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Crew Effects from Toxic Exposures on Aircraft

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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 leak 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 the 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 Federal Aviation Regulations, most notably FAR 25.831. Symptoms of immediate or short-term nature and 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, gastrointestinal, respiratory and nervous system effects, and long-term central nervous and immunological effects.

Keywords Aircraft air contamination · Substituted diphenylamine · Phenyl-alpha-naphthylamine · Tricresyl phosphate · Triorthocresyl phopshate · Organophosphate induced chronic neurotoxicity (OPICN) · Aerotoxic syndrome

Abbreviations

CAS	Chemical Abstracts Service
COPIND	Chronic organophosphate-induced neuropsychological disorder
DOCP	Di-ortho cresyl phosphate
FAR	U.S. Federal Aviation Regulation
JAR	Joint Aviation Regulation
MOCP	Mono-ortho cresyl phosphate
MSDB	Material Safety Data Bulletin
NTE	Neurotoxic esterases
OHS	Occupational Health and Safety
OP	Organophosphorus
OPICN	Organophosphorus ester-induced chronic neurotoxicity
OPIDN	Organophosphorus ester-induced delayed neurotoxicity
PAN	Phenyl-alpha naphthylamine
TCP	Tricresyl phosphate
TOCP	Tri-ortho cresyl phosphatekp

1 Introduction

As already noted in Chapters 10 and 11, 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) [1, 2]. 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.

Notwithstanding emergency situations, a range of other situations can arise whereby aircraft cabin air can be contaminated [3]. 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 auxilliary 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 transmissions,
- oil contamination from synthetic turbine oil,
- engine combustion products (for example, defective fuel manifolds, seal failures, engine leaks).

Significant contaminants include: aldehydes; aromatic hydrocarbons; aliphatic hydrocarbons; chlorinated, fluorinated, methylated, phosphate or nitrogen

compounds; esters; and oxides [4–6]. One additional problem is the lower partial pressure of oxygen that is present in the cabins of planes flying at altitude [7].

To date, most studies that have been carried out to measure atmospheric contamination in aircraft by engine oil leaks or hydraulic fluids are sufficiently flawed on procedural and methodological grounds as to render their conclusions invalid. Further, no monitoring has occurred during an oil leak.

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" [8]. 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.

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.

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, including dizziness, fatigue respiratory problems, nausea, disorientation, confusion, blurred vision and tremors [9–11]. Neurotoxicity is a major flight safety concern especially where exposures are intense [12].

2 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" [13].

Chemical exposures in aircraft are not unheard of. In 1953, the US Aeromedical Association first expressed their concerns about the toxicity risks of cabin air contamination by hydraulics and lubricants [14]. Other risks have been identified more recently, either as part of the chemicals routinely used in maintaining aircraft [15], or as toxicological factors in aviation accidents [16, 17].

A complex approval process exists for ensuring that materials used in aviation are manufactured to relevant standards, 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 additive tricresyl phosphate (TCP), 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 was recognised [18].

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.

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 [6]:

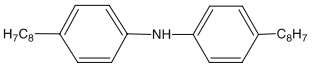
- synthetic esters based in a mixture of 95% C_5-C_{10} fatty acid esters of pentaerythritol and dipentaerythritol;
- 3% tricresyl phosphate (Phoshoric acid, tris(methylphenyl) ester, CAS No 1330-78-5);
- 1% phenyl-alpha-naphthylamine (PAN) (1-Naphthalenamine, N-phenyl, CAS No 90-30-2);
- a substituted diphenylamine;
- 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).

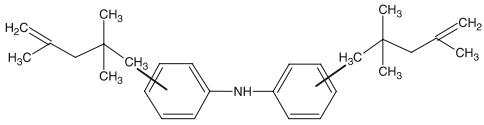
2.1 The Substituted Diphenylamine

The substituted diphenylamine is variously reported as benzamine, 4-octyl-N-(4-octylphenyl), (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 Fig. 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



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



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

Fig. 1 Substituted diphenylamines

relates to its environmental effects, such as poor biodegradability and toxicity to aquatic invertebrates [19].

2.2 N-Phenyl-alpha-naphthylamine

N-Phenyl-alpha-naphthylamine, (CAS No 90-30-2), also known as phenyl-alpha-naphthylamine (PAN), is a lipophilic solid as an antioxidant in lubrication oils and as a protective agent in rubber products (see Fig. 2). In these products, the chemical acts as a radical scavenger in the auto-oxidation 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 [20]. Both urine and faeces appear to be the main routes of excretion [21].

By single dosing, PAN has a short-term 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 [22]. This result is supported by case studies in exposed workers [23, 24]. At the concentration used (1%), Mobil Jet

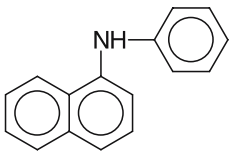


Fig. 2 N-Phenyl-1-naphthylamine

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 [21].

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 [25]. While the methodology used in this study makes evaluation of the results problematic (use of one gender, small sample sizes, limited number of dose groups, subcutaneous 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 [26]. 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 [27].

2.3 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 Fig. 3). Since the apparently different three-dimensional structures of the molecule are not chemically locked in place, they are not optical isomers. Therefore, 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);

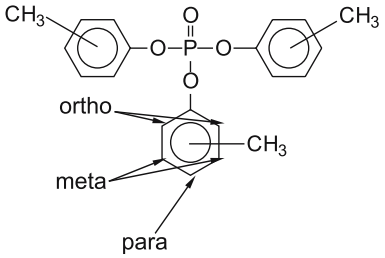


Fig. 3 Structure of Tricresyl phosphate

- 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 [28]. The study of OP toxicity is extensive, and generally characterised by a toxicity of inhibition of the esterase enzymes, most particularly cholinesterases [29] and neurotoxic esterases [30]. The mechanism of effect is phosphorylation [31].

Signs of low level intoxication include headache, vertigo, general weakness, drowsiness, lethargy, difficulty in concentration, slurred speech, confusion, emotional lability and hypothermia [32]. The reversibility of such effects has been questioned [33].

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 [34]. This is the basic site of inhibition for all OP molecules [35, 36].

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 sensorimotor mixed peripheral neuropathy, called organophosphorus-induced delayed neurotoxicity (OPIDN) [36, 37]. The mechanism of toxicity is now fairly well understood, as indeed are the organophosphorus structures which are predicted to cause OPIDN [38].

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 [39].

More recently, chronic exposure to organophosphates has been associated with a range of neurological and neuropsychological effects [40–44]. 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 organophosphate-induced neuropsychological disorder (COPIND) has been described, of neurological and neuropsychological symptoms [45]. 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 [46], or long-term low level repeated exposure with (often) no apparent signs of exposure [43]. The basic mechanism of effect is not known, although it is not believed to be related to the esterase inhibition properties of organophosphorus compounds. It is also not known if these symptoms are permanent.

In addition, since the introduction and extensive use of synthetic organophosphorus 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 [47–49]. The neuronal disorder that results from organophosphorus ester-induced chronic neurotoxicity (OPICN), which leads to long-term neurological and neurobehavioral deficits and has recently been linked to the effects being seen in aircrew despite OP levels being too low to cause OPIDN [50].

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 [50].

While the description above relate 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 [51–53]. 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 one tri-ortho (TOCP) isomer, triorthocresyl 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 neu-

rotoxicity of products containing TCP [18]. 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 [18], and questions have been raised about the lack of consistency between stated ingredient data and actual amounts of toxic isomers present in commercial formulations, and their impact on exposed individuals [6].

3 Effects of Aircraft Oil 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 [55]. 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.

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 [56], a 1983 study of Boeing 747 flight attendants in the USA (this paper linked symptoms to ozone) [57], a 1990 study of aerospace workers [58], and a 1998 study of BAe 146 flight crews in Canada over a four-month period [9]. A recent report of seven case studies considered representative of the common symptoms of irritancy and toxicity described similar symptoms [10], and a follow up survey by the same research group reported similar findings in a larger group of fifty crew respondents [59]. Two union-based studies in pilots provide additional data [60, 61].

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 1 overleaf.

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-headededness 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

Table 1 Studies reporting signs and symptoms in aircrew

Symptom cluster	Sign or symptom	Reference [56]	[57]	[58]	[9]	[10]	[59]	[60]	[61]
	Number of cases/reports 89	248	53	112	7	50	21	106	
Loss of consciousness/ 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%
Gastrointestinal 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%

Table 1 (continued)

Symptom cluster	Sign or symptom	Reference [56]	[57]	[58]	[9]	[10]	[59]	[60]	[61]
		Number of cases/reports 89	248	53	112	7	50	21	106
Neurological 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/neuropsychological 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%
	General increase in feeling unwell							21%	27%
	Behaviour modified, depression, irritability	26%	20%	60%		4/7	40%		
	Change in urine		3%	6%			4%		
	Joint pain, muscle weakness, muscle cramps		29%			2/7	38%	5%	30%

symptoms as being multitudinous and variable [62], 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 1).

4 Other Factors of Importance to the Aviation Industry

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 [63].

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 may in turn 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:

- permissible exposure standards for occupational exposures to airborne contaminants – 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 [64];
- There is "not agreement on a toxicological standard among aviation toxicologists to apply to aircraft". Exposure standards were developed by the American Conference of Industrial Hygienists (ACGIH) for the average worker at or near sea level pressure in relatively good health. Flight crew work in conditions where atmospheric pressure is reduced. [67] Most chemicals do not have exposure standards and of those that do exist most "are still regulated by voluntary standards set before 1971", when adopted uncritically and unchanged with new science having had no impact on them. [68];
- 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 more toxic than TOCP [65];
- 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 have not incorporated more recent scientific and medical evidence [64];
- ventilation rates for buildings.

Occupational exposure standards may be inadequate to protect nonworkers, for example passengers.

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 [66].

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.

5 Conclusions

What emerges in the analysis of this data 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 the nervous system, respiratory system, gastro-intestinal system, and possibly the immune system and cardiovascular system. These symptoms may be expressed specifically to these systems, or may be seen more generally, such as headache, behavioural change or chronic fatigue.

The symptoms reported by exposed individuals as shown in Table 1 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, respiratory and immunological effects (see Table 2). This syndrome may be reversible following brief exposures, but features are emerging of a chronic syndrome following significant exposures [10, 11, 59].

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:

- As shown in the section on leaks, 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. From the analysis in Chapter 11, the rates of occurrence of incidents are higher than the aviation industry admits, 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, it 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

Table 2 Aerotoxic syndrome: short- and long-term symptoms

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

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.
- Pilots continue to fly when experiencing discomfort or symptoms. 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 the 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 substantial 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.

Contaminants in the air of an occupational environment should, under normal circumstances, alert management to a potential problem [63]. 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.

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 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?

More scientific and medical research is needed on the short and long-term effects of exposure to contaminated air and, until this is completed, all areas of the aviation industry should take fume exposure events seriously; they should be seen as an important part of educating crew and the aviation industry, thereby addressing the problem.

Many of the world's leading experts who have seen aircrew from around the globe or were familiar with the issue spoke at a recent conference held in London by the British Airline Pilot Association (BALPA) looking at the issues of contaminated air by engine oils and concluded:

- There is a workplace problem resulting in chronic and acute illness amongst flight crew (both pilots and cabin crew).

The workplace in wich these illnesses are being induced is the aircraft cabin environment. This is the resulting in significant flight safety issues, in addition to unacceptable flight crew personnel healt implications.

- Further, we are concerned the passengers may also be suffering from similar symptoms to those exhibited by flight crew.

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