Aircraft cabin air contamination and aerotoxic syndrome—a review of the evidence

Frank Cannon*

Aviation Lawyer

There is growing concern that there are no plans by the aviation industry and its safety regulators to take action to end the bleed-air system, whereby the interior breathing air within airliners enters the aircraft through the jet engines. As a result fumes from engine lubricating oil, hydraulic fluid, de-icing fluids etc. routinely enter the cabin air. Aircrew and some frequently flying passengers are presenting with the symptoms associated with toxic exposure. It would be thought wise for the industry and its regulators to make timely plans for the permanent eradication of what many agree is a design fault that has lasted for too long. This is especially true now that an alternative, bleed-free, ventilation and pressurization system has been operating successfully for some time in the form of the Boeing 787. This paper reviews the evidence the industry and its regulators appear to be ignoring.

1. Background

Life is unsustainable at the altitudes at which airliners cruise. This is due to extremely low atmospheric pressure, density, temperature and humidity. The industry-preferred solution has been a system of drawing or "bleeding" high-pressure air into the aircraft through the aircraft's jet engines. This "bleed air" is used to heat and pressurize the cabin and flight deck and to pressurize the aircraft's drinking water tank and the aircraft's hydraulic system. Drawing air through the engines inevitably results in contamination by the engine lubricating oil, hydraulic fluid and other substances. One of the fundamental problems lies in the fact that *all* the constituents of jet engine lubricating oil and aircraft hydraulic fluid are harmful to humans with various degrees of toxicity [1–14]. The Boeing 787 "Dreamliner", on the other hand, uses dedicated electrical compressors with air bearings, without risk of oil or hydraulic system contamination, to pressurize, refresh and heat the aircraft interior.

The airliner cabin is a hermetically sealed pressure vessel, with an *inflow* of bleed air and a computer-controlled *outflow*, which exhausts back to the atmosphere. Jet engines operate at extremely high temperatures. The only air that enters the interior of the aircraft during operation

* E-mail: frank@cannonslaw.com

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is the bleed air from the engines. If the pilots shut the bleed air valves after the doors are closed, even with the aircraft still on the ground, all the occupants would eventually perish, when the oxygen was depleted. There is, of course, an alarm to draw such an abnormal condition to the pilots' attention; the point is made in order to emphasize the *essential* nature of the *living* space within an airliner. Engine oil seals, in particular, are far from perfect and, as this paper will demonstrate, they leak on a regular basis. In a report presented to the National Aeronautics and Space Administration (NASA), Raymond Chupp, an expert on jet engine seals and an employee of General Electric, a leading jet engine manufacturer, while discussing oil seals in particular, pointed out that "A zero-leakage seal is an oxymoron" [15].

Hydraulic systems vent to atmosphere. The atmosphere, in this case, is the interior of the aircraft. In addition, the hydraulic pumps, and some actuators, are mounted in the engines, and the bleed air is also used to prepressurize the hydraulic systems. The very high pressure of aircraft hydraulic systems (>10 MPa) creates "sweats", leaks and ruptures. The overall result is that the interior air of aircraft is routinely contaminated by hydraulic fluid in addition to the engine lubricating oil.

Once contaminated, the internal distribution ducting, buried within the aircraft's structures, becomes impossible to clean, especially the final narrow ducting that reaches out to every seat-row, and its associated "gaspers" above the crew's and passengers' heads. Black sooty deposits of uncleanable contamination were reported in a UK Civil Aviation Authority (CAA) study in 2004 [16]. Photographs from this study are shown in Figure 1.

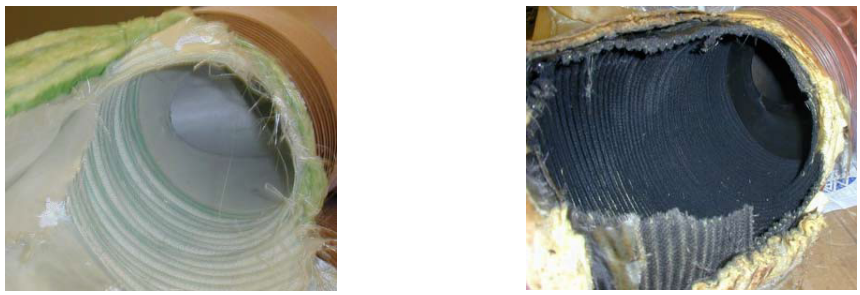

Figure 1. (left) Opened-up new ducting; (right) "in use" ducting [16].

There are two points of note associated with Figure 1; firstly, after the air distribution point depicted in these photographs there is no filtration before the air, passing through this ducting, reaches the lungs of the aircraft's occupants; secondly, the black, sooty deposits depicted in Figure 1(right) were analysed by the CAA and matched the aircraft's engine oil; that is, the oil is evidently the source of the contamination. Such accumulated contamination furthermore ensures continuous delivery of toxic substances to the occupants long after the source of the contamination has been rectified.

There are many other contaminants routinely detected in the internal aircraft cabin air that are both toxic, neurotoxic, or both [6, 14]. Metabolites of these compounds have been detected in aircrew urine specimens and fat biopsies. The parent compounds deduced from these results include de-icing fluids (ethylene or propylene glycol), as well as ultrastrong disinfectants,

anti-insect sprays (permethrin and other pyrethroids), and flame retardants (e.g., polybrominated diphenyl ether). The first-named is presumably sucked into the air intakes after deicing operations; the others are likely to originate from operations carried out inside the cabin by airline personnel. Oil residues are not the sole dangers inherent in the bleed-air architecture of aircraft. Traces of the metal alloys used in the construction of jet engines, such as titanium, beryllium and nickel, have been detected in aircrew's tissue biopsies [14].

Jet fuel metabolites are there also. Jet fuel has been shown to suppress the immune system [17–19], which may help to explain the popularly held association, in the minds of some who fly, between flying and various transient post-flight ailments.

The route of entry of jet fuel into the cabin air stream might seem something of a puzzle, because fuel is not added to the compressor airflow in the engine until after the bleed-air ports. However, the repeated finding of jet-fuel metabolites in aircrew leads to the reasonable hypothesis that fuelling operations and ground running of engines are the likely source. Additionally at busy airports, there is frequently a queue of aircraft lining up for take-off. If such a queue were to consist of, say, five aircraft, in line one after the other, on the way to the runway, then the lead aircraft is drawing in through its engines the freshest of the air. Number 2 in the queue is drawing in the exhaust of number 1, and number 3 is drawing in the air cumulatively contaminated by the exhausts of numbers 1 and 2. Number 5 is the unluckiest, because the air it is sucking in is the exhaust of number 4, which contains the *cumulative* exhausts of the three preceding aircraft. Such contamination is wholly unfiltered. Fuel-burn is optimized for cruise conditions, and therefore engine exhaust during ground operations contains an appreciable amount of unburnt fuel.

When operating on the ground, jet engines act like giant suction sweepers (vacuum cleaners) and will generally ingest anything and everything they encounter; for example, apron and runway debris, de-icing chemicals, and other surface treatments. A proportion of anything that enters the giant fan at the front of the engine will end up, unfiltered, in the lungs of the occupants. Parts of the jet engine, including the bearings supporting the jet engine's main shaft, all bathed in oil and subject to wear, are composed of alloys of *beryllium*, an extremely toxic metal [20]. In fact the aerospace industry is the biggest user of beryllium, which was detected as a DNA adduct during blood tests of several pilots in 2006 [14].

Toxicity of the aircraft cabin breathing air is not the only disadvantage of flight at high altitude. Oxygen is reduced by up to 30%, creating a danger for all those whose medical condition or age would dictate a greater need. The CAA has acknowledged that the oxygen level in aircraft cabins while cruising "*is adequate for healthy passengers*" [21]. But unhealthy people travel by air too; those who are, knowingly or unknowingly, compromised by medical conditions as well as very young or elderly people who may require a fuller amount of oxygen. The corollary of the CAA statement is that the oxygen level may be inadequate for those who are not healthy (besides, no precise definition of "healthy" accompanied the statement). Typically, blood oxygen saturation levels, in healthy adults, in cruise, are 87 to 92%, as opposed to an expected average saturation at sea level of 97 to 99%. For short-haul flights, and healthy adults, this may be acceptable; however the range of aircraft has been progressively extended over recent years, and nonstop flights in excess of 17 hours are not unusual nowadays [28].

Due to the reduced pressure and oxygen at altitude, workplace exposure limits (WELs) of toxic chemicals [22] are not valid for aircraft in flight. Nevertheless the air industry and its

regulators continue to point out, erroneously, that levels of toxicity are well below such "health limits". From a toxicological point of view, the thinner atmosphere has a number of deficits, which are wholly overlooked by the air transport industry and its regulators; to give just one example, the recommended limit for carbon monoxide (CO) exposure is halved at aircraft cruising altitude [1]. CO, a neurotoxic and potentially lethal gas, is always detected when looked for in aircraft air sampling [5, 6, 23]. Yet, surprisingly, no commercial aircraft is equipped with a detector or monitor for carbon monoxide, even although a detector or monitor for the home can be purchased very cheaply in hardware stores.

2. Jet engine lubricating oil

Jet engine lubricating oil consists of synthetic esters of pentanoic acid and pentaerythritol. It also contains an aromatic compound, tricresyl phosphate (TCP). TCP is a member of the organophosphate family of chemicals, all sharing a similar core molecular structure (i.e., a phosphoric acid ester). Organophosphates were essentially invented for only one purpose—to kill. They were first developed and used, on an industrial scale, as potent nerve agents for chemical warfare. When their killing properties became more widely known, they were used in diluted form, in solution, as an insecticide. Some nonvolatile organophosphates were found to be useful to engineers, particularly aero-engineers, for their high resilience under stress. Hence, long after the use of organophosphate pesticides was banned, on health grounds, by most enlightened nations, organophosphate use survived in the lubricating oil of jet engines.

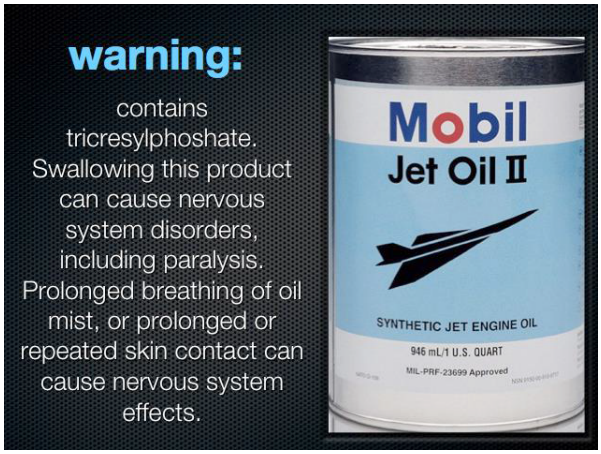

Figure 2. The warning on the back of the can of the most popular engine lubricating oil. Aircrew and passengers are unlikely to ever see this warning.

Tricresyl phosphate is present in almost all jet engine lubricating oils (a notable exception being the French Nycoil, which has enjoyed modest market penetration). As its name implies, TCP is a mixture of different isomers (chemical compounds with the same atomic formula, but with different arrangements of the atoms in the molecule). The cresyl substituent has three isomers, which are called *meta* (*m*), *para* (*p*) and *ortho* (*o*). It is a matter of simple combinatorics to deduce that, with its three cresyl substituents, TCP can have ten isomers: six *ortho* isomers, *ooo*, *oom*, *oop*, *omp, omm* and *opp*, two *meta* isomers, *mmm* and *mmp* and two *para* isomers,

ppp and *mpp*, some or all of which may be present in the substance labeled "TCP". It has been widely misunderstood that the most lethal isomer is the tri-*ortho*-cresyl phosphate (*ooo*) (T*o*CP). This was due to the notoriety of several high-profile mass poisonings, from the 1930s to the 1950s, which happened to involve the tri-*ortho* isomer. The di-*ortho* and mono-*ortho* isomers are much more toxic [8], and, as shown above, are more prevalent.

In the 1950s, only military aircraft were using bleed air from the jet engines—the first civil airliner to use bleed air was the French Sud Aviation Caravelle (1959), which had Rolls-Royce Avon engines—but governments restricted the T*o*CP content of *all* commercial TCP products, on health grounds. Accordingly, the tri-*ortho* content in jet engine lubricating oil is supposed to be less than 0.1% of the total TCP. In reality, the *ortho* proportion in aircraft cabin air has been observed to be much greater [23, 24]. The mono-*ortho* (*omm, omp* and *opp*) (M*o*CP) isomers are ten times more neurotoxic, and the di-*ortho* (*oom* and *oop*) (D*o*CP) isomers are five times more neurotoxic, than T*o*CP [8]. The *para* and *meta* isomers of the engine lubricating oil are also now recognized as being harmful [9, 11–14, 25].

The oil also contains N-phenyl-1-naphthylamine (a known carcinogen), alkylated diphenylamines and phenyldimethyl phosphate (see Table 1) [26].

Table 1. Additives in jet oil.

Reportable hazardous substances	CAS N^{o}	Concentration in the oil	Function	Health hazard
N-PHENYL-1-NAPHTHYLAMINE	90-30-2	1%	Anti-oxidant	Carcinogen
PHENYLDIMETHYL PHOSPHATE (3:1)	25155-23-1	1%	Anti-oxidant	Skin irritant
TRICRESYL PHOSPHATE	1330-78-5	3 – 5%	Anti-wear	Neurotoxin

Table 1 was compiled from the safety data sheet (SDS) [26] for the lubricating oil with the largest worldwide market share (49%)—Exxon Mobil Jet Oil II. This is, for example, the lubricating oil used by British Airways and other leading airlines. Acknowledging health concerns, the statement in the SDS confirms "*Danger of adverse health effects by prolonged exposure*" and "*This material is considered to be hazardous according to regulatory guidelines*". Chemists with a sharp eye may detect that the CAS (Chemical Abstract Service) number of tricresyl phosphate given by Exxon Mobil—CAS 1330-78-5 (in the SDS [26] bearing a revision date of 2014)—was actually replaced by two different numbers in the CAS *Designated List* in 1999 [9]. Hence there is confusion as to the true content of the oil, since the old number applied to all isomeric TCP mixtures, whereas the new numbers designate the *ortho* (CAS 000078-30-8) and *para* (CAS 000078-32-0) isomers. By continuing to provide the out-of-date CAS number the manufacturer undermines any assurance regarding the *ortho* content of the oil. This is particularly the case since a UK government-sponsored study of aircraft cabin air actually found the *ortho* content to be much higher than it is supposed to be in the original oil as supplied [23, 24].

As long ago as 2001, Chris Winder and Jean-Christophe Balouet, in their seminal work "The toxicity of commercial jet oils" [1], provided timely warning to the industry regarding what they termed the relative and effective toxicities. Mobil states that the T*o*CP, that is the tri-*ortho*, content in the oil is 0.005 parts per million (ppm). However, during the 1999, *Australian Senate Inquiry Into Contaminated Aircraft Cabin Air* Mobil's chemists revealed that the other *ortho* isomer mixtures are present at 6 parts per million (ppm) for D*o*CP and at 3070 ppm for M*o*CP. This had been entirely concealed until then. It dramatically transforms the actual toxicity of the oil. Taking into account the relative toxicities of the three different *ortho* isomers—D*o*CP is 5 times more neurotoxic and M*o*CP is 10 times more neurotoxic than the T*o*CP [8]—the *effective* toxicity has been hugely understated.

To nonscientists the dosage associated with "parts per million" (ppm) may seem so small as to be insignificant, but most powerful substances have exposure health limits and are routinely detected at levels measured in ppm.

For example, the 8-hour WEL for carbon monoxide (CO) at sea level for healthy adults is 30 ppm [27]. A similar limit for beryllium (mentioned *supra*), again at sea level and for healthy adults only, is 0.000002 ppm or 0.002 ppb (parts per [US] billion) [20, 27].

Winder and Balouet calculated the *effective* toxicity of TCP (Table 2) [1]. From this data, we see that the toxicity of the engine lubricating oil is understated by a factor of more than 30 000.

Table 2. The relative and effective toxicities of the *ortho*-isomers of TCP.

Isomer	Concentration[a] (ppm)	Relative toxicity	Effective toxicity
ToCP	0.005	1	1
DoCP	6.0	5	30
MoCP	3070	10	30 700
Total (equivalent toxicity factor of *ortho*-containing TCP)			30 731

[a] Supposed concentration in fresh engine oil, based on the composition of modern commercial TCP as admitted by Exxon's chemists.

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3. Aircraft hydraulic fluid

According to their materials safety data sheets (MSDS), aircraft hydraulic fluids can contain up to 100% tributyl phosphate (TBP), or various mixtures of TBP, dibutylphenyl phosphate (DPP) and butyldiphenyl phosphate (BDP). These constituents are chemicals of health concern [4, 9], yet they are always, as will be seen later, found in aircraft cabin air, when looked for (Figure 3).

4. Neurotoxic effects of organophosphates

TCP, TBP, DPP, BDP, triisobutyl phosphate (TIBP) and triphenyl phosphate (TPP) are all examples of organophosphates (OP). Organophosphates act in three neurotoxic modes to poison the human body:

First, cholinergic neurotoxicity caused by inhibition of acetylcholinesterase, which leads to overstimulation of muscarinic and nicotinic acetylcholine receptors, with subsequent development of cholinergic toxicity [29]. Simply explained, this mode refers to the instantaneous, short-term, interference in brain and nerve functioning (it should be borne in

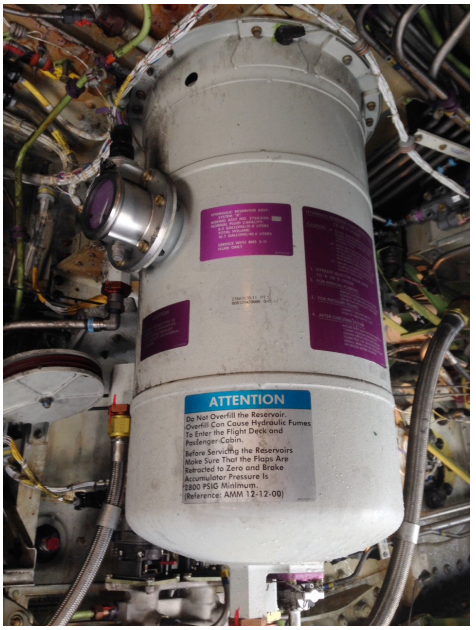

Figure 3. One of the hydraulic reservoirs of a Boeing 737. The warning reads: "Do Not Overfill the Reservoir. Overfill Can Cause Hydraulic Fumes to Enter the Flight Deck and Passenger Cabin." Notwithstanding the warning, hydraulic fumes can justifiably be said to be an almost permanent component of the interior air of aircraft.

mind that organophosphates were developed as chemical warfare agents to instantly incapacitate an enemy soldier).

Second, organophosphorus ester-induced delayed neurotoxicity (OPIDN). This is a central–peripheral axonopathy characterized by Wallerian-type axonal degeneration of the central (CNS) and peripheral (PNS) nervous systems, followed by secondary demyelination [30]. The clinical picture for OPIDN is manifested by mild sensory disturbances, ataxia, weakness and muscle fatigue and twitching, which may progress to paralysis.

Third, organophosphorus ester-induced chronic neurotoxicity (OPICN), characterized by long-term neurological and neurobehavioral deficits accompanied by brain cell death [29]. Such devastating injury has been documented *post mortem* [31].

5. Inflammatory effects of organophosphates

Organophosphates have also been shown to cause an inflammatory response [32]. The authors of this paper concluded: "There is significant experimental evidence that acute OP intoxication elicits a robust inflammatory response, and emerging evidence suggests that chronic repeated low-level OP exposure also upregulates inflammatory mediators". Myocarditis, including lymphocytic myocarditis, and other serious inflammatory conditions, have been reported [31]. There is an accepted increased risk of developing deep vein thrombosis (DVT) in flying, which has been said to be due to the inactivity and lack of exercise associated with long-haul flight. However, it has been shown that exposure to organophosphate *increases the risk of DVT at least twofold* [33], perhaps dispelling a myth.

6. Genetic variation

A fundamental mistake of the air transport industry and its regulators has been to ignore one of the most basic facts of life, namely DNA differences. Due to genetic variation within the human species some aircraft crew (and frequently flying passengers) are unable to detoxify OP poisons between flights [34–36].

Even the lubricating oil manufacturers recognize this: "Health studies have shown that chemical exposure may cause potential human health risks which may vary from person to person" [26]. As a consequence of this genetic variation, an accumulation of neurotoxic metabolites will occur in some people.

Detoxification takes place in the liver via enzymes. At least one enzyme, paraoxonase (PON1), is known to be beneficial for the detoxification of organophosphates, and if a person has a genetic deficiency in the manufacture of this enzyme, such a person will be more at risk than others [35, 36]. Any inability, even partial, to detoxify organophosphates should be viewed with serious concern. Most of the detoxification pathways involve multiple steps and, correspondingly, multiple enzymes, doubtless including members of the cytochrome P450 superfamily [34]. Some of the intermediates in the overall detoxification process are even more toxic than the initial substances. A genetic deficiency might take the form of absence of the enzyme that detoxifies the extremely toxic intermediate. In such cases, it is expected that *accumulation* will occur, in the body, of the more neurotoxic metabolites such as cresyl saligenin phosphate and phenyl saligenin phosphate [37, 38].

7. Symptoms of aerotoxic syndrome

For more than two decades, pilots and cabin crew have complained of nervous system symptoms following exposure to air emissions inside aircraft [1–7, 14]. Aerotoxic syndrome is nothing more than organophosphate poisoning resulting from exposure to bleed air. There is, as has been observed *supra*, an added component of jet fuel suppression of the immune system.

The most common but debilitating symptoms include chronic fatigue, severe headaches, cognitive dysfunction, paraesthesia, ataxia and mood-swings [2]. These are all, individually or collectively, enough to ground a pilot on the spot, and render cabin crew unfit for further duty. Remedial treatment starts with removal from the toxic environment. For aircrew, this involves cessation of employment. The longer this is delayed the more intense the affliction and the more difficult the recovery. In a lot of cases, where brain damage has been allowed to go too far, full recovery is impossible. A striking observation is the connexion between exposure and symptom onset [2]. The reverse is also striking, namely that recovery, sometimes only partial, is possible only in the absence of further exposure [2].

This should not have come as a surprise to the industry. The bleed-air system for passenger airliners was taken from the military aviation industry. In the 1950s United States Air Force pilots were complaining of a mysterious illness, which was traced to their exposure to the oil contamination of the bleed air that heated and pressurized their fighters, bombers and transport aircraft. As long ago as 1955, Henry A. Reddall, an engineer employed by an aircraft manufacturer called North American Aviation Inc., published a paper entitled "Elimination of aircraft bleed air contamination" [39]. He wrote: "To understand the methods used for elimination, consideration must first be given to the source of the contamination and the process

by which the bleed air becomes contaminated. The basic cause has been reasonably well established as leakage of engine oil into the compressor air. In the process of compression the oil and air are heated to temperatures of the order of 700 to 800 °F [370 to 425 °C]. This causes decomposition of the oil and these products are then discharged into the air-conditioning system ducting at the engine bleed air ports." He was writing exclusively about military aircraft. Civil aircraft were not yet using engine-derived bleed air (Figure 4). He advocated that the only way to eliminate the contamination of the cabin air was to draw in the air using separate compressors, in the same way as the civilian airliners of the time.

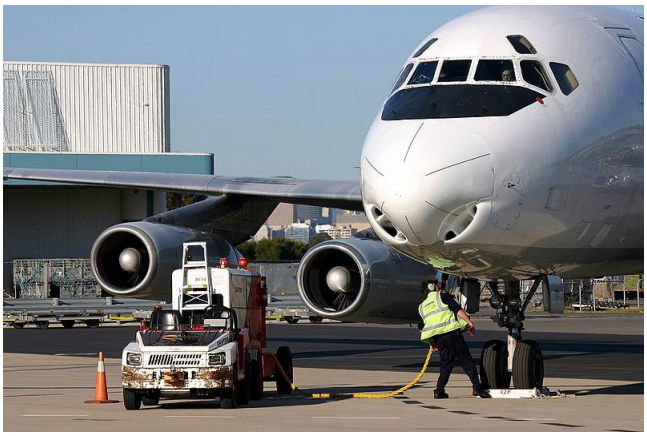

Figure 4. Non-bleed-air technology in 1960. Note the air intakes in the nose of this Douglas DC 8 aircraft. This aircraft used electrical compressors to pressurize and heat the interior.

North American Aviation later became part of the Boeing Airplane Company. It may be noted, therefore, that Boeing took more than 50 years to heed Reddall's advice, eventually producing the "bleed-free" Boeing 787 ("Dreamliner"), which uses separate electrical compressors (its first flight was in December 2009).

The human body is extensively innervated. Organophosphates act as neurotoxicants, which means they may attack the nerves, including the brain. Therefore when aircrew present with paraesthesia (numbness and tingling), a likely cause is injury to the peripheral nervous system (PNS), such as the ulnar, tibial, femoral or sciatic nerves. When aircrew present with ataxia (lack of coordination), cognitive dysfunction (fuzzy thinking) and mood swings, a likely cause is injury to the central nervous system (CNS)—the brain and spinal cord. When aircrew report shortness of breath, an unexplained cough, chest tightness, apnoeia (failure to breathe regularly when sleeping), a likely diagnosis is injury of the autonomic nervous system (ANS); and when aircrew fall sick with indigestion, diarrhoea or constipation, this may be due to damage to the enteric nervous system (ENS).

There is an obvious link between affected aircrew and agricultural workers whose health has been damaged by exposure to organophosphate pesticides. Gulf War syndrome, a similar affliction, due to exposure to military organophosphate nerve gases and other ordnance, is now fully acknowledged in the USA (but still apparently denied by the UK government).

Airlines and regulators (including the UK CAA) have stopped maintaining records of grounded aircrew "who (and, or, whose doctors) *allege* a connexion between their affliction and

the toxic contamination of bleed air." The greatest danger from such a failure of oversight is that pilots and cabin crew will remain at work for as long as their worsening symptoms will allow, with consequent impact on flight safety. There is no monitoring of the health of aircrew, even although this was one of the main recommendations of the UK Committee on Toxicity when asked for advice by the UK government in 2006. "Pilots' health should be regularly monitored particularly with regard to investigating neuropsychological impairment" they stated, continuing with: "In view of the plausible nature of evidence linking exposures to short-term health effects, further research in humans should be conducted to determine whether exposure to contaminated cabin air is responsible for the reported ill-health in aircraft crew." Both of these recommendations have been wholly ignored by the industry and, particularly, by the UK government (who had sought the advice in the first place). It might not unreasonably be suggested that the results of implementing such advice may not have been entirely welcome.

8. The continuous presence of organophosphates in aircraft cabin air

There is abundant evidence of the continuous leakage of lubricating oil and hydraulic fluid into aircraft cabin air *at all times*. This happens both on a continuous—normal—basis and on occasions characterized as "fume events". A constituent of engine lubricating oil, which is of health concern, is tricresyl phosphate (TCP) and a constituent of hydraulic fluid, which is of health concern, is tributyl phosphate (TBP); both are organophosphates. Contamination of the cabin air *simultaneously* by both organophosphates has almost universally been reported, as will be seen.

All detected and measured contamination has occurred, thus far, in routine flight, as opposed to a so-called "fume event". There has never been a measurement of contamination in a reportable fume event. It has to be accepted that on occasions when there has been obvious overcontamination in the form of smoke or a strong smoky or oily smell detectable by the unaided human senses, while the precise level of contamination has never been captured, had a measurement taken place it would doubtless have recorded contamination levels much greater than during normal operations (routine flight).

8.1 "Fume events"

The compression of the atmosphere within jet engines heats the incoming air to a high temperature. Anyone who has inflated a bicycle tyre with a hand pump will recollect the heat at the outlet of the pump. Multiply that heat many times. Liberation of hot oil past an engine oil seal and into contact with hot surfaces results in a burning oily smell, often with visible smoke in the cabin. Such events, known as "fume events", are generally acknowledged as giving rise to a "worst-case" pollution level. Fume events are unpredictable. They are said to occur within the very wide range of 0.05% to 0.5% of all flights, but more precise estimates vary, depending upon what is considered to constitute a "fume event", and whether anyone bothers to report it. Given the number of flights per day around the world, this incidence translates to *at least* three fume events per day throughout the world. It is still an unpredictable uncertainty. As a consequence, in the absence of permanent hard-wired automatic monitoring of aircraft cabin air, measurement of the actual contamination of the cabin air in such a fume event has proved elusive. In such circumstances a scientific hypothesis based on best "guesstimation" has

reasonable validity. The aerial concentration of tricresyl phosphate in a typical fume event has been reasonably estimated as 1.5 mg/m^3, of which tri-*ortho*-cresyl phosphate is 0.5 mg/m^3 [40]. This level of contamination is indeed of health concern.

The hypothesis that fume events cause greater contamination of the bleed air than has been measured during routine flight, draws support from a study (*infra*) which, cleverly, made use of the used and discarded filters that are deployed in the recirculation loop of the cabin air. A proportion of the air that passengers and cabin crew have breathed (*but not the pilots*) is captured and recirculated through a high efficiency particulate air (HEPA) filter (perhaps surprisingly the pilots are supplied with 100% unfiltered bleed air—implying clear acceptance by the industry that HEPA filters have no effect on organophosphate contamination). The authors of a study that will be described later were able to obtain 184 spent filters, of which 77 had been replaced prematurely due to suspicion of excessive "fume event" contamination. Comparative analysis of these 77 filters with the other 107 filters, which had been replaced at a normal maintenance interval, allowed the authors to conclude that "*a higher level of jet engine oil signature was seen*" in these filters [41]. In another test, where an aircraft was grounded because it had suffered excessive oil leakage into the bleed air, the opportunity was taken to sample the cabin air before and after the aircraft's offending engine was replaced. The tests were conducted in safety with the aircraft on the ground. The arithmetic mean of the tricresyl phosphate concentrations was an order of magnitude higher before (5.1 ± 1.1 μg m^{-3}, median 5.5, min–max 3.6–5.9) than after (0.47 ± 0.04 μg m^{-3}, median 0.47, min–max 0.41–0.51) replacement of the engine ($P = 0.02$). The authors were able to assert that "this difference supports a hypothesis of elevated tricresyl phosphate levels in cabin air during engine leaks resulting in smoke-in-cabin incidents" [42].

8.2 Routine contamination

There are many studies, and *ad hoc* air-sampling measurements, all demonstrating contamination of cabin air, by organophosphates, in "normal" flight operations. A few are described below.

(1) The Cranfield study (2011) [23]. Commissioned by the UK government. A total of 100 flights were sampled and in 73 of them the cabin air was contaminated by organophosphates in the form of TCP, T*o*CP, TBP or TPP either alone or in combination. It cannot be concluded that the other 27 flights were organophosphate-free, because the instruments used had minimum detection limits well above zero. Hence, all one can be sure of is that *at least* 73% had detectable levels of organophosphate during the flight.

(2) The TNO study (2013) [43]. This airline-commissioned report looked for only one of the organophosphates customarily present in aircraft cabin air, tricresyl phosphate, and *not* tributyl phosphate. Of a total of 20 flights during which samples were taken, 16 were contaminated by tricresyl phosphate. Again no proper conclusion can be drawn from "non-detects". All one can say with any degree of certainty is that *at least* 80 per cent of samples contained detectable tricresyl phosphate. This study also deployed wipe sampling in addition to air sampling. The flight-deck coaming, just ahead of the pilots' faces, was wiped and analysed for the presence of tricresyl phosphate. Of the 10 wipe samples taken in various aircraft, *all* were positive for tricresyl phosphate.

(3) The Rosenberger study (2013) [44] was an exercise carried out on behalf of the German airline, Lufthansa. It limited itself to looking *only* for tricresyl phosphate, no doubt as *the* chemical of maximum concern. Out of a total of 9 flights where air samples were taken, tricresyl phosphate was detected *in all of them*.

(4) Solbu et al. (2011) [42]. This was a peer-reviewed scientific study carried out by independent scientists and doctors in Norway. It confirmed that out of 76 flights tested by air sampling, *all of them* were contaminated by organophosphates of one kind or another, from hydraulic fluid or lubricating oil. In addition, wipe sampling was performed and *all of them* reported positive for organophosphate contamination. This study also examined and analysed six high efficiency particulate air (HEPA) filters from aircraft air-conditioning systems. These filters are designed to remove particulates. They are unable to remove organophosphates in gaseous form, but they are thought to be able to trap some organophosphates in droplet (aerosol) form. Organophosphate in the form of TCP was found *in all the filters*; this is of serious concern as the filters analysed had only been in use for between 130 to 470 hours (such filters are not normally replaced before 3000 hours). The authors concluded "TCP (tricresyl phosphate) was determined in all filter samples, supporting an assumption of the general presence of TCP in cabin and bleed air in aircraft with turbine jet engines".

When it was reported, during the course of this study, that one particular aircraft had been involved in an observed leakage of lubricating oil into the cabin air, due to a faulty engine oil seal, the authors took the opportunity of examining the internal air of that aircraft, during ground operation of the engines, *before and after* the faulty engine was replaced. The scientists reported: "A smell of burned oil was present in the cabin ground testing prior to the engine change". It was found that tricresyl phosphate was detected before the engine change at levels ten times greater than the tricresyl phosphate levels present after the engine change. The authors remarked: "This difference supports a hypothesis of elevated TCP levels in cabin air during engine leaks resulting in smoke-in-cabin incidents". The use of the word "elevated" implies that there is a more general background level of organophosphate at other times. A similar remark can also usefully be made with regards to the finding of the study by Eckels et al. (*supra et infra*)—"a higher level of jet engine oil signature was seen", implying a lower general level at all other times.

(5) Eckels et al. (2014) [41]. This was a scientific peer-reviewed study of 184 HEPA filters taken from aircraft. The authors described 107 of these as "standard", by which they meant that they had no known special contamination issues (and had therefore been replaced according to the standard (normal) maintenance interval), and 77 were "nonstandard", meaning that they were associated with known contamination issues (and had been replaced earlier than normal). The authors reported that 90% of all filters had detectable levels of tricresyl phosphate, with much higher levels of such contamination in the 77 nonstandard filters. The authors were able to assert "an ancillary conclusion of this study is that TCP at some level is relatively common in aircraft cabin air".

(6) Lyasova et al. (2011) [45] was a peer-reviewed scientific study designed to detect exposure to tri-*ortho*-cresyl phosphate in aircraft cabin air using a volunteer blood test after flight. This test looks for phosphorylated butyrylcholinesterase (BChE), an exclusive marker for

such exposure. Twelve passengers had blood samples taken post-flight. They had all been on different flights. The authors established that at least six of these passengers had BchE levels commensurate with exposure to tri-*ortho*-cresyl phosphate (T*o*CP). Four of the passengers who tested positive were retested some months after flying and no evidence of ToCP exposure was detected.

(7) A military study (2005) [46]. Pressurized military aircraft powered by jet engines suffer from the same problems as their civilian cousins. In this study, defence scientists in Australia sampled the interior air on British Aerospace Hawk trainer aircraft, the General Dynamics F 111 bomber, and the Hercules C130 transport aircraft. Of 13 samples taken in flight on the Hawks, 9 were contaminated by tricresyl phosphate and 4 were below the limit of detection. Of 19 samples collected in flight on the F111 aircraft, 14 were contaminated by tricresyl phosphate; and of 12 samples collected in ground runs, 8 were contaminated by tricresyl phosphate. On the Hercules C130 aircraft, out of 30 air samples collected, 28 were contaminated by organophosphates (TCP plus TBP).

(8) Another military study (2011) [47]. Australian defence scientists measured the air samples obtained in aircraft coded "FT" (flight trainer), "FB" (fighter bomber) and "CT" (cargo transport), no doubt equating to the BAe Hawk, the General Dynamics F111 and the Hercules C130, respectively. Of 6 samples taken on the Hawk during engine ground runs, all of them contained tricresyl phosphate. Of 2 samples obtained during engine ground runs on the C130, both contained tricresyl phosphate. Out of 4 samples obtained on the C130 during flight, 3 contained tricresyl phosphate. Out of 13 air samples captured in the F111 during fight, 12 contained tricresyl phosphate. In the same aircraft, of 7 air samples captured during engine ground runs, all contained tricresyl phosphate. In addition, *all* the samples taken from the F111 and the Hawk had significant levels of organophosphates in the form of tributyl phosphate (presumably from aircraft hydraulic fluid).

(9) Schindler et al. (2012) [48]. In this study, 332 urine samples were collected from aircrew after flight, and then tested for the presence of metabolites of organophosphates. *All* of the 332 samples tested positive for the metabolites of tributyl phosphate (TBP) and triphenyl phosphate (TPP), presumably originating from the aircraft hydraulic fluid. This demonstrates, overwhelmingly, that aircrew and passengers are exposed to these harmful toxins in all flights. Although the title of this study implies that the samples were taken following fume events, none of the flights triggered a "reportable incident" associated with a fume event. It can be reasonably suggested, therefore, that the occasions upon which the 332 samples were taken are representative of normal flights. In this connexion, the results are entirely in line with all previous studies demonstrating routine contamination of aircraft cabin air by organophosphates of one type or another.

(10) A 1983 Australian military report [49] concluded "Positive indications of turbine oil vapours were found in filter bags taken from the air-duct systems of suspect aircraft. Some traces of organo-phosphorous compounds, particularly the tricresyl phosphate additive in the oil, were found in the filter bags."

(11) In 2009 the Fresenius Institute in Germany carried out a wipe-sampling study on behalf of Condor [50], an airline which operated 3 aircraft types: the Boeing 757, the Boeing 767 and the Airbus 320. Wipe samples were taken at various locations in these aircraft after flight.

The samples were analysed for the presence of tricresyl phosphate (TCP). In the Boeing 757 fleet of 13 aircraft, TCP was at or above the lower detection limit in at least 5 of them. Of the Boeing 767 fleet of 9 aircraft, TCP was detected in at least 6 of them. In the Airbus 320 fleet of 12 aircraft, TCP was detected in at least 11 of them.

(12) WDR/ARD cabin swab sampling (2009) [51]. This was a private study undertaken by investigative journalist Tim van Beveren on behalf of the German ARD television network. Samples were independently analysed at the University of British Columbia. Of 33 wipe samples taken from a variety of locations within 12 different aircraft, 32 tested positive for tricresyl phosphate.

(13) On Monday 21 April 2008, the BBC *Panorama* programme carried out air and swab sampling as part of their investigation of contamination of bleed air (the programme fragment was entitled 'Something in the Air'). The BBC investigators took air samples and swabs from several different aircraft. They had the results analysed by the eminent toxicologist Prof. Christiaan van Netten at the University of British Columbia. *All* the samples were found to be contaminated by tricresyl phosphate, with the distinct "jet-oil signature"; that is, the particular distribution of relative abundances of the different isomers characteristic of the TCP used in jet oil, and Professor van Netten was filmed telling the investigators: "This proves that oil from the engine gets into the air and this is what you were breathing when you took your samples" [52].

Dr Susan Michaelis, in her PhD thesis *Health and Flight Safety Implications from Exposure to Contaminated Air in Aircraft* [6], lists no less than 55 studies over a 26-year period from 1983 to 2009 designed to elicit information on cabin air quality in aircraft. A few studies were carried out by, or on behalf of, airlines and manufacturers, and their results were never made public, from which an obvious inference may be drawn (one recalls that the Metropolitan Railway in London, the world's first underground railway, initially used steam locomotives to pull the trains, resulting in a rather foul atmosphere in the lengthy tunnels on the line, but it became fashionable to breathe this air as a remedy for a variety of respiratory complaints; no one seems to be suggesting that contaminated aircraft cabin air has any health benefits at all). Of 45 of these published studies that tried to determine the source of the pollution, 21 were able to identify the gas turbine (jet engine) lubricating oil as the source of contamination. Astonishingly, carbon monoxide (CO), whose lethality, or potential for serious health impairment, should never be doubted or ignored, was detected in at least 18 of these studies, the remainder being silent on the matter. Significantly, of the 4 studies undertaken during 2009, which sought to detect contaminants, *all* found TCP contamination [6]. There is no doubt that measuring equipment and techniques have improved over time, an observation supported by these results of 2009 (Dr Michaelis published her thesis in 2010).

8.3 Confirmed and documented occasions of bleed-air contamination

There are hundreds, perhaps even thousands throughout the world, of officially reported and documented occasions when normal flight has been interrupted by contamination of the cabin air. It is beyond the scope of this paper to list every instance. A few are given in this section.

(1) In 2003, G-MANS, a BAe 146 aircraft, was making ready for a departure from Belfast to Manchester. After an evacuation due to oil fumes, it was officially determined that oil had

been leaking from an oil seal in the auxiliary power unit (APU). Significantly, the UK Air Accident Investigation Branch commented "minor leakage is not atypical of this type of APU." The official report [53] declared that oil had been leaking into the internal air for some time prior to the incident (an APU is a small jet engine in the tail of the aircraft and its sole purpose is to provide supplementary electrical power and bleed air, as well as a source of such power and air when the main engines are not running).

(2) In another case in 2003, G-MIMA, also a BAe 146, arrived in a very distressed state of smoke and oil fumes at Gatwick Airport. The report determined that failure of an oil seal had "allowed hot engine oil to enter the bleed air airstream and subsequently enter the cabin as smoke/fumes" [54].

(3) In 2006, a Boeing 757 from Madrid to Heathrow declared an emergency due to smoke in the aircraft. The subsequent report found that engine oil had leaked into the breathing air of the aircraft [55].

(4) G-BYAO, a Boeing 757, was flying from Newcastle to Larnaca in 2006 when the cabin air was contaminated by blue smoke and the aircraft diverted, declaring "mayday". The cause was ultimately determined to be "a fractured number 1 bearing seal ring on one of the main engines, which had allowed engine oil to leak into the compressor airflow path and into the bleed air system, which provides air to the cabin air conditioning system". However, before that finding emerged the operator, Thomsons, had instructed the crew to "conduct a proving flight" with the empty aircraft: its take-off had to be aborted due to further oil fumes entering the cabin [56].

(5) In 2012, a Thomas Cook Boeing 757 (G-FCLA) flying from Dalaman, Turkey to Glasgow landed uneventfully. As disembarkation was about to commence, with the aircraft's APU providing electrical power and air-conditioning, smoke and an oil smell were perceived within the aircraft. They quickly became so bad that the captain ordered an emergency evacuation, which deployed all the slides. The source of the oil was determined to be a leaking oil seal on the APU. That did not present a problem to Thomas Cook Airlines, who immediately repacked all the slides and scheduled the aircraft to operate another flight the following morning to Tenerife, with the APU placarded as "inoperative". The AAIB report [57] takes up what happened next: "as the aircraft reached its cruise altitude, both pilots started to feel unwell, with some light headedness and dizziness. They donned their oxygen masks, made a "PAN" call and initiated a diversion to Manchester". The AAIB observed that the likely cause of this incident was the contaminated state of the ducting.

8.4 Pilot incapacitation

One particular report from the UK AAIB provides a comprehensive and authoritative archetype of many similar events. The report can be categorized as one of *devastating findings and yet unbelievable complacency*. This was a massive investigation following the near-loss of passenger aircraft G-JEAK, a four-engine jet, on 5 November 2000, due to pilot incapacitation brought on by contamination of the bleed air. The AAIB report [58] summarizes the incident as follows: "The incident occurred whilst on approach to Birmingham Airport. Following reports of unusual "oily petrol" smells in the cabin, the first officer, after visiting the cabin, started to feel nauseous. The first officer's condition began to decline to an extent that he had difficulty in

concentrating. The commander took over the handling duties and the first officer went onto 100% oxygen, and took no further part in the flight. The commander also felt 'light headed' and had difficulty in judging height during the ensuing approach and landing. Following a successful landing, the commander was able to taxi the aircraft and began to feel better." The cause of this near-disaster, according to the AAIB, was summarized as follows: "An engineering investigation revealed the presence of an oil leak from the auxiliary power unit (APU) generator cooling fan seal, which allowed engine turbine oil to enter the APU air inlet plenum chamber and, subsequently, fumes to enter the cabin via the Environmental Control System". The AAIB report takes up the history of events after the First Officer returned to the flight deck: "However, as he entered the flight deck, he began to feel nauseous. He sat in his seat but began to feel progressively worse, although his workload was low. He felt 'light-headed' and had difficulty in concentrating. He was aware of a tingling feeling in his fingertips and his arms started shaking. At about this time the commander also began to feel nauseous and asked the first officer how he felt. The first officer replied that he 'felt dreadful' and the commander looked at him and saw that his face was white and that his pupils appeared dilated. The commander took over the handling duties, instructed the first officer to put on his oxygen mask and called the SCA (senior cabin attendant) to the flight deck. When she arrived, the first officer was on 100% oxygen, his seat was well back from the aircraft controls and his hands were seen to be trembling. The commander instructed her to check the flight deck regularly during the descent and approach. Thereafter, the first officer took no part in the conduct of the flight although he was able to nod in response to the commander's questions. By this stage, the aircraft was at approximately FL70 (7000 feet on the standard altimeter setting), to the west of Birmingham, and positioning for an ILS (instrument landing system) approach to Runway 15. The commander was feeling progressively worse. He felt light-headed and recalled considering three aspects: landing, declaring an emergency and putting on his oxygen mask. However, he felt able to cope only with one decision and continued with his approach. The commander considered that he was subsequently able to complete all of the necessary checks and maintained normal radio contact with ATC. However, he reported that his heart was 'racing' and his mouth was dry. Additionally, when he became visual with the runway at about 1000 to 1500 feet above ground level, the commander seemed to have double vision and had difficulty in judging height. The aircraft was fully configured for landing with full flap and the commander kept the autopilot engaged until about 150 feet above ground level; he described the subsequent landing as 'firm'. The commander noted afterwards that it was all he could do just to land the aircraft as by now he felt very light headed and tired".

The AAIB supervised the strip-down of the air-conditioning system and goes on to report that "the subsequent engineering examination of the two condenser heat exchangers revealed evidence of semi-hard black deposits in the ducting of both units, and the presence of wet oil in the unit from the No 1 pack. Samples of the black deposits were analysed, and the findings indicated that these samples were consistent with the specification for Exxon 2380 (now BP Turbine oil 2380)".

The AAIB classified the incident as "Incapacitation due to the presence of oil contamination in the aircraft air".

While discussing other reported incidents where it was determined that pilot impairment had occurred, the AAIB report [58], in relation to a British Airways Boeing 757 (G-CPEL)

incident of bleed-air contamination, stated: "Towards the end of the flight, on approach to Heathrow, the crew missed numerous ATC calls, which prompted the controller to ask the crew 'if everything was all right'. In addition, the commander did not reduce aircraft speed to configure the aircraft for landing until reminded by the controller when the aircraft was at 3.7 nautical miles DME (distance measuring equipment)". Speed and configuration should have been set and stabilized well before 7 miles.

The report then discusses a further 8 incidents, over a two-year period (2001/2), occurring to British Airways Boeing 757 aircraft, where there was demonstrable bleed-air contamination.

In May 2001, the AAIB officially recommended that the Federal Aviation Agency of the USA work with Boeing to address the numerous instances of bleed-air contamination of the aircraft cabin air. In reply the FAA stated: "The team concluded that the root cause of the flight deck odor problem is oil leakage from the Rolls Royce RB211-535C engine".

A final, astonishing, observation made by the AAIB inspectors was: "The problem of oil contamination of aircraft cockpit and cabin air supplies has been known about for some years" [58].

8.5 Air industry literature

There are many examples of information publications by manufacturers, airlines and other industry stakeholders, which clearly acknowledge bleed-air contamination.

(1) Rolls-Royce. On 21 November 2008, Rolls-Royce updated an engineering circular entitled "Cabin Odour" [59]. The author was the Assistant Chief Engineer for the Trent 500 engine. Cabin odour problems were stated to be associated with lubricating oil leakage at the internal gearbox (IGB) seals and the fan bearing housing (FBH) seals. This document observed that the "primary cause of cabin odour events is oil leakage through the FBH air/oil seal due to inadequate sealing margin", and that "leakage from the front of the IGB is a secondary source of odour".

(2) Airbus. On 1 August 2012 Airbus issued a service information leaflet [60] to aircraft engineers entitled *Oil in the Air Conditioning System*. It instructed engineers how to trouble-shoot for fumes, smoke or oil smell in the cabin. Possible causes of the oil smells, said the leaflet, were external oil leaks or "engines". In another service information leaflet [61], entitled *Smoke and/or Oil Smell in the Cabin from the Engine*, possible causes are listed as "static seals".

(3) National Aeronautics and Space Administration (NASA). In November 2011, the Director of the US NASA Aviation Safety Reporting System (ASRS), Linda J. Connell, issued an ALERT Bulletin [62] to both Airbus and Boeing, entitled *Tricresyl Phosphate (TCP) Cabin Air Contamination*. This bulletin drew attention to a recent report in the following terms: "ASRS received a report from an air carrier Flight Attendant describing adverse health effects of cabin air contamination in an Airbus aircraft. The Flight Attendant stated the cabin air was noticeably 'foul' upon boarding and became worse en route. Reporter stated the aircraft was grounded after the contaminant tricresyl phosphate (TCP) was detected and the crew (flight and cabin) was taken to the hospital. Flight crew was reported to be on sick leave for ten days to two weeks and the reporter continues to experience adverse health-effects". The bulletin went on to detail three other similar instances of smoke or fumes in the cabin.

What is significant about this bulletin is that it was issued by an official arm of the US government and copied to Boeing, Airbus, the FAA and EASA (the European safety regulator). This bulletin called for corrective action by Boeing and Airbus. No action was, however, ever taken.

8.6 Long-term, low-level exposure

Where contamination of bleed air has been measured, generally the levels are found to be low. Nonetheless the effective toxicity defined by Winder and Balouet [1] should be taken into account. The industry stakeholders have seized on the relatively low levels to hypothesize that no adverse health effects could possibly result. The assertion of no harm is usually based on the postulate of "safe levels". Sometimes workplace exposure limits are used, although the scientific basis of such limits is often unclear. Such a stance ignores the obvious ill health in aircrew that is constantly emerging, notably in the absence of any other plausible or possible cause. Moreover, the industry ignores the fact that aircrew live with this low-level contamination every working day. If we recall that some aircrew may not be able to detoxify the poisons properly between flights due to their particular genetic constitution, then a *potential* formula emerges which dictates that if a crew member is unable to detoxify between flights at a rate equal to or greater than the rate of re-intoxication, ill health is inevitable. Support for such a formula is found in the theory of long-term–low-level (LTLL)-induced ill health. Exposure to a poison can be either an acute single high dose, or exposure can take place at relatively low levels over a prolonged interval of time. There is now a growing body of scientific literature [63–65] showing that LTLL has been overlooked as causative of neural damage.

Mackenzie Ross has pointed out that "The majority of well-designed studies found a significant association between low-level exposure to OPs and impaired neurobehavioral function which is consistent, small to moderate in magnitude and concerned primarily with cognitive functions such as psychomotor speed, executive function, visuospatial ability, working and visual memory" [63]. In 2005, Abou-Donia published a paper entitled "Organophosphorus ester-induced chronic neurotoxicity" [64]. He described how low-level organophosphate intoxication over a period was very much more effective in producing the delayed and chronic versions of organophosphate-induced neurotoxicity than an acute single dose. Kaplan and co-researchers reported persistent long-term cognitive dysfunction and defects in concentration, word-finding and short-term memory in individuals who had been exposed to *low* levels of the organophosphorus insecticide chlorpyrifos [67].

In 2003, research scientists belonging to the US Navy published a paper entitled "Known harmful effects of constituents of jet oil cabin smoke" [5], which concluded that "doses below the recognized threshold for acute exposure could cause an organophosphate-type poisoning if the exposure was long in duration". In addition, *in vitro* studies with mouse brain cortex demonstrated that glutamate (an important neurotransmitter) signaling was impaired by tri-*ortho*-cresyl phosphate concentrations as low as one nanomolar (a thousand millionth of a mole per cubic decimetre). The authors were able to conclude that neurite dysfunction was observed after extremely low doses before structural changes were observed [69].

8.7 Industry complacency and regulator connivance

Scrutiny of the evidence relating to contamination of aircraft cabin air and ill-health effects would not be complete without examination of industry and regulatory efforts to practise and enforce occupational health and safety.

The CAA

Within the UK, the regulator is the Civil Aviation Authority (CAA). Its Chief Medical Officer is Dr Nigel Dowdall. Before taking up this post, he was Chief Medical Officer at British Airways. Under the terms of a 2009 memorandum, the CAA agreed with the UK Health and Safety Executive (HSE) that the CAA would become the enforcer of the occupational health and safety of aircrew [70]. There is no evidence that it is performing that function.

Prior to 2013, the CAA maintained a database of pilots who had "lost their medical" and "who, and, or, whose doctors, alleged that their condition was associated with the toxicity of the aircraft cabin air". Prior to 2013, enquiries under the Freedom of Information (FOI) Act freely elicited the numbers. A similar FOI request made after that date drew the reply that "There are no persons who had lost their medical certification and who, and, or, whose doctors, alleged that their condition was associated with the toxicity of the aircraft cabin air". Upon an appeal under FOI legislation, the CAA reply was upheld as being correct, because the CAA had simply abolished the register. It was not even available as a frozen archive. The CAA had rid itself of the only means of knowing whether the "problem" was growing. This deliberate act can hardly be consonant with the duties required of an industry regulator and enforcer of occupational health and safety of aircrew.

Prior to 2006, the CAA kept a register of "fume events" categorized according to aircraft types. This allowed the worst offending aircraft types to be identified. That register no longer exists, not even in archival form.

On 17 April 2008 Dr Dowdall wrote an internal CAA memo to Sandra Webber, who was at the time head of consumer support at the CAA. The memo demonstrates what could be described as connivance with the industry in seeking to oppose and silence those who were genuinely concerned about the toxicity of bleed air and ill health. The memo states: "Dear Sandra, As I'm sure you have noticed lobby groups such as GCAQE (Global Cabin Air Quality Executive) and the Aerotoxic Association have been particularly active recently resulting in a number of newspaper articles, parliamentary questions etc. We now have the [BBC] Panorama programme scheduled for Monday evening." The memo then goes on to mention discussions on the subject by Dr Dowdall with Patrick Spink (British Airways press office) and Elaine Millar (UK government affairs). Then the memo seeks to achieve the following startling aim: "At present this issue has not gained momentum, however it is becoming a topic of increasing interest and there is a growing need to act as soon as possible to counter the impact of lobbying groups."

In 2002, the UK CAA issued a communication to airlines. It was called "Flight Operations Department Communications (FODCOM) 21/2002"—UK Civil Aviation Authority, Safety Regulation Group, Aviation House, Gatwick, West Sussex, England. It recommended that airlines ensure that pilots are instructed to wear oxygen masks immediately upon becoming

aware of an oily smell. In the absence of a proper detection system aboard airliners, it is hardly reassuring to have to depend upon the ability of the aircrew to detect "an oily smell". It has often been observed, even in official regulator, airline and government statements, that the only detection system available to save an airliner and its occupants from disaster is the captain's nose. Strange, therefore, that there is no specific test or medical requirement for a pilot to have a sense of smell at all to pass his or her initial or annual Class 1 medical certificate [71].

FAA and EASA

In 2007, the UK accident investigators, AAIB, issued a report on a serious case of pilot incapacitation resulting from cabin air contamination [70]. It recommended that the FAA (USA regulator) and EASA (pan-European regulator) require the installation of flight-deck detection and warning systems for oil smoke/mist, given evidence of compromised flight safety when pilots are exposed to oil fumes. No action was, however, taken.

British Airways

In the issue of British Airways' *Cabin Crew News* (a newsletter issued to all cabin crew) of 3 October 2003, Dr Michael Bagshaw, who was then Chief Medical Officer at British Airways and who is now Medical Advisor at Airbus, wrote (*sic*):

> Medical—Fumes in the Cabin
> Following a number of recent incidents in which oily smells have been detected in the aircraft cabin, it is recognised that there is understandable concern about the possible toxicity of these fumes. The preferred oil on the [Boeing] 757/767 fleet is Mobil Jet Oil II. This contains synthetic hydrocarbons and additives, including an organophosphate known as Tricresyl Phosphate (TCP), which acts as a high-pressure lubricant. Engine lubricating oil contains around 3% TCP. TCP is a toxic mixture that can cause a wide array of transitory or permanent neurological dysfunctions when swallowed. However there have been no recorded cases of neurological harm in humans following dermal or inhalation exposure. This means that the substance can be potentially harmful if swallowed in large enough quantity, but is not harmful if absorbed through the skin or breathed in. Exposure to large doses of some organophosphates by skin contact, inhalation or by swallowing may cause adverse effects on the nervous system. However not every organophosphate compound will cause these problems, including those used in jet engine oil.
>
> Dr Michael Bagshaw

This statement is misleading in many respects. The assertion that tricresyl phosphate (TCP) "is not harmful if absorbed through the skin or inhaled" is at odds with Mobil's own warning label on the can of oil. This label, as we have seen above, displays a prominent warning that "prolonged breathing of oil mist, or prolonged or repeated skin contact can cause nervous system effects". It also runs counter to Mobil's assertions in its Safety Data Sheet (SDS) [26]: "Danger of adverse health effects by prolonged exposure"; and "This material is considered to be hazardous according to regulatory guidelines". Dr Bagshaw's assertion also ignores the

prolonged, repetitive and cumulative exposure inherent in the working lives of aircrew. Highly significant is that this BA assurance to aircrew is contrary to the official guidance provided by the UK Government under the *Approved Supply List* (7th edition) for the purposes of the *Chemicals (Hazard Information and Packaging for Supply) Regulations 2002* [9]. This list is the official UK document complying with the Dangerous Substances Directive [67/548/EEC]. In this list the *ortho* content of TCP is classified as "toxic", and the *para* and *meta* content of TCP is classified as "harmful".

Two facts are conspicuous by their absence from Dr Bagshaw's advice. Firstly, there is no mention of the organophosphates present in the hydraulic oil, which is a permanent contaminant of aircraft cabin air. Here the *Approved Supply List* [9] classifies them as "harmful". Secondly, the advice ignores the scores of BA's own aircrew, known to BA, who have been grounded by symptoms consistent with organophosphate poisoning.

However, the most eloquent criticism of Dr Bagshaw's attempt at reassurance is the observation that it had to be written at all.

Boeing

That Boeing is well-aware of the toxicity of bleed air is indisputable. It was sued in court in the USA by a flight attendant, Terry Williams, and settled out of court in 2010 [66].

On 26 October 2007, George Bates, a senior Boeing engineer in the Environmental Controls Department, wrote an e-mail to colleagues, under the subject-matter *Toxicity*, in which he wrote:

> Hydraulic Mist is another toxic product I refuse to get involved with, even though our recirculation filters have the capability to coalesce the mist … I will add that the propulsion folks do not account or certify the bleed-air quality they feed to us. John Klym was the most recent to try to get the propulsion folks to step up to owning their system by-products. All he got was the run-around like I got in 2000 for the 747-400. … The engine specs are the hole no one has addressed … Given the number of [contamination] events for the 757/RB211-535C and 535E engines resulting from failed Fan and Forward IFC Bearing Seals allowing oil by-products in the bleed ducts, I would have thought that the FAA would have forced the issue … With all the diversions (about 1 every 2 weeks) and return to base events due to Haze in the Cabin, I would have thought the FAA would have made the Engine Manufacturers address this by now. Some of the 757 events have been pretty significant in that the crew reported blue smoke with defined waves in the smoke. The visibility was limited so that the attendants in the aft galley could not see to the mid-cabin over-wing exits. This is more than a light haze that we debate endlessly about for smoke evacuation. Who knows what the by-products are in hot synthetic Turbine Oil. The Material Data Sheet has warnings about skin contact and breathing the fumes of the oil, let alone the partial combustion products … Bottom line is I think we are looking for a tombstone before anyone with any horsepower is going to take interest**.**

Such was the complacency and sheer politico-industrial clout of the air transport industry that when this e-mail came to light, any effect it brought to bear was ignored by governments, regulators and manufacturers alike.

Airbus

For more than 3 years, since 2012, Airbus has been flying around one of its own test airliners fully equipped with computers and monitors, performing real-time monitoring of cabin air quality. No results have ever been made public, leading to speculation that if the results were good news, they would have been published before now.

9. Conclusion

It is quite obvious that the fundamental issue is not the toxicity of lubricating oil, of hydraulic fluid, nor of de-icing fluid. In fact it could be said that they all do their job very well. The fault lies in the *system* of aircraft pressurization and ventilation which, by deliberate design, allows such toxicity to reach the lungs of the occupants. It is important to bear this in mind when criticism seems to emerge of the oil and fluid products and their manufacturers. The same is true of the engine manufacturers and their products. Their sole aim must be to provide efficient and long-lasting engines.

Exxon Mobil states in its safety documentation "This product is not expected to produce neurotoxic effects under normal conditions of use." This may actually be true or, on the other hand, it may be a carefully crafted statement designed to be all things to all men.

For an industry that prides itself in the supremacy of safety and embraces the precautionary principle, the use of bleed air for the ventilation of aircraft seems incomprehensible. Aviation has a fundamental golden rule—often referred to as "Murphy's Law", but with less levity it may be known as the precautionary principle—"if it can happen, it will happen". If a wing breaks off because a bolt was inserted the wrong way round, the bolt would be redesigned so that it could never be inserted the wrong way round.

The industry and its regulators are well aware of the organophosphate contamination of bleed air. They are also aware of serious symptoms reported by aircrew, and that such symptoms are consistent with organophosphate poisoning. Their defence, without a shred of evidence to support it, is that the detected low levels of toxicity could not possibly cause the reported symptoms.

Such a defensive hypothesis is untenable for four reasons:

Firstly, some aircrew have no ability to detoxify, while others can be classified as "poor metabolizers". To a person with no ability to detoxify, even the slightest contamination is dangerous.

Secondly, such measurements as have been reported to date may underestimate the actual contamination level. While a very high proportion of all air and wipe samples ever taken have proved positive for organophosphate contamination, none have tested the air quantitatively as well as qualitatively, continuously, from engine startup to engine shutdown on the same flight, without interruption. All tests to date have deployed small portable battery-powered detectors sucking in the cabin air through a capture medium. The medium is subsequently analysed in a laboratory. It is, accordingly, impossible to determine whether such levels of contamination as have been detected are the result of a single quasi-instantaneous release of contamination, or the accumulation of background levels of contamination deposited over the entire time the detector was running. The battery power available in such hand-held detectors has been incapable of lasting a whole flight, even in short-haul operations. Such levels as have been reported have demonstrated a wide variation. As a consequence, one cannot be sure if the values detected are

the "peaks" or the "troughs" of a constantly variable rate of contamination. One thing is for sure: the levels that have so far been detected represent the minimum, and the maximum may as yet be unknown.

Thirdly, long-term–low-level (LTLL) contamination, such as that to which aircrew are exposed, has been shown to be more injurious than short-term acute exposure. Aircrew breathe the low-level toxicity every minute of their working days. When an aircraft lands the passengers disembark, but the crew turn around and do it all over again, time and time again. Perhaps there has too much emphasis on fume events, while the real damage is being done by LTLL.

Fourthly, the industry and its regulators do not maintain a database of aircrew claiming to have suffered health impairment due to the toxicity of bleed air, thus rendering any denial of causality vacuous.

The failure by the industry to fit permanent hard-wired detectors for bleed-air contamination would seem, at first glance, to be something of a puzzle. Perhaps the installation of such equipment would be wholly unpalatable for the industry. It would require the setting of a "minimum safe level", below which the detector would not set off an alarm. This might itself present a problem because in 1990 the World Health Organization (WHO) stated: "with regard to organophosphate exposure, there is no safe level".

In the light of available evidence on the seemingly perpetual toxic contamination of aircraft cabin air, in the event that such detection systems were to be fitted, no aircraft would ever be able to leave the ground because of the din of warnings going off.

On 12 December 2012, a 43 year-old British Airways pilot passed away in a distressed state of health, which had existed for at least 18 months. At autopsy, his nervous system tissues, brain and peripheral nerves were scrutinized and found to fully support a largely anamnestic diagnosis made some months earlier. Moreover, the *post-mortem* findings supported the results of a [then] newly developed test of serum autoantibodies specific to seven nervous system proteins. Differential diagnosis, *post mortem*, failed to provide an alternative cause of his illness. Morbidity pathways were clearly established. Substantial brain and heart injury were documented. The results were carefully published in a peer-reviewed scientific paper [31]. No criticism of the paper has ever emerged.

It has to be recognized that there are clearly powerful commercial interests opposed to any official acknowledgement of a link between bleed-air toxicity and aircrew—and passenger—ill health, even when it might be considered to be blindingly obvious:

- Governments will seek to protect huge industries;
- Regulators will connive with the regulated and will never admit they got it wrong;
- Airlines are hell-bent on providing mass air travel at the cheapest prices, even at the expense of crew ill health and a serious risk to flight safety;
- Manufacturers will be reluctant to change, with consequent heavy development costs;
- Insurers of airlines and manufacturers will seek to delay the floodgates of claims;
- Lastly, the trades unions throughout the world who, it might be thought, would have "done something about it before now", represent thousands of aircrew, but also represent at least ten times more aerospace workers who make the aircraft and the engines.

In 1979, a hitherto secret, and shocking, memo from within the tobacco industry emerged. Misleadingly titled *Smoking and Health Proposal*, and written by an official of Brown & Williamson Tobacco Company, it disclosed many of the tactics employed by "big tobacco" to

counter "anti-cigarette forces". In one of the paragraphs, about how to counter "anti-smoking campaigners and lobby groups", it revealed "Doubt is our product, since it is the best means of competing with the 'body of fact' that exists in the mind of the general public. It is also the means of establishing a controversy."

This is an aspect of what is often referred to as agnotology—the science of ignorance. Stanford University academics R. Proctor and L. Schiebinger assert that "ignorance is often more than just an absence of knowledge; it can also be the outcome of cultural and political struggles. Ignorance has a history and a political geography, but there are also things people don't want you to know. 'Doubt is our product' was the tobacco industry slogan" [73].

Our parting observation concerning contaminated bleed air and crew ill health is this: *The Westgate case is the elephant in the room*.

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