TRISCRESYLPHOSPHATE AND RELATED COMPOUNDS IN AIRPLANES:
POSSIBLE EFFECTS ON NERVOUS SYSTEMS OF FLIGHT CREW MEMBERS

JACOB DE BOER

Institute for Environmental studies, VU University, De Boelelaan 1087, 1981HV Amsterdam,
The Netherlands;
Email: jacob.de.boer@vu.nl

EXTENDED ABSTRACT
Triscresylphosphate (TCP), and in particular its tri-ortho substituted isomer, are being used in airplane motor oil. Bleed air, provided to the cabin and flight deck can contain traces of TCP. TCP can cause neurotoxic effects in humans. Regularly, airline pilots report suffering from neurotoxic effects. TCP was analysed and found in flight deck but the concentrations found do not exceed provisional toxicity thresholds. Specific conditions in air planes and other toxic compounds present in bleed air may be responsible for the reported neurotoxic syndromes.

Key Words: Triscresylphosphate, airplanes, bleed air, fume event

1. INTRODUCTION
Since the start of the jet era in the 1960s air supply in pressure cabins in airplanes is systematically provided by bleed air systems. Coming from outside, the cold air is heated via the engines and then supplied to the passenger cabin and the flight deck. During the passage through the engines the air can accumulate small amounts of motor oil. This motor oil can contain traces of additives that are present in motor oil to protect the engine against wear and tear and to extend its life span. Triscresylphosphate (TCP), an arylphosphate, is one of the most used motor oil additives for this purpose. Most jet oils produced since the 1990s contain 13-150 mg/L TCP (De Nola et al., 2008). However, TCP is also known to be neurotoxic. During the last decade a growing number of reports on so-called fume events have been received by authorities. In fact, every day, somewhere in the world a ‘fume event’ takes place. During such a fume event, smoke enters the flight deck because seals may leak, probably during a switching event in the engine, often at the beginning of the landing procedure. During fume events pilots need to put on their oxygen masks while trying to land the airplane safely. Apart from these fume events, several reports on health problems of pilots have been received. Pilots suffer from tunnel vision, loss of memory, headaches and other possible neurotoxic syndromes. A chronic exposure to compounds such as TCP has been suggested to cause these health problems, partly during repeated fume events, partly due to a constant leakage of small amounts of TCP during every flight. Passengers have been less frequently and less intensively exposed to TCP, as the bleed air systems to the flight deck and the passenger cabin are normally separated to give the pilots more time in case of fire. TCP (Fig. 1) consists of ten isomers that differ from each other in the way the methyl groups are situated in the phenyl rings. The tris-ortho (o,o,o) isomer is supposed to be the most toxic one, but some reports consider the toxicity of isomers with only one ortho group higher (Hanhela et al., 2005). Not all isomers are available as analytical standards, which hinders the determination of total TCP. The neurotoxicity data are limited. Rats are relatively insensitive for TCP. Therefore, an experiment has been carried out with chickens. More information is needed for a more precise calculation of the risks of TCP. Similarly to many other organophosphate compounds, TCP cannot be found in human blood because
there is a rapid metabolism. Studies on metabolites of TCP in human blood are therefore also needed. This study gives an overview of a series of first experiments and calculations to establish a link between TCP exposure and health problems of pilots.

**Figure 1.** Structure of TCP and mass spectrum of o,o,o-TCP.

**2. MATERIALS AND METHODS**

Glass tubes of 7x70 mm containing 300 mg Chromosorb 106 were used for monitoring in airplanes. Only flightdecks were monitored. Air was pumped through by a battery-driven pump with a capacity of 120 L/h (Van Netten, 2009). The columns were desorbed by 3 mL diethyl ether. TCP was analyzed by GC/EI-MS. The separation and quantification of the TCP isomers took place on a 30 m x 0.25 mm ID x 0.25 μm HP-5 (5 % phenyl methyl) capillary column. The initial oven temperature was set at 50 ºC for 0.5 min, then increased to 150 ºC at 50 ºC/min, then increased to 280 at 60 ºC/min and finally increased to 320 at 10 ºC/min and held for 5 min. The injector temperature was 275 ºC and the helium carrier gas flow was 1.0 mL/min. The samples were injected in splitless mode (1 μL). Three isomers (o,o,o, m,m,m and p,p,p) were characterized using the m/z ratio of 165, 277 and 368. The o,o,o-TCP shows higher abundance of the 165 whether the m,m,m- and p,p,p-TCP shows a higher 368 m/z ratio. The m/z ratio of 368 and 277 decreases when the ortho substituents change into the meta and para positions. All samples were analyzed with internal standard tris (2-ethylhexyl) phosphate, and the results were corrected for the column blank.

**3. RESULTS AND DISCUSSION**

The maximum total-TCP concentrations analysed were 37 and 43 ng/m³ in a Fokker F-100 and a KLM Cityhopper Fokker F-70 airplane, respectively. The maximum o,o,o-TCP concentration found was 28 ng/m³ in the F-70. The maximum allowable TCP concentration in military aircraft in Australia is 100 μg/m³, which is advised to lower to 1 μg/m³ because of the possible presence of mono-ortho-trimethylcresylphosphate (TMOC), which is ten times
more toxic than o,o,o-TCP (Hanhela et al., 2005). Newer jet oils contain less o,o,o,-TCP, but more TMOCP than o,o,o,-TCP.

Abou Donia (1981) and Craig and Barth (1999) describes neurological effects due to exposure of humans to organophosphate esters, which is called organo phosphate ester-induced chronic neurotoxicity (OPIDN). Hens used for neurotox experiments of TCP show variable response to TCP, which at least demands for a factor of 100 safety factor in risk calculations. Because of the higher toxicity of TOCP a 100-fold underestimation of OPIDN threshold is possible (Mackerer et al., 1999). Their most critical threshold value for OPIDN is 1400 mg/day chronic exposure for a person of 70 kg. Taken into account the 100-fold underestimation, this would result in a realistic threshold of 14 mg/d total TCP for a person of 70 kg. We suppose a 100% uptake of the lipophilic TCP components (worst-case). Inhalation volume of a human: ca. 10 L/min, or ca. 600 L/h, which is 0.6 m$^3$/h. The TCP concentration in the F-70: 43 ng/m$^3$ (20x lower than newest Australian limit) would then result in an uptake of ca. 25 ng/h, which is 250 ng per 10h flying time/d. This is still 56,000-fold under the most realistic threshold of Mackerer et al.

4. CONCLUSIONS
Unless TCP in the vapour phase is much more toxic than TCP when dosed orally, it is unlikely that chronic exposure to TCP levels that ‘normally’ occur at flight decks of aircrafts such as F-70 and F-100 cause neurotoxic effects such as OPIDN. However, the auxiliary power unit (APU) may cause higher TCP levels that could come into the critical range. Furthermore, it cannot be excluded that other compounds from engine oil are present that could be more critical in terms of human health effects (Lipscomb et al., 1995). Also, hitherto unknown additive or synergic effects may occur due to the presence of a multitude of chemicals in the oil, while degradation products of TCP could also show a specific toxicity. The high altitude and lower air pressure at the flight deck and in the cabin may have an influence on the toxic mechanism of TCP. Finally, TCP can also originate from the flame retardant used in seats and plastic cabin material in airplanes.

The IPCS (1990) reports: “Because of considerable variation among individuals in sensitivity to o,o,o-TCP, it is not possible to establish a safe level of exposure. It seems that at least one o-tolyl group among the three phenolic moieties of TCP is necessary to induce neurotoxic effects. When tri-substituted cresols are used in the synthesis and manufacture of other compounds, the purified meta and para isomers should be used in order to avoid the accidental synthesis of ortho-substituted products”.

5. ACKNOWLEDGMENT
The author likes to acknowledge Mr. Michel Mulder for taking samples and Mr. Brandsma, Ms. Lammertse and Ms. Van der Veen for carrying out analytical work.

6. REFERENCES
