

SUBMISSION TO THE SENATE REFERENCES COMMITTEE

RURAL AND REGIONAL AFFAIRS AND TRANSPORT

On the Inquiry into

Air Safety – BAE 146  
Cabin Air Quality

Carl R. Mackerer, Ph.D.  
Principal Toxicologist  
Mobil Business Resources Inc.  
P.O. Box 310  
Paulsboro, NJ USA 08066  
Ph: 856-224-4630  
carl\_r\_mackerer@email.mobil.com

Edwin N. Ladov  
Manager, Product Stewardship & Toxicology  
Mobil Business Resources Inc.  
P. O. Box 310  
Paulsboro, NJ USA 08066  
Ph: 856-224-4610  
ed\_n\_ladov@email.mobil.com

## Introduction

We do not believe that Mobil jet turbine oils pose any significant toxicological risk to individuals accidentally exposed to aerosols or vapors in aircraft cabins. Such exposures are not what we would refer to as "normal use" but the cabin levels that can be reached during such exposures are comprehended by our internal and published risk assessments and are considered safe. These assessments are based on Mobil toxicology testing as well as the extensive toxicology database found in the published literature.

Many allegations have been made and questions raised during these hearings concerning the toxicology and product stewardship aspects of our business relating to the safe use of our jet turbine engine oils, particularly Mobil Jet Oil II. Many of the concerns raised by scientists and other interested parties were about the composition of Mobil Jet Oil II, the relevance of the toxicology work to human health effects, and the statements on our Material Safety Bulletins and package labels. It was mentioned several times in testimony or written submissions that information related to these topics was difficult to obtain.

We would like to point out that, the desired information would not have been difficult to obtain from Mobil, but we have no record of anyone contacting Mobil, either in Australia or the U.S., to request such information with the exception of some of our airline customers, Mr. Stephen Holland of Worksafe Australia, and Dr. Steen Kristensen of NICNAS. When such requests were made, Mobil provided data, reports, relevant published papers and advice. In fact, had those who needed specialized information contacted us, we could have provided advice, particularly with regard to appropriate analytical methods, that would have provided a better scientific foundation around which to evaluate the health concerns of airline attendants and pilots.

### Mobil Jet Oil II Product Stewardship Background and History

At this time, we would like to discuss the process resulting in Mobil's decision to provide neurotoxicity warnings on product labels and safety data sheets for Mobil Jet Oils containing tricresyl phosphate (TCP), an antiwear additive used in all major synthetic jet engine lubricants. Most of this information is included in correspondence which already is in the public record.

In the late 1980's, Mobil voluntarily conducted a series of toxicological laboratory evaluations of its major product lines as part of its ongoing product safety stewardship program. These animal studies showed the jet oils tested, containing a maximum of 3% TCP, might be potentially harmful. Subsequently we updated the product Material Safety Data Sheets to include this information and recommended that exposure via skin, inhalation and ingestion be minimized. The emphasis was on ingestion as there had been reports that individuals in certain developing countries may have suffered from delayed neurotoxic effects after ingestion of foodstuffs or beverages adulterated with aryl phosphate esters. There also had been reports of neurotoxic effects suffered by a few workers involved in the manufacture of *pure* (100%) TCP.

A formal risk assessment was conducted by Mobil in 1990 which provided confirmation that ingestion was, in fact, the principal route of exposure that could potentially produce neurotoxic effects. Because of the ingestions that had earlier been reported, it was decided that communication (through labeling) of this potential ingestion hazard to individuals working directly with the jet oils was appropriate. The risk assessment clearly showed that a potentially harmful dose is not possible via inhalation at levels at or even higher than Threshold Limit Value of 5.0 mg/m<sup>3</sup> for the oil mist. These levels would produce a clearly visible oil mist. Also, an accidental contamination of the entire body surface with an oil containing 3% TCP for 6 hours would not result in the absorption of more than an estimated non-toxic single dose. Therefore, warnings on exposure via inhalation and skin were not placed on product containers.

Additional joint toxicology studies by Mobil and a major manufacturer of TCP confirmed that an oil with 3% TCP could produce neurotoxic effects in animals administered very high oral doses. This led Mobil to adopt a very conservative labeling approach for its jet oils by including language recommending minimizing exposure by all routes and emphasizing the importance of good personal hygiene practices. The decision was made in the early part of 1997 and labeling was phased in during the year.

Finally, a 1999 risk assessment published in the Journal of Toxicology and Environmental Health, concluded that it is unlikely that food such as cooking oil would be contaminated with enough Jet Oil containing 3% TCP to cause toxicity and, at the dosage required for neurotoxicity, it would be virtually impossible for a person to receive enough of the oil in the normal workplace (or in an aircraft) to cause such toxicity. Further there is no record of a jet oil formulated with modern "conventional" TCP causing human toxicity.

Mobil's decision to label these products was based solely on its own policies and product safety stewardship practices. In fact, based on the toxicological data developed over the years, Mobil Jet Oil II is a non-hazardous product based on the NOHSC document "Approved Criteria for Classifying Hazardous Substances." Based on this document, mixtures of chemicals can be classified by one of two methods: a) the mixture can be tested and classified as a whole, or b) it can be classified by consideration of the content and health effects of each of its components. Using either method, it can be concluded that Mobil Jet Oil II is non-hazardous under NOHSC.

The Worksafe Australia classification adopted by Mobil was acknowledged as correct in the correspondence between Mobil and Worksafe Australia that we are including in our submission today.

With regard to phenyl-naphthylamine, Mobil Jet Oil II contains approximately 1% of the alpha isomer. Testing has confirmed that this concentration did not cause sensitization in animals or humans. The concentration of the carcinogenic beta isomer and beta naphthylamine which might be present as impurities is negligible to non-existent in Mobil Jet Oil II.

Finally, we acknowledge that there were some inconsistencies several years ago in the classification of Mobil Jet Oil II under the European Community regulations. They were incorrectly classified as harmful in 1996 and 1997. This has been subsequently corrected.

In summary, recent changes that have been made to the label and Material Safety Data Sheets do not reflect any underlying change in product composition or any new information about health hazards. Mobil Jet Oil II has been, and continues to be, safe for its intended purposes. The changes to the label were based solely on Mobil's own product stewardship practices, and a very conservative approach to labeling. It must be emphasized that the revised labeling and MSDS statements do not reflect new information on Mobil Jet Oil II, suggesting hazard, where none existed before.

#### Possible Areas for Further Research

There has been much speculation that Mobil Jet Oil II may be the cause of the alleged adverse health effects. This is unsupported by the scientific evidence as discussed previously. The clinical symptoms reported are not the same as those that have been historically seen for TCP. The reported symptoms appear to closely match those for exposure to carbon monoxide (CO). These effects can be exacerbated by low oxygen levels and high carbon dioxide levels in the aircraft cabin. Possible sources for carbon monoxide include the ambient cabin air and, in the case of a malfunction of the aircraft mechanical systems, thermally degraded hydraulic oil and turbine oil that might enter the aircraft cabin. These oils may break down at very high temperatures and liberate carbon monoxide on contact with hot metal surfaces. Under these extreme conditions, carbon monoxide would be produced from virtually any oil and independent of any additives, including TCP, that might be present.

In summary, Mobil has conducted extensive research on its products and always has been willing to share this information with the public through a variety of communication vehicles including scientific papers, symposia, technical committees, product literature, etc. We will continue these efforts and look forward to working with interested stakeholders to help resolve the concerns and issues around the use of our products.

## TCP SUMMARY

- **TCP is a necessary component in jet engine oils.** It is used as an antiwear additive to increase load-carrying capacity and tolerance to increasing speed of rotating or sliding motion. The antiwear properties of TCP for this critical application are unique and no replacement has been identified that will meet the stringent performance requirements for jet turbine oil. For many years, it was also thought that reducing the number and variety of compounds in the TCP would also reduce its performance<sup>1,3</sup>.
- **The TCP used in jet engine oil is a very complex mixture.** The conventional TCP used in Mobil Jet II is a complex mixture prepared primarily from m and p cresol. However, other substituted phenols as well as xylenols are present in the synthesis mixture. We have identified 10 phenols and xylenols, as well as low levels of ortho cresol and phenol, in hydrolyzed conventional TCP<sup>4</sup>. Ortho cresol was present at about 0.16%, m + p cresol combined at 80% and the other phenols at 17%. Thus the number of triaryl phosphate combinations in TCP is very high and is not limited to the ten that can be formed from ortho, meta and para cresol.

It is not practical to measure all of the triaryl phosphate compounds present because standards do not exist for most of them. However, their concentrations can be computed by statistical procedures from the compounds present in hydrolysates. The various phenols and xylenols have virtually the same reactivity. This procedure has been used for many years<sup>5</sup>. In the TCP additive, TOCP levels are calculated to be < 5 ppb, mono ortho cresyl phosphate ~ 3070 ppm, and diortho cresyl phosphate ~ 6 ppm. These values are reduced by 33 fold after dilution in the oil.

In JEO 291, a recently developed low toxicity TCP<sup>4</sup> is used. The hydrolysates of this TCP are ~ 99% m and p cresol and ortho cresol is only ~ 0.06%<sup>4</sup>; virtually all of the ortho substituted phenols and xylenols are eliminated. TOCP is calculated to be < 1 ppb, mono ortho cresol TCP ~ 1760 ppm, and diortho cresyl TCP ~ 1.1 ppm. Even though molecular diversity is reduced in this TCP, its antiwear performance is excellent thus dispelling some concerns raised years ago.<sup>3</sup>

- **TCP is an organo phosphate that is very different from those used as insecticides.** Both TCP and the organo phosphate insecticides can inhibit cholinesterase enzymes in blood plasma, red blood cells and the nervous system. The blood cell enzymes have no known function in the body, and inhibition of these enzymes is indicative of exposure to an inhibitor but not of toxicity. In general, the plasma enzyme is inhibited the most and nervous system enzyme (including brain) the least. The insecticides owe their killing power to the inhibition of cholinesterase (specifically acetyl cholinesterase) in the nervous system of target insects as well as in non-target animals, including people, and these toxicants are far more potent in this behavior than is TCP. For example, parathion is more than 2500 times as acutely toxic (eg. rat oral LD50) than is TCP<sup>6</sup>. The insecticides have recently been characterized as being capable, in people, of causing lasting neurological/psychological effects on intellect, mood,

perception, behavior, etc. after overtly toxic exposures as well as after confirmed "sub clinical" exposures<sup>7</sup>. These effects evidently did not occur in any of the many poisonings studied by the authors of the published papers which describe the large number of human cases of organophosphate induced delayed neurotoxicity (OPIDN) caused by single or repeated exposures to TCP<sup>8</sup> through ingestion. Many of the TCP poisoned patients were extensively evaluated, interviewed and followed over long periods by physicians and other investigators, so there was ample opportunity for other effects to be observed or reported by the patients if they had occurred.

In 1957 Tabershaw<sup>9,10</sup> published results of a study conducted at a chemical plant that synthesized TCP and other triaryl phosphates, under unfavorable industrial hygiene conditions. Workers were exposed over a long period of time (average of 8.9 yr.) to a constant air concentration of 0.27 mg/m<sup>3</sup>, and intermittent levels of up to 3.4 mg/m<sup>3</sup> during maintenance operations. Opportunity for skin exposure was universal in the plant. There was significant depression of plasma cholinesterase activity but not of erythrocyte cholinesterase. There were mild gastrointestinal and/or neuromuscular symptoms which did not correlate with observed cholinesterase activity. The clinical effects were considered mild and did not cause any lost work time or require medical follow-up, and could not with certainty be ascribed to TCP. It was concluded that the clinical data were conspicuous by the absence of any serious disease attributable to TCP.

**Older, more toxic forms of TCP have caused paralysis.** TCP is an organophosphate compound that produces a very specific neurological effect<sup>11</sup>, now called organophosphate induced delayed neuropathy (OPIDN), when sufficiently high dose levels are reached; experimentally, this level is found to occur only after an enzyme present in the brain called neuropathy target esterase (NTE) is inhibited by about 70% in a sensitive species such as the hen<sup>4</sup>. The hen and human are about equally sensitive when dosage is computed on a per gram body weight basis<sup>4</sup>. The neuropathy occurs after a single large exposure, or multiple smaller exposures, large enough to inhibit NTE to the critical level.

In humans, after high level, acute oral poisoning, there are transient gastrointestinal symptoms (abdominal pain, diarrhea, vomiting and nausea) followed by a latency period lasting 3 - 30 days and then progressive development of muscle soreness, numbness of calf muscles, and paralysis<sup>3</sup>. In severe cases, hands and forearms may also become paralyzed. At least partial or complete recovery occurs in most cases but residual effects can last a lifetime.

It is likely that more than 60,000 people have experienced OPIDN from exposure to TCP found in a variety of lubricants and foodstuffs over the last century<sup>12</sup>. However, all but three cases were from oral ingestion of contaminated food or drink. The three exceptional cases were in working men during World War II in the UK who received massive dermal and/or inhalation exposure.

Toxicity of commercial TCPs (which also contain other aryl phosphate components) has been traced largely to the presence of 2-methyl phenol (o-cresol) and 2-ethyl phenol in the reaction mixture used for the synthesis, but 2-propyl phenol and ortho substituted xylenols



also contribute low levels of activity.<sup>4</sup> Since the 1950's, manufacturers have striven to reduce the toxicity of TCP by lowering the concentration of ortho cresol used in the synthesis. The TCP used today is, therefore, less toxic than those that produced human poisonings years ago. The neurotoxic potency of the TCP used in 1950 was 25-60 times that used today in Mobil Jet Oil II and about 415-633 times that used in Mobil Jet Oil 291. The TCP used in Jet Oil 291 does not inhibit NTE by 70% when administered to hens nor does it cause OPIDN in hen studies.<sup>4</sup>

The weak cholinesterase inhibiting activity of the TCP used in Jet Oil II also was further reduced by lowering the level of ortho cresol in the synthesis mixture. This reduction is probably the result of increased levels of triparacresyl phosphate which is not a cholinesterase inhibitor (Mackerer, 1999). TCP with decreased anticholinesterase activity (low-toxicity TCP) is used in Mobil Jet Oil 291.

- **Newer versions of TCP have markedly reduced the toxicity of Jet Engine Oils.**

In 1959, in Morocco, two jet engine oils (manufacturers unknown) containing an older more toxic TCP than used in Jet Oil II were intentionally added to cooking oil. Ingestion of this contaminated oil poisoned more than 10,000 people and produced the typical sequelae of OPIDN<sup>14</sup>; most patients recovered but some retained paralysis of hands and/or feet. (This poisoning was a key factor driving future attempts to develop a less toxic TCP for jet turbine oil applications.) These patients were closely observed by an international group of physicians and scientists, and there were no reports of cognitive, behavioral, or mood changes, that would indicate the presence of neurological deficiencies other than OPIDN.<sup>15</sup>

Mobil Jet Oil II produced symptoms of mild cholinergic stimulation (eg. lacrimation and salivation) and OPIDN when administered to hens repeatedly, by the oral route, at 2 g/kg. OPIDN was not observed at a dose of 1 g/kg/d.<sup>16,17</sup> There was a clear threshold for effect in that symptoms were only seen in hens with brain NTE inhibitions greater than 70%.<sup>4,16</sup>

A Jet Oil formulation containing the TCP used in Jet Oil 291 at 2 g/kg dose produced minimal symptoms of cholinergic stimulation than did Jet Oil II, did not inhibit NTE to more than 70%, and did not produce OPIDN.

In 1990, Mobil conducted an internal risk assessment of the potential of a release of Mobil Jet Oil II into an aircraft cabin to cause neurotoxicity in airline employees or passengers. This assessment was later recast and published<sup>12</sup>. It was concluded that even under extreme exposure conditions toxicity would not occur from release of Mobil jet engine oils. Also, Mackerer et al<sup>4</sup> concluded that cabin air concentrations of Jet Oil would not reach levels that could produce OPIDN.

## References

- Davey, W. 1950. Extreme pressure lubricants. Phosphorus compounds as additives. *Ind. Eng. Chem.* 42:1841-1847.
- Klaus, E., and Bieber, H. 1965. Effects of  $^{32}\text{P}$  impurities on the behavior of tricresyl phosphate— $^{32}\text{P}$  as an antiwear additive. *ASLE Trans.* 8:12-20.
- Godfrey, D. 1965. The lubrication mechanism of tricresyl phosphate on steel. *ASLE Trans.* 8:1-11.
- Mackerer, C. R., Barth, M. L., Kreuger, A. J., Chawla, B., and Roy, T. A. 1999. Comparison of neurotoxic effects and potential risks from oral administration or ingestion of jet engine lubricants. *J. Toxicol. Environ. Health* 57:293-328.
- Henschler, D. 1958. Tricresyl phosphate poisoning. Experimental classification of problems of etiology and pathogenesis. *Klin. Wochenschr.* 36:663-674.
- Sax, N. I. And Lewis, R. J. 1989. Dangerous properties of industrial materials. 3:2652, 3429.
- Jamal, G. A. 1997. Neurologic syndromes of organophosphorus compounds. 16:133-170.
- WHO. 1990. Effects on Humans. Environmental Health Criteria. 110:70-79.
- Tabershaw, I. R., and Kleinfeld, M. 1957. Manufacture of tricresyl phosphate and other alkyl phenyl phosphates: an industrial hygiene study. II. Clinical effects of tricresyl phosphate. *Arch. Ind. Health* 15:541-544.
- Tabershaw, I. R., Kleinfeld, M., and Feiner, B. 1957. Manufacture of tricresyl phosphate and other alkyl phenyl phosphates: an industrial hygiene study. I. Environmental factors. *Arch. Ind. Health* 15:537-540.
- Smith, M. I., Elvove, I., and Frasier, W. H. 1930. The pharmacologic action of certain phenolic esters, with special reference to the etiology of so-called ginger paralysis. *US Public Health Reports* 45:2509-2524.
- Craig, P. H. and Barth, M. L. 1999. Evaluation of the hazards of industrial exposure to tricresyl phosphate: a review and interpretation of the literature. *J. Toxicol. Environ. Health* 57: 293-328.
- Hunter, D., Perry, K. M. A., and Evans, R. B. 1944. Toxic polyneuritis arising during the manufacture of tricresyl phosphate. *Br. J. Ind. Med.* 1:227-231.
- Henschler, D., and Neumann, W. 1968. Contributions to the toxicology of the triaryl-phosphate mass poisoning in Morocco. In *Triaryl-phosphate poisoning in Morocco 1959*, eds. A. v. Albertini, D. Gross, and W. M. Zinn, pp. 20-27. New York: Intercontinental Medical Book Corporation.
- Several authors. 1968. Contributions to the toxicology of the triaryl-phosphate mass poisoning in Morocco. In *Triaryl phosphate poisoning in Morocco*, eds. A. V. Albertini, D. Gross, and W. Zinn, pp. 20-27. Stuttgart: George Thieme Verlag; New York: Intercontinental Medical Book Corp.



- 16 Freudenthal, R. I., Rausch, L., Gerhart, J. M., Barth, M. L., Mackerer, C. R., and Bisinger, E. C. 1993. Subchronic neurotoxicity of oil formulations containing either tricresyl phosphate or tri-ortho cresyl phosphate. *J. Am. Coll. Toxicol.* 12:409-415.
- 17 Daughtry, W., Biles, R., Jortner, B., and Ehrich, M. 1996. Subchronic delayed neurotoxicity evaluation of jet engine lubricants containing phosphorous additives. 32:244-249.

ATTN: Mr. J. O'KEEFE

ORIGINAL IS IN

TODAY'S MAIL.

Mobil Oil Australia

A.C.N. 004 252 999

417 St. Kilda Road

Melbourne 3004

Ph: (03) 9252 3111

Fax: (03) 9252 3589

GPO BOX 4607

Melbourne VIC 3001

TELEX 20 19 26 52

FAX: (03) 9866 9079

Web Site

<http://www.mobil.com.au>

October 7, 1999

The Secretary  
Senate Rural and Regional Affairs  
and Transport References Committee  
SG 62  
Parliament House  
Canberra ACT 2600



Dear Sir/Madam,

Thank you for the opportunity to comment on the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) document entitled "Overview of Available Scientific Background Information" on Mobil Jet Oil II ("Overview"). As we have discussed previously, Mobil Oil Australia, Ltd. (MOA) is concerned that the document presents a misleading picture of potential health and safety hazards associated with Mobil Jet Oil II ("MJO" or "the product"). We appreciate your willingness to consider the issues we raise herein on the record as part of the overall presentation of the Overview to The Secretariat of the Senate Rural and Regional Affairs and Transport Committee.

At the outset, we acknowledge that the Overview does not purport to represent an assessment of potential health risks associated with MJO (see page 2 of Overview). Still, the document does set forth detailed information on the toxicology of a number of MJO components, including constituents that may be present as impurities and only at trace levels. We are concerned that the lack of context for these toxicological profiles may result in The Secretariat (or members of the public who otherwise review the document) concluding that the product displays certain health and safety risks, when in fact it does not. When the product is viewed as a whole, MJO is not a hazardous material as classified pursuant to the National Occupational Health and Safety Commission (NOHSC) Worksafe criteria (see page 11 of Overview). Also, scientific studies, including the recent work by Mackerer et. al. (referenced in the Overview), demonstrate that exposure to jet oils does not pose a significant risk to human health.

MOA has a number of specific concerns with respect to the presentation of information in the Overview. For example, the Overview emphasizes the presence of phenyl-beta-naphthylamine (PBN) and beta-naphthylamine (BNA) based on a 1992 Material Safety Data Bulletin ("MSDB") that indicated these materials are present in the product. Information not available on the MSDB (and, therefore, not referenced in the Overview), is that each of these compounds is present in one of the primary antioxidants used in MJO, phenyl-alpha-naphthylamine (PAN). The PAN specification states that the antioxidant provided to Mobil has a maximum of 0.5% of PBN and a maximum of

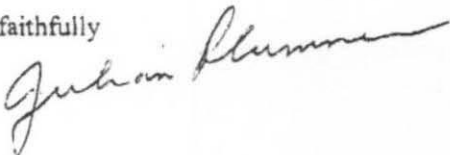
0.005% of BNA. Since PAN is used in the MJO formulation at about 1%, the *maximum* levels of PBN and BNA that could *potentially* be present are 0.005% (50 parts per million) and 0.00005%, (0.5 part per million). The actual levels present would be expected to be much lower. In fact, because of the extremely low levels of these impurities, Mobil stopped listing them in the MSDS as "ingredients" in the formulation sometime after 1992. The change in the MSDS was due solely to a reassessment of what was considered meaningful information from a hazard communication perspective. There was no change in the MJO formulation in the 1990's to remove PBN or BNA as speculated in the Overview. In view of the above, we believe the Overview could lead to undue public concern due to the statement regarding the presence of potential carcinogens in the product. In fact, these constituents are present as impurities only at trace levels and below the level at which an adverse health effect could occur.

Another concern we have is the highlighting in the Overview of the discussion of Tri-ortho-cresyl-phosphate (TOCP). Although the presentation of the material on *pure* TOCP technically may be correct, the discussion is not relevant with respect to MJO because the potential maximum concentration of this impurity in the product is around 1 part per million. In addition, although the reproductive toxicity of TOCP in animals is mentioned in the Overview, no note is made that this effect has never been seen in humans.

Finally, the section on formation of trimethylolpropane phosphate (TMPP) as a product of combustion (section 5) does not apply to MJO. Accordingly, the discussion should be removed from the document in its entirety.


In this letter, we have raised certain issues that MOA believes need to be addressed in order for the Overview to present a more realistic picture of the constituents of MJO. If you think it would be useful, we would be glad to make available to NICNAS our technical specialists for more detailed discussion of these and other issues relating to MJO.

Yours faithfully



J C Plummer  
Manager, Aviation Lubricant Sales

Endorsed:



J P Mannes  
Director, Lubricants Division  
Mobil Oil Australia Limited