Submission No 87

Inquiry into RAAF F-111 Deseal/Reseal Workers and their Families

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Submission

Dangers of Ethylene Glycol Monomethyl Ether and other products known as Fuel Systems Icing Inhibitors within the Australian Army Aviation Combat System.

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Report for F111 Deseal/Reseal Inquiry

1. History

1.1 In 1960 the modernising Australian Army created a unit dedicated to combat supply of fuels whenever the Services deployed. This unit was developed to supply fuels for Ground Vehicles including Armoured, Aviation Units including Rotary Winged and had the capacity to work with naval vessels as well

1.2 Based at Puckapunyal the unit was designated as 8 Petroleum Platoon, Royal Australian Army Service Corps (RAASC). Originally operating with English equipment it soon learnt that United States equipment held better potential and, with some adapting, began servicing the combined services of the Australian Military

1.3 In dealing with fuel the soldiers learnt about the components of each substance and some dangers. Of high emphasis was the deadly effect of Tetraethyl Lead especially when cleaning rubber storage tanks. The equipment provided for the individuals safety when doing such tasks were overalls, rubber gloves and a Commonwealth Industrial Gas Mask with a filter to reduce inhalation of fumes plus a blower at one end which inflated the rubber storage tank and allowed access for operators. Cleaning equipment was a mop and rags.

1.4 The only other precaution at this time was an extra ration on 1 litre of milk per Petroleum Operator (the trade designation for members of the unit) per

day. This was later found to have no real benefit nor could it be supplied regularly to provide any perceived benefit.

2. Fuel Systems Icing Inhibitor (FSII)

2.1 All members of the unit were aware that aviation fuel supplied was mixed with FSII usually **Ethylene Glycol Monomethyl Ether** but other substances may have been utilised from time to time. Petroleum Operators were not made aware of the dangers of this substance although they needed to ensure upon receipt of aviation fuel deliveries that this substance was included in the product or a mixing was required to enable the fuel to be used by aircraft being supplied.

2.2 Aircraft commonly worked with in the 1960's included Mirage Fighters, Canberra Bombers especially in an annual exercise using Tindal Airbase (which was nothing like the metropolis it is now), Caribou Transports, Iroquois Helicopters and Bell Sioux. The fuel remains in service to day and this product would have had residue in the F111 fuel tanks and contributed to the effects of Aircraft Fitters working within an enclosed environment.

2.3 Members of 8 Petroleum Platoon were deployed to Vietnam as part of the Australian Commitment and the major fuel supplied was Aviation Turbine Kerosene with FSII (AVTUR). Capacity at Nui Dat base alone was able to supply 40,000 litres per day at the Kangaroo Helipad. This does not include supplies of AVTUR and Aviation Gasoline(AVGAS) to 161 Recce Flight or to Petroleum Operators in the field providing 'on site' supplies where required

2.4 No protective equipment was supplied for the dispensing of this fuel except for rubber gloves used daily quality control checks. The fuel at Kangaroo Helipad or inside Nui Dat was often dispensed wearing the standard greens or even in shorts and boots on extremely hot days. In the field it was the standard Army issue greens.

3. Dangers

3.1 Since the 1980's the dangers of FSII have come to light with changes to aircraft in the civilian aviation fuel industry to allow larger aircraft to mixing the substances as required in flight but continues to be supplied for use by the military and smaller aircraft.

3.2 Accordingly in recent years various agencies have provided advice relating to the products with the following indications being common[1];

Hazards Identification

Emergency Overview

WARNING! FLAMMABLE LIQUID AND VAPOR. MAY FORM EXPLOSIVE PEROXIDES IN AIR. HARMFUL IF SWALLOWED, INHALED OR ABSORBED THROUGH SKIN. AFFECTS CENTRAL NERVOUS SYSTEM, BLOOD AND BLOOD FORMING ORGANS, REPRODUCTIVE SYSTEM AND KIDNEYS. POSSIBLE BIRTH DEFECT HAZARD. MAY CAUSE BIRTH DEFECTS BASED ON ANIMAL DATA. MAY CAUSE IRRITATION TO SKIN, EYES, AND RESPIRATORY TRACT.

SAF-T-DATA(tm) Ratings (Provided here for your convenience)

Health Rating: 3 - Severe (Life)
Flammability Rating: 2 - Moderate

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[11] Materials Handling Data Sheet (part)
http://www.jtbaker.com/msds/englishhtml/e5300.htm as at 7 July 2008

Reactivity Rating: 2 - Moderate
Contact Rating: 3 - Severe (Life)
Lab Protective Equip: GOGGLES & SHIELD; LAB COAT & APRON; VENT HOOD; PROPER GLOVES; CLASS B EXTINGUISHER
Storage Color Code: Red (Flammable)

Potential Health Effects

Inhalation:
Inhalation of vapors may cause irritation, headache, dizziness, fatigue, nausea, vomiting, and loss of appetite. Weakness, incoordination and tremors may occur.

Ingestion:
Moderately toxic. Can cause headache, fatigue, nausea, vomiting, dizziness, and weakness. Hemorrhagic gastritis, liver damage, pancreas damage and brain oedema resulting in death has occurred in human exposure of 3 g/kg. Damage to kidneys is possible from ingestion of large quantities.

Skin Contact:
May cause irritation with redness and pain. May be absorbed through the skin with possible systemic effects.

Eye Contact:
May cause irritation, redness and pain.

Chronic Exposure:
Prolonged exposure may cause injury to bone marrow, blood cells, kidney, liver and testes. A suspected human reproductive hazard and a birth defect hazard. Severe neurological disabilities has been reported from chronic industrial exposure. Symptoms have included headache, dizziness, lethargy, weakness, personality changes, apathy, unequal pupil size, and disorientation.

Aggravation of Pre-existing Conditions:
Persons with pre-existing blood or central nervous system disorders may be more susceptible to the effects of this substance.

3.3 Female Soldiers have been removed from the Petroleum Units due to dangers to their reproduction system yet the author knows of no action to protect males occurring

3.4 Further indications of the dangers of these products exist in numerous references especially concerns re cancers (see Annex A).

3.5 The illnesses indicated have been recognised in both Aircraft Fitters and Retired Petroleum Operators whether having served overseas on active duty or not. Petroleum Operators were refused permission to present information to the F111 Deseal/Reseal Inquiry yet similarities occur throughout the reports findings.

4 Conclusion

4.1 While the results of the F111 Deseal/Reseal ought to be applauded as it recognised the dangers of numerous products used in the operation the outcome should include compensation for members and families of all services who operate with the designated fuels and products relate to military aviation as the dangers are equally shared.

4.2 During any refuel process utilising fuels with FSII all possible safety equipment ought to be available to Refueller/Petroleum Operator to reduce all possible risks. This obviously has to be suitable to operational requirements but the safety of operators should be paramount. Only qualified operators should refuel aircraft after ensuring the safety of those within the vicinity including any personnel aboard aircraft especially in the hot (engines operating) refuel situation.

4.3 All Military Arms need to work together to develop safe operating procedures including regular reviews and trade information relating to equipment failure or dangerous incidents when they occur and not months or even years after the event.

ANNEX A

CHRONIC TOXICITY SUMMARY

ETHYLENE GLYCOL MONOMETHYL ETHER

(EGME; 2-methoxyethanol; 1-hydroxy-2-methoxyethane; methyl cellosolve)

CAS Registry Number: 109-86-4

I. Chronic Toxicity Summary

Inhalation reference exposure level	**60 µg/m^3 (20 ppb)**
Critical effect(s)	Testicular toxicity in rabbits
Hazard index target(s)	Reproductive system

II. Physical and Chemical Properties (HSDB, 1995)

Description	Colorless liquid
Molecular formula	$C_3H_8O_2$
Molecular weight	76.09
Density	0.965 g/cm^3 @ 20° C
Boiling point	125°C
Melting point	-85.1°C
Vapor pressure	6.2 torr @ 20°C
Solubility	Miscible with water, alcohol, benzene, ether, acetone
Conversion factor	1 ppm = 3.1 mg/m^3 @ 25°C

III. Major Uses and Sources

Ethylene glycol monomethyl ether (EGME) is used as a solvent for cellulose acetate and resins (HSDB, 1995) as well as a solvent in the semiconductor industry. It is also used in dyeing leather and in the manufacture of photographic film. EGME is used as an anti-freeze in jet fuels. Quick drying varnishes, enamels, nail polishes, and wood stains may also contain EGME. The specific annual statewide industrial emissions of EGME from facilities reporting under the Air Toxics Hot Spots Act in California based on the most recent inventory were estimated to be 7398 pounds (CARB, 1999). (Many industries did not report emissions of specific glycol ethers. Thus there were also emitted 2,922,744 pounds of the general category glycol ethers, which can include EGME.)

IV. Effects of Human Exposure

Human exposures to ethylene glycol monomethyl ether have been associated with hematological and neurological abnormalities. To determine whether employees potentially exposed to ethylene glycol monomethyl ether during manufacturing and packaging had a higher prevalence

of anemia, leukopenia, or sterility than an in-plant comparison group, a cross-sectional study was conducted. Blood samples on 65 of 97 potentially exposed and control white males, and semen samples from a subset of 15 were analyzed. No gross abnormalities or clinically meaningful differences in hematological or fertility indices were noted. Decreased testicular size was reported in workers (who were exposed to an 8-hour TWA concentration of 0.42 ppm EGME or less) but it was not statistically significant (Cook *et al.*, 1982).

Cullen *et al.* (1983) studied possible bone marrow toxicity of workplace substances including dipropylene glycol monomethyl ether, EGME, and various aliphatic, aromatic and halogenated hydrocarbons used for offset and ultraviolet cured multicolor printing. Evaluation of seven co-workers of a printer with aplastic anemia indicated normal peripheral blood, but bone marrow specimens demonstrated clear patterns of injury in three while the others had nonspecific signs of marrow effect. The authors could not assign the changes to known risk factors and concluded that further evaluation of possible bone marrow toxicity resulting from exposure to glycol ethers and ultraviolet curing printing processes was warranted. This was done to some extent in their studies on shipyard painters below.

Welch and Cullen (1988) evaluated shipyard painters exposed to ethylene glycol ethers (EGEE and EGME). Air concentrations at the workplace were estimated based on 102 samples over six shifts in Sparer *et al.* (1988). Time –weighted average (TWA) exposures to EGEE ranged from 0 to 80.5 mg/m3 with a mean of 9.9 mg/m3. TWA exposures to EGME ranged from 0 to 17.7 mg/m3 (mean = 2.6 mg/m3). The authors note that during the time period of measurement, painting activities were unusually low and previous NIOSH analyses indicated considerably higher exposures. Ninety-four painters and 55 controls answered a medical and environmental exposure questionnaire including work history and provided blood, urine, and in some cases semen samples. Mean hemoglobin levels, total cell counts and differential counts did not differ between exposed and control. However, the authors found that the lowest quartile of hemoglobin was mostly painters and the lowest polymorphonuclear leukocyte counts were in painters. Nine painters were considered anemic and five were considered granulocytopenic. The authors note that the absence of a significant difference in the group as a whole and the inability to detect a dose-response pattern in the exposed group makes a strong conclusion unwarranted.

Welch *et al.* (1988) evaluated the semen samples from the workers in the cohort from Welch and Cullen (1988). Sperm concentration, velocity, motility, morphology, morphometry, and viability were measured. Although not statistically significant, the measures of sperm count tended to be lower in the painters with a $p = 0.10$ for density and $p = 0.11$ for count. When nonsmokers were analyzed separately from smokers, the number of oligospermic painters was larger than that in controls at $p = 0.05$. There was no difference between controls and exposed men who were smokers. The authors state that although mean values of sperm count did not differ significantly between controls and exposed groups, biologically important differences were seen when the proportion of men with oligospermia was examined. The proportion of painters with azoospermia was 5% with only 1% expected based on other population surveys. The authors note that to create a dose-response model for an effect of glycol ethers on semen parameters would require description of the exposure of each individual 3 to 6 months prior to sampling.

The painters moved frequently from one exposure area to the next, making exposure assessment particularly difficult in this cohort.

Cullen *et al.* (1992) conducted a histopathologic analysis of the bone marrow and circulating blood cells in the workers previously examined in Welch *et al.* (1988). The objects of the study included : 1) to exclude other causes for granulocytopenia and depressed hemoglobin levels noted in some painters exposed to ethylene glycol ethers, 2) to determine if subclinical evidence of hematologic damage is present in healthy coworkers, and 3) to identify host or exogenous factors which may increase the risk of hematologic damage in glycol ether exposed painters. Workers were grouped as follows: Group I consisted of those painters that had anemia or granulocytopenia in the Welch and Cullen (1988) study; Group II consisted of exposed painters with normal hematology; Group III consisted of unexposed controls. A battery of hematologic and biochemical parameters were measured and a questionnaire was completed to determine occupational exposure status, health status and drug and alcohol consumption. All hematologic parameters were normal in all groups. Tests of liver, renal, and thyroid function were normal in all groups. Bone marrow histology showed no differences between groups. One biochemical parameter, pyruvate kinase activity, was lower in Group I than Groups II and III ($p = 0.05$). Depression of red cell pyruvate kinase did not vary by race and was lower in every subject in Group I by more than one standard deviation. Low pyruvate kinase is the most consistent red cell enzyme defect noted in acquired hematologic disorders.

Reversible neurological symptoms (apathy, fatigue, decreased appetite) and macrocytic anemia were observed in a worker following occupational dermal and inhalation exposure to an average concentration of 35 ppm EGME for 1-1.5 years (Cohen, 1984). The worker was also exposed to methyl ethyl ketone and propylene glycol monomethyl ether at concentrations of 1-5 ppm and 4.2-12.8 ppm, respectively.

Hematologic effects were also reported in three women employed in a factory working with glue consisting of 70% acetone and 30% EGME (Larese *et al.*, 1992). The women exhibited abnormally low white blood cell counts, relative lymphocytosis and macrocytosis. These hematological parameters returned to normal following cessation of exposure.

Older case reports support findings of neurological and hematological toxicity following occupational exposure to EGME (Greenburg *et al.*, 1938; Zavon, 1963; Parsons and Parsons, 1938).

V. Effects of Animal Exposure

A concentration dependent decrease in testes weight was observed in male rabbits exposed to 30, 100, or 300 ppm EGME 6 hours per day, 5 days per week for 13 weeks (Miller *et al.*, 1983). Degenerative changes in the germinal epithelium were observed in male rabbits of all exposed groups, but were not statistically significant at 30 ppm. Two of five male rabbits exposed to 300 ppm EGME died during the course of the study. Female rabbits were also exposed; two of five female rabbits exposed to 100 or 300 ppm EGME died during the course of the study. The animals died at different times of different causes and thus the authors were uncertain if the

deaths were treatment related. Reduced body weight gain, pancytopenia (abnormal depression of all the cellular elements of the blood), and thymic atrophy were observed in rabbits of both sexes exposed to 300 ppm EGME. No effects on the reproductive organs of the female rabbits were observed.

In the same study (Miller *et al.*, 1983) male and female rats were exposed to 30, 100, or 300 ppm EGME 6 hours per day, 5 days per week for 13 weeks. Moderate to severe degeneration of the germinal epithelium and seminiferous tubules was observed in male rats exposed to 300 ppm EGME. A significant decrease in body weight was observed in male rats exposed to 300 ppm and in female rats exposed to concentrations of EGME of 100 ppm or greater. Pancytopenia, lymphoid tissue atrophy, and decreased liver weights were observed in animals of both sexes exposed to the highest concentration. Also in the highest exposure group, mean values for total serum protein, albumin and globulins were lower than control values.

Doe *et al.* (1983) designed a two-part study to provide a rapid assessment of the effect of glycol ethers on some aspects of reproduction in the rat. Exposure to EGME was by inhalation at 100 and 300 ppm for 6 hr/day. First, pregnant females were exposed on Days 6 to 17 of gestation. Body weight gain was reduced in both groups. No litters were delivered in the 300-ppm group and only 9/20 rats in the 100-ppm group produced litters; the number, weight, and viability of the pups were reduced, but the pups appeared normal externally. Second, male rats were exposed for 10 days. There was a reduction in testicular weight accompanied by seminiferous tubular atrophy in the 300-ppm group. There were no effects at 100 ppm. Exposure at 300 ppm EGME caused significant reductions in white blood cell count, red blood cell count, hemoglobin concentration, hematocrit, and mean cell hemoglobin.

More recent data point to the immune system as a key endpoint of EGME toxicity. A statistically significant dose-related decrease in thymus weight was observed both in male rats administered drinking water containing 2000 and 6000 ppm EGME (161 or 486 mg/kg/day) and in female rats administered drinking water containing 1600 and 4800 ppm EGME (200 or 531 mg/kg/day) for 21 days (Exon *et al.*, 1991). Histopathological examination revealed thymic atrophy and loss of demarcation between the cortex and medulla. Decreased spleen cell numbers were observed in female rats at both dose levels and male rats at the high dose level. Male rats in the high dose group exhibited a statistically significant decrease in body weight gain. Testicular effects were also observed in exposed male rats.

Pregnant mice were exposed to 100, 150, or 200 mg/kg/day EGME on days 10-17 of gestation (Holladay *et al.*, 1994). Thymic atrophy and inhibition of fetal thymocyte maturation were observed in EGME-treated offspring examined on day 18 of gestation. Also, the ability of the EGME-treated fetal mouse liver cells to repopulate the spleen of irradiated mice was significantly impaired as compared to that of control fetal mouse liver cells.

VI. Derivation of Reference Exposure Level

Study	Miller *et al.*, 1983; U.S. EPA, 1995
Study population	Rats and rabbits
Exposure method	Inhalation (0, 30, 100, or 300 ppm)
Critical effects	Decreased testes weight and degenerative hanges in the testicular germinal pithelium.
LOAEL	100 ppm
NOAEL	30 ppm
Exposure continuity	6 hours/day, 5 days/week
Average experimental exposure	5.4 ppm for NOAEL group
Human equivalent concentration	5.4 ppm for NOAEL group (gas with itemic effects, based on RGDR = 1.0 using fault assumption that lambda (a) = lambda)
Exposure duration	13 weeks
LOAEL uncertainty factor	1
Subchronic uncertainty factor	10
Interspecies uncertainty factor	3
Intraspecies uncertainty factor	10
Cumulative uncertainty factors	300
Inhalation reference exposure level	0.02 ppm (20 ppb; 0.06 mg/m^3; 60μg/m^3)

The REL is based on the same study on which U.S. EPA based its RfC. However, OEHHA declined to use a modifying factor because the criteria for use of such factors are not well described by U.S. EPA. However, since rabbits were the more sensitive species and live 6 years (312 weeks), a 13 week study in rabbits merits a subchronic UF of 10.

A comparison with the proposed REL for EGME of 20 ppb (60 μg/m^3) can be made using the occupational study of Welch *et al.* (1988) of the semen of shipyard painters exposed to both EGEE and EGME. The men supplied demographic characteristics, medical conditions, personal habits, and reproductive history; underwent a physical examination; and provided a semen sample. The painters were exposed to EGEE at a TWA concentration of 0 to 80.5 mg/m^3 (mean = 9.9 mg/m^3, and to EGME at a TWA concentration of 0 to 17.7 mg/m^3 (mean = 2.6 mg/m^3). The painters had an increased prevalence of oligospermia and azoospermia and an increased odds ratio for a lower sperm count per ejaculate compared to shipyard employees who were not painters. (The results were controlled for smoking.) Adding the mean exposure levels together results in a total glycol ether concentration (EGME + EGEE) of 12.5 mg/m^3, equivalent to a continuous exposure of 4.5 mg/m^3. Division by a UF of 10 for a LOAEL and by another of 10 for human intraspecies variability results in a REL of 40 μg/m^3 (10 ppb), similar to the REL based on rabbits. Since exposure was primarily to EGEE with co-exposure to EGME, and exposure assessment ws difficult to quantify, this study was not deemed suitable for developing a REL. Nonetheless, the REL developed using this study is close in value to the proposed REL of 20 ppb.

VII. Data Strengths and Limitations for Development of the REL

The strengths of the inhalation REL for EGME include the availability of subchronic inhalation exposure data from a well-conducted study with histopathological analysis and the observation of a NOAEL. In addition, there are a number of human studies showing similar toxicological endpoints to those demonstrated in animal studies. Major areas of uncertainty are the lack of adequate human exposure data, and the lack of chronic inhalation exposure studies.

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