Current Topics in Toxicology
Vol. 3, 2006

Original Communication

# Hazardous chemicals on jet aircraft: case study – jet engine oils and aerotoxic syndrome

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## ABSTRACT

The cabin of an airplane is a specialised working environment and should be considered as such. The oils and hydraulics used in airplane engines are toxic, and specific ingredients of such materials are irritating, sensitising and neurotoxic. If oil or hydraulic fluids leaks out of engines, this contamination may be in the form of unchanged oil/fluid, degraded oil/fluid from long use in the engine, combusted oil/fluid or pyrolised oil/fluid, in the form of gases, vapours, mists and particulate matter. If leak incidents occur and the oil/fluid is ingested into bleed air and is passed to the flight deck and passenger cabins of airplanes in flight, aircrew and passengers may be exposed to contaminants that can affect their health and safety. Where contamination of air in flight deck and passenger cabin occurs that is sufficient to cause symptoms of discomfort, fatigue, irritation or toxicity, this contravenes the air quality provisions of Aviation Regulations, most notably FAR 25.831. Symptoms of immediate or short term nature reported by exposed staff in single or few leak incidents are consistent with the development of irritation and discomfort. Symptoms of a long term nature (that is, sustained symptoms for at least six months) reported by some exposed staff following small to moderate numbers of leak incidents are consistent with the development of an irreversible discrete occupational health condition, termed aerotoxic syndrome. Features of this syndrome are that it is associated with air crew exposure at altitude to atmospheric contaminants from engine oil or other aircraft fluids, temporarily juxtaposed by the development of a consistent symptomology including short-term skin, gastro-intestinal, respiratory and nervous system effects, and long-term central nervous and immunological effects.

**KEYWORDS:** aviation air quality, aviation safety, airborne contaminants, jet oils, aerotoxic syndrome

## INTRODUCTION

Air quality is an important aviation problem. Problems arise from a number of factors, including:

- The problem of hypoxia. Commercial flight levels typically range from 31,000 to 42,000 ft, above sea level and the aircraft cabin is pressurised to an hypobaric environment equivalent to 8,000 ft (2,315 m). Hypoxia may interact adversely with chemical exposures[1].
- The problem of ventilation. Studies indicate[2] that it is common that all modes of transport have ventilation rates less than current ASHRAE 62 guidelines for commercial buildings[3]. This finding, of itself, does not imply poor air quality. However, it suggests that initiatives to reduce air quality should be resisted and

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indicates that opportunities to improve air quality should be encouraged. For example, a Canadian study of one aircraft type and airline found that 245 of 333 commercial flights did not satisfy the ASHRAE air ventilation criteria of fifteen cubic feet/occupant, and that 18 of 33 flights had less than ten cubic feet/ occupant[4].

- The problem of contamination of air. Chemical exposures in aircraft are not unheard of. In 1953, The US Aero-medical Association first expressed their concerns about the toxicity risks of cabin air contamination by hydraulics and lubricants[5]. The oils and hydraulics used in aircraft engines can be toxic, and specific ingredients of oils can be irritating, sensitising (such as phenyl-alpha-naphthylamine) or neurotoxic (for example, ortho-containing triaryl phosphates such as tri-orthocresyl phosphate). If oil or hydraulic fluid leaks occur, this contamination may be in the form of unchanged material, degraded material from long use, combusted or pyrolised materials. These materials can contaminate aircraft cabin air in the form of gases, vapours, mists and aerosols. Other risks have been identified more recently, either as part of the chemicals routinely used in maintaining airplanes[6], or as products of the passengers or cargo[2].
- Problems of combustion and emergency situations[7]. Passenger protective breathing equipment tests conducted by the UK Air Accidents Investigation Branch (AAIB) identify contaminants in combustion situations such as carbon monoxide, hydrogen cyanide, hydrogen fluoride, hydrogen chloride, nitrogen oxides, sulphur dioxide, ammonia, acrolein, and other hydrocarbon compounds[8].

Notwithstanding normal operational activities or emergency situations, a range of other situations can arise whereby aircraft cabin air can be contaminated[9]. These include:

- uptake of exhaust from other aircraft or on ground contamination sources,
- application of de-icing fluids,
- hydraulic fluid leaks from landing gear and other hydraulic systems,
- excessive use of lubricants and preservative compounds in the cargo hold,
- preservatives on the inside of aircraft skin,
- large accumulations of dirt and brake dust may build up on inlet ducts where auxillary power units extract air from near the aircraft belly,
- ingestion of oil and hydraulic fluid at sealing interfaces, around oil cooling fan gaskets and in worn transitions,
- oil contamination from synthetic turbine oil,
- engine combustion products (for example, defective fuel manifolds, seal failures, engine leaks).

Other air quality problems include ethanol and acetone, indicators of bioeffluents and chemicals from consumer products[10]. One additional problem is the lower partial pressure of oxygen that is present in the cabins of planes flying at altitude[11].

International aviation legislation such as the US Federal Aviation Regulations (FAR) and airworthiness standards for aircraft air quality state "*crew and passenger compartment air must be free from harmful and hazardous concentrations of gases or vapors*"[12]. Where contamination of air in the flight deck and passenger cabin occurs that is sufficient to cause symptoms of discomfort, fatigue, irritation or toxicity, this contravenes such standards and legislation.

## The chemical products used in aviation

The aviation industry has used fuels, lubricants, hydraulic fluids and other materials that can contain a range of toxic ingredients. Aircraft materials such as jet-fuel, de-icing fluids, engine oil, hydraulic fluids, and so on, contain a range of ingredients, some of which are toxic[13,14,15,16]. Significant contaminants include: aldehydes; aromatic hydrocarbons; aliphatic hydrocarbons; chlorinated, fluorinated, methylated, phosphate or nitrogen compounds; esters; and oxides[17].

A complex approval process exists for ensuring that materials used in aviation are manufactured to relevant standards. For example, jet fuels are specified by the American Society for Testing and Materials (ASTM D 1655 Standard Specification for Aviation Turbine Fuels)[18] and the United Kingdom Ministry of Defence (MOD Standard 91-91)[19], and the jet engine oil specification of the US Navy MIL-PRF-23699 is used for jet oils. This process of approval and re-approval for new product formulations has meant that there is some resistance to modifying formulations (for example, for health and safety reasons).

Consequently, changing approved formulations is not conducted without significant justification. In the case of the jet oil additive tricresyl phosphate (TCP, discussed below), manufacturers have been reluctant to modify product formulations by substituting toxic TCP additives that perform well in critical applications. This has meant that potentially toxic products have continued to be available and used long after their toxicity has been recognised[20]. It is not known if an approved formulation containing, for example 3% tricresyl phosphate, is considered a change in formulation if the proportion of individual isomers in the TCP mixture is altered, but the 3% remains unchanged. However, as Mobil indicate, only the base stock esters have been modified over the past thirty or so years, suggesting that the mixture of isomers in TCP stock has not been changed.

**Fuels** are based on the type on engine type (piston, turbo or jet) and operating conditions. They are similar to other petroleum products that have a boiling range of approximately 150°C to 300°C. The freezing point and flash point are the principle differences between the finished fuels. The main fuels used are the kerosene based Jet A (used in the USA or Jet A-1 (used around the world). Jet B is a modified fuel for use in cold climates. Chemical additives allowed for use in jet fuel are also defined in product specifications[21].

Over two million workers are occupationally exposed each year to jet propulsion fuels. Approximately 220 billion litres of these kerosene-based jet fuels are annually consumed[22].

Kerosene-based hydrocarbon fuels are complex mixtures of over 200 aliphatic and aromatic hydrocarbon compounds ($C_6$ to $C_{17}$), including varying concentrations of potential toxicants such as benzene, n-hexane, toluene, xylenes, trimethylpentane, methoxyethanol, naphthalenes (including polycyclic aromatic hydrocarbons [PAHs], and certain other $C_9$-$C_{12}$ fractions such as n-propylbenzene, trimethylbenzene isomers). Table 1 lists some of the components of an early sample of Jet Fuel A[23].

This is consistent with proprietary commercial information, as available on product MSDS (although the aromatic fraction may have been reduced over the years (see Table 2).

**Lubricants** are classified into either:

- mineral petroleum oils either straight mineral of the appropriate viscosity or blended with additives or part synthetic multigrade oils for piston engines; or
- mineral based (mainly for earlier models of jet engines) or synthetic or turbojet, turboprop or turbofan engines.

Oil types include: mineral oils; semi-synthetic oil; synthetic oils; jet oils; turbine oils; piston engine oils, gear oils.

**Hydraulic Fluids** are usually of the mineral or synthetic, normal or superclean type.

**Greases** usually containing mineral or synthetic base oils with metal soaps or organic thickeners or inorganic fillers.

**Speciality chemicals** include antiseize compounds; bonded parts; coolants; corrosion preventatives; damping fluids; de-icing fluids; dry lubricants; instrument oils; lubricity agents; protectives; sealants, adhesives, epoxy resins; shock strut fluids.

**Table 1.** Jet A Constitution

| Constituent Composition | % Volume | |
|---|---|---|
| Simple Alkanes | | 53.7 |
| Includes: | | |
| Decane | 16.5 | |
| Undecane | 36 | |
| Methyl Alkanes | | 3.77 |
| Cycloalkanes | | 0.79 |
| Monocyclic Aromatic Hydrocarbons | | 31.8 |
| Includes: | | |
| Benzene | 0.02 | |
| Butylbenzene | 2 | |
| 1,2-Diethylbenzene | 0.24 | |
| 1,2-Diethyl-3-propylbenzene | 5.4 | |
| 1,4-Diethyl-2-ethylbenzene | 0.2 | |
| Ethylbenzene | 0.02 | |
| 1-Methyl-4-propylbenzene | 3.3 | |
| Propylbenzene | 3-5 | |
| 1,2,4,5-Tetramethylbenzene | 9 | |
| Toluene | trace | |
| 1,2,3-Trimethylbenzene | 6.6 | |
| Xylenes | 0.07 | |
| Polycyclic Aromatic Hydrocarbons | | 0.63 |
| Includes: | | |
| Naphthalene | 0.14 | |
| 2-Methylnaphthalene | 0.34 | |
| 1,3-Dimethylnaphthalene | 0.15 | |

**Table 2.** Jet A Constitution (from Product MSDS)

| Component | % present |
|---|---|
| Saturated Hydrocarbons (Paraffins and Cycloparaffins) | 70-80% |
| Aromatic Hydrocarbons | 17-20% |
| Unsaturated Hydrocarbons (Olefins) | 3-6% |

A range of aviation chemicals is shown in Table 3.

Inhalation is an important route of exposure, with exposure to uncovered skin being a second, less significant route (for example, following exposure to oil mists or vapours). Ingestion is unlikely.

A number of recently published studies reported acute or persisting biological or health effects such as human liver dysfunction, emotional dysfunction, abnormal electroencephalograms, shortened attention spans, decreased sensorimotor speed and immune system dysfunction from single, short term repeated exposure, or long term repeated exposure of humans or animals to kerosene-based hydrocarbon fuels, to constituent chemicals of these fuels, or to fuel combustion products[24, 25,26,27,28,29,30,31]. Other reports suggest that other aviation chemicals may be toxic[17,32,33].

Occasionally, such exposures may be of a magnitude to induce symptoms of toxicity. In terms of toxicity a growing number of aircrew are developing symptoms following both short term and long term repeated exposures,

**Table 3.** Aviation Chemicals

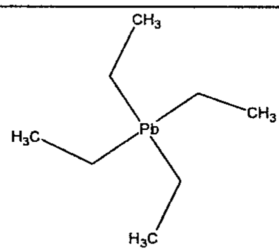
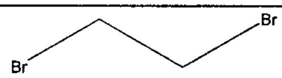
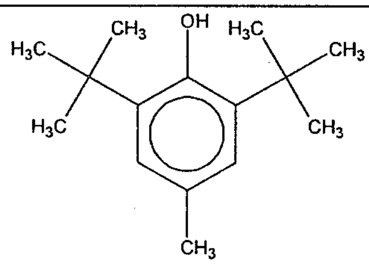
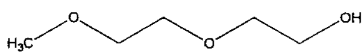
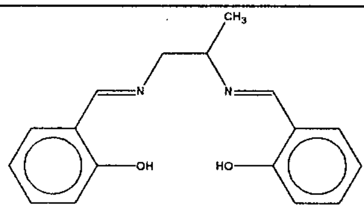
| Product | Type | Ingredients | Formula |
|---|---|---|---|
| **Jet Fuels** | | | |
| | Jet A and Jet A-1 | A kerosene based fuel, based on ASTM Specification D1655) | Varies, depending on manufacturer |
| | Jet B | A wide cut blend of gasoline and kerosene, rarely used except in very cold conditions | Varies, depending on manufacturer |
| | Aviation gasoline | | Varies, depending on manufacturer |
| **Aviation fuel additives** | | | |
| | Anti-knock additives | Tetra-ethyl lead (TEL) |  |
| | | Ethylene dibromide |  |
| | Anti-oxidants | 2,6-ditertiary butyl-4-methyl phenol |  |
| | Electrical conductivity/ static dissipater additives | Stadis®450 | Proprietary mixture |
| | Corrosion inhibitor/ lubricity improver | "DCI-4a" | Proprietary mixture |
| | Anti-icing additives | Di-ethylene glycol monomethylether |  |
| | Metal deactivators | N,N'-disalicylidene-1,2-propane diamine |  |
| | Biocides | | |
| | Thermal Stability Improver additives | (mainly military applications) – "+100" | Proprietary mixture |
| | Leak detection | Tracer A® | Proprietary mixture |

Table 3 continued..

| | | | |
|---|---|---|---|
| Lubricants, based on | | | |
| | Mineral oils | | Proprietary mixtures |
| | Synthetic oils | | Proprietary mixtures |
| Hydraulic fluids | | | |
| | Mineral types | | Proprietary mixtures |
| | Synthetic types | | Proprietary mixtures |
| Greases | | | |
| Speciality Chemicals | | | |
| | Antiseize compounds | | Proprietary mixtures |
| | Coolants | | Proprietary mixtures |
| | Corrosion preventatives | | Proprietary mixtures |
| | Damping fluids | | Proprietary mixtures |
| | De-icing fluids | | Proprietary mixtures |
| | Dry lubricants | | Proprietary mixtures |
| | Instrument oils | | Proprietary mixtures |
| | Lubricity agents | | Proprietary mixtures |
| | Protectives | | Proprietary mixtures |
| | Sealants, adhesives, epoxy resins | | Proprietary mixtures |
| | Shock strut fluids | | Proprietary mixtures |
| | Bonded parts | | Proprietary mixtures |

including dizziness, fatigue, nausea, disorientation, confusion, blurred vision, lethargy and tremors[34,35,36]. Neurotoxicity is a major flight safety concern especially where exposures are intense[37].

Taken together, these indicate that air quality on aircraft is a significant aviation safety issue.

**Case study: the toxic ingredients of jet oils**

The engine oils that are used in jet engines are precision oils that need to operate in extreme conditions. Some commercial jet oils have been in use as engine oils in aviation for decades. For example, Mobil USA note that Mobil Jet Oil II (a jet oil with close to half the market share) "has been essentially unchanged since its development in the early 1960s" and "most changes have involved slight revisions of the ester base stock due to changes in raw material availability"[39].

Therefore, jet oils are specialised synthetic oils used in high performance jet engines. They have an appreciable hazard based on toxic ingredients, but are safe in use by engineering personnel who handle the product routinely provided that:

- health and safety information such as labels, material safety data sheets, manufacturers manuals and the like are obtained and consulted;
- a suitable risk assessment is carried out that identifies hazards and assesses risks, and recommends suitable controls and precautions;

- maintenance personnel follow the appropriate controls and safety precautions as recommeded in health and safety information and risk assessments; and
- the oil stays in the engine.

Aircraft engines that leak oil may expose others to the oils through uncontrolled exposure. Airplanes that use engines as a source of bleed air for cabin pressurisation may have this source contaminated by the oil, if an engine leaks. If such leaks occur, exposed crew and passengers do not have access to the health and safety information, risk assessments or advice on controls that engineering staff have; where such information or advice is lacking, they may be at additional risk.

Using a typical commercial Jet Oil (Mobil Jet Oil II), various sources, such as the supplier's label on the cardboard box the cans are shipped in, the product Material Safety Data Bulletin (MSDB), and information from the manufacturer, list the following ingredients[17]:

- synthetic esters based in a mixture of 95% $C_5$-$C_{10}$ fatty acid esters of pentaerythritol and dipentaerythritol;
- 1% of a substituted diphenylamine;
- 3% tricresyl phosphate (Phoshoric acid, tris(methylphenyl) ester, CAS No 1330-78-5);
- 1% phenyl-alpha-naphthylamine (PAN) (1-Naphthalenamine, N-phenyl, CAS No 90-30-2);
- a last entry "ingredients partially unknown" is also noted on some documentation.

Of these ingredients, the most toxicologically significant components are the substituted diphenylamine, phenyl-alpha-naphthylamine (PAN) and tricresyl phosphate (TCP).

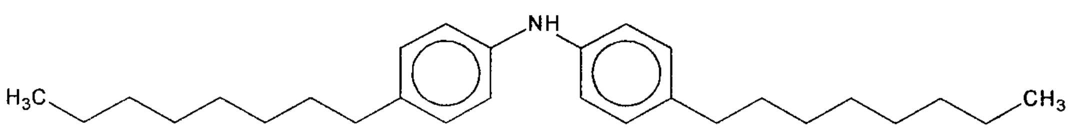
### The substituted diphenylamine

The substituted diphenylamine is variously reported as Benzamine, 4-Octyl-N-(4-Octyl-phenyl), (CAS No 101-67-7) or 0.1-1% N-Phenyl-benzeneamine, reaction product with 2,4,4-Trimethylpentene (CAS No 68411-46-1), and used as an antioxidant, in concentrations not greater than 1% (see Figure 1).

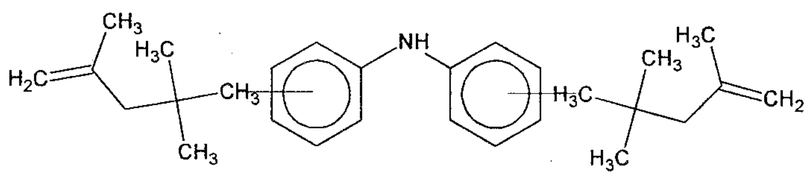
There is little toxicity data available for this ingredient, although it is not believed to be toxic by single exposure (no data on long term exposure). The disclosure of this ingredient in hazard communication by identity probably relates to its environmental effects, such as poor biodegradability and toxicity to aquatic invertebrates[40].

### N-Phenyl-alpha-naphthylamine

**N-Phenyl-alpha-naphthylamine,** (CAS No 90-30-2), also known as Phenyl-alpha-naphthylamine (PAN), is a lipophilic solid used as an antioxidant used in lubrication oils and as a protective agent in rubber products (see Figure 2). In these products, the chemical acts as a radical scavenger in the auto-oxidation




Benzamine, 4-Octyl-N-(4-Octylphenyl)




N-Phenyl-benzeneamine, reaction product with 2,4,4- Trimethylpentene

**Figure 1.** Substituted Diphenylamines.

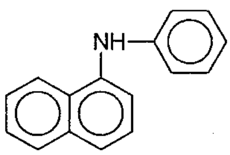



**Figure 2.** N-Phenyl-1-naphthylamine.

of polymers or lubricants. It is generally used in these products at a concentration of about 1% (its concentration in jet oils). The commercial product has a typical purity of about 99%. Named impurities are: N-Phenyl-2-naphthylamine (CAS No 135-88-6, 500 to below 5000 ppm), 1-Naphthylamine (below 100-500 ppm) and 2-Naphthylamine (below 3 to 50 ppm), aniline (below 100 to 2500 ppm), 1-naphthol (below 5000 ppm), 1,1-dinaphthylamine (below 1000 ppm).

PAN is readily absorbed by mammalian systems and rapidly biotransformed[41]. Both urine and feces appear to be the main routes of excretion[42].

By single dosing, PAN has a low toxicity, with $LD_{50}$s above 1 g/kg. The chemical has a similar mechanism of toxicity to many aromatic amines, of methaemoglobin production. PAN is not irritating in primary skin and eye irritation studies. However, in a guinea pig maximisation test, PAN was shown to be a strong skin sensitiser[43]. This result is supported by case studies in exposed workers[44,45]. At the concentration used (1%), Mobil Jet Oil II meets cut off criteria (1%) for classification as a hazardous substance in Australia for sensitisation properties.

Most genotoxicity studies report negative results, suggesting little genotoxicity potential[42].

Most repeated dose toxicological studies focus on its potential carcinogenicity. An experimental study, using both PAN and the related compound N-phenyl-2-naphthalenamine administered subcutaneously to mice found a heightened incidence of lung and kidney cancers[46].The methodology used in this study makes evaluation of the results problematic (use of one gender, small sample sizes, limited number of dose groups, sub-cutaneous administration as an inappropriate route of exposure, and so on). A high incidence of various forms of cancer was also found among workers exposed to antirust oil containing 0.5% PAN[47]. While these animal and human results offer only limited information, they are at least supportive of a mild carcinogenic effect.

This must be contrasted with the results of long term carcinogenicity bioassays in rats and mice conducted by the US National Toxicology Program with the structurally related N-phenyl-2-naphthylamine (studies were not carried out on PAN), which have not reported any carcinogenic potential for this chemical[48].

## Tricresyl phosphate

**Tricresyl phosphate**, (CAS No 1330-78-5) is also known as Phosphoric acid, tris (methylphenyl) ester or Tritolyl phosphate. TCP is a blend of ten tricresyl phosphate isomer molecules, plus other structurally similar compounds, including phenolic and xylenolic compounds. TCP is a molecule comprised of three cresyl (methylphenyl) groups linked to a phosphate group. The location of the methyl group in the cresyl group is critical for the expression of neurotoxicity, with ortho-, meta- or para- prefixes that denote how far apart the hydroxyl and methyl groups are on the cresol molecule. Technically, there are 27 ($3^3$) different combinations of meta, ortho and para cresyl groups in TCP (see Figure 3). Since the apparently different three-dimensional structures of the molecule are not chemically locked in place, they are not optical isomers. Therefore,

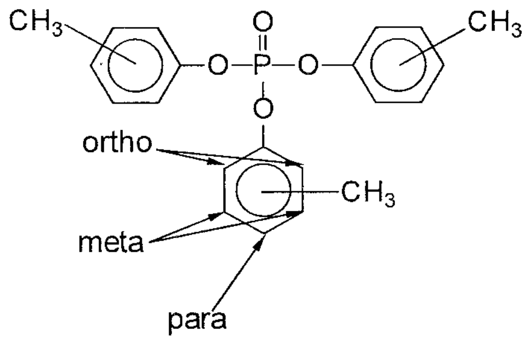



**Figure 3.** Structure of Tricresyl Phosphate. TCP molecule showing designation of o, m and p cresyl groups.

structures with similar numbers of cresyl groups (such as ppm, pmp and mpp) are considered the same molecules. This gets the apparent 27 structures down to the real ten isomers conventionally described.

CAS Number descriptors for tricresyl phosphate chemicals have been introduced to differentiate between ortho-cresyl and non-ortho-cresyl isomers:

- CAS No 78-30-8 Tricresyl phosphate (containing o-o-o, o-o-m, o-o-p, o-m-m, o-m-p, o-p-p isomers);
- CAS No 78-32-0 Tricresyl phosphate (containing m-m-m, m-m-p, m-p-p, p-p-p isomers).

TCP is a compound with a toxicity typical of the organophosphorus compounds. Human toxicity to organophosphorus (OP) compounds has been known since at least 1899, when neurotoxicity to phosphocreosole (then used in the treatment of tuberculosis) was reported[49]. The study of OP toxicity is extensive, and generally characterised by a toxicity of inhibition of the esterase enzymes, most particularly cholinesterases[50] and neurotoxic esterases[51]. The mechanism of effect is phosphorylation[52].

Signs of low level intoxication include headache, vertigo, general weakness, drowsiness, lethargy, difficulty in concentration, slurred speech, confusion, emotional lability and hypothermia[53]. The reversibility of such effects has been questioned[54].

Signs of poisoning are usually foreshadowed by the development of early symptoms related to acetylcholine overflow and include salivation, lacrimation, conjunctivitis, visual impairment, nausea and vomiting, abdominal pains and cramps, diarrhoea, parasympathomimetic effects on heart and circulation, fasciculations and muscle twitches[55]. This is the basic site of inhibition for all OP molecules[56,57].

A second reaction with certain OPs (including TCP) leads to further neurotoxic and neuropathological changes. This is inhibition of neurotoxic esterases (NTE) which produces a progressive distal symmetrical sensori-motor mixed peripheral neuropathy, called organophosphorus induced delayed neurotoxicity (OPIDN)[57,58]. The mechanism of toxicity is now fairly well understood, as indeed are the organophosphorus structures which are predicted to cause OPIDN[59].

OPIDN has a severe pathology. It is quite likely that such a severe condition would be presaged with a range of clinical and pre-clinical signs and symptoms. These have been reported extensively, and an "intermediate syndrome" was defined in 1987[60].

More recently, chronic exposure to organophosphates has been associated with a range of neurological and neuropsychological effects[61,62,63,64,65]. Such symptoms (mainly neurological and neurobehavioural symptoms) may also be seen in exposed individuals who have been sufficiently fortunate in not having exposures that were excessive enough in intensity or duration to lead to clinical disease.

A distinct condition - chronic organo-phosphate neuropsychological disorder (COPIND) has been described, of neurological and neuropsychological symptoms[66]. These include:

- diffuse neuropsychological symptoms (headaches, mental fatigue, depression, anxiety, irritability);
- reduced concentration and impaired vigilance;
- reduced information processing and psychomotor speed;
- memory deficit and linguistic disturbances;

COPIND may be seen in exposed individuals either following single or short term exposures leading to signs of toxicity[62], or long term low level repeated exposure with (often) no apparent signs of exposure[64]. The basic mechanism of effect is not known, although it is not believed to be related to the esterase inhibition properties of organo-phosphorus compounds. It is also not known if these symptoms are permanent.

In addition, since the introduction and extensive use of synthetic organo-phosphorus compounds in agriculture and industry half a century ago, many studies have reported long-term, persistent, chronic neurotoxicity symptoms in individuals as a result of acute exposure to high doses that cause acute cholinergic toxicity, or from long-term, low-level, subclinical doses of these chemicals[67,68,69]. The neuronal disorder that results from organophosphorus ester-induced chronic neurotoxicity (OPICN), which leads to long-term neurological and neuro-behavioral deficits and has recently been linked to the effects being seen in aircrew despite OP exposures being too low to cause OPIDN[70].

Furthermore, OPICN induced by low-level inhalation of organophosphates present in jet engine lubricating oils and the hydraulic fluids of aircraft could explain the long-term neurological deficits consistently reported by crewmembers and passengers, although organophosphate levels may have been too low to produce OPIDN[71].

While the description above relates to the general toxicity of OPs, they are characteristic of exposure to tricresyl phosphate. The ten isomers that make up TCP are toxicologically different, and it is well established that the ortho containing isomers are the most toxic[72,73,74]. Of the ten isomers of TCP, six contain at least one ortho-cresyl group: three mono-ortho (MOCP) isomers, two di-ortho (DOCP) isomers and tri-orthocresyl phosphate (TOCP). Other, similar ortho-containing chemicals, such as the xylenols and phenolics, are also present in commercial TCP formulations in small amounts. Manufacturers of TCP have reduced the levels of ortho-cresyl and ortho-ethylphenyl isomers to reduce the potential for neurotoxicity of products containing TCP[20]. How much these refinements had removed the toxic impurities outlined above is not known. Indeed, toxicity was still being detected in commercially available products in 1988[75], and questions have been raised about the lack consistency between stated ingredient data and actual amounts of toxic isomers present in commercial formulations, and their impact on exposed individuals[17].

In evidence to the Australian Senate Aviation Inquiry in 1999, Mobil USA noted that Mobil Jet Oil II contains less than 5 ppb (0.005 ppm) TOCP[76]. This is an impressively low amount, and suggests that the neurotoxic potential from a chemical containing such a low level would be vanishingly small.

However, concentrations from other neurotoxic ingredients are not so readily available. In the Mobil USA evidence to the Australian Senate Aviation Inquiry, it became apparent that DOCPs were present in TCP at a concentration of 6 ppm, and MOCPs were present at a concentration of 3070 ppm. As these ingredients are present in higher concentrations than TOCP, and have a significantly higher toxicity than TOCP, it is suggested that a statement of low TOCP content is misleading as it underestimates the toxicity of the –OCP ingredients by a factor of 30,000 (see Table 4)[77].

Tricresyl phosphate will also contain mixed esters of orthophosphoric acid with different cresyl radicals, of the mono- and di-cresyl types[71]. The important issue with this data is that the level of all orthocresyl phosphates should impact on the regulatory classification of materials containing TCP.

## Monitoring studies

Most studies that have been carried out to measure atmospheric contamination in aircraft

**Table 4.** Tricresyl Phosphate: Toxicity of Isomers

| Isomer | Concentration (ppm) | Relative Toxicity | Equivalent Toxicity |
|---|---|---|---|
| TOCP | 0.005 | 1 | × 1 |
| DOCP | 6 | 5 | × 30 |
| MOCP | 3070 | 10 | × 30700 |
| | | **Total** | **× 30731** |

by engine oil leaks or hydraulic fluids are sufficiently flawed on procedural and methodological grounds as to render their conclusions invalid[78]. For example, they feature:

- an inability to collect poorly volatile contaminants
- inadequate sample collection times;
- inadequate sample volume;
- inappropriate storage of samples;
- no chain of custody;
- not taking account of altitude;
- studies were conducted on the ground (sometimes with airplane doors open).

Better techniques are required for capture and analysis of airborne contaminants of gas, vapour, mist and particulate types in aircraft leak incidents[79].

Further, no monitoring has occurred during an oil leak[10].

**Numbers of leaks**

There is a spectrum of defects and malfunctions in an airplane engine ranging from the trivial, to the serious, to the catastrophic. As trivial malfunctions can escalate into serious events, it is necessary to ensure that all types of malfunctions are identified, investigated and rectified.

The aviation industry itself acknowledges that air quality exposure events are primarily due to oil leaking into the air supply. All parties acknowledge that a problem exists, and has existed for a long time[80,81]. However, they then paradoxically deny that leaks are a serious matter, suggesting that it is not it is an air safety issue, rather an OHS, general health or comfort issue[82]. Regulatory agencies indicate that "serious impairment" includes the loss of crew's ability to see flight deck instrumentation or perform expected flight duties. However, they also suggest this excludes purely psychological aspects of the concern of odours, and concerns about long-term exposure.

When a leak occurs, it may be dismissed by the pilot as being a nuisance, in that it appears to have no apparent effect. Or it may be considered minor and reported within the company and fixed without record (anecdotally, some pilots report leak events to ground crew verbally or unofficially, for example, on scrap paper or even cocktail napkins). In this, there is inappropriate subjective interpretation of the terms "undue discomfort" and "harmful or hazardous levels of gases or vapours" specified in aviation regulations, and this interpretation errs on the side of convenience. Or a record may be made, but not considered sufficiently serious to report to aviation regulators, either voluntarily or as part of mandatory requirements. Lastly, as aviation regulations impose strict guidelines on how aircraft defects are defined, must be reported, investigated and dealt with, some leaks may actually be reported to aviation regulators[83]. These reports tend to cover the serious problems, but not always so. However, with substantial under-reporting and a culture of complacency between operators and regulators, no aviation regulatory authority can honestly consider that the reports they receive from the industry represent anything other than a very small tip of a very large iceberg of leak events.

From review of available sources and reported and accessible information, it is apparent that only a small fraction of the known incidents are reported[83]. Table 5 shows an analysis of various voluntary and mandatory sourced collections of leak events (taken from[83]).

Some of the more significant data includes:

- One BAe 146 operator reports oil/fumes every 66 flights in 1992 reducing to every 131 flights in 1999; and 775 mandatory aircraft technical log reports in two and a half years[84].
- The British Airline Pilots Association (BALPA) survey of B757 pilots showed that 106 pilots reported in excess of 1667 exposure events mostly thought to be associated with oil contamination of the air supply[85].
- FAA Service Difficulty Reports Search (SDRS) - 8268 cases of smell, fume, odour, gas, toxic fume, or toxic gas from 1986 to 2000[86].

- o 760 reports of contamination at one US airline on the MD80 aircraft from 1989 - 1998[87].
- o BAe 146 odour occurrence report - 791 optional reports[88].
- o BAe reports from aircraft operators - 439 reports from 1986 to 2000, including 212 from one operator in over three years[89].

Although the data in Table 5 is unlikely to be complete, it is not possible to conclude that leak rates are so low that they should be dismissed.

In conclusion, evidence is available that suggests that there are a substantial number of leak incidents on airplanes, especially on certain models of aircraft. Many of these leaks go unreported to aircraft operators. Of those leak incidents that are reported to aircraft operators, many are not reported to regulatory authorities. Of those leak incidents that are

**Table 5.** Rates of Aircraft Smoke/Fume/Oil and Other Fluid Contamination

| Report type/ Country | Year(s) | Aircraft type | Number of reports | Comment |
|---|---|---|---|---|
| BALPA/UK | 2001 | B757 | 1667+ | 1667+ reports of smoke or fumes mostly thought to be from oil in air conditioning system |
| MOR*/ UK CAA | 1988-<br>Jan 2004 | B757 | 104+ | "Smoke and or fumes" – oil/smoke/fumes/de-icing/ hydraulic fluid<br>• 16 reports 1988 - 1998<br>• 88 reports 1999 – January 2004 |
| MOR*/ UK CAA | 1985-2003 | BAe 146 | 85+ | "Smoke and or fumes" – oil/smoke/fumes/de-icing/hydraulic fluid<br>• 11 reports 1985 - 1995<br>• 68 reports 1996 – 2003 |
| Other UK data | 1998-2004 | B757 | 47 | Reports sent via email or airline reports (but not on CAA data base) |
| Other UK data | 2002-2004 | BAe 146 | 23 | Airline reports not on CAA data base |
| UK CAA | 1989-1999 | 5 Jet types | 128 | Smoke/gas fumes (non-mandatory)<br>• 1 event every 22,265 flights<br>• B757 (21), BAe 146 (17) |
| UK AAIB | 2000-2002 | BAe 146/ B757 | 19 | Smoke/fumes incidents<br>• B757 -10<br>• BAe 146 - 9+ |
| BAe/UK | 1985-2000 | BAe 146 | 439 | • 36 operators report 227 cases of contaminated air -1985 - 2000<br>• 1 operator reports 212 cases of tainted cabin air 1996 -1999 |
| Aircraft Defect Reports/ Australia | 1991-1999 | BAe 146 | 775 | Mandatory reports in aircraft technical log. Number of reports<br>• 1992 – 418 reports = 1 every 66 flights<br>• 1997 - 189<br>• 1999 (6 months)- 168 reports = 1 every 131 flights |

Table 5 continued..

| Report type/ Country | Year(s) | Aircraft type | Number of reports | Comment |
|---|---|---|---|---|
| Odour Occurrence Reports/ Australia | 1991-2000 | BAe 146 | 791 | Optional (voluntary) BAe 146 odour occurrence reports. |
| CASA/ Australia | 1996-2002 | BAe 146 | 22 | Examples of oil seal bearing defects, fumes and crew impairment |
| ATSB/ Australia | 1991-2002 | BAe 146 | 32 | Oil/hydraulic fume - smoke or odour incidents |
| FAA/US | 1986-2000 | Various | 8268 | SDRS - Smell, fume, odour, gas, toxic fume, or toxic gas |
| AFA/US | 1989-1998 | MD80 | 760 | 900 reports at 1 airline - (73% on MD80) |
| FAA/US | 1989-1999 | Various | 167 | Accidents and Incidents Data Systems (AIDS)<br>• 23 (14%) - Air quality events connected to air contaminants in ventilation system – 1 every 3,590,000 departures<br>• 60 events of ventilation toxic contaminant events + smoke in cockpit/cabin - (1978-1999) |
| NTSB/US | 1990-2000 | Jet transport | 5 | Smoke/fumes |
| TSB/Sweden | 1999 | BAe 146 | 1 | All crew members "temporarily effected by probably polluted cabin air" |

reported to regulatory authorities, not all are added to relevant databases. Ultimately, only a very small number of leak incidents are investigated fully.

Further, as already noted, this information must also be evaluated against substantial under reporting. The information available clearly varies greatly dependent on the source. It can be seen that there are a substantial number of reports on particular types of aircraft.

### Effects of hazardous chemicals leaks on crew

Where exposure may be to high levels of airborne contaminants, it is not unreasonable for signs of irritancy and discomfort to be observed. Similarly, it is not unreasonable to consider that a person exposed to a chemical that contains 1% of a sensitiser and 3% of a neurotoxicant might show signs of irritancy and neurotoxicity. These symptoms are often reported in air crew who may be exposed to aircraft fluids.

The earliest case found in the literature of toxicity following jet oil exposure and adverse health problems in air crew was reported in 1977[90]. A previously healthy member of an aircraft flight crew was acutely incapacitated during flight with neurological impairment and gastrointestinal distress. His clinical status returned to normal within a day. The aetiology of his symptoms was related to an inhalation exposure to aerosolised or vapourised synthetic lubricating oil arising from a jet engine of his aircraft. This paper notes that analysis of two samples of military specification oil contained less than 3.5 ppm and 140-175 ppm of TOCP. Analysis of oils from commercial airlines were found to contain 3.5

to 56 ppm of TOCP[99]. This data confirms high levels of TOCP in early oils. Bearing in mind Table 4 above show that the concentrations of MOCP and DOCP isomers are orders of magnitude above the TOCP concentrations, the true –OCP concentration of exposures to these oils is severely underestimated by being expressed in TOCP concentrations alone.

Other studies of exposures in aircraft exist in the literature, including a 1983 study of eighty nine cases of smoke/fumes in the cockpit in the US Air Force[91], a 1983 study of Boeing 747 flight attendants in the USA (this paper linked symptoms to ozone)[92], a 1990 study of aerospace workers[93], and a 1998 study of BAe 146 flight crews in Canada over a four-month period[34]. A recent report of seven case studies considered representative of the common symptoms of irritancy and toxicity described similar symptoms[35], and a follow up survey by the same research group reported similar findings in a larger group of fifty crew respondents[94]. Two union based studies in pilots provide additional data[85,95].

These studies investigated different exposures and situations, and the range of symptoms in these studies was quite broad, affecting many body systems. However, there are common themes in symptom clusters in these studies, as shown in Table 6.

**Table 6.** Studies Reporting Signs and Symptoms in Aircrew from Jet Oil Leaks

| **Symptom Cluster** | **Sign or Symptom** — **Reference** / **Number of cases** | **100** **89** | **101** **248** | **102** **53** | **38** **112** | **39** **7** | **103** **50** | **104** **21** | **94** **106** |
|---|---|---|---|---|---|---|---|---|---|
| Loss of conscious-ness/ Inability to function | Fainting/loss of consciousness/grey out | 4% | 4% | | | 3/7 | 14% | | |
| | Respiratory distress, shortness of breath, respiration requiring oxygen | | 73% | | 2% | 4/7 | 62% | 26% | 4% |
| Symptoms of direct irritation to eye, airways or skin | Irritation of eyes, nose and throat | | | | | 7/7 | | 32% | 37% |
| | Eye irritation, eye pain | 35% | 74% | 57% | 24% | 4/7 | 76% | | |
| Respiratory symptoms secondary to irritation | Sinus congestion | 35% | 54% | | 5% | 2/7 | | | |
| | Nose bleed | | 17% | | | 1/7 | 4% | | |
| | Throat irritation, burning throat, gagging and coughing | 2% | 64% | 57% | 43% | 2/7 | 76% | | |
| | Cough | | 69% | | | 2/7 | 12% | | |
| | Difficulty in breathing, chest tightness | | 68% | | | 3/7 | 62% | | |
| | Loss of voice | | 35% | | | 1/7 | | | |
| Skin symptoms secondary to irritation | Rashes, blisters (on uncovered body parts) | | | 36% | | 4/7 | 48% | 16% | 8% |

Table 6 continued..

| Symptom Cluster | Sign or Symptom / Reference Number of cases | 100<br>89 | 101<br>248 | 102<br>53 | 38<br>112 | 39<br>7 | 103<br>50 | 104<br>21 | 94<br>106 |
|---|---|---|---|---|---|---|---|---|---|
| Gastro-intestinal symptoms | Nausea, vomiting, gastrointestinal symptoms | 26% | 23% | 15% | 8% | 6/7 | 58% | 5% | 15% |
| | Abdominal spasms/cramps/ diarrhoea | 26% | | | | 3/7 | 20% | 5% | 16% |
| Neurotoxic symptoms | Blurred vision, loss of visual acuity | 11% | 13% | | 1% | 4/7 | 50% | 5% | 4% |
| | Shaking/tremors/tingling | 9% | | | 3% | 3/7 | 40% | | |
| | Numbness (fingers, lips, limbs), loss of sensation | | | 8% | 2% | 4/7 | | 10% | 12% |
| Neuro-logical symptoms related to basal nervous system function | Trouble thinking or counting, word blindness, confusion, coordination problems | 26% | 39% | 42% | | 6/7 | 58% | 21% | 22% |
| | Memory loss, memory impairment, forgetfulness | | | 42% | | 7/7 | 66% | 26% | 11% |
| Cognitive/ neuropsycho-logical symptoms related to higher nervous system function | Disorientation | 26% | | | 15% | 4/7 | | 16% | 8% |
| | Dizziness/loss of balance | 47% | | | 6% | 4/7 | 72% | 16% | 3% |
| | Light-headed, feeling faint or intoxicated | 35% | 54% | | 32% | 7/7 | | 21% | 33% |
| Nonspecific general symptoms | Chest pains | 7% | 81% | | 6% | 2/7 | 22% | | |
| | Severe headache, head pressure | 25% | 52% | | 26% | 7/7 | 86% | 21% | 33% |
| | Fatigue, exhaustion | | | | | 7/7 | 62% | 21% | 30% |
| | Chemical sensitivity | | | 32% | | 4/7 | 72% | 26% | 10% |
| | Immune system effects | | | | | | | 21% | 3% |
| | Behaviour modified, depression, irritability | 26% | 20% | 60% | | 4/7 | 40% | | 27% |
| | Change in urine | | 3% | 6% | | | 4% | | |
| | Joint pain, muscle weakness, muscle cramps | | 29% | | | 2/7 | 38% | 5% | 30% |

While this Table shows a long list of symptoms, it is possible to characterise many symptoms more consistently. For example, different papers report dizziness or loss of balance or light-headedness or feeling faint or feeling intoxicated or disorientation. It would be incorrect to regard such symptoms as being entirely different from each other – they point to a basic neuropsychological dysfunction affecting balance. But rather than dismissing such symptoms as being multitudinous and variable,[96,97] it may be more appropriate to re-categorise symptoms with clearer definitions, so that the artificial distinctions between symptom reporting can be clarified, and a shorter list of "symptom clusters" be developed (as shown in the first column of Table 6).

**The cockpit/cabin environment**

The cockpit or cabin of an aircraft is a unique environment. It is a specialised working environment for the air crew that cannot (indeed, must not) be equated with workplaces at sea level, or workplaces where specialised ventilation and escape are possible[98].

The process of aircraft pressurisation means that the working environment is hypoxic. Flying crew are required to conduct complex operations requiring high order cognitive skills and coordination expertise. Flight attendants may be required to direct emergency procedures requiring composure and confidence. Anything that may have an impact on the delivery of these tasks can have serious consequences.

A lowered level of oxygen in turn may have an impact on the emergence of adverse health problems to toxic exposures.

For these reasons, the application of conventional occupational health and safety procedures to this specialised environment are inappropriate. Examples of these include:

- ventilation rates for buildings;
- absence of safety information, risk assessment and advice on control of risks;
- the use of permissible exposure standards. A common assertion by aviation companies is that "all chemical exposures are within acceptable TWA exposure standards". However, these[99]:
  - apply only to the specified chemical;
  - protect 'nearly all workers', not all workers;
  - cannot protect sensitive workers;
  - are NOT no effect levels;
  - poorly consider periods of peak exposure;
  - ignore skin exposure;
  - ignore exposures to other contaminants;
  - must not be applied to people other than workers (ambient standards for the general public are often 100-1000 times lower);
  - must not be applied to unusual environments (for example, the cabin of an airplane)[100];
- extenuating circumstances on board aircraft (including humidity and cabin pressure) have not been studied to the extent that a suitable exposure standard can be identified that incorporates these factors or identifies interactions between factors[101];
- it is incorrect to assume the exposure standard for TOCP as being "adequately protective" for a TCP containing mixture of TCP isomers as other ortho isomers (MOCPs, DOCPs) are at least 5-10 times more toxic than TOCP[102];
- procedures for assessing the risks of exposures to more than one chemical, that may act in synergy to produce toxicity (for example, carbon monoxide and lowered oxygen);
- under circumstances of exposure to mixtures of contaminants, levels may be well below recommended levels in currently accepted exposure standards - yet still generate complaints or signs and symptoms, because they act in synergy with other contaminants or because some standards may be outdated and not have incorporated more recent scientific and medical evidence[101];

Occupational exposure standards must also not be applied to non-workers, for example passengers.

### Combustion and pyrolysis processes

Further, an oil leak from an engine at high pressure and temperature may burn or pyrolise before it enters the cabin. This produces carbon-containing materials which, in the presence of energy and oxygen, produce the two oxides of carbon: Carbon dioxide ($CO_2$) and Carbon monoxide (CO). The first of these ($CO_2$) is produced in the presence of an abundance of oxygen, the second (CO), where stoichiometric concentrations of oxygen are lacking (usually in conditions of incomplete combustion). Both of these oxides are gases, one (Carbon monoxide) is quite toxic at low concentrations, causing toxic asphyxiation. Single or short term exposure to CO insufficient to cause asphyxiation produces headache, dizziness, and nausea; long term exposure can cause memory defects and central nervous system damage, among other effects[103].

Many combustion and pyrolysis products are toxic. The toxic asphyxiants, such as carbon monoxide, have already been introduced above. Some thermal degradation products, such as acrolein and formaldehyde are highly irritating. Others, such as oxides of nitrogen and phosgene, can produce delayed effects. Still others, such as particulate matter (for example, soot) can carry adsorbed gases deep into the respiratory tract where they may provoke a local reaction or be absorbed to produce systemic effects.

A leak of such an oil from an engine operating at altitude would see most of the oil pyrolise once it leaves the confined conditions of temperature and pressure operating in the engine. While it seems reasonable that any ingredients with suitable autoignition or degradation properties that allow such a transformation after release from the engine could be radically transformed, it is possible to speculate in only general terms about the cocktail of chemicals that could form. Presumably it would include carbon dioxide, carbon monoxide, partially burnt hydrocarbons (including irritating and toxic by-products, such as acrolein and other aldehydes, and TCP (which is stable at high temperatures). These contaminants will be in gas, vapour, mist and particulate forms. These contaminants could not be classified as being of low toxicity. The possible problems that might arise from exposure to such a cocktail cannot be dismissed without proper consideration.

### Aerotoxic syndrome

What emerges in the analysis of this information, is a pattern of symptoms related to local effects to exposure to an irritant, overlaid by development of systemic symptoms in a number of body systems, including nervous system, respiratory system, gastro-intestinal system, and possibly immune system and cardiovascular system. These symptoms may be expressed specifically to these symptoms, or may be seen more generally, such as headache, behavioural change or chronic fatigue.

The symptoms reported by exposed individuals as shown in Table 6 are sufficiently consistent to indicate the development of a discrete occupational health condition, and the term aerotoxic syndrome is introduced to describe it (Etymology: aero refers to aviation, toxic to toxicity of exposure and associated symptoms). Features of this syndrome are that it is associated with air crew exposure at altitude to atmospheric contaminants from engine oil or other aircraft fluids, temporarily juxtaposed by the development of a consistent symptomology including short-term skin, gastro-intestinal, respiratory and nervous system effects, and long-term central nervous and immunological effects (see Table 7).

This syndrome may be reversible following brief exposures, but features have emerged of a chronic syndrome following significant exposures[35,36,94].

More recent research has established a long term syndrome of toxicological[78,79], medical[104,105], respiratory[106], neuropsychological[107] and psychological[108] effects.

**Table 7.** Aerotoxic Syndrome: Short and Long Term Symptoms

| Short term exposure | Long term exposure |
|---|---|
| o **Neurotoxic symptoms:** blurred or tunnel vision, nystagmus, disorientation, shaking and tremors, loss of balance and vertigo, seizures, loss of consciousness, parathesias;<br>o **Neuropsychological or Psychotoxic symptoms:** memory impairment, headache, light-headedness, dizziness, confusion and feeling intoxicated;<br>o **Gastro-intestinal symptoms:** nausea, vomiting;<br>o **Respiratory symptoms:** cough, breathing difficulties (shortness of breath), tightness in chest, respiratory failure requiring oxygen;<br>o **Cardiovascular symptoms:** increased heart rate and palpitations;<br>o **Irritation** of eyes, nose and upper airways. | o **Neurotoxic symptoms:** numbness (fingers, lips, limbs), parathesias;<br>o **Neuropsychological or Psychotoxic symptoms:** memory impairment, forgetfulness, lack of co-ordination, severe headaches, dizziness, sleep disorders;<br>o **Gastro-intestinal symptoms:** salivation, nausea, vomiting, diarrhoea;<br>o **Respiratory symptoms:** breathing difficulties (shortness of breath), tightness in chest, respiratory failure, susceptibility to upper respiratory tract infections;<br>o **Cardiovascular symptoms:** chest pain, increased heart rate and palpitations;<br>o **Skin symptoms:** skin itching and rashes, skin blisters (on uncovered body parts), hair loss;<br>o **Irritation** of eyes, nose and upper airways;<br>o **Sensitivity:** signs of immunosupression, chemical sensitivity leading to acquired or multiple chemical sensitivity<br>o **General:** weakness and fatigue (leading to chronic fatigue), exhaustion, hot flashes, joint pain, muscle weakness and pain. |

## CONCLUSIONS

The presence of contaminants in flight decks and passenger cabins of commercial jet aircraft should be considered an air safety, occupational health and passenger health problem:

- Incidents involving leaks or engine oil and other aircraft materials into the passenger cabin of aircraft occur frequently and are "unofficially" recognised through service bulletins, defect statistics reports and other sources. The rates of occurrence of incidents are higher than the aviation industry cares to admit, and for some models of aircraft are significant. These need appropriate reporting, follow up investigations and health investigations for those exposed.
- The oils used in aircraft engines contain toxic ingredients which can cause irritation, sensitisation and neurotoxicity. This does not present a risk to crew or passengers as long as the oil stays in the engine. However, if the oil leaks out of the engine, contaminated bleed air may enter the air conditioning system and cabin air. Where these leaks cause crew or passenger discomfort, irritation or toxicity, this is a direct contravention of the US Federal Aviation Authority's and the European Joint Aviation Authorities' airworthiness standards for aircraft ventilation (FAR/JAR 25.831).
- As indicated by manufacturer information and industry documentation, aviation

materials such as jet oils and hydraulic fluids are hazardous and contain toxic ingredients. If such fluids leak into the air supply, cabin and flight deck, toxic exposures are possible. Presently, the aircraft manufacturers, airline operators and the aviation regulators deny that this is a significant problem.

- Leaks of oil and other fluids into aircraft may be considered of a nuisance type, but where they affect the health and performance of crew, or the health of passengers, this is to be considered a flight safety and health issue and must be given appropriate priority. The aviation industry presently denies that any problem exists.
- Pilots continue to fly when experiencing discomfort or irritation or symptoms of toxicity. There is a lack of understanding by pilots regarding the toxicity of the oil leaks, occupational health and safety (OHS) implications and the necessity to use oxygen. This is further compounded by airline health professionals who, when confronted with a pilot who has been exposed in a fume event and who is concerned about its consequences, have a poor understanding of the short and long-term medical issues that may arise and tend to be dismissive about their implications.
- Attempts by the industry to minimise this issue, such as acceptance of under-reporting of incidents, inadequate recognition of the extent of the problem, inadequate adherence/interpretation of the regulations, inadequate monitoring, inappropriate use of exposure standards and care provided to crew reporting problems, have perpetuated this problem.
- The health implications both short and long-term, following exposure to contaminants being reported by crew and passengers must be properly addressed. A syndrome of symptoms is emerging, called aerotoxic syndrome, suggesting these exposures are common and a sufficiently large enough group of affected individuals exists.
- Where contaminants impair the performance or affect the ability of pilots to fly planes, as has been reported for a number of incidents, this is a major safety problem. Where contaminants cause undue discomfort or even transient health effects in staff and passengers, this is a breach of FAR 25.831 and other regulations.

Statements by organisations in the aviation industry have attempted to deal with this problem reactively and somewhat flexibly, as evidence emerged:

- "There are no engine oil leaks".
- "Well, there may be some engine oil leaks, but they are very uncommon".
- "Well, there more a few engine oil leaks than we would like, but the oil is safe under normal conditions of use".
- "Well, the oil may contain hazardous ingredients, not at levels that it affects the health of crew".
- "The health problems being reported by our workers are not related to the leaks".
- "Well, if there are health problems, they are related to some other health condition".
- "Well, there may be a few health problems from exposure to oil leaks, but they are transient or mild, and are reversible".

As noted above, where contamination of air in flight deck and passenger cabin occurs, or where this is sufficient to cause symptoms of discomfort, fatigue, irritation or toxicity, this contravenes air quality provisions of Aviation Regulations, most notably FAR/JAR 25.831.

In fact, contaminants in the air of an occupational environment (specialised or not) should, under normal circumstances, alert management to a potential problem[98]. There is a nested hierarchy of factors which influence the genesis of aerotoxic syndrome, from design of aircraft, engines and oils, to operational aspects and organisational culture, through to injury and disease (see Figure 4).

However, much of the focus is at the lower levels of this hierarchy, with action basically

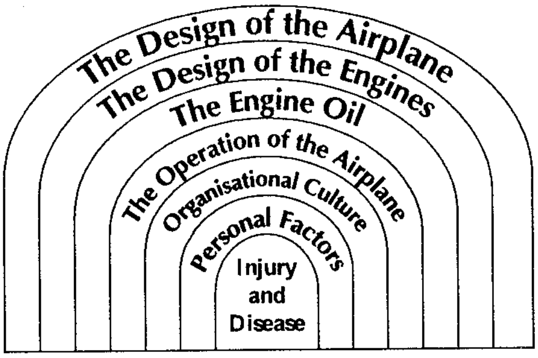



**Figure 4.** The Genesis of Aerotoxic Syndrome.

targeting affected workers. Better attention further up the hierarchy might be more useful.

Rather than just responding to a problem reactively, this needs action on a number of fronts:

1. Better designed aircraft, engines and APUs are needed that don't leak.
2. Better designed aircraft environmental systems are needed that do not rely on bleed air.
3. Better, more safer, chemical products are needed to be used in this industry.
4. Standard, open, non-retributive systems for the reporting of leaks are needed.
5. Organisations in this industry need to acknowledge their occupational health and safety responsibilities as mandated by legislation and should develop and implement appropriate systems that allow those responsibilities to be met (because their existing systems don't).
6. All reports of leaks should be recorded and all such records should be openly available.
7. Risk assessments of exposures are needed that are inclusive, not exclusive, of workers and passengers.
8. Better health systems are needed that treat affected employees with sympathy and respect and not contempt.
9. Better models are needed for monitoring, diagnosis, treatment, rehabilitation and compensation of affected workers. This is urgently needed for the legacy that already exists of pilots and flight attendants who have been affected, forced out of the industry and have been in the wilderness ever since.
10. And of course, research. Research is needed into better engineering systems, less toxic chemicals, better diagnosis, better treatment, better risk assessments and representative epidemiological surveys of employees in the industry. Proper medical and scientific research needs to be undertaken in order to help airline management and crew to better understand both the short-term and long-term medical effects of being subjected to air contamination. This research must be independently funded and objectively reported. At best, it must be free of bias from vested interests that are so skilful at obscuring the issue.

Over the past fifty years, the concept of duty of care has emerged as one of the most important legal responsibilities for employers. In the workplace, the duty of care of an employer to its workers has been crystallised into OHS legislation. Aviation safety is something that a person outside of the industry would understand to cover all aspects of safety, including the health and safety of its workers. However, this does not seem to be how all industry insiders see it. Many in the industry see aviation safety as being about making sure the planes keep flying. Both the aviation regulators and the airlines themselves think that OHS is not their business - which is strange, because if they do not look after the health and safety of workers in the industry, then who will?

## ACKNOWLEDGEMENTS

I gratefully acknowledge the support and ongoing contributions of my colleagues Dr. Jean-Christophe Balouet, Ms. Susan Michaelis and Captain Tristan Lorraine in the development of this paper.

## REFERENCES


1. Balouet, J.-C., Kerguelen, M., and Winder, C. 2001, Toxicology, 164, 164.
2. Spengler, J., Burge, H., Dumyahn, T., Muilenburg, M., and Forester, D. 1997, Environmental Survey on Aircraft and Ground Based Commercial Transportation Vehicles. Harvard School of Public Health, Boston.
3. ASHRAE. 1990, Ventilation for Acceptable Indoor Air Quality: ANSI/ASHRAE Standard 62. American Society of Heating, Refrigerating and Air Conditioning Engineers, Atlanta.
4. O'Donnell, A., Donnini, G., and Nguyen, V. 1991, ASHRAE J. 42.
5. CAT, AMA. 1953, Aviation Toxicology: An Introduction to the Subject and a Handbook of Data. Committee of Aviation Toxicology, Aero Medical Association, Blakiston.
6. Tupper, C. R. 1989, Aviat., Space Environm. Med., 60, 73.
7. Smith, P, W., Lacefield, D. J., and Crane, C. R. 1970, Aerosp. Med., 41, 760.
8. Trimble, E. J. 1996, Toxicology 115, 41.
9. ASHRAE. 1999, Air Quality Within Commercial Aircraft: ASHRAE Standard 161. American Society for Heating, Refrigeration, Airconditioning and Energy, Atlanta.
10. Nagda, N. L., and Rector, H. E. 2003, Indoor Air, 13, 292.
11. Balouet, J.-C., Kerguelen, M., and Winder, C. 2001, Toxicology, 164, 164.
12. FAA. 1965, Federal Air Regulations, Transport Category Airplanes, Airworthiness Standards, Section 25.831. Washington: US Federal Aviation Authority.
13. Healy, C. E., Nair, R. S., Ribelin, W. E., and Bechtel, C. L. 1992, Am. Indust. Hyg. Assoc., J. 53, 175-180.
14. Carpenter, H. M, Jenden, D. J., Shulman, N. R., and Tureman, J. R. 1959, AMA Arch. Industr. Health, 20, 234-252.
15. Mattie, D. R., Hoeflich, T. J., Jones, C. E., Horton, M. L., Whitmire, R. E., Godin, C. S., Femming, C. D., and Andesen, M. E. 1993, Toxicol. Indust. Health, 9, 995.
16. Hewstone, R. K. 1994, Sci. Total Environ., 156, 243.
17. Winder, C., Balouet, J.-C. 2002, Environ. Res., 2002; 89, 146.
18. ASTM. 2003, ASTM D 1655-04a Standard Specification for Aviation Turbine Fuels. American Society for Testing and Materials. ASTM International, West Conshohocken, PA.
19. MOD. 2005, Defence Standard 91-91: Turbine Fuel, Aviation Kerosine Type, Jet A-1, NATO Code: F-35. UK Ministry of Defence, London.
20. Mackerer, C. R., Barth, M. L., Krueger, A. J., Chawla, B., Roy, T. A. 1999, J. Toxicol. Environm. Health, 57, 293.
21. White, R. D. 1999, Drug Chem. Toxicol., 22, 145.
22. Ritchie, G., Still, K., Rossi, J., III, Bekkedal, M., Bob, A., and Arfsten, D. 2003, J. Toxicol. Environm. Health B, Crit. Rev., 6, 357.
23. Schlaifer, R., and Heron, S. 1950, Development of Aircraft Engines and Fuels. Harvard University Press, Boston.
24. Stewart, P. A., Lee, J. S., Marano, D. E., Spirtas, R., Forbes, C. D., and Blair, A. 1991, Brit. J. Indust. Med., 48, 531.
25. Ritchie, G. D, Still, K. R., Alexander, W. K., Nordholm, A. F., Wilson, C. L., Rossi, J.III, and Mattie, D. R. 2001, J. Toxicol. Environm. Health B: Crit. Rev. 4, 223.
26. Kanikkannan, N., Burton, S., Patel, R., Jackson, T., Shaik, M. S., and Singh, M. 2001, Toxicol. Lett., 119, 133.
27. Harris, D. T., Sakiestewa, D., Titone, D., Robledo, R. F., Young, R. S., and Witten, M. 2001, Toxicol. Indust. Health, 16, 261.
28. Kabbur, M. B., Rogers, J. V., Gunasekar, P. G., Garett, C. M., Geiss, K. T., Brinkley, W. W., and McDougal, J. N. 2001, Toxicol. Appl. Pharmacol., 175, 83.
29. Ru, R. H., Mitchell, C. S., Kay, G. G., and Risby, T. H. 2004, Aviat., Space Environm. Med., 75, 49.
30. Monteiro-Riviere, N. A., Inman, A. O., and Riviere, J. E. 2004, Toxicol. Appl. Pharmacol., 195, 339.

31. Tesseraux, I. 2004, Toxicol. Lett., 149, 295.
32. Hobson, D. W., D'Addario, A. P., Bruner, R. H., and Uddin, D. E. 1986, Fund. Appl. Toxicol., 6, 339.
33. Geiss, K. T., and Frazier, J. M. 2001, Sci. Total Environ., 274, 209.
34. van Netten, C. 1998, Appl. Occup. Environm. Hyg. 13, 733.
35. Balouet, J.-C., and Winder, C. 1999, American Society of Testing and Materials (ASTM) Symposium on Air Quality and Comfort in Airliner Cabins New Orleans, USA.
36. Winder, C., and Balouet, J.-C. 2001, J. Occup. Health Safety – Aust. NZ. 17, 471.
37. BASI. 1997, British Aerospace plc BAE 146-300 10 July 1997: Preliminary Report 9702276. Canberra: Bureau of Air Safety Inspection, 23 October 1997.
38. Michaelis, S. 2002, J. Occup. Health Safety – Aust. NZ 18, 291.
39. Buck, W. H., Mobil Oil Corporation, 27 July 1999.
40. ACC. 2001, High Production Volume Chemical Submission, Substituted Diphenylamines. American Chemistry Council, Arlington VA, 18 December 2001. At: http://www.epa.gov.gov/chemrtk/subdiapha/c13378.pdf
41. Miyazaki, K., Kawai, S., Sasayama, T., Iseki, K., and Arita, T. 1987, Yakuzaigaku (Arch Pract. Pharm.) 47: 17 (English abstract).
42. IPCS. 1998, CICAD No 9: N-Phenyl-1-naphthylamine. International Programme on Chemical Safety, Geneva.
43. Boman, A., Hagelthorn, G., Jeansson, I., Karlberg, A.-T., Rystedt, I., and Wahlberg, J. E. 1980, Cont. Dermat. 6, 299.
44. Kalimo, K., Jolanki, R., Estlander, T, and Kanerva, L. 1989, Contact Dermatitis 20, 151.
45. Carmichael, A. J., and Foulds, I. S. 1990, Cont. Dermat. 22, 298.
46. Wang, H.-W., Wang, D., and Dzeng, R.-W. 1984, Cancer Res. 44, 3098.
47. Jarvholm, B., and Lavenius, B. 1981, Scand. J. Work Environ. Health 7, 179.
48. US NTP. 1988, Toxicology and Carcinogenesis Studies of N-Phenyl-2-naphthylamine in F344/N Rats and B6C3F1 Mice (feed studies). US National Toxicology Program. http://ntp-server.niehs.nih.gov/htdocs/lt-studies/tr333.html
49. Echobion, D. J. 1993, In: Echobion, D. J., and Joy, R. M., editors. Pesticides and Neurological Diseases, second edition. CRC Press, Boca Raton.
50. Aldridge, W. N. 1954, Biochem., J. 56, 185.
51. Johnson, M. K. 1975, Biochem Pharmacol., 24, 797.
52. Earl, C. J., and Thompson, R. H. S. 1952, Brit. J. Pharmacol., 7, 685.
53. Eyer, P. 1995, Hum. Exp. Toxicol., 14, 857.
54. Kilburn, K. H. Environm. Epidemiol. Toxicol., 1999, 1, 153.
55. Minton, N. A., Murray, V. S. G., Med. Toxicol., 1988, 3, 350.
56. Johnson, M. K. 1975, Arch. Toxicol., 34, 259.
57. Metcalf, R. L. 1982, Neurotoxicology, 3, 269.
58. Baron, R. L. 1981, Ann. Rev. Entolomol., 26, 29.
59. Johnson, M. K. 1990, Toxicol. Appl. Pharmacol., 102, 385.
60. Senanayake, N., and Karalliede, L. 1987, N. Engl. J. Med., 316, 761.
61. Dille, J. R., and Smith, P. W. 1964, Aerosp. Med., 35, 475.
62. Savage, E. P., Keefe, T. F., Mounce, L. M., Heaton, J. A., and Burcar, P. J. 1988, Arch. Environm Health, 43, 38-45.
63. Rosenstock, L., Keifer, M., Daniell, W. E., McConnell, R., and Claypoole, K. 1991, Lancet, 338, 225-227.
64. Steenland, M. 1996, Brit. Med. J., 312, 1311.
65. UK DoH. 1999, Toxicology of OPs and the mechanisms involved. Chapter 5 in: Organophosphates. Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment. UK Department of Health, HMSO, London.

66. Jamal, G. A. 1997, Adv. Drug React. Toxicol. Rev., 16, 133.
67. Gershon, S., and Shaw F. B. 1961, Lancet; 1, 1371.
68. Metcalf, D. R., Holmes, J. H. 1969, Ann. N. Y. Acad. Sci., 160, 357.
69. Callender, T. J., Morrow, L., Subramanian, K. 1994, J. Toxicol. Environm. Health 41, 275.
70. Abou-Donia, M. B. 2004, Arch. Environm. Health 58, 484.
71. Abou-Donia, M., Contaminated Air Protection Conference: Proceedings of a Conference held at Imperial College, London, 20-21 April 2005, Winder, C., editor. BALPA/UNSW, Sydney, 59.
72. Smith, M. I., Elvove, I., Valaer, P. J., Frazier, W. H., and Mallory, G. E. 1930, US Pub. Health Rep., 45, 1703.
73. Henschler, D., and Bayer, H. H. 1958, Arch. Experim. Pathol. Pharmacol., 233, 512.
74. Henschler, D. 1958, Klin. Wochens. 36, 663.
75. Daughtrey, W., Biles, R., Jortner, B., and Erlich, M. 1996, Fundam. Appl. Toxicol., 32, 244.
76. Mackerer, C. R., and Ladov, E. N. 1999, Mobil USA Submission to the Australian Senate Inquiry into Air Safety – BAe 146 Cabin Air Quality.
77. Winder, C., and Balouet, J.-C. 2001, Toxicology, 164, 215.
78. Winder, C., and Michaelis, S. 2005, In: Air Quality in Airplane Cabins and Similar Enclosed Spaces, Hocking, M., editor. Handbk. Environm. Chem. 4 Part H, 212.
79. van Netten, C. 2005, In: Air Quality in Airplane Cabins and Similar Enclosed Spaces, Hocking, M., editor. Handbk. Environm. Chem., 4 Part H, 193.
80. BAe. Service Bulletin SB 21-150: Air Conditioning - To Inspect Engine Oil Seals, APU and ECS Jet Pump and Air Conditioning Pack for Signs of Oil Contamination. British Aerospace Systems, Hatfield, 20 March 2001.
81. Best, R., Michaelis, S. 2005, In: Air Quality in Airplane Cabins and Similar Enclosed Spaces, Hocking, M., editor. Handbk. Environm. Chem., 4 Part H, 249.
82. Hamilton, T. 2004, Letter to British Airline Pilots Association (BALPA) Aircraft Environment Task Group. UK Civil Aviation Authority, London.
83. Winder, C., and Michaelis, S. 2005, In: Air Quality in Airplane Cabins and Similar Enclosed Spaces, Hocking, M., editor. Handbk. Environm. Chem. 4 Part H, 221.
84. Ansett Australia. 2000, Submission and Evidence by Ansett Australia to the Australian Senate Inquiry into Air Safety (1999-2000) BAe 146 Cabin Air Quality. Parliament of Australia, Canberra.
85. Michaelis, S. 2003, J. Occup. Health Safety – Aust. NZ 19, 253.
86. AAFA. 2003, Aircraft Air Quality: What's Wrong with it and What Needs to be Done. American Association of Flight Attendants: Submission to the Aviation subcommittee of the Transportation and Infrastructure Committee of the US House of Representatives, Washington, June, 2003.
87. O'Brien, K. BAe 146 Aircraft: Q398. Hansard, Australian Parliament House, Canberra, 2 December 2002.
88. FAAA. 1999, Submission and Evidence by Ansett Australia by the Flight Attendant Association of Australia to the Australian Senate Inquiry into Air Safety (1999-2000) BAe 146 Cabin Air Quality. Parliament of Australia, Canberra.
89. SHK. 1999, Report RL 2001:41e: Incident Onboard Aircraft SE-DRE During Flight Between Stockholm and Malmö, M County, Sweden, on 12 November 1999. Swedish Statens Haverkommission (Swedish Board of Accident Investigation), Stockholm.
90. Montgomery, M. R., Wier, G. T., Zieve, F. J., and Anders, M. W. 1977, Clini. Toxicol., 11, 423.
91. Rayman R. B., and McNaughton G. B. 1983, Aviat., Space Environm. Med., 67, 738.
92. Tashkin, D. P., Coulson, A. H, Simmons, M. S., and Spivey, G. H. 1983, Arch. Occup. Environment. Health, 52, 117.
93. Sparks, P. S., Simon, G. E., Katon, W. J., Altman, L. C., Ayars, G. H., and Johnson, R. L. 1990, West. J. Med. 158, 28.

94. Winder, C., Fonteyn, P., and Balouet, J-C. 2002, J. Occup. Health Safety – Aust. NZ 18, 321.
95. Cox, L., and Michaelis, S. 2002. J. Occup. Health Safety – Aust. NZ 18, 305.
96. NAS. 2001, The Airliner Cabin Environment and the Health of Passengers and Crew. US National Academy of Science, National Academy Press, Washington.
97. Rayman, R. B. 2002, Aviat., Space, Environm. Med., 73, 211.
98. Singh, B. 2004. Aviat. Safety Spotl., 10.
99. Winder, C. Misuse of the exposure standard concept. Journal of Occupational Health and Safety – Australia and New Zealand 14: 107-110, 1998.
100. Cone, J. E. 2005, In: Air Quality in Airplane Cabins and Similar Enclosed Spaces, Hocking, M., editor. Handbk. Environm. Chem., 4 Part H, 293.
101. Fox, R. 1997. Air Quality Testing Aboard Ansett Airlines BAe 146 Aircraft: Final Report. Allied Signal Aerospace.
102. Craig, P., and Barth, M. 1999, J. Toxicol. Environm. Health B Crit. Rev., 2, 281.
103. WHO. 1986, Early Detection of Occupational Diseases. World Health Organization, Geneva, 154.
104. Harper, A. 2005, In: Contaminated Air Protection Conference: Proceedings of a Conference held at Imperial College, London, 20-21 April 2005, Winder, C., editor. BALPA/UNSW, Sydney, 43.
105. Somers, M. 2005, In: Contaminated Air Protection Conference: Proceedings of a Conference held at Imperial College, London, 20-21 April 2005, Winder, C., editor. UNSW, Sydney, 2005, 129.
106. Burdon, J., and Glanville, A. R. 2005, In: Contaminated Air Protection Conference: Proceedings of a Conference held at Imperial College, London, 20-21 April 2005, Winder, C., editor. UNSW, Sydney, 2005, 53.
107. Heuser, G., Aguilera, O., Heuser, S., and Gordon, R. 2005, In: Contaminated Air Protection Conference: Proceedings of a Conference held at Imperial College, London, 20-21 April 2005, Winder, C., editor. UNSW, Sydney, 2005, 43.
108. Coxon, L. 2002, Neuropsychological assessment of a group of BAe aircraft crew members exposed to jet engine oil emissions. J. Occup. Health Safety-Aust. NZ, 18, 313.