set to avoid inhalation of hydrocarbon mists that could accumulate in the lungs and cause pneumonitis. In other words a mist, not vapor, is the concern in terms of any inhalation hazard.

Based on the physical properties of the LPA solvents and the NIOSH Criteria Document, we believe that low temperature use of LPA Solvents, without significant aerosolizing, would present little or no inhalation exposure potential to personnel.

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WASTE CLASSIFICATION

The federal standards for waste classification are found in 40 CFR, Part 261. In general, wastes are defined by being specifically listed by name or exhibiting the characteristic of ignitability, corrosivity, reactivity or EP toxicity. EP toxicity is determined by analysis of the liquid, or a water extract for solids, for specific listed chemicals. If the analyte contains more than a

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specified concentration of one out of eight metals or six pesticides, the chemical or substance is hazardous.

Vista LPA solvents are not listed as hazardous, nor do they exhibit a characteristic that classes them as hazardous. Mineral spirits are ignitable by nature and exhibit a hazardous characteristic. I don't believe they would exhibit toxicity based on the current EP toxicity test.

However, a toxicity characteristic (TC) has been proposed by EPA that will include more substances, including benzene and toluene. This revision, when effective, may cause mineral spirits to exhibit toxicity for waste classification purposes.

As to waste disposal of solvents, there is a general ban on disposing of liquids in landfills unless they have been adsorbed, fixed, or otherwise rendered nonliquid. This applies to hazardous and nonhazardous liquids. The other options for disposal of waste liquids, such as fuel burning programs, are available for hazardous and nonhazardous liquids. However, the administrative burden for disposing of hazardous liquids is generally greater.

LPA could potentially migrate to groundwaters if disposed of improperly. The presence of contaminants of any kind in groundwater is a concern. It is simply a matter of relative risk of the contamination, based on toxic potential and concentration. LPA is of a low order of toxicity and would be of less concern than mineral spirits, all other factors being equal. Based on this, we cannot confirm that LPA products do not endanger water resources if discarded in small amounts in landfills.

VOC STATUS

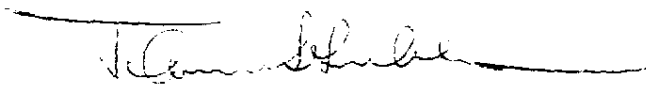
The definition and regulatory status of VOC's is variable from state to state in most cases. As you are aware, the state of California rules are often used as a benchmark.

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Typically, VOC's are defined based on a vapor pressure measurement. Lower vapor pressure products are less likely to be covered or impacted by VOC rules. Often the aromatic content is what drives the vapor pressure. LPA is low in aromatics and vapor pressure, and therefore has a lower potential to be impacted by VOC regulations or restrictions.

Sincerely,



Thomas G. Grumbles, C.I.H.
Environmental Quality Manager

dlj

cc: J. Lopez, P. Douvry, C. Piersen

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VISTA LPA-140 SOLVENT HAZARD DETERMINATION

Vista LPA-140 Solvent is a hydrotreated, predominately paraffinic, hydrocarbon solvent. Based on our hazard determination, under OSHA's Hazard Communication Standard (29 CFR 1900.1200), LPA-140 Solvent only presents the physical hazard of combustibility.

A chemical is considered a physical hazard if there is scientifically valid evidence that it is a combustible liquid, compressed gas, explosive, flammable liquid or solid, organic peroxide, oxidizer, pyrophoric, unstable (reactive), or water-reactive. The flash point of LPA-140 Solvent is approximately 140°F making it a combustible liquid.

A chemical presents a health hazard if one statistically significant study done in accordance with established scientific protocol indicates an effect may occur in exposed populations. Based on acute mammalian testing done on LPA-140; including dermal LD₅₀, primary skin irritation, primary eye irritation, inhalation LC₅₀ for 1 hour exposures, and oral LD₅₀, LPA-140 Solvent has been determined not to be a health hazard as defined by the OSHA Hazard Communication Standard.

Vista LPA-140 Solvent contains no OSHA defined carcinogens, at levels greater than 0.1%. As for the broad categories of "minerals oils", petroleum oils, or other generic classifications of oils which have been indicated to have a carcinogenic potential by various studies we believe the following to be true in respect to LPA-140 Solvent. IARC Monograph 33, a compilation of carcinogenic evidence for Mineral Oils (lubricant base oils and derived products) classifies the carcinogenicity of petroleum derived oils based on the increasing severity of processing or refinement. This monograph deals with oils that are predominately lubricant base oils with initial boiling points exceeding 750°F and having carbon number distributions of 15 to 50. The monograph also lists specific oils as defined by CAS numbers assigned by the U.S. EPA. LPA-140 Solvent has an IBP of 360°F and has a carbon number distribution predominately in the C₁₂-C₁₆ range. Also the CAS number descriptive of LPA-140 Solvent is not listed in the monograph. Based on these facts, Monograph 33 does not contain evidence relevant to LPA-140 Solvent.

Of those oils discussed in the monograph, LPA-140 Solvent is closest to the white oil (Class 5) classification, for which the only evidence for carcinogenicity is of questionable significance to human exposures.

VISTA LPA SOLVENT HAZARD DETERMINATION

Vista LPA Solvent is a hydrotreated, predominately paraffinic, hydrocarbon solvent. Based on our hazard determination, under OSHA's Hazard Communication Standard (29 CFR 1900.1200), LPA Solvent only presents the physical hazard of combustibility.

A chemical is considered a physical hazard if there is scientifically valid evidence that it is a combustible liquid, compressed gas, explosive, flammable liquid or solid, organic peroxide, oxidizer, pyrophoric, unstable (reactive), or water-reactive. The flash point of LPA is approximately 144°F making it a combustible liquid.

A chemical presents a health hazard if one statistically significant study done in accordance with established scientific protocol indicates an effect may occur in exposed populations. Based on acute mammalian testing done on LPA; including dermal LD₅₀, primary skin irritation, primary eye irritation, inhalation LC₅₀ for 1 hour exposures, and oral LD₅₀, LPA Solvent has been determined not to be a health hazard as defined by the OSHA Hazard Communication Standard.

Vista LPA Solvent contains no OSHA defined carcinogens, at levels greater than 0.1%. As for the broad categories of "minerals oils", petroleum oils, or other generic classifications of oils which have been indicated to have a carcinogenic potential by various studies we believe the following to be true in respect to LPA Solvent. IARC Monograph 33, a compilation of carcinogenic evidence for Mineral Oils (lubricant base oils and derived products) classifies the carcinogenicity of petroleum derived oils based on the increasing severity of processing or refinement. This monograph deals with oils that are predominately lubricant base oils with initial boiling points exceeding 750°F and having carbon number distributions of 15 to 50. The monograph also lists specific oils as defined by CAS numbers assigned by the U.S. EPA. LPA Solvent has an IBP of 370°F and has a carbon number distribution predominately in the C₁₂-C₁₆ range. Also the CAS number descriptive of LPA Solvent is not listed in the monograph. Based on these facts, Monograph 33 does not contain evidence relevant to LPA Solvent.

Of those oils discussed in the monograph, LPA Solvent is closest to the white oil (Class 5) classification, for which the only evidence for carcinogenicity is of questionable significance to human exposures.

LPAHAZ.400

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NIOSH estimates that about 600,000 workers in the United States are potentially exposed to all "specialized" naphthas.

(c) Varnish Makers' and Painters' Naphtha

Varnish makers' and painters' (VM and P) naphtha is a mixture of hydrocarbons that has a boiling range of approximately 95-160 C (203-320 F) [1,10,17,18]. It is sometimes known as benzine, Naphtha 76, ligroin, or high boiling petroleum ether [10,18].

Physically, VM and P naphtha is a colorless to yellow liquid that has an aromatic odor [18]. It has a flashpoint (closed cup) of -7 to 13 C (20-55 F) and is classified as a type I B flammable liquid [18]. Its mean molecular weight ranges from 87 to 114 and it is composed of about 45-60% paraffins, 30-45% naphthenes, and 5-13% aromatics [11-14]. A sample of VM and P naphtha analyzed by Carpenter et al [17] showed 55% paraffins, 30% monocycloparaffins, 2% dicycloparaffins, and 12% alkylbenzenes. The hydrocarbon chain ranges chiefly from C7 to C11 [4]. Additional physical and chemical properties are given in Table XIV-1.

VM and P naphtha can be produced from a straight run distillate of paraffinic or mixed base crude [2]. It is used as a quick-evaporating paint thinner of moderate solvent power [2].

NIOSH estimates that about 600,000 workers in the United States are potentially exposed to all "specialized" naphthas.

(d) Mineral Spirits

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Mineral spirits are a mixture of hydrocarbons that have a boiling range of 150-200 C (302-392 F) [1,19]. These compounds have also been termed white spirits, petroleum spirits, and light petrol [5,19,20].

Stoddard solvent is considered by some investigators to be synonymous with mineral spirits [19,21].

Mineral spirits are clear, colorless liquids with a "pleasant, sweetish odor," and are very slightly soluble in water [5]. They do not blacken or corrode a clean metallic copper strip in 30 minutes at the boiling point of the mineral spirits [22]. The solvent contains about 30-65% paraffins, 15-55% naphthenes, and 10-30% aromatic hydrocarbons [11-14]. Additional chemical and physical properties are given in Table XIV-1.

This solvent is produced from straight-run naphtha derived from a paraffin-base or mixed-base crude [2]. Mineral spirits are used as a general-purpose thinner, a solvent for paint and varnish industries, and a drycleaning agent [2,5,20].

NIOSH estimates that 61,000 workers in the United States are potentially exposed to mineral spirits.

(e) Stoddard Solvent

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Stoddard solvent is a mixture of hydrocarbons, predominantly C9 to C11, that has a boiling range between 160 and 210 C (320-410 F) [1,21,23]. It is a clear, colorless liquid and has a minimum flashpoint of 38 C (100 F) [19,24]. The dry point ranges from 166 to 210 C (330-410 F). Its Kauri-Butanol value ranges from 27 to 45. It is insoluble in water but readily soluble in most organic solvents [19]. Stoddard solvent must be negative on a specific test for mercaptans, and the copper strip corrosion test (ability to blacken or corrode a strip of polished copper placed in the solution) for 3 hours at 100 C (212 F) must be negative [25]. Chemically, Stoddard solvent is a mixture of 30-50% straight and branched chain paraffins, 30-40% naphthenes, and 10-20% aromatic hydrocarbons

therapy. These symptoms were eliminated by the use of a sedative-anti-spasmodic drug. All the individuals returned to work 30 minutes after their arrival to the health center except for one older person who had remained in the vapors longer than the others. He was free of symptoms within 30 minutes but was observed a little longer as a safety precaution. Blood counts and urinalyses were made on all of the exposed individuals and the results were all normal. A followup examination after 5 years showed no medical problems that could be attributed to the petroleum naphtha vapor exposure.

(d) Mineral Spirits

In 1958, Kegels [47] reported the effects of white spirits on a 36-year-old man who cleaned floors with copious amounts of the solvent. He had no previous history of serious illness. The man and several women had been exposed to white spirits for at least 4 months. He was constantly surrounded by "clouds" of solvent, but neither airborne measurements of white spirits nor daily exposure times were quantitated. When some women complained of nausea and vomiting, the use of white spirits was stopped. The author indicated that the white spirits boiled between 153 and 185 C and contained 83% paraffins and 17% aromatics.

The man showed no blood abnormalities when medically examined, but 3 months later, he complained of fatigue and pallor [47]. A physician's diagnosis was that he was suffering from overwork. Several months later, the patient still had abnormal symptoms. A second physician performed a blood analysis and sternal puncture and indicated that the subject had either aplastic anemia or aleukemic leukemia. Subsequently, he was

diagnosed as having aplastic anemia with thrombocytopenia and leukopenia (80% lymphocytes and 20% neutrophils).

Treatment consisted of three blood transfusions, but his condition did not improve and he was admitted to a hospital [47]. Signs of purpura on the skin and mucous membranes now appeared. The initial blood analysis showed decreased erythrocytes (2,480,000/cu mm), leukocytes (2,300/cu mm), platelets (34,000/cu mm), and hemoglobin (45%). After several blood transfusions and iron and liver extract injections, the employee's condition improved and he returned to work. However, shortly thereafter, he developed articular rheumatism which was treated with cortisone. He was treated continuously for aplastic anemia and rheumatism by blood transfusions and iron, liver extract, and adrenocorticotrophic hormone injections.

About 2 years later, after several intermittent periods of infection, the patient again felt fatigued and weak [47]. Hematologic examination showed severe decreases in erythrocytes (1,710,000/cu mm) and leukocytes (2,800/cu mm). A sternal puncture biopsy examination showed marked hypocellularity. Urobilinogen was present in the urine, indicating signs of liver dysfunction probably caused by hemosiderosis. The subject died a few months later from septicemia. Kegels indicated that this subject probably had a sensitivity to white spirits, since other workers exposed to white spirits did not develop aplastic anemia. The possible role of benzene in the etiology of the disease should be considered, even though the boiling range of white spirits should preclude any benzene being present, since trace amounts of benzene could have been present in the solvent as contaminants.

In 1975, Astrand and associates [48] examined the effects of white spirits on human alveolar air and blood solvent concentrations during rest and exercise. The white spirits used in the study consisted of 83% aliphatic and 17% aromatic components.

Fifteen men, 20-34 years of age, were used in the study [48]. In initial trials, subjects were exposed to 2,500 or 5,000 mg/cu m of white spirits. The concentrations of mineral spirits were determined by gas chromatography. The duration of exposure was not reported. Nausea and vertigo were apparent at both concentrations.

The authors [48] decided that subsequent experiments should be conducted with white spirits at concentrations that ranged from 1,000 to 2,500 mg/cu m to reduce the discomfort of the men and to accurately analyze the solvent content in alveolar air and blood. Five subjects each inhaled both 1,250 and 2,500 mg/cu m of white spirits for 30 minutes at rest and then during exercise at an intensity of 50 watts. Four subjects were exposed at 1,250 mg/cu m for 30 minutes during rest and in three 20-minute exercise periods at intensities of 50, 100, and 150 watts which are equivalent to the amount of energy used in light industrial work, manual labor, and heavy manual work, respectively [49]. Two subjects were first exposed for 30 minutes to 2,500 mg/cu m of white spirits in atmospheric air (20.90% oxygen, 0.04% carbon dioxide, and 79% nitrogen) and then to the solvent in a mixture of 21% oxygen, 4% carbon dioxide, and 75% nitrogen during a 30-minute rest period and a 30-minute exercise period (intensity of 50 watts). Two subjects were exposed to white spirits at 1,250 mg/cu m in the air for 30 minutes during rest followed by three 30-minute exposures during exercise at an intensity of 100 watts. The two remaining subjects

were each exposed at rest to 1,000, 2,500, 1,500, and 2,000 mg/cu m for one 30-minute period. The white spirits concentration or work rate changes were made after each 30-minute period without interrupting exposure. Alveolar air samples were collected during exposure, and arterial and venous blood samples were taken from preplaced catheters in the brachial artery and medial cubital vein, respectively. Heart rate and blood lactic acid content were determined in some subjects at the end of each exposure period. Cardiac output, oxygen uptake, and volume of expiratory and alveolar air were determined in all subjects after 20 minutes of each exposure period.

During exercise, three subjects had occasional premature atrial beats as shown by electrocardiograms; however, they were of the same type as those observed during rest [48]. One man developed premature atrial beats exclusively in conjunction with solvent exposure (concentration not reported). Another displayed gradual flattening, and, ultimately, inversion of the T wave during exposure to white spirits. This subject had no symptoms, and the electrocardiogram became normal a few days after solvent exposure (concentration not reported). No differences were noted in heart rate, alveolar ventilation, or oxygen uptake either at rest or during exercise at an intensity of 50 watts during exposure at 1,250 and 2,500 mg/cu m of white spirits. Cardiac output was normal at rest and increased in a normal manner as work increased during exposure at 1,250 and 2,500 mg/cu m of white spirits. Blood lactate content was unaffected by white spirits exposure.

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The authors [48] found that the concentrations of aliphatic and aromatic white spirits components in the air and blood differed

considerably. After a 30-minute exposure at rest to white spirits at 1,250 mg/cu m (1,038 mg/cu m of the aliphatic component), the concentration of the aliphatic component in the alveolar air was 255 mg/cu m (25% of the concentration in the inhaled air). The corresponding arterial and venous blood concentrations were 1.7 and 1.3 mg/kg, respectively. During the 50-watt exercise, the alveolar aliphatic component concentration increased to about 515 mg/cu m or about 50% of the concentration in the inhaled air. The arterial and venous blood concentrations were 3.5 and 2.4 mg/kg, respectively. During exposure at rest to about 210 mg/cu m of the aromatic components of white spirits (at a white spirits concentration of 1,250 mg/cu m), the aromatic concentration in the alveolar air after 30 minutes was about 30 mg/cu m, about 15% of the concentration in the inhaled air. The corresponding arterial and venous blood concentrations were both 0.2 mg/kg. During exercise at 50 watts, the alveolar concentration of the aromatic components increased to about 20% of the concentration in the inhaled air. The aromatic component concentrations in the arterial and venous blood were 0.9 and 0.6 mg/kg, respectively.

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Exposure to white spirits at a concentration of 2,500 mg/cu m (2,075 and 425 mg/cu m of the aliphatic and aromatic components, respectively) at rest produced alveolar concentrations of the aliphatic and aromatic components of 563 and 56.4 mg/cu m, respectively [48]. The arterial and venous aliphatic component concentrations were 3.4 and 2.2 mg/kg, respectively, while the arterial and venous aromatic components concentrations were 0.6 and 0.4 mg/kg. Alveolar air and arterial blood concentrations of the aliphatic components after white spirits exposure at 2,500 mg/cu m during exercise were approximately double those at the

resting level. The aromatic components in the alveolar air during exercise, however, were only 50% of those at the resting level. The increases in the aromatic components of the arterial and venous blood during exercise were similar to those seen for the aliphatic components.

When exercise intensity was increased successively to 150 watts during exposure to white spirits at a concentration of 1,250 mg/cu m, the alveolar aliphatic component concentration rose in steps from 256 to 622 mg/cu m, while the aromatic components rose from 28 to 59 mg/cu m [48]. In general, the alveolar concentrations of the aliphatic and aromatic components of white spirits in these studies leveled off after 10 minutes exposure and remained relatively unchanged during the rest of the exposure period. In contrast, the arterial and venous blood concentrations rose continuously throughout each exposure. The blood concentrations did show a plateau only after exposure at 1,250 mg/cu m of white spirits for 90 minutes.

In these studies, the authors [48] found a linear relationship between the arterial and alveolar concentrations of the aliphatic and aromatic components; the venous concentrations paralleled arterial concentrations. The total uptake of the aliphatic and aromatic components by the subjects was determined at rest during four consecutive 30-minute exposure periods. The uptake values were 59, 53, 47, and 46% of the total amount of the aliphatic components and 70, 64, 59, and 58% of the total amount of the aromatic components. During 50-watt exercise, the uptake was about 39% for the aliphatic components and 69% for the aromatic components. Thus, the proportion of aliphatic to aromatic components taken up decreased during exercise. However, the total uptake, measured in milligrams/period

of exposure, was slightly greater during exercise than at rest for both the aliphatic and aromatic components.

Astrand et al [48] concluded that measuring the solvent content of the inhaled or alveolar air was less reliable than measuring the blood concentration of aliphatic and aromatic components when assessing uptake, since the aliphatic components reacted as if they were not very soluble in blood while the aromatic components were relatively soluble. The authors noted that more solvent reached the blood during exercise than during rest. Thus, as would be expected, the degree of physical activity associated with a worker's job may influence solvent vapor toxicity.

In 1975, Gamberale and coworkers [50] studied the effects of exposure to white spirits (mineral spirits) on humans. Performance tests were conducted in perceptual speed, reaction time, short-term memory, numerical ability, and manual dexterity. Two sets of experiments were performed. In the first experimental series, 14 men, 18-34 years of age, were separated into 2 equal groups. One group was first studied under experimental conditions with exposure to white spirits and then, 7 days later, with exposure to air. The subjects in the second group were studied in a similar manner but in the reverse order. Under the experimental conditions, the first group was exposed to white spirits at 625, 1,250, 1,875, and 2,500 mg/cu m for four consecutive 30-minute periods. The concentration was increased after each 30-minute period without interrupting exposure. Gas chromatography was used to determine the white spirits concentration. The white spirits were supplied through a low air resistance breathing valve and mouthpiece. The presence or absence of white spirits was disguised by the introduction of menthol crystals into

the mouthpiece, since previous studies indicated that the subjects might taste and smell the solvent [48] which might, therefore, influence the experimental results. The five performance tests were always carried out in the same sequence during the final 20 minutes of each exposure period. Alveolar air samples were taken every 5 minutes and heart rate was monitored. At the termination of the experiment, the subjects were asked several questions to ascertain their perception of the experimental conditions.

From the answers to these questions, the authors [50] concluded that exposure to white spirits probably did not affect the subjective reactions in the psychologic experimental series. No difference in the heart rate of the subjects was noted between treatment and control situations. There was no impairment of the five performance tests as a result of solvent exposure. The resulting air concentrations of aliphatic components of the white spirits were about 175, 300, 450, and 600 mg/cu m at the 625, 1,250, 1,975, and 2,500 mg/cu m exposures, respectively; aromatic component concentrations at these exposures were about 25, 40, 50, and 75 mg/cu m. The solvent vapor concentrations were determined by gas chromatography.

In the second experiment [50], eight of the subjects who participated in the first experiment were exposed to 4,000 mg/cu m of white spirits for 50 minutes. During the final 20 minutes of exposure, the same performance tests used in the first experiment were performed. The subjects were also studied under control conditions without exposure to white spirits, as in the previous experiment. Half of the subjects were studied during control conditions 2 days before and the other half 2 days after the experimental trial. White spirits had no effect on perceptual speed, numerical ability,

and manual dexterity [50]. There was, however, a definite prolongation of reaction time and a possible impairment of short-term memory as a result of exposure at 4,000 mg/cu m. The alveolar concentrations of the aliphatic and aromatic components of the white spirits were about 850 and 100 mg/cu m, respectively.

(e) Stoddard Solvent

In 1940, Braunstein [51] reported that a 26-year-old man who worked in a drycleaning factory and had his forearms and hands wetted with or immersed in Stoddard solvent during most of the workday developed follicular dermatitis of the exposed skin after 2 weeks of employment. In the following 1 or 2 weeks, he felt nauseated after inhaling the fumes in the workroom. He continued to work in the factory for about 8 weeks longer before seeking medical aid after yellowing of the skin and four or five vomiting episodes had occurred. He was admitted to a hospital about 3 months after his first exposure to solvent.

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The patient felt weak and had lost 6 pounds during the previous 2 months; however, he regained this loss by the time of discharge from the hospital 1 month later [51]. Abnormalities shown by physical examination were jaundiced skin and eyes and a moderately enlarged liver. Temperature, pulse, and respiration values were normal. A roentgenogram of the abdomen was normal. He had an increased serum icteric index, decreased glucose tolerance, and increased erythrocyte resistance to hemolysis. The urine had traces of albumin, sugar, and urobilin; the feces contained bile. The blood urea nitrogen (BUN) content was increased; total erythrocyte count and hemoglobin value were decreased; total leukocyte count was normal; and some abnormal-sized erythrocytes were noted. A skin sensitization test

concentration than air controls and naive controls (animals never used in the experiment). Animals dying during or shortly after exposure showed marked congested or hemorrhagic lungs, whereas survivors after 24 hours showed no remaining irritation.

The authors [17] concluded that, since exposure of rats and dogs at 2,800 mg/cu m did not produce treatment-related signs of toxicity, and acute exposure of humans at 4,100 mg/cu m, but not at 660 mg/cu m, produced eye, nose, and throat irritation, a hygienic standard for VM and P naphtha should be set at 2,000 mg/cu m (430 ppm).

(d) Mineral Spirits

In 1966, Rector and coworkers [82] reported the effects of a paint thinner on five species of animals exposed continuously (23.5 hours/day, 7 days/week) for 60-90 days or intermittently for 8 hours/day, 5 days/week, for a total of 30 exposures. The mineral spirits used in this study were obtained from a Navy supply depot under the listing of "Paint Thinner, Mineral Spirits Grade I." This type of mineral spirits had a boiling point range of 140-190 C, a mean molecular weight of 144-169, and a specific gravity of 0.786-0.787. The compound was analyzed as a complex mixture of 80-86% saturated hydrocarbons, 1% olefins, and 13-19% aromatics.

Air concentrations of mineral spirits for this study [82] were calculated from nominal input since no satisfactory analytical procedure was available at the time the experiments were begun. Later in the study, the airborne concentration of mineral spirits was monitored and found to be 95% of the calculated nominal input with an average deviation of 4.8%. The concentration of airborne mineral spirits remained relatively constant in monitored experiments.

In continuous 90-day exposure experiments, rats, guinea pigs, rabbits of both sexes, and male dogs and monkeys were exposed to mineral spirits with concentrations ranging from 114 to 1,271 mg/cu m except for approximately 30 minutes/day, which were required for feeding and servicing of cages and chambers [82]. Exposure of the dogs, monkeys, and rabbits to the mineral spirits at any of these concentrations failed to produce death. An occasional death was noted in the rats exposed to the mineral spirits but the number of deaths (3/106) was similar to that of the controls (6/224). In contrast, guinea pigs were very susceptible to the mineral spirits with deaths occurring in all groups exposed at a concentration of 363 mg/cu m or more. The rate of body weight gain was generally similar in test animals and in controls, except in guinea pigs and monkeys. The decline in body weight became apparent in guinea pigs at 619 mg/cu m and in monkeys at 555 mg/cu m, in continuous exposure experiments. Continuous exposure of the guinea pigs and monkeys to mineral spirits at a concentration of 1,271 mg/cu m for 90 days resulted in body weight losses of 4 and 9%, respectively.

In dogs exposed to mineral spirits at 238 and 619 mg/cu m, a marked alteration between the preexposure and postexposure leukocyte counts was found [82]. The differential leukocyte counts and hematocrit and hemoglobin values were within normal limits. No consistent pattern of dose-response blood relationships was found except for a minor, but consistent, increase in the postexposure leukocyte counts in both rabbits and guinea pigs. This change was seen in both exposed animals and chamber controls. Hematologic data from the animals exposed at 1,271 mg/cu m were not reported.

Gross examinations of all animals were conducted at the end of each study [82]. While no remarkable changes were noted, irritation and congestion of the lungs were commonly found in all species. The severity of the irritation and the number of animals involved appeared to be dose-related. The livers of several guinea pigs appeared "discolored and wrinkled." No gross abnormalities were seen in the spleen, kidneys, or heart of any other species that could be attributed to solvent exposure.

Microscopic examination of the heart, lungs, liver, spleen, and kidneys was carried out on all surviving dogs, rabbits, and monkeys, and on 50% of the surviving guinea pigs and rats [82]. In general, congested lungs were found only in animals exposed to mineral spirits at a concentration of 1,271 mg/cu m. At this level, the lung tissue of all species showed evidence of bronchitis and mixed inflammatory cell infiltration. While occasional signs of lung irritation were seen at lower concentrations, the number of animals involved was such that the lung irritation could not be definitely related to the exposure. Microscopic examination of the heart, spleen, and kidney sections showed no findings that could be related to the exposure, but examinations of the liver yielded a mixed pattern. Focal necrosis, sometimes associated with worms, was seen in the livers of rats, rabbits, dogs, and monkeys. Mild-to-moderate vacuolar changes in the plate cells were noted in some guinea pigs and monkeys exposed at 363 mg/cu m and higher. This finding, however, was variable, and the incidence of liver damage did not correlate well with the exposure concentration. The vacuolar changes seen in the livers of guinea pigs were distributed in the peripheral area of lobules, although central and diffuse changes were also commonly seen. While no peripheral vacuolar

changes in the hepatic plate cells were seen in the control guinea pigs, some patchy vacuolar changes were observed. The authors concluded that only the hepatic changes noted in the animals exposed at 513 and 1,271 mg/cu m of mineral spirits were likely to have been caused by solvent exposure.

To find the explanation for the high mortality seen in guinea pigs exposed to mineral spirits at concentrations of 550 mg/cu m, Rector et al [82] investigated the serum alkaline phosphatase activity, serum isocitric dehydrogenase activity, liver lactate production, and serum or plasma urea levels in 10 treated and 10 control guinea pigs. However, the results of this experiment did not explain the high mortality in the guinea pigs.

The authors [82] also conducted three intermittent exposure studies in which the same five species, viz, rats, guinea pigs, rabbits, dogs, and monkeys, were exposed 8 hours/day, 5 days/week, for 30-60 exposures to mineral spirits at concentrations of 593, 596, or 1,353 mg/cu m. In the first study, animals exposed at 1,353 mg/cu m for 6 weeks showed no toxic signs. Body weight gains and blood values were similar in both exposed and control animals. No consistent microscopic changes were found except for possible lung irritation in guinea pigs. Seven of eight guinea pigs exposed at 1,353 mg/cu m showed some lung congestion and emphysema, which were not seen in the controls, and one of seven showed vacuolar changes in hepatic plate cells. In the second study, animals exposed at 596 mg/cu m for 6 weeks showed no signs of toxicity, and body weights and the hematocrit, hemoglobin, and total leukocyte count were all within normal limits. One-half of the rats and guinea pigs were killed and their tissues were examined microscopically. No noteworthy changes were found. After a

2-week nonexposure interval, the remaining half of the rats and guinea pigs were reexposed for a second series of 30 exposures at 593 mg/cu m. No noticeable signs of toxicity occurred during this second reexposure period, and the hematocrit, hemoglobin content, and total leukocyte count were within normal limits. Following a 17-day observation period, the animals were killed for necropsy. Microscopic findings indicated greater focal lymphocytic involvement in the lungs of several exposed guinea pigs than in the controls. No noteworthy microscopic changes were reported in any other tested species. There were no deaths in the animals exposed at 593 mg/cu m of mineral spirits.

Of the five species examined in this study, the guinea pigs were found to be particularly susceptible to mineral spirits [82]. As the air concentration of mineral spirits increased above 363 mg/cu m, guinea pigs began to die. No deaths occurred in guinea pigs during continuous exposures at 114 and 238 mg/cu m; no adverse changes were noted at these levels in any other species exposed. From these short- and long-term inhalation studies, the authors recommended that a guideline for a 90-day exposure period in submarines to mineral spirits containing 15-20% aromatic hydrocarbons be set at 40 mg/cu m.

In 1974, Gillespie et al [83] reported the effects of various paint components, including mineral spirits, on inflammatory response and tissue resistance to infection. Albino rabbits (2-3 kg) were used to assess the inflammatory response to the various paint components. A subcutaneous injection of mineral spirits at a dose of 0.1 ml was made into a shaved portion of the rabbit's back, and 4 days later the diameter of the indurated margins of the skin was measured. The indurated margin was about

2.4 cm in diameter and was greater than the indurated margins caused by most of the other paint components. The assessment of the resistance of tissue to infection was accomplished by subcutaneous injection of 0.1 ml of bacterial inoculum (10,000 or 100,000 *Staphylococcus aureus*) and a mineral solvents sample (injected at an unknown volume) and by measuring the inflammatory responses in terms of amount of induration and pus formation 4 days later. Mineral spirits caused increased induration, inflammation, and tissue necrosis but did not impair the ability of the wound to resist infection. Similar results were seen with other paint solvents and pigments.

(e) Stoddard Solvent

In 1974, Grant [54] reported that Stoddard solvent was essentially innocuous to the rabbit cornea. No details were given.

In 1975, Carpenter et al [21] reported the effects on rats of the inhalation of Stoddard solvent at 2,400, 4,600, or 8,200 mg/cu m (420, 800, or 1,400 ppm), based on a mean molecular weight of 144 calculated from mass spectrometry data and analyzed by gas chromatography, for a single 8-hour period. Groups of 15 rats were used at each exposure level. Exposure to 8,200 mg/cu m of Stoddard solvent resulted in the death of one of the rats at the termination of the inhalation period. Eye irritation, slight loss of coordination, and a bloody exudate around the nostrils were noted. A concentration of 4,600 mg/cu m produced similar signs but no loss of coordination. Inhalation of Stoddard solvent at 2,400 mg/cu m for 8 hours failed to cause any visible response during or after exposure; body weight gains were normal during the subsequent 14 days.

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reticulocyte counts at the 5,800 and 1,300 mg/cu m concentrations. The authors felt that the above changes were unimportant and could possibly be experimental artifacts rather than serious deleterious effects.

(d) Mineral Spirits

In 1975, Astrand et al [48] reported on the effects of white spirits (mineral spirits) on human alveolar air and blood solvent concentrations during rest and exercise. The white spirits used in the study consisted of 83% aliphatic and 17% aromatic components. In the initial trials, men were exposed at 2,500 or 5,000 mg/cu m for an unspecified period of time. Nausea and vertigo were apparent at both concentrations. No differences were noted in heart rate, alveolar ventilation, or oxygen uptake either at rest or during exercise at an intensity of 50 watts during exposure at 1,250 and 2,500 mg/cu m of white spirits. In addition, their studies [48] indicated that more solvent reaches the blood during exercise than during rest. This finding, in the absence of changes in alveolar ventilation, suggests changes in the respiratory transport.

In 1975, Gamberale et al [50] reported the effects of exposure to white spirits (mineral spirits) on humans. Performance tests were conducted in perceptual speed, reaction time, short-term memory, numerical ability, and manual dexterity. Men exposed to white spirits at 625, 1,250, 1,875, and 2,500 mg/cu m for four continuous 30-minute periods showed no impairment of the five performance tests. Exposure to 4,000 mg/cu m of white spirits for 50 minutes had no effect on perceptual speed, numerical ability, and manual dexterity. There was, however, a definite prolongation of reaction time and a possible impairment of short-term memory as a result of exposure at 4,000 mg/cu m. The authors concluded that there was a risk

of subjective distress and adverse effects on psychomotor and intellectual functions in a worker exposed to 2,500 mg/cu m who is doing light industrial work, since the alveolar air concentrations of white spirits in workers at rest exposed at 4,000 mg/cu m was similar to the white spirits alveolar air concentration of workers exposed at 2,500 mg/cu m doing light physical activity [48,50].

Rector et al [82], in 1966, described the effects of mineral spirits on five species of animals exposed continuously for 60-90 days or exposed intermittently, 8 hours/day, 5 days/week, for a total of 30-60 exposure periods. In continuous 90-day exposure experiments, rats, guinea pigs, rabbits, dogs, and monkeys were exposed to mineral spirits of concentrations ranging from 114 to 1,271 mg/cu m (18-200 ppm, assuming a molecular weight of 156). Exposure of the dogs, monkeys, and rabbits to the mineral spirits at all concentrations tested failed to induce mortality. An occasional death was noted in the rats at all concentrations, but the number of deaths was similar to that of the controls. In contrast, the guinea pigs were very susceptible to the mineral spirits with deaths occurring in all groups subjected at a concentration of 363 mg/cu m (60 ppm) or greater. No deaths occurred in the guinea pigs exposed at 114 or 238 mg/cu m (18 or 37 ppm). The rate of body weight gain generally was similar in test animals and in controls except in guinea pigs and monkeys exposed at the highest concentration of 1,271 mg/cu m. In the overall study, no consistent pattern of dose-response hematologic relationships was found, and, therefore, some of the alterations seen in preexposure and terminal leukocyte counts could not be attributed to solvent exposure. Although no remarkable changes were noted

on gross examination of all animals, lung irritation and congestion were observed in all species. In general, the observations of congested lungs were substantiated microscopically in only those animals exposed to mineral spirits at 1,271 mg/cu m, where lung tissue showed evidence of bronchitis and mixed inflammatory cell infiltration. Microscopic examination of the heart, spleen, and kidneys did not show adverse findings that could be attributed to solvent exposure.

Rector et al [82] also conducted three intermittent exposure studies in which the same five species were exposed 8 hours/day, 5 days/week, for 30-60 exposures to mineral spirits at concentrations of 593-596 or 1,353 mg/cu m (93-94 or 212 ppm). The animals exposed at 1,353 mg/cu m for 6 weeks showed no toxic signs and body weight patterns and hematologic values were similar to those of the controls. No consistent microscopic changes were found except for possible lung irritation and liver damage in guinea pigs exposed at 1,353 mg/cu m. Animals exposed at 596 mg/cu m for 6 weeks showed no signs of toxicity and body weight gains and hematologic parameters were all within normal limits. No noteworthy microscopic tissue changes were reported. After a 2-week recovery interval, several rats and guinea pigs who were exposed previously to 596 mg/cu m were reexposed for a second series of 30 exposures at 593 mg/cu m. There were no noticeable signs of toxicity during this second reexposure and hematologic parameters were within normal limits. After a 17-day observation period, the animals were killed and autopsied. The only noteworthy microscopic finding was focal lymphocytic involvement in the lungs of some exposed guinea pigs.

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(4) Mineral Spirits

While mineral spirits and Stoddard solvents are not always considered the same petroleum products and are used differently for different purposes in industry, their boiling ranges (Stoddard solvents, 160-210 C; mineral spirits, 150-200 C) [1] are almost identical, and, therefore, their chemical compositions are similar. In fact, many investigators use the terms mineral spirits and Stoddard solvents interchangeably [3,4,18,19,21].

White spirits (mineral spirits) at concentrations of 2,500 mg/cu m or greater have been shown to cause nausea and vertigo in humans [48]. Concentrations of 625-2,500 mg/cu m (about 98-392 ppm) of white spirits for periods up to 2 hours had no effect on performance tests, such as perceptual speed, reaction time, short-term memory, numerical ability, and manual dexterity [50]. Exposure to white spirits at 4,000 mg/cu m (627 ppm) for 50 minutes caused a prolongation of the reaction time and a possible impairment of short-term memory [50]. The concentration of white spirits in the alveolar air during exposure to 4,000 mg/cu m, at rest, was similar to the alveolar air concentration of this solvent when the white spirits concentration was 2,500 mg/cu m and the subject was doing light manual work [48,50]. Thus, as should be expected, physical activity can increase the solvent concentration in the lungs and magnify its toxic potential.

Rector et al [82] observed that five species of animals exposed continuously for 60-90 days or exposed intermittently for 8 hours/day, 5 days/week, for 6 weeks to mineral spirits at 1,271 mg/cu m (200 ppm) showed no consistent pattern of dose-related hematologic relationships or any

remarkable gross changes except for lung irritation. Deaths were not seen at any concentration in rats, rabbits, dogs, or monkeys; however, some guinea pigs exposed at concentrations of 363 mg/cu m (57 ppm) or greater died. No guinea pigs died as a result of exposure at 288 mg/cu m (37 ppm). Animals intermittently exposed at 593-1,353 mg/cu m (93-212 ppm) displayed no adverse toxic signs except for slight lung irritation. The lung irritation was seen mainly in the animals exposed at 1,353 mg/cu m for 8 hours/day, 5 days/week, for 6 weeks.

Unless workers are several times more sensitive to intoxication by these hydrocarbons than the animals tested by Rector and coworkers [82], these animal data do not argue for a limit as low as those for the previously discussed solvents, 350 mg/cu m, inasmuch as the exposures were continuous rather than the intermittent type of exposure encountered in occupational exposure. Other tests with humans [48,50], limited mainly to acute sensory or performance responses, also suggest that a higher limit might be acceptable. However, none of these data give assurance that chronic intoxication, such as the polyneuropathy associated with one or more of the lower molecular weight hydrocarbons, might not occur. Exposures to jet fuels, ie, to mixtures of kerosene and gasoline, have caused polyneuropathy [77]. While the C5-C8 alkanes present in the jet fuel might have caused the neurologic effects, a contribution by higher boiling fractions seems possible. Since the workers described did not have eye irritation, it seemed to the authors that kerosene, which, unlike gasoline, is not a significant eye irritant, was the major component in the exposure mixture. In view of the uncertainties, it is proposed that a more conservative approach be followed until more definitive data are available,

specifically, that the same limit proposed for lower boiling fractions be recommended, viz, 350 mg/cu m as a TWA concentration.

Mineral spirits have been shown to cause dermatitis [72], and Stoddard solvents have been recognized as being capable of causing skin irritation [51,75] and possibly aplastic anemia [52,53] after dermal exposure. Of these several cases of aplastic anemia, one may have been from myelodepressant drugs. While benzene may not be expected to be an important contaminant of mineral spirits or Stoddard solvents, because of the boiling range, there is no other evident cause of anemia. From present knowledge, there seems to be no reason in addition to a small number of case histories to suggest that aliphatic hydrocarbons can cause aplastic anemia, but further research on the point seems warranted. Since there is a similar composition between mineral spirits and Stoddard solvents, it is recommended that dermal exposure to mineral spirits be avoided. The benzene content of mineral spirits should be evaluated to ensure that the federal exposure limit for benzene is not exceeded.

(5) Stoddard Solvents

There are four classes of Stoddard solvents that are used in the drycleaning industry: regular Stoddard solvent, 140 flash solvent, odorless solvent, and low end point solvent [24]. Inhalation toxicologic data exist for only two of the four classes: regular Stoddard solvent [21] and 140 flash solvent [56]. The studies [51-53,55] that did not specify the specific class of this solvent were considered to be regular Stoddard, since it is the solvent primarily used by the drycleaning industry and is commercially known as "Stoddard solvent" [25]. Henceforth in this

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STODDARD SOLVENT

Mineral Spirits; White Spirits

$C_{10}H_{22}$

TLV, 100 ppm ($\approx 525 \text{ mg/m}^3$)

STEL, 200 ppm ($\approx 1050 \text{ mg/m}^3$)

Stoddard solvent is a mixture of straight and branched chain paraffins, naphthenes (cycloparaffins) and aromatic hydrocarbons. It is a colorless liquid with a kerosene-like odor. Using the chemical formula above, the molecular weight would be 128.25. The reported specific gravity is 0.79, boiling range from 154 to 202° C and a flash point greater than 100° F. Insoluble in water, Stoddard solvent is miscible with benzene, absolute alcohol, ether, chloroform, carbon tetrachloride and carbon disulfide.

It is used as a diluent in paints, coatings and waxes; as dry cleaning agent; as a degreaser and cleaner in mechanical shops; and as a herbicide.

Aspiration⁽¹⁾ of the liquid results in diffused chemical irritation of the lungs resulting in edema, a few milliliters may be fatal in these incidents.

Carpenter *et al.*⁽²⁾ found that inhalation of 8200 mg/m³ (1400 ppm), substantially air saturation at 25° C, caused death of 1 or 15 rats in 8 hours. Beagle dogs and cats had spasms and died at this concentration. There were no significant effects in dogs that inhaled 330 ppm, 190 ppm and 84 ppm, 6 hours daily, 5 days/week for 13 weeks (65 exposure days). However, rats at 330 ppm for 65 days showed slight kidney damage. The rats from 300 ppm group had an increase in blood-urea-nitrogen after 65 days. This may be associated with masked tubular regeneration and dilation of the loops of Henle as noted above for this level.

In 15-minute inhalation period for people, slight eye irritation was reported in 1 of 6 at 150 ppm.

Rector *et al.*⁽³⁾ exposed rats, guinea pigs, rabbits, dogs, and monkeys 8 hours/day, 5 days/week for 30 exposure days and also for 90 days continuously to vapors of mineral spirits used as a paint thinner by U.S. Navy. Their samples met the Stoddard solvent specifications. In an 8-hour exposure at 290 ppm there was minor congestion and emphysema in guinea pigs lungs only. The rats did not show kidney lesions, which was different than Carpenter's findings for his strain of rats. None of the other test species showed any signs of physiological damage at 290 ppm.

Because Stoddard solvent contains 65% or more C_{10} and higher molecular weight hydrocarbons⁽⁴⁾ proper recognition of decane and its homolog's hazardous nature should be taken.

Nau *et al.*⁽⁵⁾ found n-decane had an LC_{50} of 540 ppm for mice exposed for 3.75 hours. This level was borne for 18 hours/day, 7 days/week by rats without significant effects after 123 days. Also, the n-decane was skin absorbed where application of 16.33 grams (total) was applied 3 times

weekly for 50 weeks. The kidneys and lung were the most seriously effected. When benzene in like manner was tested, it gave no evidence of skin absorption⁽⁴⁾ and the LC_{50} for benzene was found to be 1300 ppm. In comparison, other studies have indicated lethal concentrations for mice to be 16,000 ppm for heptane,⁽¹⁾ 13,500 ppm for octane,⁽⁵⁾ and 3200 ppm (LC_{50} 4 hours)⁽⁶⁾ for nonane.

Millions of industrial and domestic workers have been exposed to Stoddard solvent with minimal evidence of serious health effects, apart from its defatting and irritating action on the skin.

Relatively few data are available on the actual concentrations of vapor such exposures have involved, however. Oberg, in a survey of 30 dry cleaning plants in Detroit, found an average exposure of 65 ppm, with a TWA of 35 ppm, for Stoddard solvents with flash points of about 105° F.⁽⁷⁾ The worst plant had an estimated average exposure of 135 to 200 ppm.

Carpenter *et al.*⁽²⁾ suggested 200 ppm as a hygienic standard for man.

NIOSH, on the other hand, proposed a workplace environmental standard of 350 mg/m³, corresponding to about 60 ppm.⁽⁸⁾ The NIOSH recommendation, on a mg/m³ basis, is the same for all refined petroleum solvents, from petroleum ether to mineral spirits and Stoddard solvents, including 140 Flash aliphatic solvent. The latter is not included in this discussion. NIOSH also proposed a 15 minute ceiling of 1800 mg/m³, or about 310 ppm.

It is recommended that the current TLV of 100 ppm be retained for Stoddard solvent. This limit was calculated from data on the toxicities of its major ingredients, and was designed primarily to prevent the irritative and narcotic effects of the vapors. Subsequent data, although somewhat difficult to interpret, tend to confirm this value, in the opinion of the Committee. The occurrence of questionable kidney injury from exposures not greatly in excess of 100 ppm⁽²⁾ provides additional evidence of the desirability of a lower TLV than that which existed prior to the adoption of the 100 ppm value.

A STEL of 200 ppm, somewhat lower than the ceiling limit recommended by NIOSH, is suggested.

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