Aerotoxic Syndrome
An Emerging Risk?
Aerotoxic Syndrome: A Collection of Articles from BC Disease News
BC Legal's Reporting on a Possible Emerging EL/PL Risk

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This publication presents a collection of BC Disease News articles on so-called ‘aerotoxic syndrome’, which we have been monitoring as an emerging health risk for more than 5 years (since September 2014). Naturally, there is some repetition, as we consistently refer back to previously written articles in consolidation of present day knowledge. Nevertheless, this Guide is a comprehensive examination of condition, potential causes and scope for future EL/PL claims.

Feature:
Are Aerotoxic Syndrome Claims in the Air? – Part 1
Edition 64 of BC Disease News (19 September 2014)

INTRODUCTION
A developing field of interest for disease practitioners is so-called ‘aerotoxic syndrome’, a condition that is said to arise from exposure to contaminants in the air of aircraft cabins that have their source in aircraft engines. This series will explore the background to the issue, whether it can be said ‘aerotoxic syndrome’ exists and the obstacles any future claim will have to surmount.

The first part of this series focuses on the background to the issue and whether there is such a thing as ‘aerotoxic syndrome’.

BACKGROUND
As de Graaf et al explain, although the air from the turbine engines of commercial jet aircraft is used primarily for the propulsion of the aircraft, some is also used to refresh and replenish air in the cabin of the aircraft. The air in the cabin (including the cockpit) is a mixture of air from the outside environment, recirculated filtered air from the cabin, and air bled off from the engines – known as ‘bleed air’. This is the case with almost all modern commercial aircraft, although a notable exception is the new Boeing 787 Dreamliner, which does not draw any cabin air from the engines. As a result of oil-seal leakage in the engine system and auxiliary powers units (APU) (which provide power to aircraft when the engines are not running), engine oil and hydraulic fluid, additives present in these products and the products of their pyrolysis (thermal decomposition) can contaminate the ‘bleed air’ entering the cabin air and consequently be inhaled by passengers and crew alike – these are ‘fume events’. These contaminants in the air include volatile organic compounds (VOCs), low molecular weight organic acids, esters, ketones, and organophosphates. Organophosphates are the chief concern – and in particular tricresyl phosphate (TCP) isomers – since they are highly toxic and can result in neurotoxicity (nerve damage), causing pain and serious paralysis of limbs, and bowel and lung disorders, often with a degree of permanent disability.

It is said the presence of organophosphates in the cabin air environment can result, both in cases of short and long-term exposure, in ‘aerotoxic syndrome’. The syndrome is especially likely to affect cabin crew owing to the statistical probability of them being exposed more frequently for longer periods of time, but passengers can also be affected.

While all aircraft employing bleed air systems may be responsible for fume events – and therefore contaminants in the air – particular concern has arisen in relation to the BAe 146 series of aircraft (which includes the subsequent Avro RJ series) and the Boeing 757 series of aircraft (particularly those 757s fitted with Rolls Royce RB211-535C engines).

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3 Committee on Toxicity, ‘Statement on the Review of the Cabin Environment, Ill-Health in Aircraft Crews and the Possible Relationship to Smoke/Fume Events in Aircraft’ (COT Statement 2007/06, September 2007) [3].
4 Ibid [37].
5 House of Lords Science and Technology Committee, ‘Air Travel and Health: An Update’ (1st Report of Session 2007-08, HL Paper 7) [4.39].
7 Aerotoxic Association (n 2) ‘Can Anyone be Affected’.
8 COT (n 3) [20], [66].
WHAT IS ‘AEROTOXIC SYNDROME’?

‘Aerotoxic syndrome’ is an unrecognised medical term introduced by Winder and Balouet in 2000. According to the Aerotoxic Association, which supports those allegedly affected by the condition, the syndrome can entail the following symptoms, either acutely or chronically:

- Fatigue – feeling exhausted, even after sleep;
- Blurred or tunnel vision;
- Shaking and tremors;
- Loss of balance and vertigo;
- Seizures;
- Loss of consciousness;
- Memory impairment;
- Headache;
- Tinnitus;
- Light-headedness, dizziness;
- Confusion/cognitive problems;
- Feeling intoxicated;
- Nausea;
- Diarrhoea;
- Vomiting;
- Coughs;
- Breathing difficulties (shortness of breath);
- Tightness in chest;
- Respiratory failure requiring oxygen;
- Increased heart rate and palpitations; and
- Irritation of eyes, nose and upper airways.

HOW MANY ARE AFFECTED?

If ‘aerotoxic syndrome’ is a condition, how many are affected by it? As it has already been noted, air crew are statistically most likely to be affected. With respect to passengers, in 2011 the United Kingdom’s Civil Aviation Authority (CAA) figures showed that out of a total of 48,000 written passenger complaints in the 10 years from January 2001, just 244 (0.5%) were categorised as medical. Of those, the main health problems were pregnancy issues, skiing injuries, infectious diseases, allergies (typically from peanuts), food poisoning and passengers being scalded by hot drinks. Therefore a low number of passengers appear to be affected by ‘fume events’.

DOES ‘AEROTOXIC SYNDROME’ EXIST?

‘Aerotoxic syndrome’ is not presently a recognised medical condition. However, are ‘fume events’ capable of inducing ill health as a result of exposure to organophosphates?

The issue received some initial consideration by the House of Lords Science and Technology Committee in 2000. The Committee concluded that there was no evidence of harmful contaminants (or harmful levels of contaminants where they were present). Thus, concerns about significant risk to the health of passengers and crew were found not to be substantiated. Despite that finding, it recommended that airlines should continue to assess air quality.

In February 2004, the CAA published a detailed review of the toxicology of pyrolised aircraft engine oil and measurements of cabin air quality in response to a number of symptoms and incidents reported by aircraft crew. The CAA concluded that there were no identifiable components of pyrolysed engine oil which had the potential to cause the symptoms reported by

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11 House of Lords Science and Technology Committee (2000) (n 6) [1.68]-[1.73].
12 ibid [5.48]-[5.51]
In 2007, the Department for Transport asked the Committee on Toxicity (COT) to undertake an independent scientific review of data submitted by the British Airline Pilots Association (BALPA) relating to concerns about the possible health effects from ‘fume events’ on commercial aircraft. Pilots had estimated that ‘fume events’ occurred on approximately 1% of all flights. However, the COT investigation found that ‘fume events’ occur on approximately 0.05% of all flights (that is 1 in 2000 flights), although that might vary depending on airframe, engine type and servicing. It is worth noting by way of an aside that more recent Department for Transport figures show that in 2010 there were ‘fume events’ on just 0.018% of flights (that is one in 5,555 flights).\(^\text{15}\) The COT considered as a general point that it would be prudent to take appropriate action to prevent ‘fume events’.\(^\text{16}\) However, it concluded, on the basis of the available evidence, that there was no causal association between ‘fume events’ (either generally or following incidents) and ill-health in any commercial aircraft crews. But it did note a number of ‘fume events’ where the temporal relationship between reports of exposure and acute health symptoms provided evidence that an association was plausible, though it did not say it was established.\(^\text{17}\) Moreover, it recommended further investigation of neuropsychological impairment in pilots.\(^\text{18}\)

The AHWG also commissioned the Institute of Occupational Medicine (IOM) to determine whether there was contaminant residue which had accumulated over time on the internal surfaces of cabins. The study focused on the residue of 4 organophosphates – TCP, TBP, butyl diphenyl phosphate (BDPP) and dibutyl phenyl phosphate (DBPP). A total of 17 aircraft, 5 airport-based vehicles and 2 offices were evaluated, with a total of 86 locations sampled. The report was published in March 2012.\(^\text{23}\) TCP, BDPP and DBP measured in the surface deposits from aircraft cockpits were generally higher than those in offices, with the exception of the BAe 146. The levels of TCP or other TCPs were found. TBP was detected more routinely, but not in the majority of samples; TBP levels were highest during first engine start.\(^\text{21}\) Levels of other substances, such as carbon monoxide, toluene and xylenes, were comparable to levels of indoor pollutants seen in domestic homes.\(^\text{22}\)

The report, published in March 2011, found that no guidelines or standards were exceeded. Specifically, the Workplace Exposure Limits (WELs) established by the Health and Safety Executive (HSE) for organophosphates – including tri-orthocresyl phosphate (TOCP), the most toxic form of TCP, and tributyl phosphate (TBP) – were not breached.\(^\text{20}\) Indeed, it was noted that in 95% of the cabin air samples, no detectable amounts of TOCP or other TCPs were found. TBP was detected more routinely, but not in the majority of samples; TBP levels were highest during first engine start.\(^\text{21}\) Levels of other substances, such as carbon monoxide, toluene and xylenes, were comparable to levels of indoor pollutants seen in domestic homes.\(^\text{22}\)

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- Proximity to oil sources;
- The presence of electronic equipment;
- Cleaning regimes;
- External sources of organophosphates; and
- Lighting levels, which may promote the decomposition of the organophosphate compounds.


\(^{14}\) Ibid Chapter 1, Section 5.

\(^{15}\) Department for Transport (n 10).

\(^{16}\) COT (n 3) [85].

\(^{17}\) Ibid [86].

\(^{18}\) Ibid [94].


\(^{20}\) Ibid.

\(^{21}\) Ibid.

\(^{22}\) Ibid.

The amount of TCP measured was highest in planes. Estimates were then made of the theoretical maximum airborne concentrations of TCP and TBP; these estimated concentrations were low and comparable to earlier studies.

In April 2013, Schindler et al investigated the levels of organophosphate metabolites in aircrews by analysing urine samples.24 Levels of some organophosphates – but not TCP – were found to be significantly higher compared with unexposed persons. None of the samples contained TOCP metabolites above the limit of detection. It was concluded that elevated metabolite levels could be due to ‘fume events’ or due to release of commonly used flame retardants from the highly flame protected environment in the aircraft. In any event, there was a slight occupational exposure of aircrews to organophosphates.

In December 2013, after discussing the issue and considering the research, the COT adopted an agreed position paper with respect to cabin air. It concluded:

‘More generally, the Committee considers that a toxic mechanism for the illness that has been reported in temporal relation to fume incidents is unlikely. Many different chemicals have been identified in the bleed air from aircraft engines, but to cause serious acute toxicity, they would have to occur at very much higher concentrations than have been found to date (although lower concentrations of some might cause an odour or minor irritation of the eyes or airways). Furthermore, the symptoms that have been reported following fume incidents have been wide-ranging (including headache, hot flushes, nausea, vomiting, chest pain, respiratory problems, dizziness and light-headedness), whereas toxic effects of chemicals tend to be more specific. However, uncertainties remain, and a toxic mechanism for symptoms cannot confidently be ruled out’.25

The CAA endorses this position.26

In January 2014, Schindler et al sought to determine whether aircraft technicians were exposed to organophosphates. Levels of metabolites were established from urine samples. Again, no TCPs were detected, but there were significant increases in other organophosphates post-shift compared with pre-shift. It was concluded there was occupational exposure to organophosphates and further studies were necessary to collect information on sources, routes of uptake and varying exposures during different work tasks, possible health effects and protective measures.27

In June 2014, de Boer et al highlighted the current gaps in understanding concerning cabin air quality.28 They noted that measured levels of possibly toxic substances in cabins contain a very high level of uncertainty and there are no proper studies considering doses during an actual ‘fume event’. Further, they noted that studies so far have failed to consider individual sensitivities.

DISCUSSION AND FURTHER DEVELOPMENTS

There does not appear to be, on the evidence so far, any proven risk to those on aircraft from organophosphate exposure as a result of ‘fume events’ – either direct or aerotoxic syndrome does not appear to exist. Indeed Professor Michael Bagshaw, a professor of aviation medicine at King’s College London, concluded in August 2013 that ‘so far as scientific evidence has been able to establish to date, the amounts of organophosphates to which aircraft crew members could be exposed, even over multiple, long-term exposures, are insufficient to produce neurotoxicity’.29

Despite the COT concluding that there is unlikely to be any toxicity arising from ‘fume events’, it has not ruled out the possibility. Indeed, there are considerable gaps in knowledge as de Boer et al have identified. Moreover, it has previously endorsed ongoing research. The COT’s position paper is unlikely to quell the view of those who firmly believe in the existence of such an illness.20

of ‘aerotoxic syndrome’. The Aerotoxic Association is one such example, as noted above. Moreover, following the release of the COT’s position paper, Dr Sarah Mackenzie Ross, 30 a consultant clinical neuropsychologist at UCL, wrote to the COT on 13 May 2014, criticising the way in which her own study had been dismissed by the Committee and its failure to properly evaluate her study. 31

With respect to further developments, on 14 July 2014, British Airways circulated an email to crew updating them on the latest evidence and providing incident reports to report any ‘fume events’/ill-health effects. 32

On 31 August 2014, it was reported that medical experts believe that former British Airways pilot, Richard Westgate, who died aged 43 in December 2012, died of sustained exposure to organophosphates. 33 Westgate suffered symptoms including headaches, loss of memory and numbness in his limbs. Abou-Donia et al carried out a number of tests pre- and post-mortem and noted:

‘Differential diagnosis showed that the work environment, clinical condition, histopathology and serum biomarkers for nervous system injury are consistent with organophosphate-induced neurotoxicity. The results also showed that exposure to organophosphates rendered the nervous system and heart tissue sensitive and predisposed to further injury’.

They concluded:

‘One is drawn to the conclusion that the most likely cause of the subject’s illness was organophosphate-induced neurotoxicity’. 34

Abou-Donia said Westgate’s case was ‘one of the worst cases of organophosphate poisoning’ he had ever seen. He said:

‘In all my specialised tests for neuro-specific autoantibodies he was the worst by far…The air transport industry constantly overlooks vital components of organophosphate poising: the combined effects of multiple compound exposure – repeated low dosage exposure is just as dangerous as a single large dose (often more so) – and the genetic predisposition to toxicity of the individual’s genes’. 35

An inquest into Westgate’s death is ongoing, the outcome of which may have significant ramifications. 36

On 4 August 2014, the Daily Express published an article where it was claimed by TravelWatch that travellers need more warnings on toxic air after 15 passengers apparently suffered stinging eyes, sore throat, coughing and nausea after a ‘fume event’ on a flight from Bulgaria to Manchester. 37

As to the position of pilots, BALPA states, in a position statement of 1 January 2013, that their thinking is aligned with that of the European Cockpit Association and Building Research Establishment (ECA). 38 The ECA’s position dates from January 2012. It says:

30 An expert heavily involved in research on organophosphate sheep dips, pesticides, Gulf War syndrome and now aerotoxic syndrome. She was one of the claimant experts in the original Organophosphate Litigation concerning sheep dips, which concluded in the Court of Appeal as Snell v Robert Young & Co Ltd [2002] EWCA Civ 1644, [2003] CP Rep 25.
36 See Learmount (n 33). It is worth noting that Professor Abou-Donia and Dr Mackenzie Ross are the claimant experts of choice in the fields of organophosphate sheep dips, pesticides, Gulf War syndrome and now aerotoxic syndrome, alongside Dr Peter Julu and Dr Goran Jamal.
‘Cabin air contamination by chemicals from the engine oil, is a known problem that can cause short term health effects which compromise flight safety when a fume event occurs. ECA wants to raise awareness with regulatory bodies at EU level that improvements can be made to existing procedures. At the same time ECA calls for continuous development of new technologies that can assist in further reducing the occurrence and effects of fume events. Studies need to be run to identify if long-term health effects exist.35

Although the evidence indicates toxicity is unlikely, the COT has nevertheless advised it would be desirable to prevent ‘fume events’. What progress has been made in this regard? As to the aircraft manufacturing industry, it has largely failed to implement any preventative measures. With respect to Airbus, it appears to believe there is no issue with air contaminants. John Leahy, Chief of Operations, referred to the possibility of air cabin contaminants as ‘absurd’; he confirmed none of Airbus’ aircraft will be ‘bleed air’ free.40 Indeed, its new A350 XWB aircraft, which is in the late stages of flight testing and due to be first delivered later this year,41 has adopted a ‘bleed air’ system for cabin air.42 Meanwhile, Boeing has taken some measures, introducing the 787 aircraft which does not use ‘bleed air’ for cabin air. That said, the removal of the ‘bleed air’ system does not appear to have been related to concerns about cabin air contaminants; instead, Boeing says the system was so designed to improve fuel consumption.43 Moreover, a ‘bleed air’ system remains on Boeing’s other new aircraft – the latest incarnation of the 747, the 747-8.44 With respect to the airlines, some action has been taken. For example, in 2012, Lufthansa announced that it was working to reduce ‘fume events’ on its fleet of Airbus A380 aircraft.45 The airline said that it had experienced an unusual number of such events, particularly when outbound from Singapore – the airline said it suspected that climate conditions might have had a causal role. It insisted, however, that ‘fume events’ do not cause health problems and that measures were being taken to avoid unpleasant odours in the cabin. It subsequently installed protective covers in front of the ‘bleed air’ inlets inside the Rolls Royce Trent 900 engines to prevent ‘fume events’. It also commissioned the installation of sensors in cockpits to record concentrations of substances in the cabin air once pilots notice an unusual odour. Could it be that, in the fullness of time, the positions adopted by manufacturers and airlines have an impact on any future claims? We will consider this issue in the next part of the series.

CONCLUSIONS

Current evidence indicates that the occupants of aircraft can be exposed to organophosphates. The apparent source of this exposure is contaminated air as a result of ‘fume events’. However, the levels of contaminants are not anything like the known hazardous levels. Accordingly, the current medical and scientific evidence indicates that ‘aerotoxic syndrome’ does not exist. If it does exist, it has not yet been proven. There remain considerable gaps in our knowledge, particularly the levels of contaminants during actual ‘fume events’. Could it be that these as yet unknown levels are high enough to induce neurotoxicity? Perhaps legal claims are in the air after all. With this in mind, in the next part of the series we consider the obstacles that would have to be surmounted in any future claim.

Feature:
Aerotoxic Syndrome Claims: in the Air? – Part 2
Edition 65 of BC Disease News (26 September 2014)

INTRODUCTION

A developing field of interest for disease practitioners is so-called ‘aerotoxic syndrome’, a condition that is said to arise from exposure to contaminants in the air of aircraft cabins that have their source in aircraft engines. This feature series explores

the background to the issue, whether it can be said that ‘aerotoxic syndrome’ exists and the obstacles any future claim will have to surmount. The first part of this series focused on the background to the issue and whether there is such a thing as ‘aerotoxic syndrome’. It concluded that, on the current evidence, it cannot be said that ‘aerotoxic syndrome’ exists. Nevertheless, the possibility of claims is clear. The second part of this series therefore considers the obstacles that future claims will have to surmount to succeed.

THE APPLICABILITY OF ENGLISH LAW

Intuitively, any claim brought by an employee member of aircrew for organophosphate-induced neurotoxicity is likely to be presented as a claim for negligence or breach of statutory duty. But a preliminary issue that requires determination before those claims are considered is whether English law applies to the claim at all. After all, while some of the ‘fume events’ that are alleged to result in neurotoxicity will occur in British airspace, where they will be subject to English law,46 many will inevitably occur in international airspace or airspace belonging to another nation. Does English law apply in these situations?

One option of course is that the parties to the claim – the employee aircrew member and the employer airline – will have freely contracted in advance for any disputes to be resolved according to English law. Similarly, the parties may contract after-the-event that any dispute is to be resolved according to English law. That is an entirely acceptable resolution that is endorsed by article 14 of Regulation 864/2007/EC (the ‘Rome II Regulation’ on the law applicable to non-contractual obligations).

Beyond an agreement to be bound by English law, it might be thought that the applicable law could be derived from the Convention for the Unification of Certain Rules for International Air Carriage (1999) (the ‘Montreal Convention’),50 since it lays down rules for the carriage of persons by air. However, it is clear from the terms of the Convention that the relevant rules only apply to the passengers on board the aircraft, not the aircrew. Therefore the Convention will not regulate a claim made by a member of aircrew against their employer for organophosphate-induced neurotoxicity.

The solution to the issue appears to reside in the Rome II Regulation, which seemingly applies to events giving rise to damage that occur after 19 August 2007, where the applicable law is determined by the court on or after 11 January 2009.48 Thus, it would apply to ‘fume events’ occurring from 20 August 2007 onwards, where the applicable law was determined after 10 January 2009. Although the Regulation does not make express provision for torts committed on aircraft, it has been submitted that such torts fall within the general rule of the Regulation.49 The general rule is that the applicable law is the law of the country in which the damage occurs (manifests itself),50 irrespective of the country in which the event giving rise to the damage occurred and irrespective of the country or countries in which the indirect consequences of that event occur: article 4(1). However, where the claimant and the person alleged to be liable both have their habitual residence in the same country at the time when the damage occurs, the law of that country applies: article 4(2).51 Finally, where it is clear from all the circumstances of the case that the tort is manifestly more closely connected with a country other than that which is indicated by articles 4(1) and 4(2), the law of that country applies: article 4(3).

Applying those rules to an ‘aerotoxic syndrome’ claim, it will not always be clear where the organophosphate-induced neurotoxicity/aerotoxic syndrome occurred, since it entails a number of symptoms which do not necessarily immediately result from exposure; there may be a latency period in those cases where there is prolonged low-level exposure. It is suggested, therefore, that damage ‘occurs’ when it is medically diagnosed, which will ordinarily be in England or Wales for aircrew that live in England or Wales. Thus, English law would apply, as that is the country in which the damage ‘occurs’. In any event, it will ordinarily be the case that both the claimant member of aircrew and defendant employer airline will be habitually resident in England or Wales, therefore English law would apply. Finally, it will also ordinarily be the case that the alleged breach of duty will manifestly be most closely connected with England and Wales, where the aircraft will normally be maintained, the airline will have a base, and the member of aircrew will be based. Accordingly English law would also apply in consequence of that formulation.

46 English law would apply since Britain has complete and exclusive sovereignty over the airspace above its territory: article 1 of the Convention on International Civil Aviation (1944) (the ‘Chicago Convention’), given effect to domestically by the Civil Aviation Act 1982 and the orders and regulations made under it. A nation’s territory includes its territorial waters: article 2 of the Chicago Convention. British territorial waters extend 12 nautical miles from baselines established by Order in Council, which are normally the low-tide mark: see the Territorial Sea Act 1987.

47 Enacted in the Carriage by Air Act 1961, Schedule 18.

48 See Bacon v Nacional Suiza Cia Seguros Y Reseuros SA [2010] EWHC 2017 (QB) [61].


50 That occurrence should be treated synonymously with manifestation appears to be clear from the Supreme Court’s decision in Durham v BAI (Run off) Ltd (in scheme of arrangement) [2012] UKSC 14, [2012] 1 WLR 867.

51 The habitual residence of a company, such as an airline, is the place of its central administration: article 23 of the Regulation.
LEGAL OBSTACLES

Having determined that English law would apply to a potential claim made in England or Wales, attention can now be given to the legal issues that would have to be surmounted in particular claims. As it has already been said, such claims are likely to be for negligence and/or breach of statutory duty, both of which require proof that a duty of care was owed – that this duty was breached – and that the breach caused damage in consequence. Issues may arise with each of these elements.

DUTY OF CARE

With respect to the duty of care in negligence, it is trite law that a duty only arises when the risk of harm is reasonably foreseeable; there must be knowledge of the risk or the risk ought to have reasonably been foreseen. In an ‘aerotoxic syndrome’ claim, what the defendant airline would know or could be expected to know would depend wholly on the state of the knowledge at the material time. This was established by Swanwick J, in Stokes v Guest, Keen and Nettlefold (Bolts and Nuts Limited).

‘The overall test is still the conduct of the reasonable and prudent employer, taking positive thought for the safety of his workers in the light of what he knows or ought to know; where there is a recognised and general practice which has been followed for a substantial period in similar circumstances without mishap, he is entitled to follow it, unless in the light of common sense or newer knowledge it is clearly bad; but, where there is developing knowledge, he must keep reasonably abreast of it and not be too slow to apply it; and where he has in fact greater than average knowledge of the risks, he may be obliged to take more than average or standard precautions’.

Applying this test in light of knowledge (or lack of knowledge) about the toxicity of cabin air (discussed above), it becomes clear that it would be difficult for a claimant member of cabin crew to establish that a duty of care arises in respect of ‘fume events’, and certainly in relation to exposure to organophosphates. While it is foreseeable that ‘fume events’ occur, the evidence suggests they do not cause exposure to levels of organophosphates that can induce neurotoxicity. Since the evidence has not established that the risk of this type of harm is reasonably foreseeable, it is arguable no duty of care arises in respect of it. Moreover, even if a duty could be established, it might be possible to establish it only in relation to particular aircraft which are known to suffer ‘fume events’ more often, resulting in more frequent – and possibly higher level – exposures to organophosphates. This would likely require statistical and engineering evidence that particular aircraft or engines, or indeed specific combinations of particular aircraft and engines, are more susceptible to ‘fume events’. For example, of the aircraft identified as being of particular concern, the Bae 146 and the Boeing 757, there a multiple airframe iterations and engine options. As to the Bae 146, airframes include the Bae 146-100, -200, and -300, as well as the Avro RJ70, RJ85 and RJ100.

Engines include the Lycoming/AlliedSignal/Honeywell ALF 502R-3, ALF 502R-5, and the LF-507. With respect to the Boeing 757, airframes include the 757-200 and 757-300, while engines include the Rolls-Royce RB211-535E4, and the Pratt and Whitney PW2037, PW2040 and PW2043. It may be that only specific combinations of aircraft and engines could give rise to a duty of care.

As to statutory duties, the Control of Substances Hazardous to Health Regulations 2002 apply, since organophosphates are captured by the definition of ‘substance hazardous to health’. Regulation 7(1) of the 2002 Regulations provides that every employer must ensure that the exposure of his employees to substances hazardous to health is either prevented, or where that is not reasonably practicable, adequately controlled. Specifically, regulation 7(7)(b) provides that control of exposure will only be regarded as adequate if any workplace exposure limits are not exceeded. Workplace exposure limits have been set both for tricresyl phosphate (TCP) and tributyl phosphate (TBP) in the HSE’s EH40/2005 publication. In the case of TCP, the long-term exposure limit (over an 8-hour time-weighted average reference period) is 0.1 mg/m3; the short-term exposure limit (over a 15 minute reference period) (STEL) is 0.3 mg/m3. The long term exposure limit for TBP is 5 mg/m3 and the STEL is also 5 mg/m3. Thus, the duty is to ensure that exposure to organophosphates is prevented, or where that is not practicable, to at least below the workplace exposure limits. Although the 2002 Regulations apply, it is important to note that breaches of them from 1 October 2013 are no longer actionable, following the commencement of section 69 of the

52 [1968] 1 WLR 1776, 1783.
55 See regulation 2(1).
57 Ibid.
Enterprise and Regulatory Reform Act 2013, which amended section 47 of the Health and Safety at Work etc. Act 1974 to abolish civil liability; accordingly, the Regulations only impose an actionable duty for pre-1 October 2013 exposures.\(^{54}\)

Another statutory duty that might be applicable is regulation 6 of the Civil Aviation (Working Time) Regulations 2004, which provides that an employer must ensure that each crew member, employed by the employer is, at all times during the course of their employment, provided with adequate health and safety protection and prevention services or facilities appropriate to the nature of the employee’s employment. Unlike the 2002 Regulations, the 2004 Regulations were not promulgated under the Health and Safety at Work etc. Act 1974 and are therefore unaffected by the abolition of civil liability under that Act. However, it is not at all clear that a breach of regulation 6 of the 2004 Regulations attracts civil liability; whether it does depends upon whether the intention of the Regulations, considered as a whole and in the circumstances in which they were made and to which they relate, was to impose an enforceable duty.\(^ {55}\)

**BREACH OF DUTY**

Turning to the issue of breach of duty, if it is assumed for a moment that a common law duty of care was established, could it be said that it had been breached by the defendant airline if aircrew were exposed to organophosphates during ‘fume events’? Put differently, would it be a breach of duty not to prevent exposure to organophosphates? For example, would it be incumbent on all airlines to fit protective covers in front of the ‘bleed air’inlets inside of engines to prevent ‘fume events’, as Lufthansa has done with its A380 aircraft? Or, would it be incumbent on airlines to use oils and lubricants that do not contain TCP, such as French company Nyco’s Turbonycoil 600?\(^60\) Certainly these measures could eradicate organophosphate exposure, as the Committee on Toxicity has recommended, but would failure to adopt these measures fall short of the standard of care? Given that the evidence indicates no harmful levels of organophosphates enter the cabin air during ‘fume events’ anyway, it is certainly arguable that no particular measures need to be taken to meet the standard of care, beyond ensuring that aircraft are well-maintained so that oil seal failures – and the resulting ‘fume events’ – remain the rare occurrences that the statistics indicate they are. As the evidence presently shows that there is no established danger from ‘fume events’, it is arguable that no specific action needs to be taken in relation to them. It is by no means clear that failing to eradicate ‘fume events’ (or removing organophosphates from contaminants) would amount to a breach of duty.

With respect to the breach of statutory duties, regulation 7 of the Control of Substances Hazardous to Health Regulations 2002 requires exposure to organophosphates to be prevented or, where that is not reasonably practicable, at least kept to below the workplace exposure limits. All of the evidence indicates that the workplace exposure limits are not exceeded during ‘fume events’; so the exposure is at least adequately controlled. The issue is whether it is reasonably practicable to prevent exposure altogether? It is certainly possible in engineering terms, and it is at least arguable that it would not be unduly onerous for airlines to install protective covers inside engines or to opt for oils and lubricants that do not contain organophosphates to prevent exposure altogether. There is a prospect that this duty would be found to have been breached in any future claim.

Finally, as to the 2004 Working Time (Civil Aviation) Regulations, regulation 6 requires employers to ensure that each crew member, employed by the employer, is, at all times during the course of their employment, provided with adequate health and safety protection. Given that the evidence indicates there is no established risk of harm from ‘fume events’ generally, or from exposure to the low levels of organophosphates during ‘fume events’, it is certainly arguable that no specific protection needs to be provided to aircrew to guard their health or safety. Merely maintaining the aircraft in the ordinary way could be said to provide adequate protection.

**CAUSATION**

The greatest obstacle for any claimant member of aircrew would undoubtedly be causation. The claimant would have to prove, on the balance of probabilities, that, but for the exposure to organophosphates during ‘fume events’, they would not have sustained harm. This requires them to prove that it is more likely than not that, firstly, their condition is the consequence of exposure to organophosphates, and secondly, that they were exposed to sufficient quantities of organophosphates on aircraft (rather than from another source) such as to induce their condition. On the present evidence, proof of these elements is most unlikely. As was noted in part 1 of this series, the Committee on Toxicity has concluded that the symptoms reported following ‘fume events’ are wide-ranging and do not bear the specific hallmarks of neurotoxicity, and the evidence indicates

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\(^{54}\) See Enterprise and Regulatory Reform Act 2013 (Commencement No.3, Transitional Provisions and Savings) Order 2013, article 2(f).


that the levels of organophosphates present during ‘fume events’ are not nearly high enough (according to current knowledge) to induce neurotoxicity. That being the case, it is difficult to see how a claimant member of aircrew could prove causation based on current evidence. In short, ‘aerotoxic syndrome’ claims would be most unlikely to succeed for want of proving causation.

‘AEROTOXIC SYNDROME’ CLAIMS IN PRACTICE

Having identified the difficulties in bringing a claim for ‘aerotoxic syndrome’, it is unsurprising to note that there have been no known successful claims in the UK for ‘aerotoxic syndrome’. However, there is certainly the prospect of future claims being brought. The family of former British Airways pilot, Richard Westgate, have indicated they will be bringing a claim, and the firm of solicitors acting for them, Cannons Law, has indicated it is handling around 50 similar cases.61

Meanwhile, there has been litigation in other countries. Of particular interest is the Australian decision of the New South Wales Dust Diseases Tribunal in Turner v Eastwest Airlines Ltd.62 The claimant brought a claim against her former employer, Eastwest Airlines, for injuries allegedly sustained after being exposed to thick smoke during a 20 minute ‘fume event’ on a BAe 146 flight to Brisbane, in 1992. The claimant, who was 5 months pregnant at the time of the incident, said she experienced coughing, a burning throat, sore eyes and headache. Her cough became chronic and she brought a claim, contending her former employer had negligently exposed her to fumes, chemicals and dust, resulting in the chronic cough. The tribunal agreed, holding that the ‘fume event’ was foreseeable and that, on the facts, reasonable care had not been taken to prevent it. It also held the toxic particles of vapourised Mobil Jet Oil caused her respiratory problems. The claimant was awarded approximately $129,000 USD in total. The defendant airline appealed to the New South Wales Court of Appeal and then to the High Court of Australia, but lost both appeals.63 While this claim was successful, it is important to note that it was not presented as an organophosphate-induced neurotoxicity/aerotoxic syndrome claim. The court accepted that pyrolysed oil was harmful to the respiratory system, not that it was neurotoxic.

There has also been litigation in the United States. In 2002, a jury in Seattle rejected a claim brought by 26 Alaska Airlines flight attendants against Boeing and Honeywell, contending that exposure to toxic contaminants during ‘fume events’ on McDonnell Douglas/Boeing MD-80 aircraft had caused their illnesses, ranging from flu-like symptoms to brain damage. The jury said causation had not been established.64

In 2007, the California Workers’ Compensation Board found in favour of flight attendant, Ruth Medina, against her employer, Northwest Airlines. The claimant alleged that exposure to contaminants, including organophosphates, during a ‘fume event’ caused injury to her respiratory system, immune system, head, and neurological system. The Board agreed that exposure had resulted in respiratory illness, but not damage to the immune system or neurological system; there was no medical evidence to support damage to the immune or neurological systems. She was awarded damages for her respiratory disability and other economic losses.65 Again, like the Australian decision in Turner, the court only accepted that pyrolysed oil was harmful to the respiratory system, not that it was neurotoxic.

However, in 2011, former American Airlines flight attendant, Terry Williams, was believed to be the first person in the US to settle an ‘aerotoxic syndrome’ claim against Boeing. She contended that the aircraft manufacturer knew its MD-82 aircraft and ‘bled air’ system were defective, but did nothing to prevent ‘fume events’, or exposure to toxic contaminants, as American Airlines flight 843 taxied to the gate at Dallas Airport, on 11 April 2007. The ‘fume event’ allegedly caused her to suffer tremors, memory loss and severe headaches. Settlement was confidential.66

The preceding comparative analysis shows that there has not, so far, been a single successful claim for ‘aerotoxic syndrome’. While some courts have accepted that ‘fume events’ can result in respiratory harm, none have been prepared to accept they are neurotoxic. Further claims will, however, indubitably follow.

61 Learmount (n 33).
CONCLUSIONS

Current evidence indicates that the occupants of aircraft can be exposed to organophosphates during ‘fume events’. The apparent source of this exposure is contaminated air as a result of ‘fume events’. However, the levels of contaminants are not anything like the known hazardous levels. Accordingly, the current medical and scientific evidence indicates that ‘aerotoxic syndrome’ does not exist. If it does exist, it has not yet been proven. There remain considerable gaps in our knowledge, particularly the levels of contaminants during actual ‘fume events’. Could it be that these as yet unknown levels are high enough to induce neurotoxicity? It has been seen that, on the current evidence, claims for ‘aerotoxic syndrome’ will have to surmount considerable obstacles, particularly in respect of causation, if they are to succeed. Only further evidence in time will tell if ‘aerotoxic syndrome’ claims really are in the air.

Feature:
Aerotoxic Syndrome Revisited
Edition 86 of BC Disease News (6 March 2015)

INTRODUCTION

We initially covered the issue of ‘aerotoxic syndrome’ in detail in editions 64 (here) and 65 (here) of Disease News. We now revisit the topic in the wake of recent, extensive news coverage.

BACKGROUND

It will be recalled from our earlier articles that the issue concerns whether the occupants of aircraft are exposed to organophosphates during ‘fume events’ on aircraft, resulting in illness. ‘Fume events’ occur because of oil seal failures in the ‘bleed air’ supply, which draws hot air from aircraft engines into the cabin air supply, with the result that engine oils and lubricants – which can contain organophosphates – can contaminate the cabin air.

Having considered the array of research on this issue, we concluded that the existence of ‘aerotoxic syndrome’ was unlikely on the current evidence. The culmination of the research was perhaps best summarised by the Committee on Toxicity in 2013, when it said:

‘More generally, the Committee considers that a toxic mechanism for the illness that has been reported in temporal relation to fume incidents is unlikely. Many different chemicals have been identified in the bleed air from aircraft engines, but to cause serious acute toxicity, they would have to occur at very much higher concentrations than have been found to date (although lower concentrations of some might cause an odour or minor irritation of the eyes or airways). Furthermore, the symptoms that have been reported following fume incidents have been wide-ranging (including headache, hot flushes, nausea, vomiting, chest pain, respiratory problems, dizziness and light-headedness), whereas toxic effects of chemicals tend to be more specific. However, uncertainties remain, and a toxic mechanism for symptoms cannot confidently be ruled out’.

Alongside the evidence, we also considered the prospects of success of a claim for ‘aerotoxic syndrome’ by an employee member of aircrew against their employer. We concluded that, to demonstrate the existence of a duty of care, breach of that duty and causation would all be problematic. Indeed, we suggested that claims would be unlikely to succeed, especially on account of the issue of causation.

RECENT DEVELOPMENTS

In our earlier articles, we considered the case of Richard Westgate, a former British Airways pilot, who died aged 43 in December 2012, noting that medical experts had reported that it was believed he died of sustained exposure to organophosphates. After pre- and post-mortem tests they concluded:

Westgate had started flying in 1996 and reportedly experienced ‘fume events’ regularly on BAe ATP aircraft. By 1999, he reported feeling that his brain was slower than normal, and began to suffer confusion. By 2008, he reported numbness in his hands and feet, up to his elbows and knees. His last flight was on 2 September 2011. He was referred for in-patient psychiatric treatment, in January 2012, but discharged himself 1 month later with no diagnosis ever having been made. A fat biopsy showed the presence of organophosphate metabolites. In April 2012, he was consulted in the Netherlands. He was staggering, with a heavy gait, and had difficulty walking. There was severe and constant pain, tremors and a decline in mental acuity. An MRI scan showed no structural defects to explain his symptoms. He was found dead on 12 December 2012. Shortly before he died, it was diagnosed that he had suffered the consequences of exposure to organophosphates.

We noted, in our earlier articles, that an inquest was ongoing. There have now been developments in that case that have captured the attention of the national press.

In a report, dated 16 February 2015, Sheriff Stanhope Payne, the coroner in the case and the senior coroner for Dorset, said that his inquiries had ‘revealed matters giving rise to concern’. Specifically, he said that it was of concern that organophosphate compounds are present in cabin air and that the occupants of cabins are exposed to organophosphate compounds ‘with consequential damage to their health’. Moreover, it was of concern that no account is taken of genetic variation in the human species, such as would render individuals tolerant or intolerant of the exposure, and that there is no real-time monitoring to detect organophosphates in cabin air. Finally, it was of concern that impairment to the health of those controlling aircraft may lead to the death of occupants. The coroner said that, in his opinion, ‘there is a risk that future deaths will occur unless action is taken’ and ‘urgent’ action should be taken. The report has been sent to the Chief Executive of British Airways and to the Chief Operating Officer of the Civil Aviation Authority, who each have until 13 April 2015 to respond with details of action taken or proposed to be taken, or an explanation of why no action is proposed.

Although the report can be said to be the first official recognition in the UK of ‘aerotoxic syndrome’, it is important to note that it provides no further evidence in support of the existence of the syndrome. Moreover, it does not identify ‘aerotoxic syndrome’ as the cause of Westgate’s death. It notes that the post-mortem examinations gave causes of death of either pentobarbital toxicity (drug induced toxicity) or lymphocytic myocarditis, individually or in combination, and that lymphocytic myocarditis can result from organophosphate exposure. That is some distance from identifying ‘aerotoxic syndrome’ as the cause of death, given that lymphocytic myocarditis does not exclusively result from exposure to organophosphates, and the death may have resulted from pentobarbital toxicity in any event. Westgate’s post-mortem reported the presence of pentobarbital at a potentially lethal level. There was no evidence that Westgate had ever been prescribed pentobarbital.

Notwithstanding those criticisms of the report, it nevertheless garnered significant attention in the media. In the Sunday Telegraph’s front-page story on the coroner’s report, it was reported that Frank Cannon, the solicitor acting for Westgate’s family, said:

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69 Ibid.
71 Ibid.
72 Ibid.
73 Ibid.
74 Ibid.
75 Ibid.
76 Ibid. While it is accepted that acute exposure to organophosphates can result in myocarditis, it is less clear whether chronic, low-level exposure can induce myocarditis. See, for example: Christopher N Banks and Pamela J Lein, ‘A Review of Experimental Evidence Linking Neurotoxic Organophosphorus Compounds and Inflammation’ (2012) 33 Neurotoxicology 575.
'This report is dynamite. It is the first time a British coroner has come to the conclusion that damage is being done by cabin air, something the industry has been denying for years...I see this as an impending tsunami for the airline industry – it's been ignored for so long.'

He continues to represent in the region of 50 individuals in other alleged cases of 'aerotoxic syndrome'. Meanwhile, a spokesman for British Airways said it would consider the coroner’s report, while the CAA said it would also consider the report in detail. The CAA added that the report was 'nothing that passengers or crew should be overly concerned about'.

In other recent developments, in November 2014, a book entitled 'Aerotoxic Syndrome: Aviation’s Darkest Secret' was released. Written by former Pilot, John Hoyte, who founded the Aerotoxic Association and claims to suffer from 'aerotoxic syndrome', it is an account of the alleged evasion of the issue by the aviation industry. On 27 February 2015, a film, entitled 'A Dark Reflection' was released, which is said to be based on real events and explores the issue of 'aerotoxic syndrome'; it was produced by Tristan Loraine, another former British Airways pilot who claims to suffer from 'aerotoxic syndrome'.

On 3 March 2015, Robert Goodwill MP, Parliamentary Under Secretary of State at the Department for Transport, responded to a question from Angus MacNeil MP, who asked why it was not a requirement to monitor in real-time for organophosphates in cabin air. Goodwill responded:

'It has not been shown that cabin air exposures, either in general or following specific incidents, cause ill-health. Responsibility for introducing any additional monitoring or detection system on board a commercial aircraft is the responsibility of the European Aviation Safety Agency (EASA).'

**DISCUSSION AND CONCLUSIONS**

While recent events have attracted considerable publicity, no new scientific or medical evidence has been released that substantiates the existence of 'aerotoxic syndrome'. Publicity may, of course, increase the likelihood of claims being brought, but it will not strengthen any potential claim. In that regard, the position has not changed; claims are unlikely to succeed on the basis of existing evidence. Our conclusions reached in our initial treatment of this topic remain unaltered.

**Feature:**

**Aerotoxic Syndrome – Further Developments**


**INTRODUCTION**

We initially covered the issue of 'aerotoxic syndrome' in detail in editions 64 and 65 of Disease News, and again in edition 86. We now revisit the topic to detail further recent developments.

**BACKGROUND**

It will be recalled from our earlier articles that the issue concerns whether the occupants of aircraft are exposed to organophosphates during 'fume events' on aircraft, resulting in illness. 'Fume events' occur because of oil seal failures in the bleed air supply, which draws hot air from aircraft engines into the cabin air supply. Consequently, engine oils and lubricants – which can contain organophosphates – can contaminate the cabin air.

Having considered the array of research on the issue, we concluded that the existence of aerotoxic syndrome was unlikely on the current evidence. The culmination of the research was perhaps best summarised by the Committee on Toxicity in 2013, when it said:

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79 Ibid.
80 Ibid.
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RECENT DEVELOPMENTS – THE CORONER’S REPORT

In edition 86, we revisited the case of Richard Westgate, a former British Airways pilot, who died aged 43 in December 2012, noting that medical experts had reported they believed he died of sustained exposure to organophosphates. After pre- and post-mortem tests, they concluded:

‘One is drawn to the conclusion that the most likely cause of the subject’s illness was organophosphate-induced neurotoxicity’.85

Shortly before he died, he was diagnosed as suffering the consequences of exposure to organophosphates.86

We returned the ongoing inquest in respect of Westgate’s death in our previous article. In a report, dated 16 February 2015, Sheriff Stanhope Payne, the coroner in the case and the senior coroner for Dorset, said that his inquiries had ‘revealed matters giving rise to concern’.87 Specifically, he said that it was of concern that organophosphate compounds are present in cabin air and that the occupants of cabins are exposed to organophosphate compounds ‘with consequential damage to their health’.88 Moreover, it was of concern that no account is taken of genetic variation in the human species, such as would render individuals tolerant or intolerant of the exposure, and that there is no real time monitoring to detect organophosphates in cabin air.89 Finally, it was of concern that impairment to the health of those controlling aircraft may lead to the death of occupants.90 The coroner said that in his opinion, ‘there is a risk that future deaths will occur unless action is taken’ and ‘urgent action should be taken’.91

The report received significant media attention, as we noted at the time. For example, in the Sunday Telegraph’s front-page story on the coroner’s report, it was reported that Frank Cannon, the solicitor acting for Westgate’s family, said:

‘This report is dynamite. It is the first time a British coroner has come to the conclusion that damage is being done by cabin air, something the industry has been denying for years … I see this as an impending tsunami for the airline industry – it’s been ignored for so long’.92

He continues to represent in the region of 50 individuals in other alleged cases of ‘aerotoxic syndrome’.

Notwithstanding those comments, we offered a number of criticisms concerning the report. Firstly, we noted, importantly, that it provided no further evidence in support of the existence of the syndrome. Secondly, it did not identify ‘aerotoxic syndrome’ as the cause of Westgate’s death. Instead, it said that the post-mortem examinations gave causes of death of either pentobarbital toxicity (drug induced toxicity) or lymphocytic myocarditis, individually or in combination, and that lymphocytic myocarditis can result from organophosphate exposure.93 That was some distance from identifying ‘aerotoxic syndrome’ as the cause of death, given that lymphocytic myocarditis does not exclusively result from exposure to

86 Ibid.
87 Ibid.
88 Ibid.
89 Ibid.
90 Ibid.
91 Ibid.
93 Ibid. While it is accepted that acute exposure to organophosphates can result in myocarditis, it is less clear whether chronic, low-level exposure can induce myocarditis. See, for example: Christopher N Banks and Pamela J Lein, ‘A Review of Experimental Evidence Linking Neurotoxic Organophosphorus Compounds and Inflammation’ (2012) 33 Neurotoxicology 575.
organophosphates, and the death may have resulted from pentobarbital toxicity in any event. Westgate’s post-mortem reported the presence of pentobarbital at a potentially lethal level. There was no evidence that Westgate had ever been prescribed pentobarbital.94

RECENT DEVELOPMENTS – THE RESPONSES

The report was sent to the Chief Executive of British Airways and to the Chief Operating Officer of the Civil Aviation Authority, who each had until 13 April 2015 to respond with details of action taken or proposed to be taken, or an explanation of why no action is proposed.95

In its response explaining why no further action was proposed, British Airways said that the evidence did ‘not support the conclusion that there is a risk that future deaths will occur unless action is taken’, that the evidence the coroner was presented with (leading to his report) was ‘selective’ and that it was provided only by one interested party.96 It added that it follows the ‘guidance and research to which it is subject’ and ‘keeps abreast of research … and has in place a system of monitoring such events’.97

Similarly, in its response proposing no further action, the CAA said:

‘… there is no positive evidence of a link between exposure to contaminants in cabin air and possible acute and long-term health effects, although such a link cannot be excluded’.97

If said it would review its position following the results of the European Aviation Safety Agency’s research (on which, see below).99 Furthermore, the CAA said the evidence upon which the report was based was ‘selective’, and that it was ‘inappropriate’ to issue the report without first inviting submissions from the CAA.100

The inquest itself has not been scheduled.

OTHER RECENT DEVELOPMENTS

Aside from the ongoing Westgate case, it has now become clear that further research is to be conducted on the issue. On 17 March 2015, it was announced that the European Aviation Safety Agency (EASA) had awarded contracts for research on cabin air quality.101 Preliminary research will identify instrumentation to perform cockpit and cabin air contamination measurements, and provide initial indications of air quality levels, before a larger-scale programme of air quality testing on commercial aircraft with measurements to be taken during all phases of flight.102 The project will have a total duration of 20 months, with the final results expected in October 2016.103

Elsewhere, the union, Unite, has now waded into the issue, saying that it would like to see a public inquiry address the concerns. Len McCluskey, Leader of Unite, said:

‘Literally all of our cabin crew members will have experienced a “fume event” at some time. It occurs not regularly, but it occurs sufficiently often for people to be concerned about… Our intention is to make certain that aerotoxic syndrome doesn’t become a silent killer’.104

No action has been announced in response to Unite’s call.

95 Ibid.
96 Letter from BLM on behalf of British Airways to Sheriff Stanhope Payne (13 April 2015).
97 Ibid.
98 Letter from the CAA to Sheriff Stanhope Payne (10 April 2015).
99 Ibid.
100 Ibid.
102 Ibid.
103 Ibid.
CONCLUSION

The issue of 'aerotoxic syndrome' is now an increasingly fluid one, with developments becoming progressively frequent. While there has been no new evidence that more convincingly demonstrates the existence of the syndrome, the increasing attention given to the issue does increase the likelihood of claims being presented in the future. That is particularly so, following the involvement of Unite. Defence practitioners should ensure they are well-prepared for any claims that might now be forthcoming.

As ever, BC Disease News will update on the dynamic landscape as developments occur.

Feature:

Aerotoxic Syndrome – Further Developments

Edition 145 of BC Disease News (17 May 2016)

INTRODUCTION

For many years now, there have been concerns raised about possible health effects arising from exposure to the air in the cabins of commercial aircraft. These symptoms have been branded as 'aerotoxic syndrome'. We have previously featured this issue in editions 64 (here), 65 (here), 86 (here) and 95 of BC Disease News (here). We now revisit the issue to look at recent developments.

It will be recalled from our earlier articles that the issue concerns whether the occupants of aircraft are exposed to a cocktail of toxic chemicals during 'fume events' on aircraft, resulting in ill health. 'Fume events' are said to occur as a result of 'bleed air', which feeds the cabin via the aircraft engines, becoming contaminated by engine oil and hydraulic fluid, additives present in these products and the products of their pyrolysis (thermal decomposition). The contaminants in the air are said to include volatile organic compounds, low molecular weight organic acids, esters, ketones, and organophosphates. Organophosphates are the chief concern and in particular, tricresyl phosphate (TCP) isomers, since they are highly toxic and can result in neurotoxicity causing pain and serious paralysis of limbs and bowel and lung disorders. Exposure to such chemicals is said to give rise to a wide range of illnesses and symptoms, collectively labelled as 'aerotoxic syndrome'.

'Aerotoxic syndrome' was extensively reviewed by the UK Committee on Toxicity (COT) in 2013, which said in a Position Statement:

'More generally, the Committee considers that a toxic mechanism for the illness that has been reported in temporal relation to fume incidents is unlikely. Many different chemicals have been identified in the bleed air from aircraft engines, but to cause serious acute toxicity, they would have to occur at very much higher concentrations than have been found to date (although lower concentrations of some might cause an odour or minor irritation of the eyes or airways). Furthermore, the symptoms that have been reported following fume incidents have been wide-ranging (including headache, hot flushes, nausea, vomiting, chest pain, respiratory problems, dizziness and light-headedness), whereas toxic effects of chemicals tend to be more specific. However, uncertainties remain and a toxic mechanism for symptoms cannot confidently be ruled out'.

More recently, in May 2015, the Aviation Policy Division of the Department for Transport (DfT) stated that:

'Given the current understanding of the level of risk from fume events, DfT does not plan to undertake any additional research on this issue'.

This contrasts with the position of the European representative body for some 38,000 pilots in the EU and their national pilot associations-the European Cockpit Association (ECA), which said in December 2015:

'Cabin air contamination by chemicals from the engine and/or hydraulic oil, is a known problem that can cause serious short-term health effects which compromise flight safety when a fume event occurs. ECA calls for improvements to be made to existing flight and reporting procedures as well as introducing appropriate job specific training for all stakeholders. It calls for continuous development and application of new technologies that can assist in further reducing the occurrence and

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105 An independent scientific committee advising government on matters concerning the toxicity of chemicals.
effects of fume events. Studies need to be run to ascertain whether long term health effects do exist. In the meantime, the As Low As Reasonably Achievable (ALARA) principle should guide action and measures in this area.'

The UK Civil Aviation Authority also released a press summary, in June 2015, which stated:

'We rely on guidance from scientific experts based on the results of a number of independent studies and evidence reviews – including Government commissioned research. The overall conclusion of those studies is that there is no positive evidence of a link between exposure to contaminants in cabin air and possible acute and long-term health effects, although such a link cannot be excluded. Accordingly, we support the steps being taken by the European Aviation Safety Agency (EASA), which maintains responsibility for approving the safety of aircraft and setting aviation standards for European airlines, and is carrying out further research into cabin air quality'.

The EASA announced, in March 2015 that their 'Cabin Air Quality' research contract was awarded to a Consortium by ITEM & MHH, both research institutes based in Hannover, Germany. The research consists of a preliminary in-flight measurement campaign which is intended to put into place the adequate instrumentation to perform cabin/cockpit air contamination measurements and provide some first indications of the cabin or cockpit air quality level. It also intends to prepare a larger-scale campaign envisaged in the near future on board commercially-operated, large transport aeroplanes. The results of these studies are due to be published in October 2016.

Whilst it appears that ‘fume events’ can occur on aircraft (COT estimate the incidence at being 1 in every 2000 flights) the very existence of 'aerotoxic syndrome' and how the wide range of purported symptoms could be caused by any exposures remain controversial.

However, it was reported as recently as last week that a ‘fume event’ on a flight from Cologne to Leipzig forced the aircraft to return to Cologne for a safe landing, 25 minutes after departure.

In April 2016, a Lufthansa Airbus, flying from Frankfurt to Dusseldorf, experienced a similar incident. A smell of ‘old socks’, ‘sweaty feet’ and ‘old nappies’ was noticed by the occupants in the cabin and the passengers were instructed to close their air conditioning outlets. Subsequently one of the flight attendants noticed tingling arms and fingers and dizziness. Blood and urine samples detected a number of solvents which are said to be typical of contaminated ‘bleed air’.

**RESEARCH DEVELOPMENTS**

A study by Reneman and colleagues, published in June 2015, compared a group of 12 aircrew (with on average of 8,130 flying hours) reporting cognitive complaints to 11 controls (with on average of 233 flying hours). The participants underwent a number of tests to assess cognitive function and mood and it was found that the aircrew had a significantly higher number of tests scored in the impaired range. However, it was noted that the extent of the observed cognitive problems was quite limited. MRI scans of the participants’ brains showed defects in the white brain matter of the aircrew, which were not found in the controls. The extent of cognitive impairment was strongly associated with white matter integrity, but the estimated number of flight hours was not associated with cognitive impairment, nor with reductions in white matter structure. Limitations of this study include the small sample size and the self-selection of the aircrew participant group. As no association was observed between cognitive impairment and the number of flight hours, further studies with larger samples and more robust designs are needed to investigate whether there is a link with exposure to toxic cabin air.

Further to this, a review published in February 2016 compared the literature of exposure to various agents and health outcomes for aircraft crew and office workers. The maximum daily intakes were estimated and compared with reference values, or tolerable daily intake values. The frequency of oil and smoke-related incidents in aircraft, as well as measured concentrations of triorthocresyl phosphate poisoning (ToCP) in aircraft, were discovered in detail. It was concluded that, in

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view of the infrequent short-term exposure, which may be related to 'smoke/smell incidents' (though not necessarily to ToCP exposure), the available evidence indicated that ToCP does not pose a health risk.

A link between 'fume events' and 'aerotoxic syndrome' appears to be no further established by the scientific evidence.

INDUSTRY POSITION

Notwithstanding the increased health concerns and media interest, regarding 'aerotoxic syndrome', the aircraft manufacturing industry has been accused of largely failing to implement any preventative measures. With respect to Airbus, it appears to believe there is no issue with air contaminants. John Leahy, Chief of Operations, referred to the possibility of air cabin contaminants as ‘absurd’ – he confirmed that none of Airbus’ aircraft will be bleed air free.111 Indeed, its new A350-900 aircraft, which was first delivered on 22 December 2014 to Qatar Airways,112 has adopted a ‘bled air' system for cabin air.113 Meanwhile, Boeing has taken some measures, introducing the 787 aircraft as an example, which does not use ‘bled air' for cabin air. That said, the removal of the ‘bled air' system does not appear to have been related to concerns about cabin air contaminants; instead, Boeing said the system was so designed to improve fuel consumption.114 Moreover, a ‘bled air' system remains on Boeing’s other new aircraft – the latest incarnation of the 747, the 747-8.115

With respect to the airlines themselves, some action has been taken. For example, in 2012, Lufthansa announced that it was working to reduce ‘fume events' on its fleet of Airbus A380 aircraft.116 The airline said that it had experienced an unusual number of such events, particularly when outbound from Singapore – it said it suspected that climate conditions might have had a causal role.117 It insisted, however, that ‘fume events' do not cause health problems: the measures were being taken to avoid unpleasant odours in the cabin.118 It subsequently installed protective covers in front of the ‘bled air' inlets inside the Rolls Royce Trent 900 engines to prevent ‘fume events'.119 It also commissioned the installation of sensors in cockpits to record concentrations of substances in the cabin air once pilots notice an unusual odour.120

LEGAL DEVELOPMENTS

In editions 86 and 95, we revisited the case of Richard Westgate, a former British Airways pilot who died aged 43, in December 2012, from alleged exposure to toxic cabin air. At the time, we noted that in a report, dated 16 February 2015, Sheriff Stanhope Payne, the coroner in the case and the senior coroner for Dorset, said that his inquiries had ‘revealed matters giving rise to concern': Specifically, he said that it was of concern that organophosphate compounds were present in cabin air and that the occupants of cabins were exposed to organophosphate compounds ‘with consequential damage to their health'. He listed five concerns:

1. That ‘organophosphate compounds' are present in aircraft cabin air.
2. That people in aircraft cabins are exposed to them, with consequential damage to their health.
3. That impairments to the health of those controlling the aircraft - i.e. the pilots - may lead to the death of the occupants.
4. That there is no real-time monitoring to detect such compounds in the cabin air.
5. That no account is taken of genetic variation in humans, such as would render individuals tolerant or intolerant of exposure.

The coroner said that in his opinion, ‘there is a risk that future deaths will occur unless action is taken' and ‘urgent' action should be taken. The report was sent to the Chief Executive of British Airways and to the Chief Operating Officer of the Civil Aviation Authority, who each had until 13 April 2015 to respond with details of action taken or proposed to be taken, or an

explanation of why no action is proposed. Both British Airways and the CAA concluded that there was no positive evidence of a link between exposure to contaminants in cabin air and ill health and so no action was taken.121

The inquest itself is yet to be scheduled and the pre-inquest review has been adjourned from March 2016 to 21 July 2016. In addition to this, a second inquest is due to open into the case of Matthew Bass, an air steward for British Airways, who died suddenly, aged 34, in January 2014, after suffering unexplained health problems. The preliminary inquest, at Reading Court, heard that a specialist post-mortem found evidence of chronic exposure to organophosphates.122 123 An article in the Mirror, on 15 July 2015, reported that Berkshire coroner, Peter Bedford, said that he needed ‘independent evidence to rule out a more straightforward cause of death before bringing into question toxic air syndrome’ and that the inquest was postponed for 6 months.124 The Daily Mail reported the following day that Mr Bedford compared the death to Westgate’s.125

The pre-inquest review has been adjourned until June 2016 to allow for time to locate medical samples and instruct experts.

David Platt, QC, who acts for British Airways in this matter, was reported as saying that there is ‘no evidence that Aerotoxic Syndrome even exists’; adding:

‘No health agencies and no governments are accepting this exists. It must be seen as a highly controversial assumption to make.’

However, following a third death of an air crew member, Warren Brady, shortly after Matthew Bass, which is suspected to be related to ‘aerotoxic syndrome’, Cannons Law Practice LLP announced, in July 2015, that they were preparing a class action in England from 40 affected individuals, including both members of the crew and passengers.126

Likewise, Britain’s largest union, Unite, revealed in March 2016 that it was pursuing legal action against a number of UK airlines on behalf of 61 cabin crew, after they were exposed to ‘toxic cabin air’ while working on board aircraft. In the press statement, Unite stated that concern had been mounting over ‘fume events’ and exposure to contaminated cabin air, with the number of legal cases pursued by Unite increasing from 17 to 61, amid calls by the union for an independent inquiry.127 Howard Beckett, Unite Executive Director for Legal Affairs, said:

‘The issue of toxic cabin air is so serious that our cabin crew members are likening it to the impact of asbestos in the building industry. Increasing numbers of our members have come forward, seeking help and advice since we set up our toxic air helpline a few months ago. Some have been involved in one-off ‘fume events’ while others fear they have suffered long-term exposure to contaminated cabin air’.

Unite is also working with the family of Matthew Bass, who is represented by Leigh Day solicitors.

Unite’s comments preceded a House of Commons debate on the issue, held on 17 March 2016,128 in which Jonathan Reynolds (MP for Stalybridge and Hyde) requested an independent inquiry into the risks and hazards associated with contaminated aircraft cabin air, and that efforts should be made to install cabin air monitoring and detection systems in aircraft that operate using ‘bled air’. In the same debate, Henry Smith MP called for the Government to investigate what he termed ‘a deadly illness affecting cabin crew members and frequent flyers’. He went on to outline his concerns that the symptoms linked with ‘aerotoxic poisoning’ could be misdiagnosed as other conditions, stating:

‘The symptoms that affect many cabin crew can be confused with other conditions such as Crohn’s disease’.129

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121 Letter from BLU on behalf of British Airways to Sherif Stanhope Payne (13 April 2015).
However, Robert Goodwill, the Minister for the Department of Transport, referring to the comments made by the Coroner for the Westgate case, said that it would be inappropriate for the Government to comment before the verdict. He said that:

‘For the industry to drastically change the way the aircraft are air-conditioned, or, indeed, to change the lubricants, there would have to be clear evidence that shows that cabin air quality is harmful to crew and passengers.’

It is evident that, in the UK, despite increased attention on the issue of ‘aerotoxic syndrome’, there appears to be little progress of claims being brought against airline companies. Additionally, there appears to be little appetite for change amongst the airline manufacturers in relation to implementing any of the so-called preventative measures.

Can the same be said for other jurisdictions?

OTHER DEVELOPMENTS OUTSIDE THE UK

Outside of the UK, there have been no successful claims for ‘aerotoxic syndrome’. In the U.S., 16 U.S. Airways pilots and flight attendants sued a U.S. Airways contractor, ST Aerospace Mobile, over what they said was a failure in maintenance at its Mobile, Alabama servicing centre. They claimed that this resulted in 6 fume events on-board the same Boeing 767 from 28 December 2009 to 25 April 2010. Among the claimant’s symptoms were headaches, sore throats, eye irritations, dizziness and nausea. Some also complained of fatigue and cognitive difficulties. At least 2 of the claimants were pilots who lost their medical clearances. In August 2015, the union representing 6,900 U.S. Airways flight attendants sent a letter to FAA Administrator, Randy Babbitt, seeking an investigation of 87 purported air supply contamination events in 2009 and 2010, 41 of which were confirmed with mechanical records as oil-contamination events.

Elsewhere in the U.S., it was reported, in June 2015, that 4 flight attendants were suing Boeing, following a flight that made an emergency landing. 4 attendants were taken ill after complaining of fumes in the cabin.132 3 of them stated that tremors, neurological and memory problems prevented them from returning to work. Also, it was reported, in February 2016, that one of the 4 flight attendants has won a pay-out from Boeing after being exposed to toxic fumes and claiming to have suffered, among other symptoms, memory loss, tremors and speech and vision impairment. Whilst the extent and conditions of her out-of-court settlement remain confidential, Boeing and the airline industry maintain that cabin air, compressed air pumped or ‘bled’, from the plane’s engine, is safe. Breaches are extremely rare with short-term exposure to the tiny amounts of toxic substances posing no health risk.

In Australia, it has been accepted in the courts that ‘fume events’ can result in respiratory damage.133 The same has been accepted in California.134 Crucially, however, it was not accepted that ‘fume events’ can be neurotoxic.

CONCLUSION

The issue of ‘aerotoxic syndrome’ is becoming increasingly dominant in the media but there still appears to be no credible evidence to demonstrate the existence of the condition or to causally relate the same to alleged toxic contaminants in ‘bled air’ during ‘fume events’.

As ever, BC Disease News will update on this dynamic landscape as developments occur.

Aerotoxic Syndrome Inquest

So called ‘Aerotoxic Syndrome’ has been discussed at length in previous editions of BC Disease News. It will be recalled from our earlier articles that the issue concerns whether the occupants of aircraft are exposed to a cocktail of toxic chemicals during ‘fume events’ on aircraft, resulting in ill health.

‘Fume events’ are said to occur as a result of the ‘bled air’, which feeds the cabin via the aircraft engines, becoming contaminated by engine oil and hydraulic fluid, additives present in these products and the products of their pyrolysis.

(thermal decomposition). The contaminants in the air are said to include volatile organic compounds, low molecular weight organic acids, esters, ketones, and organophosphates. Organophosphates are the main concern and, in particular, tricresyl phosphate (TCP) isomers, which are highly toxic and can result in neurotoxicity causing pain and serious paralysis of limbs and bowel and lung disorders. Exposure to such chemicals is said to give rise to a wide range of illnesses and symptoms, collectively labelled as ‘Aerotoxic Syndrome’.

However, extensive medical research into this condition has not established a link between ‘fume events’ and ‘aerotoxic syndrome’ and the aircraft manufacturing industry does not consider that there is an issue with air contaminants.

Despite this lack of evidence, in editions 86 (here), 95 (here) and 145 (here), we outlined the case of Richard Westgate, a former British Airways pilot who died in December 2012 from alleged exposure to toxic cabin air. Mr Westgate’s family submitted that Mr Westgate was receiving treatment in The Netherlands for ‘Aerotoxic Syndrome’. However, at the pre-inquest review, held in July 2016 by Dr Simon Fox QC, it was held that there was no evidence to suggest that Mr Westgate’s treatment was linked to exposure to organophosphates and to conclude such would be speculation. He went on to say that:

‘Even if there was evidence to demonstrate that he was taking pentobarbital because of symptoms related to a neuropathy, I do not consider that it would be appropriate to include in the scope of my inquiry whether that condition should properly in life have been labelled as “aerotoxicity” or otherwise as a neuropathy caused by occupational exposure to organophosphates. The correctness of a diagnosis made in life may be a matter for the family to investigate and challenge in the Civil Courts; however, a Coroner’s Inquest is, in my view, not a proper forum for that investigation when there is only such a possible or speculative indirect and remote link at most between the condition and the cause of the deceased’s death’.

As such, it was held that the question of whether Mr Westgate’s death was caused by ‘Aerotoxic Syndrome’ would not be considered at the full inquest due to a lack of evidence supporting a link between the two.

It is now known that the final inquest into the cause of Mr Westgate’s death will be heard in April 2017.133

Cabin Air Quality Studies Published

In recent years, there have been numerous reports of ‘fume events’ on aircraft, while flight crews have reported a range of acute and chronic health effects, some of which have been attributed to cabin air contamination. There are a number of potential sources of cabin air contamination, including:

- Exhaust gases;
- Volatile organic compounds from cleaning products;
- Ozone;
- De-icing fluids;
- Particulate organic matter;
- Organophosphates (OPs) from lubricants;
- Hydraulic fluids; and
- Engine oils.

OPs are part of the same molecular family implicated in causing ill-health in sheep dip users and have been suggested as a potential cause of the reported health effects.

On 17 March 2015, it was announced that the European Aviation Safety Agency (EASA) had awarded cabin air quality research contracts to a consortium of the Fraunhofer Institute for Toxicology and Experimental Medicine and the Hannover 133

Medical School. EASA published the results, along with another study, which characterised the chemical composition of some turbine engine oils, including pyrolysis breakdown products.

The objective of the cabin/cockpit air study was to determine whether there are cabin air contaminants that present safety and/or potential long/short-term health risks. In total, 69 flight measurements were performed on 8 types of aeroplane/engine configurations. This included 61 flights on aeroplanes equipped with engine ‘bleed air’ systems (the main study) and 8 flights on the Boeing 787, which does not have a ‘bleed air’ system. For all flights, measurement equipment was installed in the cockpit and cabin. At defined flight phases (taxi-out, take off and climb, descent and landing, complete flight), samples were taken and then analysed using high sensitivity techniques. The results show that the air quality is similar or better than what is observed in normal indoor environments (offices, schools, kindergartens or dwellings). No occupational exposure limits and guidelines was exceeded. Special attention was paid to organophosphates, in particular, the isomers (forms) of tricresyl phosphate (TCP), which is suspected to be a cause of symptoms reported by flight crew. OPs were found in all samples, but reported concentrations were well below exposure limits.

According to the study findings, TCP concentrations in the cabin, if introduced as a continuous ‘bleed air’ contamination, ought to be constant. However, this is not the case for the individual aircraft included in the study. OPs and other contaminants were generally at the highest levels during taxi-out and at lower levels during take-off/climb and descent/landing. The researchers attribute the difference in concentrations to the rate of exchange of the cabin air. It is remarkable that TCP was detected in the non- ‘bleed air’ driven B787, especially as we have, in previous editions of BCDN, featured the comments of Boeing claiming to have alleviated any risk of ‘bleed air’ exposure. In the B787, TCP also displays changes in concentration with flight phase. The hypothesis that TCP in the cabin air of aircraft derives from ‘bleed air’ contamination must therefore be questioned, because this study did not detect permanent TCP/engine oil entry through ‘bleed air’ and found that there were sources, other than ‘bleed air’ containing TCP, in aircraft cabins. However, for 3 flights, each with a ‘bleed air’ supply, the concentration of TCP did not follow the same pattern and there were increased concentrations of TCP during particular flight phases. The increased TCP levels were unrecognised by the occupants of the aircraft. Typical oil odour sensations in other cases, however, had no concrete analytical outcome.

Overall, the results of this campaign are consistent with findings obtained through other published studies on cabin air quality. The observed frequency, pattern and concentration levels were similar to findings of other indoor environments. The study suggests that chronic exposure to ‘everyday’ levels of OPs in the cabin would not be expected to cause health problems. A number of ideas have been suggested for future studies that investigate cabin air contamination events, including human exposure studies, such as biomonitoring of blood/urine and testing for neurotoxic effects.

The second study, published by EASA, hoped to characterise the toxic effects of chemical compounds that are released into the cabin or cockpits of transport aircraft. Experimental work was performed using 2 generally used brands of oil. Oil and vapours were characterised when the oil was heated in combination with purified air and under pyrolysis (decomposition) conditions. The flight stages, from ground level to top of climb and cruising altitude were simulated. Also, toxic effects were studied, using the vapour from pyrolysis of the oil samples and an in vitro model of the human lung with an air-liquid interface.

TCP was present in the analysed oils. However, no ortho-isomers could be detected. The study concluded that neuroactive products were present, but that their concentration in the presence of an intact lung barrier was too low to be a major concern for neuronal function. However, it could not be ruled out that higher concentrations might affect neuronal activity. Furthermore, exposure for up to 48 hours resulted in decreased neuronal activity and it is therefore possible that effects of pyrolysis products develop after prolonged exposure alone. The investigators also considered the variation of human sensitivity to certain compounds and were unable to rule out that some symptoms could not be explained by actual exposure levels. It is also suggested that differences in coping strategies are well-known factors that enhance stress reactions, which, in their own right, can lead to acute health complaints and long-term health effects.

In summary, these studies do not provide evidence that OPs in cabin air are responsible for the symptoms reported by flight crew.

We have previously discussed the issue of contaminated cabin air on planes and ‘aerotoxic syndrome’ in editions 64, 65, 86, 95, 145 and 170 of BC Disease News.

Proponents of Aerotoxicity to hold Conference on Aircraft Air Contamination


Between 19 and 20 September 2017, leading proponents of aerotoxicity, such as the Global Cabin Air Quality Executive (GCAQE) and the British Professional Pilots’ Union, will hold an aviation-related health and safety conference with 6 specific objectives:

1. To provide a historical overview of the contaminated air issue and its causes.
2. To map out the flight safety aspects of contaminated air through case studies, discussion and air accident investigation findings.
3. To disseminate the latest medical and scientific theories and findings on the health aspects of exposure to contaminated air.
4. To offer guidance on the regulatory aspects of cabin air quality.
5. To examine the latest development towards bleed air filtration, contaminated air warning sensor systems and other potential solutions.
6. To provide an opportunity for networking and sharing good practice, to facilitate better inter-agency working.

The legitimacy of so-called ‘aerotoxic syndrome’ as an occupational disease is highly questionable and an issue regularly covered in BCDN. Most recently, in edition 179 of BCDN, we reported on the results of a study carried out by the European Aviation Safety Agency (EASA), which sought to delve deeper into issues surrounding air contaminants, such as organophosphates, which allegedly present safety and/or potential long/short-term health risks. EASA concluded that chronic exposure to ‘everyday’ levels of organophosphates in the cabin is unlikely to be the source of cabin crew complaints.

We will be reporting further following this conference.

Feature:

New Aerotoxicity Study Purports to Show Causation

Edition 191 of BC Disease News (30 June 2017)

INTRODUCTION

Last week, a new study of aircraft cabin air and acute and chronic symptoms in flight crew was published in Public Health Panorama, a journal of the World Health Organisation (WHO). The study was carried out by researchers from the University of Stirling and the University of Ulster with a consultant respiratory physician from Melbourne.

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This study has received widespread media attention, and has been reported by The Independent, The Sun, The Guardian, The Daily Mail and The BBC. Though the media articles tend to report the risks for passengers, e.g. ‘Flying should “come with a health warning” as toxic fumes contaminate air in cabins leading to serious health problems, research suggests’ (The Sun), the research was actually carried out on airline staff. The NHS website reported that the study found that there is a link between exposure to contaminated air and short-term problems such as drowsiness, loss of consciousness, headache and tremors, and longer-term issues such as problems with memory or concentration and fatigue. However, in this article we outline why this study in fact does not show a clear causative link between symptoms experienced and contaminated air on flights, by considering the flaws in the study, along with the particular biases of the authors. We suggest that the aim of this study was to present evidence that supports the preconceived belief that a range of symptoms reported by flight crew are due to exposure to engine oil fumes.

Research into the health of airline staff and cabin air fumes is of interest to the thousands of airline employees that may be exposed, and also to those employed in the agricultural sector and others who may be or have been exposed to organophosphates (OPs), as they have been suggested as a possible source of adverse health effects resulting from cabin air contamination. The quality of aircraft air and ‘aerotoxic syndrome’ have been discussed previously in issues 64, 65, 86, 95, 145, 170, 179 and 181 of BCDN.

BACKGROUND

It will be recalled from our earlier articles that the issue concerns whether the occupants of aircraft are exposed to organophosphates during ‘fume events’ on aircraft, resulting in various illnesses collectively labelled as ‘aerotoxic syndrome’. ‘Fume events’ on certain aircraft are said to occur because of oil seal failures in the ‘bleed air’ supply, which draws hot air from aircraft engines into the cabin air supply, with the result that engine oils and lubricants – which can contain organophosphates – can contaminate the cabin air.

Having considered the array of research on the issue, we concluded that the existence of ‘aerotoxic syndrome’ was unlikely on the current evidence. The culmination of the research was perhaps best summarised by the Committee on Toxicity in 2013, when it said:

‘More generally, the Committee considers that a toxic mechanism for the illness that has been reported in temporal relation to fume incidents is unlikely. Many different chemicals have been identified in the bleed air from aircraft engines, but to cause serious acute toxicity, they would have to occur at very much higher concentrations than have been found to date (although lower concentrations of some might cause an odour or minor irritation of the eyes or airways). Furthermore, the symptoms that have been reported following fume incidents have been wide-ranging (including headache, hot flushes, nausea, vomiting, chest pain, respiratory problems, dizziness and light-headedness), whereas toxic effects of chemicals tend to be more specific’.

More recently, in March 2017, the European Aviation Safety Agency (EASA), concluded, after having carried out 2 studies on cabin air quality and the chemical composition of engine oils (including pyrolysis breakdown products):

‘Research and scientific reviews conducted over the past decades have concluded that a causal link between exposure to cabin/cockpit air contaminants and reported health symptoms is unlikely’.

So does this new study tell us anything different?


THE STUDY

This was a combination of 2 studies, 1 involving a survey of pilots from the UK and the second being an analysis of 15 case reports of potential cabin air quality incidents. Both studies aimed to look at the circumstances and symptoms of aircrew working in the pressured air environment of aircraft, and to determine whether reported symptoms and diagnoses are consistent with exposure to engine/aircraft fumes.

The first study was a survey of UK British Airways pilots between 2005 and 2009. Lists of all known UK certified ‘BAe 146’ pilots were obtained, and 274 (14%) responded to a telephone interview or written questionnaire. They were asked whether they were aware of exposure to contaminated air, how they thought the contaminated air affected them and about any medical diagnoses they had. Data were collected on demographics, flying history, flight desk air quality history, health effects and other comments. Of the 274 who agreed to participate, 88% were aware of exposure to aircraft contaminated air, 34% reported frequent exposures and 7% reported visible smoke or mist. Overall, 63% reported immediate (i.e. acute, occurring during the flight) adverse health effects; 44% reported acute or short-term effects (lasting for days to weeks) and 32% reported medium term (lasting for weeks to months) chronic effects ‘consistent with suspected contaminated air exposures’. 142 reported specific symptoms and diagnoses, 30 reported adverse health effects, but provided no detail, while 77 reported no health effects and 25 failed to advise either way. Adverse effects included cardiovascular, gastrointestinal, general (fatigue, performance decrement) irritant, neurobehavioral, neurological and respiratory effects. Of the 274 pilots, 36 (13%) had died or had experienced chronic ill health, leading to permanent loss of fitness to fly.

The article also reports that ‘The chronic cohort (13%) reported ill health at 37-433% above the controls.’ However, there is no mention of the recruitment of a non-pilot control group, so one can only assume that the ‘control group’ is those pilots who did not report exposure to fumes in the flight deck. Further, there is no discussion that the ‘control group’ were comparable in terms of age, overall health, smoking, lifestyle and demographic factors, etc, in order to reduce the effect of confounding factors.

The second study involved analysis of 15 case reports of ‘fume incidents’ from Australia, the USA, Germany and the UK. These particular cases were chosen because the health problems reported, such as acute hyperventilation and hypoxia, were suggestive of exposure to contaminated air. Extensive data on the aircraft flight history, medical diagnoses and maintenance findings were collated. This study included specific symptoms reported per incident, rather than per person. The incidents occurred in seven different aircraft types and 87% (13 out of 15) were linked to maintenance findings of oil leakage. Symptoms ranging from in-flight incapacitation to impairment were reported in 93% of events, with the majority (73%) involving pilots and 33% including full or partial incapacitation of 2 pilots. In all, 53% of events included long-term adverse effects in one or more crew members. In total, 73% of events were associated with some form of medical investigation soon after the incident. Chronic medical findings/diagnoses were found for two-thirds of events, including cardiovascular, neurobehavioral, neurological and respiratory symptoms, chronic fatigue, multiple chemical sensitivity, aerotoxic syndrome, cancer, soft tissue damage, and chemical exposure. 9 pilots either became unfit to fly or died.

In the discussion section of the paper, the ‘bleed air’ system is described and implicated as the source of oil leakage products entering the flight deck or cabin. It is stated that chronic exposure is caused by tiny amounts of oil vapours released by oil leaking continuously over the seals during engine power changes (e.g. climbing). The researchers conclude that the population exposed to low-level oil fumes is considered to comprise all crew and passengers. It is mentioned in the discussion that, ‘The debate on cabin air contamination commonly focuses on ad hoc air-monitoring findings undertaken during normal flight operations’. However, there is no discussion of the findings from such studies. Instead, previous case studies of ‘fume events’, in which symptoms were reported, are discussed.

The discussion also outlines the known health effects of acute OP exposure, such as inhibition of acetylcholinesterase, suggesting that the authors assume that OPs are the component of engine oil fumes that is responsible for the symptoms reported, and refers to a study in which chronic symptoms due to OP exposure are supposedly reported. There is no mention or discussion of the overall unusual working conditions of air crew, which includes changes in temperature, pressure, gravitational forces, radiation and exposure to low air pressure. They also experience unusual routines, shift work, long duty hours and time zone changes.

The article concludes that:

‘Aircraft air supplies contaminated by pyrolysed engine oil and other aircraft fluids can reasonably be linked to acute and chronic symptoms, findings and diagnoses, thus establishing causation’.
It appears that the authors have reviewed data on the health effects of exposure to substances in engine oils, particularly OPs, searching for specific data in which such symptoms were found among flight and cabin crew, and then attributed the symptoms to oil exposure. Statements, such as ‘Numerous arguments have been used to deny the recognition of aerotoxic syndrome as a new occupational disease’, suggest that the authors have a pre-conceived notion that ‘aerotoxic syndrome’ does indeed exist. The discussion of alternative findings, i.e. that there is a lack of evidence for the existence of ‘aerotoxic syndrome’, is limited to statements, such as:

‘… the effects are said to be inconsistent with tri-o-cresyl phosphate-associated, OP-induced delayed neuropathy, while ignoring all other indicators of toxicity …’

The lack of inclusion of counter-arguments in the discussion and the apparent ‘cherry-picking’ of data that supports their hypothesis gives a rather one-sided view.

CAUSATION CLAIM

Despite the authors’ claim that they have demonstrated cause and effect, these studies do not demonstrate causality. The first study did not link the reported symptoms with on-board air samples. The possibility that the acute symptoms experienced may have an alternative cause was not sufficiently addressed; it is noted that hyperventilation and hypoxia (insufficient oxygen levels in tissues) have been suggested by other studies as a cause of the reported symptoms, but the article notes this in the context that the current authors disagree with these findings. In addition, no attempts are made to offer or eliminate alternative causes of chronic effects. The data in the pilot survey was self-reported, which is subject to bias for several reasons. Participants may simply not remember, their awareness of being exposed to contaminated air may not accurately reflect their actual exposure, health effects may be exaggerated and they may even be involved in current litigation. The proportion of pilots who agreed to participate was extremely low (14%), and it is likely that those who didn’t participate had not experienced any health problems. The authors do not comment on the low response rate, or that the small number of participants may have had different experiences compared to the large number of non-respondents.

In the second study, the criteria for a case to be included was that symptoms consistent with exposure to contaminated air were reported and that other data, such as maintenance data, was available. However, there was no measured air quality data from the flight deck or cabin, and so the levels of contaminants, and thus the amount of exposure, due to the ‘bleed air’ supply, are unknown. There was no comparison with, for example, reported oil leaks with no reported symptoms. 87% of symptoms were related to oil leakage, but no explanation was offered for the remaining 13%. This study suggests that oil leakage may be a cause of some of the acute symptoms reported, but that there are additional causes of such symptoms. Physical data, such as blood test results, were not available to determine whether symptoms were specifically caused by OPs in the 87% of cases with oil leaks.

STUDY QUALITY

In general, the discussion section of a journal article usually includes detailed reports of all the possible caveats within the study – however, there is no such discussion in this study. Even the obvious limitations, such as the self-reports of cabin air quality in the first study and the low participation rate, are not mentioned.

Without objective measurement of exposure, it is very difficult to determine that contaminated air is to blame. EASA, in its study on cabin air quality, states that cabin/cockpit air quality is similar or better than what is observed in normal indoor environments (offices, schools, kinder gardens or dwellings). No occupational exposure limits and guidelines were exceeded.

Unsurprisingly, the website of the Aerotoxic Organisation, aerotoxic.org, features numerous articles reporting this new study, including links to all the sources mentioned at the beginning of this article. The limitations of the study outlined here are not mentioned in any of the articles featured by the Aerotoxic Organisation.

A testimony by the lead author of the paper, Susan Michaelis, appears on aerotoxic.org.¹⁴⁸ She is a former airline pilot who stopped flying due to ill-health, which she attributes to exposure to oil fumes. She says that she regularly experienced short-term symptoms while flying the ‘BAe 146’ and could smell fumes when the air supply was switched on:

‘it was clear that the symptoms were related to oil contamination as they occurred soon after turning the air supply on…’

She describes a range of symptoms and that they became more intense and more frequent, but disappeared within 3 hours of finishing work. On 1 occasion, her symptoms continued after finishing work, and worsened. She now experiences a number of chronic symptoms, which are listed in the testimony, and claims:

‘These findings have been clearly linked by experts to my repeated exposure on BAe 146 aircraft’.

She claims that, regarding the aviation industry’s knowledge of the link between ill-health and oil fumes, ‘… the level of knowledge dates back for decades and is extremely extensive’. She took legal action against National Jet Systems and BAe Systems, though her case never went to trial. She also makes various claims relating to hidden or secret documents. She undertakes consultancy work for Michaelis Aviation Consulting, with the aim of improving aviation safety and providing guidance on cabin air quality. Several published papers are listed on the website, including a 2005 paper cited only 6 times,¹⁴⁹ and these generally argue a link between oil exposure and health symptoms with little scientific content. She has also written a PhD thesis on this topic and used some of this data in the current study. Her personal experiences and claims suggest that she is not researching this topic with an impartial view and her background includes no training in epidemiology or statistics, which are essential tools for determining cause and effect.

Dr Jonathan Burdon

Dr Jonathan Burdon, a consultant respiratory physician, is also featured on Aerotoxic.org,¹⁵⁰ and has written papers arguing the existence of ‘aerotoxic syndrome’.¹⁵¹ He is of the opinion that the air transport industry and its regulators are, ‘… apparently ignoring the problem or alternatively finding other more attractive explanations for the presenting symptom complex’.

Professor C Vyvyan Howard

Professor C Vyvyan Howard is a toxicopathologist, specialising in the action of toxic substances on the foetus and infant.¹⁵²

A large part of his current research is the investigation of toxicology of nanoparticles, and he has addressed the House of Lords Select Committee on Science and Technology investigating the use of nanotechnology in food. He also completed 6 years as a toxicologist on the DEFRA Advisory Committee on Pesticides. He is quoted by The Independent as saying, in the new study:

‘The airline industry is not seeing it this way and ignores all research that is inconvenient to them. However, this is the most comprehensive study done to date and should not be ignored’¹⁵³

THE AUTHORS

Susan Michaelis

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THE JOURNAL

Public Health Panorama is the journal of the WHO Regional Office for Europe.1 It has existed since June 2015 and, ‘...provides a platform to scientists and public health practitioners for the publication of lessons learned from the field, as well as original research work, to facilitate the use of evidence and good practice for public health action.’ According to its website, the mission of Public Health Panorama is ‘...to contribute to improving health in the Region by publishing and providing timely and reliable research, evidence, information and data for public health decision making’.

SUMMARY

In summary, it appears that the aim of this study was to present evidence that supports the hypothesis that a range of symptoms, reported by flight crew, are due to exposure to engine oil fumes. The first study does not provide evidence of a link between air quality (data regarding which is self-reported) and symptoms. The second study provides some evidence that oil fumes may contribute to acute symptoms, but does not rule out other causes of acute symptoms, provide evidence for the cause of chronic symptoms, or determine which component of the oil fumes is responsible (if any). The authors are known proponents of ‘aerotoxic syndrome’. Though it received widespread media coverage, most articles discussing this study did not mention its limitations (the exception to this being the article on the NHS website) or its authors’ backgrounds. Thus, the public, including potential future claimants, have been given inaccurate and incomplete information regarding the health effects from aircraft cabin air.

Aircraft Cabin Air Quality Conference 2017

Edition 201 of BC Disease News (22 September 2017)

We have reported several times in BC Disease News on the condition known as ‘Aerotoxic Syndrome’. It will be recalled from these articles that the issue concerns whether the occupants of aircraft are exposed to organophosphates during ‘fume events’ on aircraft, resulting in illness. Fume events occur because of oil seal failures in the ‘bleed air’ supply, which draws hot air from aircraft engines into the cabin air supply. The result of this is that engine oils and lubricants – which can contain organophosphates – may contaminate the cabin air. The aviation industry has never recognised ‘Aerotoxic Syndrome’ as a legitimate illness caused by the air in cabins. Instead, putting illnesses down to several other causes.

However, it was announced earlier this week that EasyJet airline will be the first airline to fit ‘test’ filters into its planes, in order to prevent toxic fumes entering the cabins and cockpits. EasyJet have joined up with commercial supplier, Pall Aerospace, to develop and design a new cabin air filtration system for testing. The filter was officially presented at the International Aircraft Cabin Air Conference this week by Pall, which BC Legal attended and is discussed in greater detail, below.

This development has been hailed by organisations such as the Aerotoxic Association and the Global Cabin Air Quality Executive (GCAGE), as a huge step forward for the recognition that toxic cabin air has been causing passengers and cabin crew ill health. However, in an EasyJet statement, it was made clear that the airline was still retraining from taking a position on Aerotoxic Syndrome which they said, ‘remains an area of scientific uncertainty’.1

Elsewhere this week, BC Legal attended the International Aircraft Cabin Air Conference, held at Imperial College London over two days. The conference was organised by Captain Tristan Loraine BCAI, of GCAGE and endorsed by Pall Aerospace, the European Sealing Association (ESA), UNITE, the International Joint Policy Committee of the Societies of Epidemiology (IJPCSE) and several other organisations.

The two-day event included speakers from the scientific community, academics, engineers, researchers, aircraft and engine manufacturers, politicians, air accident investigators and air cabin crew. The topics discussed, included a historical overview of the contaminated air issue, case studies of those affected, regulatory aspects of cabin air quality and the latest medical and scientific research.


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New Research Disputes Theory on ‘Aerotoxic Syndrome’ Phenomenon
Edition 258 of BC Disease News (18 January 2019)

In edition 191 of BC Disease News [here], we reviewed the validity of a study, which, according to the NHS, purported to show causation between exposure to polluted commercial aircraft cabin air and symptoms of so-called ‘Aerotoxic Syndrome’. These include drowsiness, loss of consciousness, headache, tremors, loss of memory or lapses of concentration and fatigue.

Nevertheless, the condition has been disputed among medical and aviation industry experts. In previous editions, we provided frequent updates in regards to the inquest of former British Airways pilot, Richard Westgate, who believed that his ill-health was the result of inhaling contaminated ‘bleed air’ from aircraft engines during ‘fume events’.

In April 2017, the Coroner ruled that Mr Westgate’s ‘excruciating pain’, digestive problems, fatigue, headaches, loss of cognitive ability, clumsiness and inability to sense temperature had been the consequence of an unintended sleeping tablet (pentobarbital) overdose, as opposed to an industrial disease.156

Since edition 201 [here], when we reported that the Aircraft Cabin Air Quality Conference had taken place, it is noticeable that the hysteria surrounding ‘Aerotoxic Syndrome’ has tapered off.

In November 2018, however, researchers at Manchester Metropolitan University’s Ecology and the Environment Research Centre revealed that components of low toxicity ‘bleed air’ are not converted into more harmful chemicals when they mix with engine lubricant vapours.157 Their findings have been published in the Chemosphere journal.158

The study was funded and co-authored by Frank Cannon, an aviation lawyer, former pilot and former airline owner, who has experience of representing ex-pilots and ex-cabin crew in ‘Aerotoxic Syndrome’ litigation against airline employers.

In earlier research, lead author, Dr David Megson, observed that aircraft oil does not contain a group of toxic organophosphates, called ortho-substituted tricresylphosphates (ooo-TCPs). Organophosphates are considered to be the predominant source of ‘Aerotoxic Syndrome’.

In the latest study, the Senior Lecturer in Chemistry and Environmental Forensics sought to investigate whether less harmful TCP isomers (TCP molecules with the same number of atoms, but in a different arrangement) could become ooo-TCPs by way of transisomerisation.

It was hypothesised, pre-investigation, that transisomerisation could occur as ‘bleed air’ passes through the palladium catalytic systems of aircraft cabins.

Having replicated in-flight conditions, by heating oil to 400°C in a laboratory catalytic converter, the team of researchers was able to identify that transisomerisation did not take place.

Accordingly, Dr Megson acknowledged that:

‘It was ... important for our study to establish that the oil does not appear to be the source of ooo-TCP and more focus should be placed on investigating other potential sources’.

Unite Issues Call for Public Inquiry into ‘Aerotoxic Syndrome’
Edition 261 of BC Disease News (8 February 2019)

Last week, Unite, the trade union, reiterated its call for a full public inquiry into so-called ‘aerotoxic syndrome’. Various experts believe that long-term exposure to contaminated air, which enters aircraft cabins through unsealed jet engines (also known as ‘bleed air’) during ‘fume events’ causes ‘aerotoxic syndrome’. The Civil Aviation Authority (CAA) recognises the condition as ‘aerotoxicity’.

Common symptoms of ‘aerotoxic syndrome’ include itching or soreness of the eyes, nasal discharge, sore throat, coughing, nausea, dizziness and cognitive impairment, while some consider the condition to be potentially fatal.

Throughout the inquest into the death of former British Airways (BA) pilot, Richard Westgate, it was submitted that ‘aerotoxic syndrome’ was the lethal cause, though these accusations were not tested by the Coroner at Swindon Coroners Court, in April 2017.

Following a more recent post-mortem inquest into BA cabin crew member, Matt Bass, who died aged 34, the Senior Coroner overseeing the inquest wrote an unprecedented letter of concern to the Chief Coroner. As a result, all coroners are now obliged to call for additional tests in suspected cases of ‘aerotoxic syndrome’.

On 29 January 2019, Unite was prompted to call for a public inquiry into the controversial industrial disease, when news surfaced that a ‘fume event’ had occurred on a return flight from Boston to London Heathrow (BA212), one week prior.

On the outbound BA flight from Heathrow (BA213), cabin crew complained of a ‘strange toxic smell’ when the plane landed on US soil. An official Air Safety Report logged this incident, but the aircraft was pronounced ‘safe for take-off’ by BA engineers in Boston, after having completed ‘thorough checks’.

However, 1 hour and 41 minutes post-departure, flight BA212 was forced to divert back to Boston, after smoke flooded the cabin at 30,000 ft. The pilot declared a ‘May Day’ emergency. 10 crew members were taken to hospital for medical assessment, of whom 2 crew members were ‘seriously ill’ and 1 was ‘violently unwell’.

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The Boeing 747-400 remained grounded for 3 days, while it was flown back to London without passengers. Almost immediately after arrival, the aircraft was cleared to embark on a commercial flight to New York.

‘Fume events’, like the incident on flight BA212, are not uncommon. As a matter of fact, in the past year alone, there have been multiple reported ‘fume events’ on BA flights. In August 2018, a ‘fume event’ occurred at 20,000 ft, 20 minutes into an Airbus A320 flight (BA345) from London Heathrow to Nice. Just one month later, another ‘fume event’ occurred at on an A320 flight (BA2960) from London Gatwick to Glasgow.

It is accepted that toxic organophosphates, known as ortho-substituted tricresylphosphates (ooo-TCPs), are found in toxic ‘bleed air’. What is not accepted, however, is the source of contaminants in ‘bleed air’.

Toxic organophosphates are not detected in engine oil itself and, in edition 258 of BC Disease News (here), we analysed the results of Manchester Metropolitan University research, which discovered that transisomerisation, a process that occurs in aircraft air conditioning systems, does not convert harmless organophosphates into ooo-TCPs. Ultimately, the study findings suggest that more emphasis should be placed on identifying alternative ‘bleed air’ pollutants, such as hydraulic fuel and anti-freezing fluid.

BA bosses have attributed the ‘fume event’ during flight BA212 to de-icing chemicals, used on the Boston runway in negative 15°C temperatures.

According to a BA spokesperson, employees are encouraged to report safety incidents on flights and details of these incidents are passed on to the CAA. However, the spokesperson told Sun Online Travel that:

‘There has been substantial research into questions around cabin air quality over many years. In summary, the research has not shown that exposure to potential chemicals in the cabin causes long-term ill health.’

Not all airlines have downplayed the effects of ‘aerotoxic syndrome’, however, with EasyJet announcing, in 2017, that it would be installing new Pall Aerospace air filters on its fleet to reduce the risk of exposure to ‘toxic air’.

Amid fears that ‘fume events’ have been under-reported, Unite has created a ‘Fume Event Register’ (see here).

Will the Government submit to unrelenting trade union pressure and launch a public inquiry into ‘aerotoxic syndrome’?

As an emerging employers’ liability (EL) and public liability (PL) risk, readers can expect sustained scrutiny of ‘aerotoxic syndrome’. Will the Government submit to unrelenting trade union pressure and launch a public inquiry into ‘aerotoxic syndrome’?

More Than 50 Airline Employees Commence ‘Aerotoxic Syndrome’ Claims

Edition 269 of BC Disease News (5 April 2019)

Earlier this year (here), we reported that Unite, the trade union, had reiterated its call for a full public inquiry into so-called ‘aerotoxic syndrome’, after a ‘fume event’ occurred on British Airways (BA) flight 212 from Boston to London Heathrow, in January 2019.

Engineers completed ‘thorough checks’ of the grounded Boeing 747-400, which led company bosses to conclude that this specific ‘fume event’ was caused by residual de-icing chemical exposure. Of course, proponents of aerotoxicity believe that ‘fume events’ occur when the cabin is filled with toxic ‘bleed air’, which enters through unsealed jet engine systems.

BC Legal has been monitoring ‘aerotoxic syndrome’ as an emerging risk for over 4 years and, in anticipation of employers’ liability and public liability (EL/PL) claims, we published our expansive collection of articles in edition 265 of BC Disease News [here].

Last week, Unite confirmed that 4 pilots and 47 cabin crew have initiated proceedings against 5 of the UK’s largest airlines:

- EasyJet;
- BA;
- Thomas Cook;
- Virgin Atlantic; and
- Jet2.  

80% of these claims affect BA, which has the largest fleet of aircraft of all UK airlines (see the table below).

Unite has specified that ‘legal action’ has been ‘served’, though it is unclear whether this refers to service of the Letter of Claim or service of the Claim Form. Regardless, Unite has intimated that it could take ‘up to a year’ for the cases to be heard at trial, which infers that these ‘aerotoxic syndrome’ claims will eventually litigate, if they have not already.  

In support of the claimants’ legal action, Howard Beckett, Unite’s Assistant General Secretary for Legal Services, has stated that:

‘Independent expert evidence concludes that air on board jet planes can contain a toxic mix of chemicals and compounds that potentially damage the nervous system and may lead to chronic irreversible health problems in susceptible individuals ... how many more must be put at risk before the airline industry cleans its act up?’

Reading between the lines, it is likely that the claimants will argue an association between long-term exposure to contaminants in ‘bleed air’, e.g. the organophosphate compound, tricresyl phosphate (TCP), and the onset of chronic irreversible neurological damage, i.e. symptoms of ‘aerotoxic syndrome’.

Accordingly, aerotoxic claims will not focus on occupational exposure in Boeing’s ‘Dreamliner’ (787-9 and 787-8) – a unique feature of this aircraft, as is evident from the table below, is that it boasts a ‘no-bleed’ electrical systems architecture.  

This system was designed to:

- Improve consumption and efficiency;
- Reduce costs;
- Enhance reliability; and
- Extend range.

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### Table: Airline Fleets Facing Aerotoxic Syndrome Claims

<table>
<thead>
<tr>
<th>AIRCRAFT</th>
<th>CURRENT FLEET PER AIRLINE (SOURCE: PLANESPOTTERS.NET)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>EASYJET</td>
</tr>
<tr>
<td>MODEL (LONGEST FIRST)</td>
<td>BLEED AIR SYSTEM?</td>
</tr>
<tr>
<td>AIRBUS A340-600 (75 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 777-300 (74 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS 380-800 (72.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 747-400 (71 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS A330-300 (64 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 777-200 (63.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 787-9 (63 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS A330-200 (59 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 787-8 (57 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 757-200 (47 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS 321-200 (44.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS 321-200NEO (44.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 737-800 (39.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS 320-200 (37.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS 320-200NEO (37.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>EMBRAER 190 (36 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS 319-100 (34 M)</td>
<td>✓</td>
</tr>
<tr>
<td>BOEING 737-300 (33 M)</td>
<td>✓</td>
</tr>
<tr>
<td>AIRBUS 318-100 (31.5 M)</td>
<td>✓</td>
</tr>
<tr>
<td>EMBRAER 170 (30 M)</td>
<td>✓</td>
</tr>
</tbody>
</table>

However, claimants may struggle to demonstrate causation on this basis for the following reasons.

In edition 179 (here), we reported that the European Aviation Safety Agency (EASA) had identified that cabin air quality on aeroplanes equipped with engine ‘bleed air’ systems was similar to (or better than) air quality in normal indoor environments (offices, schools, kindergartens or dwellings) and organophosphate concentration levels were well below the workplace exposure limit.\(^{169}\) What is more, in a finding that was contrary to expectation, Boeing ‘Dreamliner’ cabin air samples detected TCP, despite having no ‘bleed air’ system, implying that bleed air is not the only source of organophosphate exposure.

In another EASA study, analysis of pyrolysed engine oil corroborated the presence of neuroactive products, but concentrations were so low that an intact lung barrier could protect against the risk of neuronal impairment.\textsuperscript{170}

Disagreement over a causative relationship between engine oil fumes and neurotoxicity was further intensified by more recent Manchester Metropolitan University research (reported \textsuperscript{here}), which concluded that engine oil ‘does not appear to be the source of ooo-TCP’ (ortho-substituted tricresylphosphates), the harmful TCP isomer considered to be the predominant cause of ‘aerotoxic syndrome’. The researchers established that less harmful TCP isomers (contained in engine oil) are not converted into ooo-TCP when mixed with palladium catalysts (present in aircraft air conditioning units) in a process called transisomerisation. Hence, it was determined that ‘more focus should be placed on investigating other potential sources’ than engine oil.

Unite’s decision to single out ‘bleed air’ systems and organophosphate-containing engine oil fume exposure as the underlying cause of airline employees’ personal injuries seems, in the face of scientific uncertainty, to be presumptuously premature. It will be interesting to see whether the ‘independent expert evidence’ it claims to have obtained is new supportive evidence, or merely an alternative interpretation of previously reviewed scientific research.

In response to news of ‘aerotoxic claims’, EasyJet has expressed confidence in its operation of ‘one of the world’s most modern fleets’, with ‘fully compliant’ air quality and air-conditioning standards. While the company admitted that minor acute symptoms had been associated with ‘one-off fume events’, it submitted that there is ‘no proof that long term health issues arise from cabin air quality’. BA reacted by citing the EASA study as evidence of ‘substantial research conducted over many years’, which shows that exposure to cabin air (it did not specify ‘bleed air’) does not cause long-term ill health.\textsuperscript{171}

Elsewhere, a spokesperson for Virgin Atlantic has assured that:

’As with all British airlines we operate to the strict regulations and standards set out by the UK CAA and the European Aviation Safety Agency’.

All 3 airlines have guaranteed that the safety of their flight crews are their priority and maintained that any health concerns raised are taken seriously. Further, they certify that any aircraft posing a health and safety risk to passengers or crew would be non-operational.

If the Government succumbs to Unite’s request for an open investigation, we may soon be in a better position to confirm or deny the benefits of ‘using different oils to lubricate jet engines, better monitoring of cabin air, installing air filters and manufacturing planet that bring compressed air straight from the atmosphere’. By consequence, we may also be in a better position to predict the success or failure of aerotoxic claims which, at present, would appear to have a strong defence.

\section*{Aircraft Cabin Air Litigation’ Soon to Take-Off}

\textit{Edition 270 of BC Disease News (12 April 2019)}

In the wake of last week’s article (\textsuperscript{here}), wherein we reported that Unite, the trade union, was aware of 54 individual ‘aerotoxic syndrome’ claims, we now report that group litigation, known as the ‘Aircraft Cabin Air Litigation’, may see 94 industrial disease claims assessed collectively.\textsuperscript{172}

Last week, Judge Barbara Fontaine presided over a London High Court hearing to order collective case management. She confirmed that the ‘majority’ of claims have been brought against British Airways (BA) and that ‘many other claims’ have not yet been issued.


\textsuperscript{171} Andy Bounds, ‘Five airlines face toxic cabin air claims in UK, union says’ (28 March 2019 \textit{Financial Times})<https://www.ft.com/content/329368b6-5161-11e9-b401-8d9ef1626294> 1 April 2019.

BA employs 16,500 cabin crew and 3,900 pilots. It is estimated that this wave of claims could cost the company (and by extension, their insurer) tens of millions of pounds.

In past editions of BC Disease News, we frequently updated our readers on the progress of a Coroner’s inquest into the death of BA pilot, Richard Westgate. In April 2017, it was found that Mr. Westgate’s cause of death, aged 43, had been the consequence of an unintended sleeping tablet (pentobarbital) overdose, as opposed to ‘aerotoxic syndrome’.

It is our understanding the estate of Mr. Westgate will be one of the lead claims in the class action lawsuit and is seeking £500,000 in compensation. Claimant counsel, Michael Rawlinson QC, referred to Mr. Westgate’s case as:

‘...just one of a number of cases where aerotoxicity is alleged to have either caused injury or materially contributed to a cause of death’.

David Platt QC, instructed on behalf of British Airways PLC, argued that any Order made should be restricted to claims involving his client in isolation, in order to avoid complicating proceedings with issues affecting particular airlines and aircraft types.

He further submitted that BA denies liability on issues of breach and causation, dismissing this litigation as ‘weak and speculative’:

‘...it challenges a technical, scientific and industry consensus through the propositions upon which the claimants rely’.

Having monitored this issue for over 4 years, we agree with David Platt QC that there is a dearth of medical literature which proves that ‘bleed air’ is not neurotoxic.

Theoretically, if ‘bleed air’ systems were judged to be at fault for toxic aircraft cabin exposure, BA would be forced to retrofit non-‘bleed air’ systems (those found in its Boeing 787’s) at a speculative cost of £6 million (£25,000 per aircraft).

Confusingly, alleged ‘fume events’ have been nicknamed ‘Eau de Boeing’ by proponents of this litigation, on account of their ‘malodorous background smell’. However, this adds confusion to the cause, as not all Boeing aircraft are fitted with ‘bleed air’ systems. It also allows Airbus to escape all association with aerotoxicity.

Judge Fontaine specified that up to 8 cases, including Mr. Westgate’s case, will return to Court for a full trial.

We will endeavour to disseminate the Group Litigation Order once it has been finalised and is publicly available.

Preliminary Results Published on Airline Employee Brain Damage, with Full Results to Follow
Edition 271 of BC Disease News (26 April 2019)

In a Belgian newspaper (Het Laatste Nieuws), earlier this month, it was reported that neurophysiologist and psychologist, Daniel Dumalin, had started analysing brain damage in ‘(ex-) cabin crew members and (ex-) pilots’, amid concerns of so-called ‘aerotoxic syndrome’.

In the last edition of BC Disease News, we reported that 94 claimants, including relatives of the deceased British Airways (BA) pilot, Richard Westgate, were seeking to obtain an order for collective case management of occupational ‘aerotoxic syndrome’ claims, known as the ‘Aircraft Cabin Air Litigation’.

Defendant counsel for BA, David Platt QC, argued before Senior Master Barbara Fontaine, at the High Court, that this litigation must be ‘viewed as weak and speculative, given that it challenges a technical, scientific and industry consensus’.

If claimants are to be successful at future trials of liability, they will need to prove that contaminants in aircraft cabin air cause neurotoxic injury – a connection that has not been consistently established in medical literature to-date (see our Collection of Articles for comprehensive analysis of what is known about the condition).

Nevertheless, Mr. Dumalin’s research may add weight to the claimant’s arguments on medical causation. The Belgian scholar has publicly compared the current position of the aviation industry with the problems that the asbestos and tobacco industries faced in the 1960’s and 1970’s, respectively; they were no-longer able to continue ‘playing down the health risks’ once the ‘scientific evidence became overwhelming’.176

It can be inferred, therefore, that Mr. Dumalin is a proponent of ‘aerotoxic syndrome’. His investigation should therefore be observed with a critical eye, given the risk of positive bias.

Ultimately, his research looks to measure brain damage following ‘repeated exposure to low doses of toxins in the air’.

Where previous studies, e.g. Christiansson et al. (2008)176 and Strid et al (2014)177 (these were referred to in a European Aviation Safety Agency report178), used biomonitoring techniques, such as blood analysis, to measure the health impact of aircraft cabin air ingestion, Mr. Dumalin is analysing the neurotoxic effects of aircraft cabin air by comparing quantitative electroencephalograph (QEEG) scans of ‘(ex-) cabin crew members and (ex-) pilots’ against ‘healthy people’.

QEEG scanning can identify the amplitude, location and normalcy of brainwaves, as well as the coherence (quality of communications), phase (thinking speed) and network integration of neural pathways – all of these factors are crucial for the optimal operation of mental functioning.179

For completeness, Mr. Dumalin has assured that:

‘If a test person had previous MRI- or PET-scans ... [he] ... will include those for a thorough analysis’.

Based on his preliminary examination of 7 airline employees, Mr. Dumalin has described his findings as 'remarkable', as each scan has shown evidence of brain damage resembling the type of damage commonly seen in individuals exposed to organophosphates (the alleged exposure source in ‘aerotoxic syndrome’ claims).

[The damage] relates to damage in brain areas that control cognitive processes. This causes e.g. concentration disorders, memory problems or a hypersensitivity to trigger signals. Even more striking is that in people who haven’t been flying any more for over 10 years, the damage is as evident as in people who flew recently.

Mr. Dumalin considers it to be ‘too early to draw conclusions’ from his initial study results, but plans to submit another 4 airline employees to the same method of examination in the coming weeks. In total, he is aiming to inspect the neural activity of 50 individuals:

‘At that point the conclusions would be rather solid. And besides, after that one could repeat the same research in other countries’.

He has further emphasised that a ‘big advantage’ of QEEG testing is that it is more cost-effective than MRI-research.

‘Thus, in a few years’ time and on a world wide scale, we could acquire a large number of test persons in a relatively simple and cheap way. That would only reinforce the evidence’.

We will continue to follow the progress of this Belgian study with interest.

However, it is important to highlight that, even though this study could show that organophosphates are a definitive cause of neurotoxicity, it will not pinpoint organophosphates as the definitive exposure source of ‘aerotoxic syndrome’. This preserves the assertion that ‘toxic air from the engines enters cabin and cockpit via the air conditioning system’ of aircraft with ‘bleed-air systems’ as an as-yet uncredited association. John Leahy, Chief of Operations at Airbus, previously referred to the possibility of aircraft cabin contaminants as ‘absurd’.

Sealed Collective Case Management Order for ‘Aerotoxic Syndrome’ Group Litigation

Edition 279 of BC Disease News (5 July 2019)

So-called ‘Aerotoxic Syndrome’ claims are being brought by airline employees who have sustained personal injuries (including neurological damage), allegedly as a result of exposure to toxic substances in aircraft cabin air. For more information, read our comprehensive claims handling publication: Aerotoxic Syndrome: An Emerging Risk? [here]

In edition 270 of BC Disease News [here], we reported that a collective case management hearing had taken place, before Senior Master Barbara Fontaine, in regards to ‘Aerotoxicity Claims’.

On 17 June 2019, the Order was, at long last, sealed by the Court.

The effect of this agreement is to transfer a ‘significant’ number of litigated industrial disease cases to the Royal Courts of Justice (RCJ) in London, ‘achieving greater certainty of outcome and an overall reduction in costs’.

It also allows the RCJ to manage cases that have not yet been issued or served (‘intimated’ and ‘future’ claims), though the Order expressly invites claimant firms to ‘use their best endeavours’ to send letters of claim for all aerotoxicity cases that they intend to advance ‘as soon as practicable’, in compliance with Paragraph 6 of the Pre-Action Protocol for Disease and Illness Claims.

Another feature of the Order is that it protects the claimants’ position (especially those who have only sent a letter of claim) on limitation, by introducing a ‘standstill date’ (‘intimated’ cases – the date of the Order or, if later, the date of the letter of claim / ‘issued’ cases – the date of issue):

‘The effect of this agreement is to prevent the accrual of time between the standstill date and the termination of this agreement [the Order], being taken into consideration at any limitation trial’.

By the end of this month, solicitors’ practices, acting for (prospective) defendants to ‘Aerotoxicity Claims’, are expected to have attended a meeting with claimants’ solicitors, in order to determine:

- Which defendant firm will be the nominated ‘liaison solicitor’;
- Which of the remaining cases will join Westgate & Love v British Airways and White v British Airways as lead cases;
- The terms upon which the claims will be stayed;
- The limits and dates for disclosure;
- What the preliminary issues are and in what order;
- Proposed timetabling;

6 LETTER OF CLAIM

6.1

Where a decision is made to make a claim, the claimant shall send to the proposed defendant two copies of a letter of claim, as soon as sufficient information is available to substantiate a realistic claim and before issues of quantum are addressed in detail. One copy is for the defendants, the second for passing on to his insurers.

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- The terms upon which the claims will be stayed;
- The limits and dates for disclosure;
- What the preliminary issues are and in what order;
- Proposed timetabling;
- How a 'docket', or register, system can be set up to capture 'future claims' (those that have not been intimated to defendants) in the litigation;
- What will be the latest date that a claim may be admitted for inclusion in collective case management; and
- Future costs (costs budgeting).

The Order further directs that, by no later than 28 days before the meeting, the parties should have circulated to all attendees, in respect of each claim:
- The initials of the individual claimant;
- The date of birth of the claimant;
- The employment dates of the claimant with the various defendants;
- The dates of any 'fume events' being relied upon;
- The broad nature of the allegation of chronic exposure;
- The date of onset of symptoms;
- A broad description of the nature of the symptoms;
- The claimant's current employment status and the nature of any claim for loss of earnings; and
- The date of issue.

In addition, no later than 14 days before the meeting (this is understood to be the stage that the parties are currently approaching), Position Statements should be circulated, indicating a provisional choice of lead cases, all of which should allege 'chronic exposure' and, when considered in the round, should 'consist of a fair spread of facts'.

If, prior to the date of the meeting, consensus is achieved on lead cases and any other issues to be discussed, then the meeting will not take place.

Contentious Documentary on ‘Aerotoxic Syndrome’ Premieres at Raindance Film Festival
Edition 290 of BC Disease News (4 October 2019)

We have consistently reported, in BC Disease News articles, that a number of former pilots and cabin crew believe that air, fed to the cabin and cockpit of aircraft, is insufficiently filtered and contains toxic fumes.

All commercial aircraft, with the exception of the Boeing 787 Dreamliner, incorporate ‘bleed air’ systems within jet engines [and auxiliary power units (APU)], with the objective of supplying breathable air at high altitude.

As outside air is sucked in through a jet engine’s compression section, some of the air is ‘bled off’, while the remainder fuels the combustion reaction that powers the turbine.

Some argue that extracted ‘bleed air’, i.e. air that doesn’t reach the combustion chamber, can be contaminated, e.g. with by-products of heated lubricating oils.

*Drawing: Bleed Air Systems*
If this is indeed the case, could cumulative workplace exposure to a mixture of pollutants cause individuals to develop an occupational disease, called ‘aerotoxic syndrome’?

Although the Civil Aviation Authority (CAA) has previously acknowledged that ‘a link between exposure to contaminants in cabin air and possible long-term health effects … cannot be excluded’, it has also stated that there is no scientific evidence to show that ‘aerotoxic syndrome’ exists.180

Click here, to access our comprehensive publication on the subject: Aerotoxic Syndrome: A Collection of Articles from BC Disease News.

At the 2019 Raindance Film Festival, on Tuesday and Wednesday of last week, ticket-holders were able to watch the world premiere of Everybody Flies.

What is the significance of this film (watch the trailer here)?

The 2-hour feature-length documentary was directed and written by former British Airways (BA) Captain, Tristan Loraine, who began learning to fly at the age of 17 and was forced to prematurely retire as a result of ill-health, when he was just 44-years-old.

His website claims that:

‘His health had deteriorated due to repeated exposure to oil fumes onboard the aircraft he flew’.

In support of this accusation, tricresyl phosphate (TCP), an organophosphate constituent of jet engine oil, was identified in his blood and fat. He also claims to have suffered numbness in his fingers and feet, chronic chest infections, fatigue, nausea and a chemical blister on the side of his nose.181

Throughout his career as a pilot, spanning 20-years, he flew over 10,000-hours on various commercial aircraft, including:

- Lockheed L-1011 TriStar (top-left);
- Boeing 737 (top-right);
- Boeing 757 (bottom-left); and
- Boeing 767 (bottom-right).

[Sources: Wikimedia Commons (L-1011, 737 and 757) and Flickr (767). For L-1011 – Author: Kambui; Date: June 1989; and Description: ‘London - Heathrow (LHR / EGLL)’. For 737 – Author: Aero Icarus; Date: 21 September 2011; and Description: ‘British Airways Boeing 737-200’. For 757 – Author: Aero Icarus; Date: 24 September 1995; and Description: ‘British Airways Boeing 757-236’. For 767 – Author: Aero Icarus; Date: 13 April 1996; and Description: ‘British Airways Boeing 767-300’.]

In fact, Mr. Loraine boasts having previously flown to as many as 50 airports (in America) in a single day.

Besides flying, he became a health and safety representative for the British Airline Pilots Association (BALPA), in 1998, where he was tasked with examining colleague reports of contaminated air. Moreover, he co-chaired the Global Cabin Air Quality Executive (GCAQE), up until 2016, which hosts an annual Aircraft Cabin Air Conference.

Since 2006, Mr. Loraine’s production company, Fact Not Fiction Films Ltd ©, has made movies and documentaries that project his suspicions that the aviation industry has known about the risk of ‘aerotoxic syndrome’ for over half a century:

- ‘Welcome Aboard Toxic Airlines’ (2007) – a documentary that triggered 2 calls for a public inquiry into the issues of contaminated air on aircraft, in the UK. This undoubtedly influenced his nomination and receipt of a British Citizen Award for services to industry (BCAI), in the UK House of Lords.
- ‘Angel Without Wings’ (2010) – a documentary shadowing pilot, Susan Michaelis, who was ostensibly affected by ‘aerotoxic syndrome’.
- ‘Broken Wings’ (2011) – a documentary on an Australian Senate investigation of an incident, involving the BAe 146/RJ, which was suspected to be the cause of toxic fumes.
- ‘Pilot Fatigue’ (2012) – a documentary on the alarming incidence of fatigue among pilots, which is asserted as a symptom of ‘aerotoxic syndrome’.
- ‘American 965’ (TBC) – a documentary recording an incident, which occurred on American Airlines flight 965 (20/12/1995), with assumed involvement of toxic fumes.

His latest picture, ‘Everybody Flies’, received the backing of Unite the Union, prior to being released.183

It is the accumulation of over 4-years of work, disclosing never-before-seen documentation, scientific research, and personal testimonies of whistle-blowers. Mr. Loraine even claims that a Head Doctor in the airline sector was seeking to influence the UK Government’s sensitivity to the issue.184

For these reasons, Mr. Loraine has formed the disparaging view that the ‘Government works to protect the aerospace industry, rather than protect the public health’.185

‘We chose this documentary because it affects an incredible number of people constantly flying everywhere. Taking planes is part of our lifestyle and thinking that the air we breathe in planes could be poisonous is very scary’, said Suzanne Ballantyne, Head of Programming at Raindance.186

‘Everybody Flies’ is scheduled for cinema release in the New Year (potentially March 2020), at Everyman Cinema venues.

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183 ‘EVERYBODY FLIES’ RECEIVES STANDING OVATION AT WORLD PREMIERE IN LONDON’ (25 September 2019 Film Industry Network) <https://filmindustry.network/everybody-flies/?fbclid=IwAR3sLEJxJQ_dEpCIw0ULYH0oyfcU4SR93KXzHmSb3gQUhZCgl8QKBnIS5U> accessed 3 October 2019.
Disclaimer

This newsletter does not present a complete or comprehensive statement of the law, nor does it constitute legal advice. It is intended only to provide an update on issues that may be of interest to those handling occupational disease claims. Specialist legal advice should always be sought in any particular case.

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