

UNITED STATES AIR FORCE ARMSTRONG LABORATORY

INHALATION TOXICITY OF VAPOR PHASE LUBRICANTS

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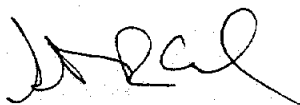
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The animal use described in this study was conducted in accordance with the principles stated in the "Guide for the Care and Use of Laboratory Animals", National Research Council, 1996, and the Animal Welfare Act of 1966, as amended.

This report has been reviewed by the Office of Public Affairs (PA) and is releasable to the National Technical Information Service (NTIS). At NTIS, it will be available to the general public, including foreign nations.

This technical report has been reviewed and is approved for publication.

FOR THE DIRECTOR



STEPHEN R. CHANNEL, Maj, USAF, BSC
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Air Force Armstrong Laboratory

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| 13. ABSTRACT (Maximum 200 words) Vapor phase lubrication is a relatively new lubrication concept which shows excellent potential for high temperature applications. One fluid which has received extensive evaluation is tri-cresyl phosphate (TCP). When heated to the vapor state, TCP undergoes repolymerization and possibly other chemical changes. TCP is known to have a toxic ortho-isomer (TOCP). A single exposure to a neurotoxic organophosphorous (OP) compound can produce damage to nerves after a delay of 8 to 10 days. This condition is known as organophosphorus-induced delayed neuropathy (OPIDN), and is characterized by axonal degeneration. OPs that cause axonal pathology interact with the enzyme NTE. Changes in the activity of this enzyme NTE is the initial step in the delayed neurotoxicity response. Exposure to all triaryl phosphate vapor phase lubricants resulted in NTE inhibition. The level of NTE inhibition generally accepted to be predictive of OPIDN in hens is 70% [1]; however, hens were notably impaired three weeks after administration of OPIDN-inducing compounds when spinal cord NTE was inhibited more than 40% [5]. Additionally, NTE inhibition as low as 31% has been shown to be associated with development of severe spinal cord damage in rats [3]. Thus, the threshold for OPIDN may well be below a 70% inhibition of NTE. Exposure to these lubricants in the vapor phase inhibits NTE. Thus, the process of vaporization is causing a change in the compound, resulting in the potential to produce neurotoxicity. Therefore, caution must be used when working with triaryl phosphate vapor phase lubricants. | | | | |
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PREFACE

This is a technical report describing the acute hazard information on triaryl phosphate vapor phase lubricants. Acute inhalation studies were performed to provide toxicity information on accidental exposure by this route.

The animals used in this study were handled in accordance with the principles stated in the Guide for the Care and Use of Laboratory Animals, prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council, Department of Health and Human Services, National Institute of Health Publication #86-23, 1985, and the Animal Welfare Act of 1966, as amended.

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ABBREVIATIONS

| | |
|------------|--|
| TCP | tricresyl phosphate |
| TOCP | triorthocresyl phosphate (tri-o-tolyl phosphate) |
| OP | organophosphorus |
| NTE | neurotoxic esterase (neuropathy target esterase) |
| OPIDN | organophosphorus-induced delayed neuropathy |
| DURAD 620B | Tert-butylated triphenyl phosphate |
| DURAD 125 | Triaryl phosphate |

INTRODUCTION

Vapor phase lubrication is a relatively new concept that shows excellent potential for high temperature applications. To generate a vapor phase lubricant, conventional liquid lubricants or lubricant anti-wear additives are heated above the boiling point of the fluid and the resulting vapors are transported to a hot bearing surface. To date, the fluid which has received the most extensive evaluation is tricresyl phosphate (TCP). When heated to the vapor state TCP is thermally decomposed. It may undergo repolymerization and possibly other chemical changes, although very little is known about the exact nature of this process.

TCP is known to have a neurotoxic ortho-isomer, triorthocresyl phosphate (TOCP), that is normally minimized in the manufacturing process. However, when TCP is used as a vapor phase lubricant, chemical reactions occur and there is concern that additional amounts of the ortho-isomer or other toxic phosphate compounds may form. Some triaryl phosphates such as TOCP are known esterase inhibitors and have been found to cause delayed neurotoxic effects in humans [1]. A single exposure to a neurotoxic organophosphorous (OP) compound can produce damage to nerves after a delay of 8 to 10 days [1]. This condition is known as organophosphorus-induced delayed neuropathy (OPIDN), and is characterized by axonal degeneration. OPs that cause axonal pathology interact with the enzyme neurotoxic esterase (NTE). Changes in the activity of the enzyme neurotoxic esterase (NTE) is the initial step in the delayed neurotoxicity response. This occurs within hours of exposure to a neurotoxic chemical [2], and changes in NTE activity can be measured in brain, spinal cord, and other nerve tissue.

Rodents have been thought to be relatively insensitive to the clinical effects of TOCP [1]. However, recent studies indicate that if morphological and NTE activity assays rather than locomotor or behavioral endpoints are used, the rat may be a more sensitive indicator of OP induced delayed neuropathy [3].

To evaluate vapor phase triaryl phosphates for neurotoxicity, 4-hr inhalation limit tests were performed on male rats exposed to a nominal vapor concentration of 5 mg/L in air. Two days after vapor exposure, the rats were examined for NTE activity. Untreated and triorthocresyl phosphate (TOCP) treated rats were used as negative and positive controls, respectively. Results showed a statistically significant difference in NTE activity between control rats and those exposed to vapor phase triaryl phosphates or treated with or TOCP, which produced 35-50% and 88% inhibition of NTE activity, respectively.

MATERIALS AND METHODS

Materials

The synthetic and natural lubricants were supplied by Wright Laboratory, Aero Propulsion and Power Directorate, Fuels and Lubrication Division, Lubrication Branch (WL/POSL). Pertinent chemical and physical properties of the test compounds are listed below.

- (1) ASHLAND Natural TCP: TRICRESYL PHOSPHATE
NO MSDS AVAILABLE

- (2) FMC Synthetic TCP: DURAD ® 125 TRIARYL PHOSPHATE

Synonyms: tricresyl phosphate, tritolyl phosphate
CAS #: 68952-35-2
Empirical Formula: $C_{21}H_{21}O_4P$
Density (GMS/ML): 1.165-1.180 @ 20 °C

- (3) FMC Synthetic TCP: DURAD 620B TERT-BUTYLATED TRIPHENYL PHOSPHATE

Synonyms: tert-butyl phenyl phenyl phosphate
CAS #: 68937-40-6
Empirical Formula: $(RO)_3 PO$
Density (GMS/ML): 1.124

- (4) KODAK TOCP

Synonyms: tri-o-tolyl phosphate
CAS #: 78-30-8
Empirical Formula: $(CH_3C_6H_4O)_3 P:O$

- (5) MOBIL OIL MOBIL OIL SHF 21 (PAO)

Synonyms: polyalpha olefins
CAS #:
Empirical Formula:

- (6) Mixture of: 15% Durad 620B (t-butylated triphenyl phosphates) and
85% Mobil SHF 21 polyalpha olefins (2 cSt PAO)

Figure 1. Tricresyl phosphate (TCP)

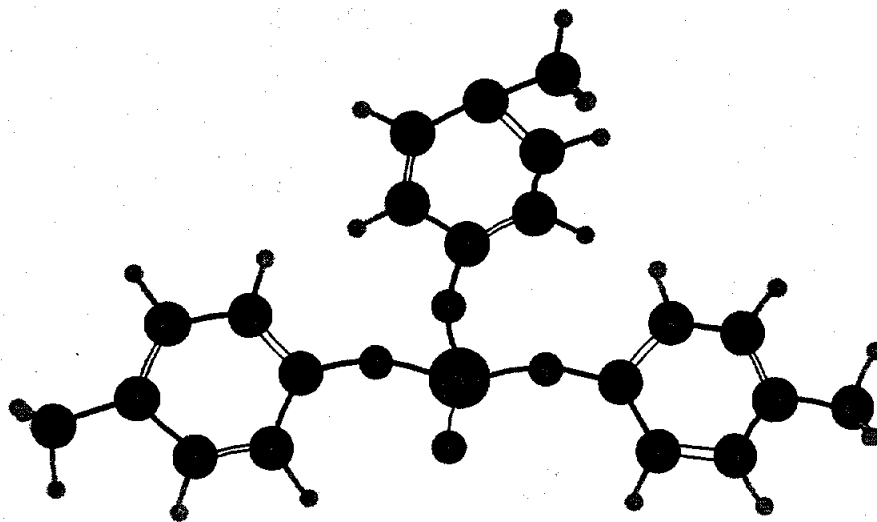


Figure 2. Triorthocresyl phosphate.

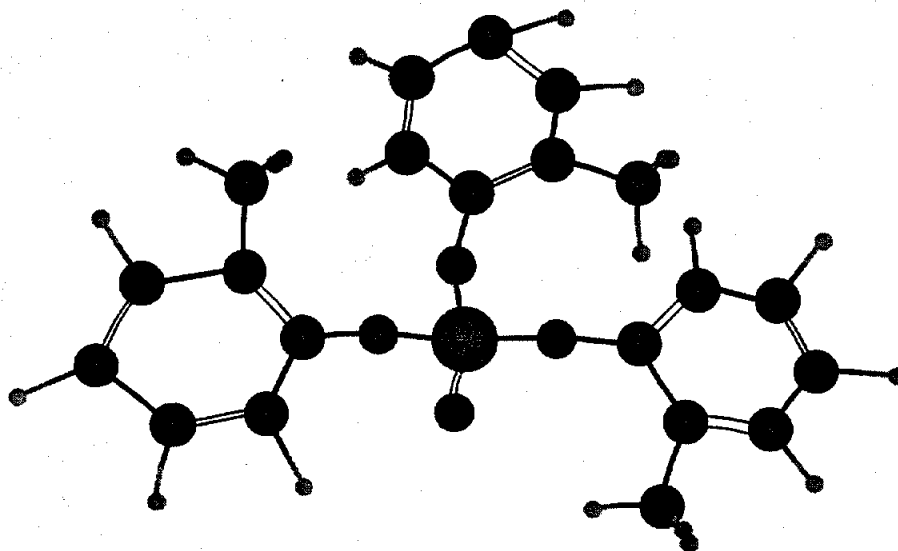
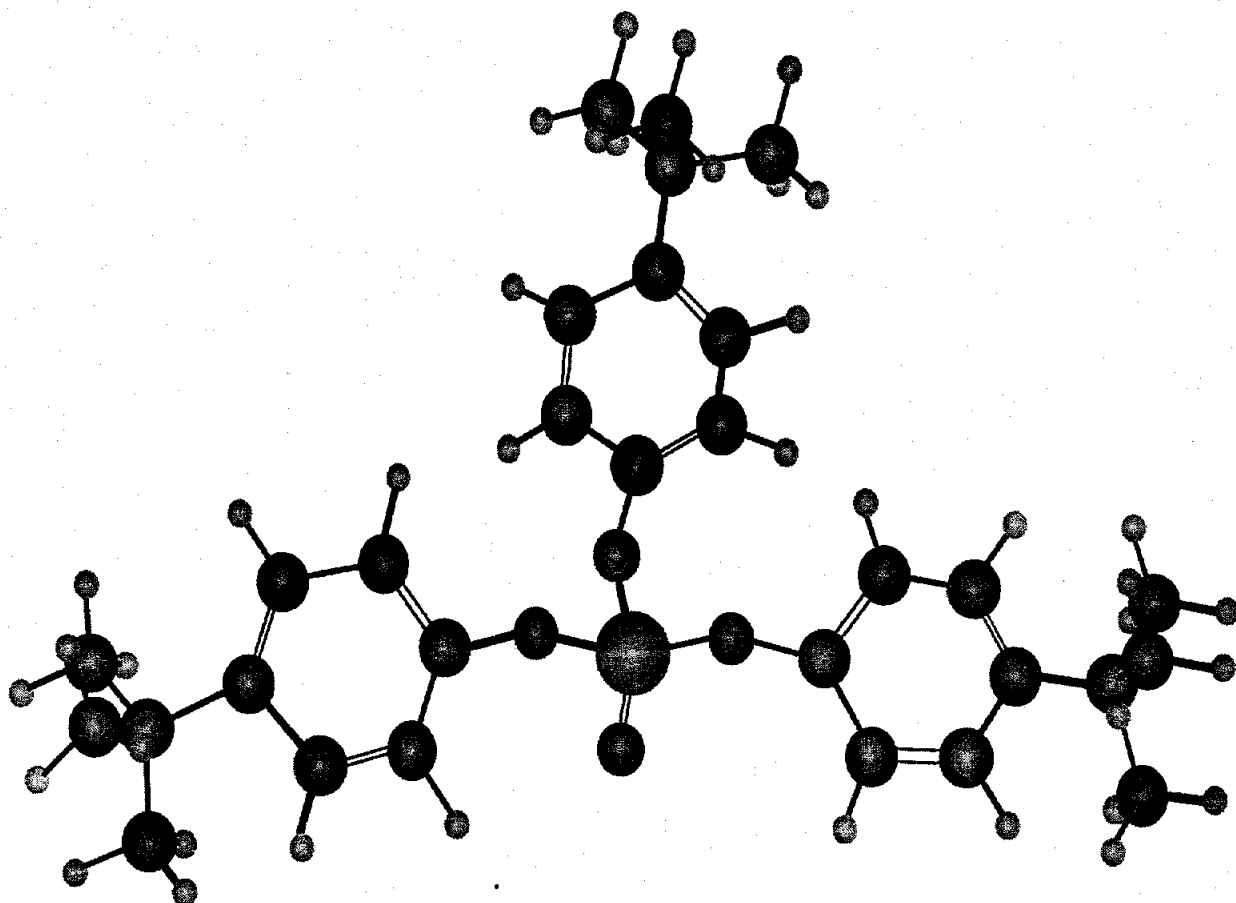


Figure 3. Durad 620B (t-butylated triphenyl phosphate)



Methods

Test animals and treatment

Male Long-Evans rats of at least 6 months of age (375-450 g) were obtained from Charles River Breeding Laboratories, Raleigh, NC and maintained on Purina #5002 lab chow *ad libitum*. Upon arrival to the laboratory the animals were acclimated to the laboratory environment for 2-weeks prior to exposure.

A series of inhalation limit tests was performed on male rats with lubricant vapors generated by test equipment supplied by the sponsor (WL/POSL). Each test consisted of a 4-hour exposure to a nominal vapor concentration of 5mg/L, which is considered the upper "limit" for a realistic exposure. The animals were placed in holders designed for nose-only exposures to the lubricant vapors to insure the animal breathed the generated vapor concentration.

TOCP (tri-o-tolyl phosphate, >99% pure, Eastman Kodak Company Rochester, New York) was used as a positive control for the target organ effect being measured in this study, neurotoxic esterase activity. The TOCP was administered by oral gavage. Because TOCP can produce dangerous cholinergic side effects at the dose level used in this study (i.e., 1.193 g/kg), the animals were pretreated 10 min. prior to oral dosing, and 4 hours post dosing with atropine sulfate (7.5 mg/kg, subcutaneous) to protect against these side effects.

A third group of animals breathed room air only. Since they received no vapor exposure they served as negative controls for the brain NTE assay.

Vapor generation

The vapor generation system consisted of a syringe pump connected to a stainless steel tube that was heated to 650° C by an electric heater furnace to vaporize the lubricant fluid (see Figure 4). Vapor condensation was prevented by keeping the transfer line heated by use of an oven.

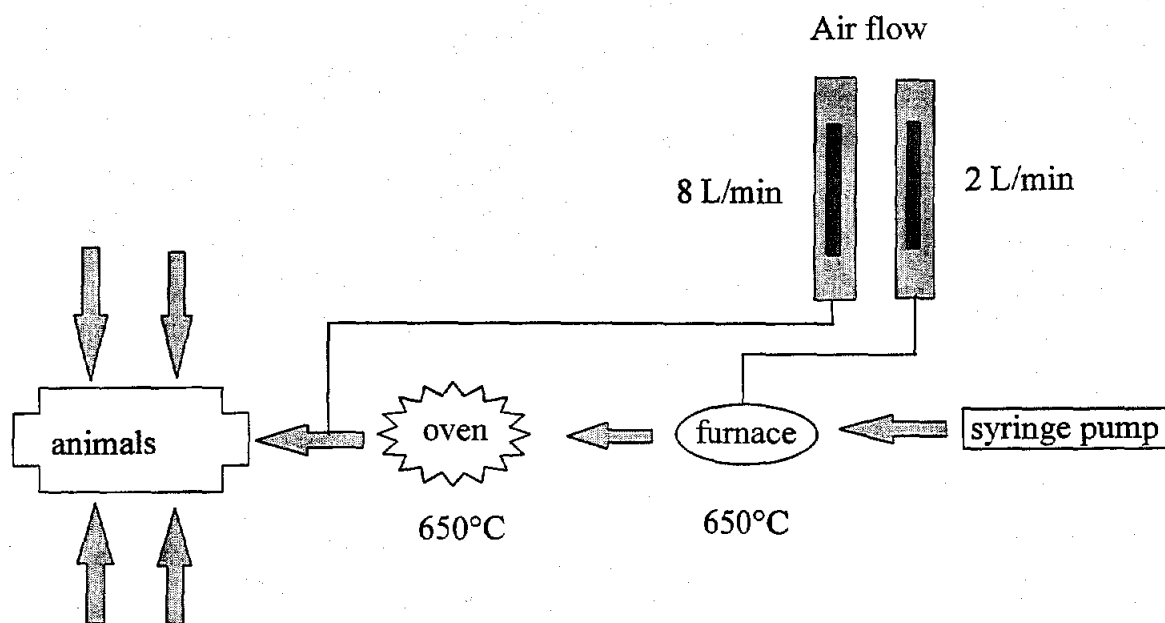
The test compound was fed into the tube with the syringe pump at a rate of 3.3 mL/hour. The density of the fluid was used to convert flow rate from a volumetric flow rate to a mass flow rate, which was used to calculate chamber concentration according to the following formula:

$$\frac{\text{DENSITY (g/ml)} \times \text{INJECTION RATE (ml/hr)}}{60 \text{ min./hr} \times \text{total air flow (i.e., 10.0 L/min.)}} = \frac{\text{g/min.}}{10\text{L/min.}} = \text{mg/L}$$

For example, the 0.0618 g/min. mass flow rate for Durad 620B was initially mixed with a carrier air flow of 2.0 L/min. This concentrated vapor flow was further diluted with an additional 8.0 L/min. air flow to provide a nominal chamber concentration of 6.18 mg/L. This is the concentration to which the animals were exposed.

An additional benefit provided by the second air stream was temperature control. During the experiment the vapor generator and transfer line temperature was maintained at 650°C; however, at no time did the animal exposure chamber exceed 30°C. This was largely a result of the addition of dilution air to the concentrated vapor immediately before entry to the animal exposure chamber.

Figure 4. Vapor Generation and Animal Exposure System.



NTE analysis

Two days after exposure, whole brains and the spinal cord including the C-4 region were surgically removed from vapor exposed, TOCP treated (positive control), and untreated control rats. The brains were weighed and 1:150 homogenates of brain tissue in 50 μ M tris: 0.02 mM EDTA, pH 8.0 buffer were prepared.

NTE activity was measured using a modification of the colorimetric assay of Johnson [4]. 50 μ l aliquots of brain homogenate were incubated with and without 25 μ l of 12.7 μ M mipafox and 100 μ M paraoxon in microliter wells. After 20 min. incubation, 50 μ l of 11.19 μ M phenylvalerate was added as the substrate and an additional incubation period of 15 min. was provided for the reaction. The reaction was terminated by the addition of 50 μ l of cocktail containing 0.008% 4-amino antipyrine, 0.08% potassium ferricyanide and 2% sodium dodecylsulfate. Condensation of phenol with 4-amino antipyrine in the presence of the oxidant potassium ferricyanide caused an orange color to develop. The intensity of the orange color was measured at 510 nm on a microplate spectrophotometer. Substrate and tissue blanks were included for each assay. NTE activity was calculated on the basis of the difference in the absorbance between the incubated samples with and without mipafox in the presence of paraoxon. NTE activity was expressed as nanomoles of phenol formed per minute per milligram brain protein.

NTE was calculated on the basis of difference in the absorbance between the incubates with and without mipafox in the presence of paraoxon. Paraoxon is used to inhibit acetylcholinesterase activity in brain, in order to measure NTE enzyme activity. The enzyme activity is calculated based on the phenol standard curve and expressed as nmol of phenol formed/15 min./mg protein.

RESULTS AND DISCUSSION

Results

Analysis of NTE activity showed a statistically significant difference between negative control rats and those treated with vapor phase lubricants or orally administered TOCP (positive control). The percent NTE inhibition for each compound tested is presented in the appendix along with body weight data, and is summarized in Table 1, below. It can be seen from Table 1 that exposure to all triaryl phosphate vapor phase lubricants resulted in NTE inhibition. Durad 620B produced the least effect, with a 34.7% inhibition of NTE activity. Durad 125 produced the greatest NTE inhibition (49.3%).

Table 1. Neurotoxic Esterase Activity (in nM of phenol/mg of protein/15 minutes) and Percent NTE Inhibition from control levels.

| Compound | Vapor Conc. (mg/L) | NTE activity mean (std.dev.) | Percent inhibition from control |
|------------------------------|--------------------|------------------------------|---------------------------------|
| Ashland (natural TCP) | 6.4 | 12.130 (1.009) | 43.5% |
| Durad 125 (synthetic TCP) | 6.4-6.5 | 12.297 (1.244) | 49.3% |
| Durad 620B | 6.2 | 16.058 (0.926) | 34.7% |
| TOCP (positive controls) | N/A | < 3.3 | >86% |
| Untreated (negative control) | N/A | > 23 | N/A |

All animals exposed to the mixture of 15% Durad 620B and 85% Mobil SHF 21 polyalpha olefins died in the first two and a half hours of the 4-hour exposure. Histological findings showed moderate diffuse perivascular edema of the lung, moderate multifocal acute-necrotizing rhinitis of the nasal turbinates with the liver and heart essentially normal. Follow on testing will evaluate gas composition to explain the acute lethality of this compound.

Discussion

The level of NTE inhibition generally accepted to be predictive of OPIDN in hens is 70%[1]; however, hens were notably impaired three weeks after administration of OPIDN-inducing compounds when spinal cord NTE was inhibited more than 40% [5]. Additionally, NTE inhibition as low as 31% has been shown to be associated with development of severe spinal cord damage in rats [3] (see Table 2, below). Thus, the threshold for OPIDN may well be below a 70% inhibition of NTE.

Table 2. Relationship between mean NTE inhibition in the brain and severe spinal cord damage in Long-Evans rats exposed to TOCP (modified from Padilla and Veronesi, 1985).

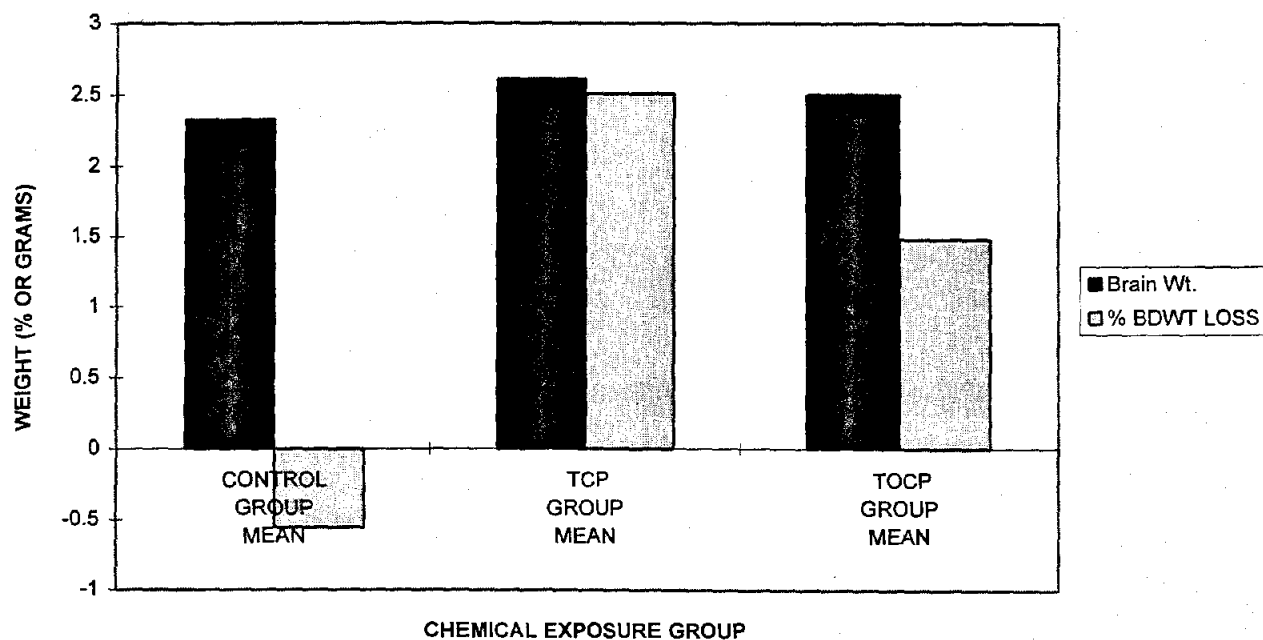
| Inhibition of Brain NTE activity (% of control values) | Severe Spinal Cord Damage (% of group) |
|---|---|
| 77-99% | 100% |
| 61-71% | 90% |
| 51-63% | 15% |
| 31-41% | 7.5% |
| 15-21% | 0 |

Recently completed studies with a mixed t-butylphenyl phosphate compound administered by oral gavage did not inhibit NTE, alter motor activity, or produce neuropathology characteristic of delayed neurotoxicity at a single doses of 2 g/kg in hens [6] or in rats (personal communication with Mr. D. Placek, FMC Corporation). When contrasted with our data, it can be shown that exposure to these lubricants in the vapor phase inhibits NTE. Thus, the process of vaporization is causing a change in the compound, resulting in the potential to produce neurotoxicity. Therefore, caution must be used when working with triaryl phosphate vapor phase lubricants.

ASHLAND NATURAL

| Animal I.D. | ANIMAL WEIGHT | | | Brain Wt. | % BDWT LOSS AT 48-HR |
|---------------------------|----------------|----------------|----------------|---------------|-------------------------|
| | 0-hour | 24-hour | 48-hour | | |
| | 1/4/95 | 1/5/95 | 1/6/95 | 1/6/95 | |
| Control 7 | 498.5 | 491.8 | 496.2 | 2.12 | |
| Control 8 | 539.2 | 531.7 | 547.3 | 2.54 | |
| CONTROL GROUP MEAN | 518.9 g | 511.8 g | 521.8 g | 2.33 g | -0.56 |
| TCP 1 | 466.1 | 453.8 | 449.9 | 2.70 | |
| TCP 2 | 484.9 | 467.3 | 471.5 | 2.52 | |
| TCP 4 | 519.5 | 526.4 | 512.2 | 2.42 | |
| TCP GROUP MEAN | 490.2 g | 482.5 g | 477.9 g | 2.61 g | 2.51 |
| TOCP 5 | 551.1 | 552.1 | 557.3 | 2.51 | |
| TOCP 6 | 594.7 | 572.8 | 571.4 | 2.48 | |
| TOCP GROUP MEAN | 572.9 g | 562.5 g | 564.4 g | 2.50 g | 1.48 |
| | | | | | |
| | | | | | |
| | | | | | |
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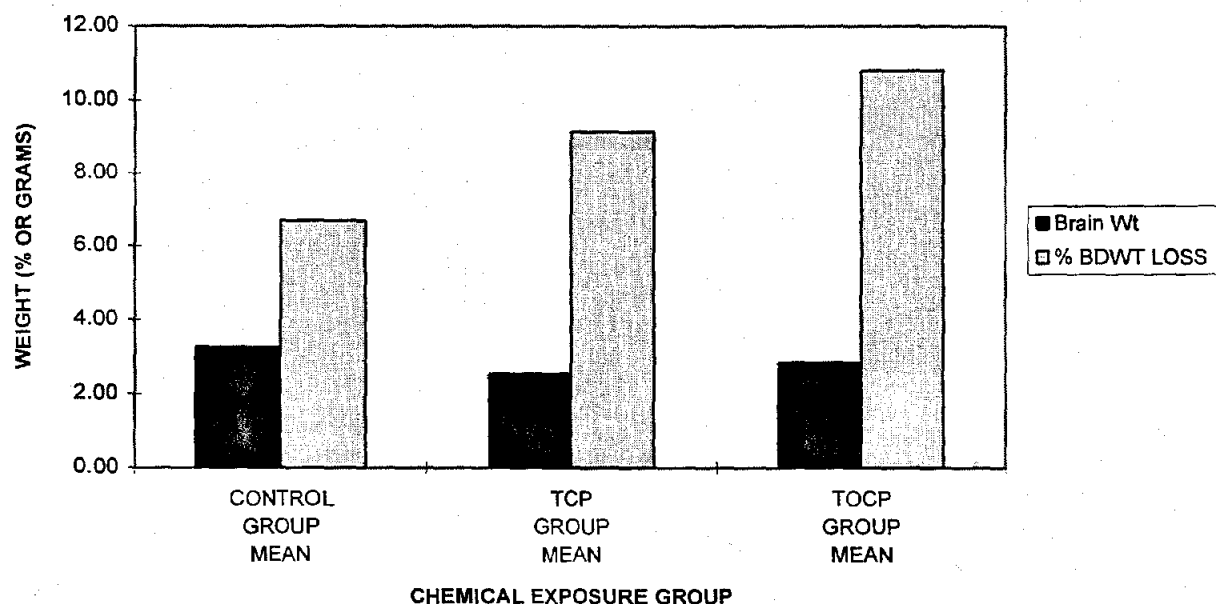
ASHLAND



DURAD 125

| Animal I.D. | ANIMAL WEIGHT | | | Brain Wt | % BDWT LOS AT 48-HR |
|---------------------------|----------------|----------------|----------------|---------------|------------------------|
| | 0-hour | 24-hour | 48-hour | | |
| | 1/9/95 | 1/10/95 | 1/11/95 | 1/11/95 | |
| Control 16 | 546.8 | 525.6 | 504.1 | 3.03 | |
| Control 17 | 541.8 | 514.2 | 524.1 | 3.39 | |
| Control 18 | 545.1 | 514.2 | 496.2 | 3.33 | |
| CONTROL GROUP MEAN | 544.6 g | 518.0 g | 508.1 g | 3.25 g | 6.70 |
| TCP 10 | 525.4 | 488.5 | 471.1 | 2.52 | |
| TCP 11 | 514.1 | 472.9 | 451.8 | 2.67 | |
| TCP 12 | 531.5 | 495.7 | 475.4 | 2.42 | |
| TCP GROUP MEAN | 513.0 g | 476.4 g | 466.1 g | 2.54 g | 9.14 |
| TOCP 13 | 604.2 | 557.7 | 533.3 | 2.94 | |
| TOCP 14 | 536.2 | 497.6 | 479.5 | 2.70 | |
| TOCP 15 | 535.8 | 503.8 | 482.0 | 2.88 | |
| TOCP GROUP MEAN | 558.7 g | 519.7 g | 498.3 g | 2.84 g | 10.82 |

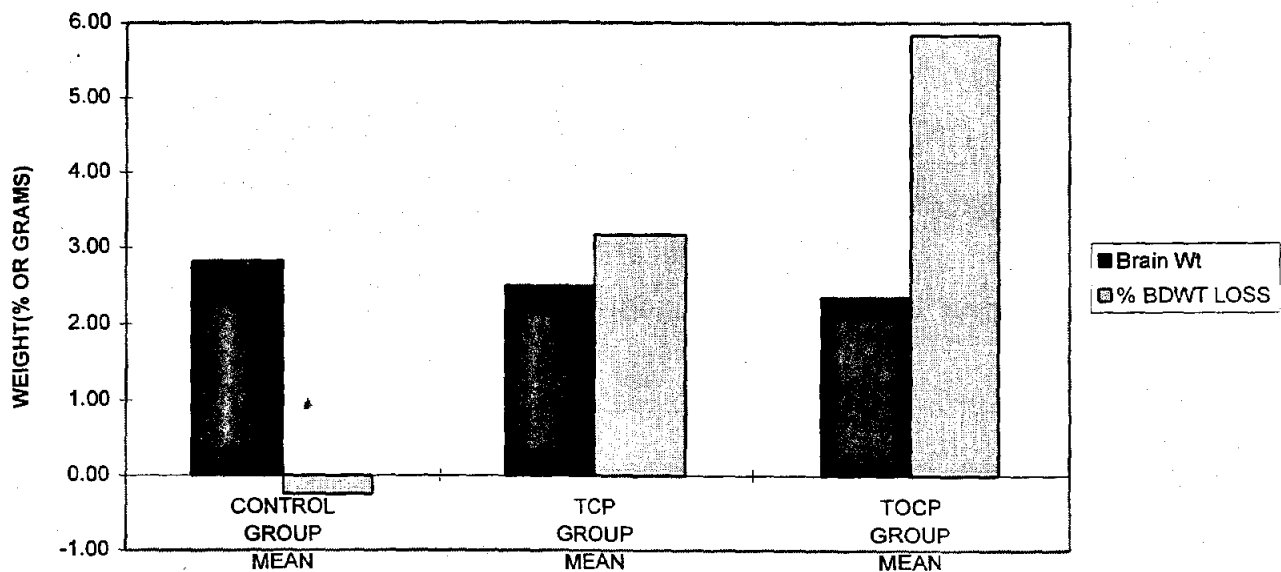
DURAD 125



DURAD 620B

| Animal I.D. | ANIMAL WEIGHT | | | | Brain Wt | % BDWT LOSS |
|---------------------------|----------------|----------------|----------------|---------------|----------|--------------|
| | 0-hour | 24-hour | 48-hour | | | |
| | 3/14/95 | 3/15/95 | 3/16/95 | 3/16/95 | | |
| | no weight | 520.2 | 524.4 | 2.87 | | |
| Control 15 | | | | | | |
| Control 16 | 491.3 | 501.2 | 495.8 | 2.77 | | |
| Control 17 | 554.1 | 548.5 | 547.9 | 2.85 | | |
| Control 18 | 556.5 | 558.3 | 552.0 | 2.79 | | |
| Control 19 | 469.0 | 481.9 | 475.1 | 2.86 | | |
| CONTROL GROUP MEAN | 517.7 g | 522.0 g | 519.0 g | 2.83 g | | -0.25 |
| TCP 5 | 505.5 | 499.6 | 506.1 | 2.75 | | |
| TCP 6 | 525.4 | 508.3 | 522.1 | 2.49 | | |
| TCP 7 | 562.0 | 500.7 | 504.4 | 2.35 | | |
| TCP 8 | 518.5 | 517.5 | 512.0 | 2.42 | | |
| TCP GROUP MEAN | 527.9 g | 506.5 g | 511.2 g | 2.50 g | | 3.16 |
| TOCP 21 | 513.0 | 479.7 | 467.5 | 2.75 g | | |
| TOCP 22 | 559.9 | 558.3 | 531.7 | 2.12 | | |
| TOCP 23 | 564.6 | 554.6 | 534.3 | 2.60 | | |
| TOCP 24 | 548.7 | 528.3 | 525.1 | 1.90 | | |
| TOCP GROUP MEAN | 546.6 g | 530.2 g | 514.7 g | 2.34 g | | 5.84 |
| | | | | | | |
| | | | | | | |

DURAD 620B



| DATE: January 6, 1995 | | | | Neurotoxic Esterase Activity (NTE) in Nanomoles of Phenol/mgm of protein/15 minutes in Control, Ashland Natural (TCP) and Triorthocresyl phosphate administered in rats | | | | | | | | | |
|-------------------------|--|--------------------------------|--------------------------|--|-------------------|---|---|-------|--|--|--|--|--|
| Animal I.D. | Difference in Absorbance between the incubates with Paraxon and incubates with Paraxon & Mipafox | Phenol conc. in ugm/50ul | Phenol conc. in uM | Phenol absolute amount in Nano Moles | Protein in mgm | NTE activity in Nano moles of Phenol/mgm of protein/ 15 minutes | Mean NTE Activity in Nano moles/ mgm of protein/ 15 minutes | STD | | | | | |
| Control 7 | 0.21 | 0.135 | 28.225 | 1.411 | 0.07 | 20.16 | | | | | | | |
| Control 8 | 0.256 | 0.284 | 59.2 | 2.96 | 0.13 | 22.77 | | | | | | | |
| Control Mean | | | | | | | 21.465 | 1.846 | | | | | |
| TCP 1 | 0.207 | 0.125 | 26.075 | 1.303 | 0.1 | 13.03 | | | | | | | |
| TCP 2 | 0.191 | 0.074 | 15.457 | 0.772 | 0.07 | 11.04 | | | | | | | |
| TCP 4 | 0.19 | 0.07 | 14.785 | 0.739 | 0.06 | 12.32 | | | | | | | |
| TCP Mean | | | | | | | 12.13 | 1.009 | | | | | |
| %inhibition of control | | | | | | | 43.50% | | | | | | |
| TOCP 5 | 0.173 | 0.016 | 3.427 | 0.171 | 0.07 | 2.448 | | | | | | | |
| TOCP 6 | 0.173 | 0.016 | 3.36 | 0.168 | 0.07 | 2.44 | | | | | | | |
| TOCP Mean | | | | | | | 2.444 | 0.006 | | | | | |
| % inhibition of control | | | | | | | 88.61% | | | | | | |

| DATE: Jan 11,1995 | | | | Neurotoxic Esterase Activity (NTE) in Nanomoles of Phenol/mgm of protein/15 minutes in Control, Durad 125 (TCP) and Triorthocresyl phosphate administered in rats | | | | | | | | | | |
|-------------------|--|--------------------------------|--------------------------|--|-------------------|---|-------------------------|---|-------|--|--|--|--|--|
| Animal I.D. | Difference in Absorbance between the incubates with Paraxon and incubates with Paraxon & Mipafox | Phenol conc. in ugm/50ul | Phenol conc. in uM | Phenol absolute amount in Nano Moles | Protein In mgm | NTE activity in Nano moles of Phenol/mgm of protein/ 15 minutes | | Mean NTE Activity in Nano moles/ mgm of protein/ 15 minutes | STD | | | | | |
| Control 16 | 0.263 | 0.307 | 64.04 | 3.202 | 0.13 | 24.632 | | | | | | | | |
| Control 17 | 0.249 | 0.26 | 54.23 | 2.711 | 0.11 | 24.65 | | | | | | | | |
| Control 18 | 0.245 | 0.248 | 51.74 | 2.587 | 0.11 | 23.52 | | | | | | | | |
| | | | | | | | Control Mean | 24.26 | 0.647 | | | | | |
| TCP 9 | 0.19 | 0.071 | 14.784 | 0.739 | 0.07 | 10.56 | | | | | | | | |
| TCP 10 | 0.206 | 0.121 | 25.403 | 1.27 | 0.1 | 12.7 | | | | | | | | |
| TCP 11 | 0.21 | 0.135 | 28.225 | 1.411 | 0.105 | 13.5 | | | | | | | | |
| TCP 12 | 0.205 | 0.119 | 24.865 | 1.243 | 0.1 | 12.43 | | | | | | | | |
| | | | | | | | TCP Mean | 12.297 | 1.244 | | | | | |
| | | | | | | | %Inhibition of control | 49.31% | | | | | | |
| TOCP 13 | 0.175 | 0.022 | 4.704 | 0.235 | 0.09 | 2.613 | | | | | | | | |
| TOCP 14 | 0.176 | 0.025 | 5.376 | 0.268 | 0.091 | 2.954 | | | | | | | | |
| TOCP 15 | 0.176 | 0.024 | 5.04 | 0.252 | 0.09 | 2.8 | | | | | | | | |
| | | | | | | | TOCP Mean | 2.789 | 0.17 | | | | | |
| | | | | | | | % inhibition of control | 88.50% | | | | | | |

| Neurotoxic Esterase Activity (NTE) in Nanomoles of Phenol/mgm of protein/15 min In Control, Durad 620B (TCP) and Trlorthocresyl phosphate administered in rats | | | | | | | | | | |
|---|---|--------------------------------|--------------------------|---|-------------------|---|---|-------|--|--|
| DATE: March 23, 1995 | | | | | | | | | | |
| Animal I.D. | Difference in Absorbance between the incubates with Paraoxon and Incubates with Paraxon & Mipafox | Phenol conc. in ugm/50ul | Phenol conc. in uM | Phenol absolute amount in Nano Moles | Protein in mgm | NTE activity in Nano moles of Phenol/mgm of protein/ 15 minutes | Mean NTE Activity in Nano moles/ mgm of protein/ 15 minutes | STD | | |
| Control 15 | 0.187 | 0.261 | 54.315 | 2.716 | 0.115 | 23.615 | | | | |
| Control 16 | 0.18 | 0.236 | 49.107 | 2.455 | 0.097 | 25.313 | | | | |
| Control 17 | 0.185 | 0.254 | 52.827 | 2.641 | 0.106 | 24.919 | | | | |
| Control 18 | 0.182 | 0.243 | 50.595 | 2.53 | 0.105 | 24.093 | | | | |
| Control 19 | 0.188 | 0.264 | 55.06 | 2.753 | 0.11 | 25.027 | | | | |
| | | | | | | Control Mean/STD | 24.59 | 0.71 | | |
| Air Control 1 | 0.178 | 0.229 | 47.619 | 2.381 | 0.095 | 25.063 | | | | |
| Air Control 2 | 0.183 | 0.246 | 51.339 | 2.567 | 0.105 | 24.447 | | | | |
| Air Control 3 | 0.175 | 0.218 | 45.387 | 2.269 | 0.092 | 24.667 | | | | |
| Air Control 4 | 0.178 | 0.229 | 47.619 | 2.381 | 0.096 | 24.802 | | | | |
| | | | | | | Air Mean/STD | 24.74 | 0.257 | | |
| TCP 5 | 0.157 | 0.154 | 31.994 | 1.6 | 0.1 | 15.997 | | | | |
| TCP 6 | 0.148 | 0.121 | 25.298 | 1.265 | 0.086 | 14.777 | | | | |
| TCP 7 | 0.15 | 0.129 | 26.786 | 1.339 | 0.08 | 16.741 | | | | |
| TCP 8 | 0.155 | 0.146 | 30.506 | 1.525 | 0.091 | 16.725 | | | | |
| | | | | | | Durad 620B Mean/ST | 16.058 | 0.928 | | |
| | | | | | | % Inhibition of Control | 34.70% | | | |
| TOCP 21 | 0.125 | 0.039 | 8.185 | 0.409 | 0.12 | 3.41 | | | | |
| TOCP 22 | 0.12 | 0.021 | 4.464 | 0.223 | 0.07 | 3.189 | | | | |
| TOCP 23 | 0.122 | 0.029 | 5.952 | 0.298 | 0.09 | 3.307 | | | | |
| TOCP 24 | 0.119 | 0.018 | 3.72 | 0.186 | 0.06 | 3.1 | | | | |
| | | | | | | TOCP Mean | 3.251 | 0.136 | | |
| | | | | | | % Inhibition of control | 86.86% | | | |

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