(ii) Summary

Richard Mark Westgate died on 12 December 2012, in Holland. He was found dead in the bed of his hotel room at about 8 am. Attention was drawn to his room because a "pillow-angel" alarm he slept on, was sounding. He was pronounced dead at the scene by the duty medical examiner and paramedics who were called by the hotel staff and the police. No attempt at resuscitation was carried out; nor was it indicated.

At the time of his death he was a pilot in the employment of British Airways Plc. He had gone to Holland, in April 2012, because he was trying to find a cure for an illness he was suffering from.

For some time before his death, the deceased suffered pain and discomfort. This included frequent severe headaches, fatigue, paraesthesia (numbness and "pins and needles"), ataxia (clumsiness and awkward gait), and cognitive deficits (poor memory and thought processes). In addition he suffered chest pain, and experienced bouts of sleep apnoea.

In September 2011 the deceased's medical certification as a pilot had been withdrawn. He was in great pain. He sought diagnosis and treatment from his GP Practice, The Marlborough Medical Practice, George Lane, Marlborough, Wiltshire, SN8 4BY. Although he attended upon the doctors and various specialists, no diagnosis was achieved. His GP case notes are included as evidence.

Before his death in Holland, Richard was diagnosed as suffering from nerve and brain injury caused, most probably, by neurotoxic contamination of the air inside his aircraft. Copy Dr Mulder's case notes are included as evidence.

An autopsy ("NFI autopsy") was carried out by direction of the Dutch Minister For Justice and Security. A second autopsy ("Symbiant autopsy") was performed on behalf of the deceased's family. Both autopsy reports confirmed the presence of lymphocytic myocarditis. Both autopsy reports also confirmed the presence in the blood of the deceased of pentobarbital at a potentially lethal dose. There was no apparent use of force. This drug required a medical prescription, although it can be purchased through the internet without prescription. There is no record of the deceased having been prescribed pentobarbital, and no further trace of the substance was discovered in his belongings. However the Symbiant autopsy report revealed evidence of repetitive therapeutic dosage in analysis of his hair. The deceased's family reasonably assume that he was taking such medication to help alleviate his pain and suffering. There was no other reason for him to have taken this drug. In particular, there is no evidence that the deceased was ever provided with instructions, either verbal or written, as to the appropriate dosage of this medication. Both autopsy reports are included as evidence, the NFI one being in original Dutch as well as a translated English version.

The deceased had been an athletic person who neither smoked nor drank. He was of athletic build, and enjoyed most forms of physical pursuits, including paragliding, at which he excelled.

The Symbiant autopsy, including pathology and microscopy disclosed evidence of:

(i) lymphocytic myocarditis consistent with toxic attack; and
(ii) extensive injury to the deceased's peripheral nervous system (PNS) in the form of lymphocytic infiltration, axonal blockage and demyelination of various nerves of his PNS, which is consistent with exposure to toxic and neurotoxic exposure.

After the deceased's death, Professor Abou-Donia performed a specialised newly developed test, using the deceased's *ante mortem in vivo* blood which demonstrated very extensive injury to the PNS and central nervous system. His test report is included as evidence.

Further, histology tests carried out by Professor Abou-Donia on the brain tissue, reported:

(a) neuronal cell loss and prominent spongiosis of the prefrontal and frontal cortex;
(b) demyelination of the cortex;
(c) spongiosis and demyelination of the hippocampus of the brain;
(d) substantial cell death in, and demyelination of, the cerebellum of the brain; and
(e) lymphocytic invasion and demyelination of the spinal cord.

These findings are consistent with organo-phosphate exposure. Such injuries result in weakness and deficits of posture, locomotion, fine movement, cognition, learning, memory, skilled movements, gait and coordination of movement of limbs. They are also consistent with symptoms reported by flight crews who were confirmed as having been exposed to known and documented contaminated cabin air events while flying. Professor Abou-Donia's Preliminary Histology report is included as evidence, and a further Final Histology Report is awaited and will be included as evidence when available.

This brain and nerve damage was the cause of the deceased's chronic symptoms prior to death, for which he seems to have been of taking pentobarbital. One of the fundamental and proximate causes of his death, therefore, was the nerve and brain damage caused by the toxic and neurotoxic exposure in the aircraft he flew.

(iii) STATEMENT OF EVIDENCE ON BEHALF OF THE FAMILY OF THE DECEASED, RICHARD MARK WESTGATE.

1 The family of the Deceased are aware that it is not the purpose of an Inquest to lay or apportion blame, but to uncover the facts surrounding a particular death. However, insofar as such a search for those facts raises a concern that future deaths may occur needlessly and for want of precautionary steps, Her Majesty's Coroner has a duty to make a report to a person who has the power to take appropriate action. This statement of evidence seeks to address such a concern**.**

2 Richard Mark Westgate was a British Airways Senior First Officer (SFO) on the Airbus 319–321 fleet of aircraft based at London's Heathrow Airport. He flew on routes within Europe and beyond [***Deceased's flying log-book Vol 1 Tab 13***]**.** As a young man he studied and worked very hard to obtain an Air Transport Pilots Licence (ATPL). As an airline pilot Richard Westgate required to hold a Class 1 Medical Certificate. ***[Guidance for Medical Certification of Aircrew Vol 2 Tab 5].*** A medical examination has to be carried out every year thereafter. Every airline pilot has to be, and then remain, "free of any condition, mental or physical, which is likely to interfere with the safe exercise of the privileges of his or her licence" (CAA Guidance on Medical Standards). These conditions, among others, include neurological, respiratory, cardiac and psychological. It is reasonable to assume, therefore, that Richard Mark Westgate commenced his life as an airline pilot as a very healthy person, both physically and psychologically. It is also known that he was very athletic, and enjoyed paragliding in particular ***[An appreciation of Richard Westgate – Copy from SE Wales Hang-Gliding and Paragliding Community Website Vol 2 Tab 6].*** We can take it that for a reasonable period of his life and career, the Deceased kept excellent health. At some point his health started to degenerate, still at a young age.

3 Aircrew and passengers in all airliners (with the exception of the Boeing 787 'Dreamliner', together with a very small number of aircraft, including private business jets, that are fitted with special bleed-air filters) are routinely exposed to neurotoxic substances in the interior air. ***[Excerpt from Cranfield***

report Vol 2 Tab 7] **[***ASHRAE Request to the FAA and EASA for routine monitoring to be installed on all aircraft Vol 1 Tab 21] [SAE Aerospace Information Report Vol 1 Tab 22***] [***CAA Paper 2004/04 Cabin Air Quality Vol 1 Tab 23] [Federal Aviation Administration Report to Congress "Aviation Safety" AVS-1 Vol 1 Tab 24] [ICAO Working Paper 16 September 2013 A38 – WP/245 Vol 1 Tab 25] [Solbu 2011 O***rganophosphates in aircraft cabin and cockpit air Vol 2** *Tab 8] [***Picture of typical warning on hydraulic reservoir Vol 2** *Tab 9] [Eckels et al 2014 Aircraft recirculation filter for air quality and incident assessment Vol 2 Tab 10] [Lyasova et al Exposure to tri-o-cresyl phosphate detected in jet airplane passengers Vol 2 Tab 11] [Boeing 787 Statement to the FAA in support of Type Certification Vo 2 Tab 12] [AAIB Bulletin Boeing-757-236-G-CPET Vol 2 Tab 13]***.** The last cited report demonstrates the likely incidence of repetitive undetected routine exposure. Being repetitive, exposure is also, for the crew at least, cumulative, because when the flight ends, and the passengers disembark, the crew turn around and do it all again, time and again. The only air that enters aircraft after the doors are closed is drawn from the jet engines, or the auxiliary power unit (APU), a small jet engine in the tail of the aircraft. This air is hot and pressurised and is known as "bleed air". In the course of climb to high altitude and to cruise at those levels, airliners require to pressurise the interior of the aircraft. At an altitude of, say, 39,000 feet the interior is actually at a pressure and density equivalent to 6,000 to 8,000 feet (depending on aircraft type – 8,000 for older types and 6,000 for newer types). This is because life cannot otherwise be sustained at the altitudes, pressures and temperatures at which aircraft travel. The interior of a jet airliner is a hermetically sealed unit, with ingress of heated and pressurised air, and egress, or outflow, of expelled air. The interior air is fully changed about every 3 to 5 minutes (depending on aircraft type) with fresh bleed-air. The bleed-air problem has subsisted since the 1960's when bleed-air took over from ram-air and mechanical compressors, in early second-generation jets. Captain Susan Michaelis, herself an affected and grounded pilot, has produced two seminal and comprehensive published volumes on the problem, which are much too large to reproduce here. However they can be obtained through ***www.susanmichaelis.com***.

4 The engines and the APU use synthetic lubricating oils. This oil contains tri-*cresyl*-phosphate (TCP) at 3–6% measured by weight ***[MSDS TCP Vol 2 Tab 14],*** as well as N-phenyl-1-naphthylamine (PAN), alkylated di-phenyl-amines and phenol dimethyl-phosphate (DMP). The base stock of the oil is composed of pentaerythritol and pentanoic acid, with small variances depending on brand. Mobil Jet Oil II, which has more than 50% of market share, is used by British Airways. ***[MSDS Vol 2 Tab 15]***. Aircraft cabin air is also routinely contaminated with the constituents of aircraft hydraulic fluid. ***[Cranfield Excerpt from Final Report Vol 1 Tab 20] [Solbu 2011 Organophosphates in aircraft cabin and cockpit air Vol 2 Tab 8].*** Hydraulic fluid contains tributyl-phosphate (TBP), dibutyl-phenyl-phosphate (DPP), or butyl-diphenyl-phosphate (BDP) or a mixture of all three. [***MSDS for Skydrol Vol 2 Tab 16] [Wikipedia entry for Skydrol Vol 2 Tab 17]*** TCP, DMP, TBP, DPP and BDP are all organophosphates (OP). The Cranfield report confirmed that 73% of all flights tested by them had OP contamination. These contaminants are all part of a family of chemicals that started life as pesticides and nerve gasses. TCP, in particular, is an outstandingly efficient neurotoxin – meaning it damages nerves. That is generally what organophosphates were designed to do – to kill ***[Wikipedia – Organophosphates Vol 2 Tab 18].*** It was later found that Organophosphates OP's had special engineering qualities, and hence why they are used in the lubrication of jet engines.

5 In normal daily use the lubricating oil leaks into the bleed-air as an oil mist and so too does the hydraulic fluid. There is then no barrier, filter or other mechanism to prevent this contamination being breathed by all on board. The human lung is actually the first filter encountered by bleed-air. There is no other source of interior air once the doors of the aircraft are closed (unless the aircraft is and remains stationary before engine start and connected to a Ground Power Unit GPU). In a typical airliner this bleed air is fed into the airliner at ceiling height and extracted at floor level. Some of the bleed-air is then recycled, and mixed with new bleed-air. Some aircraft have a particulate filter in the recirculation loop only, but this filter does not, and cannot, remove the OP contamination. The particulates in these filters do, however,

become contaminated with TCP and TBP. An American scientist, Steven Eckels and his colleagues tested 185 used bleed-air filters and detected TCP in 90% of them. This supports the assertion that bleed-air is routinely neurotoxically contaminated. He states: "*An ancillary conclusion of this study is that TCP at some level is relatively common in aircraft cabin air*." **[*Eckels et al 2014 Aircraft recirculation filter for air quality and incident assessment Vol 2 Tab 10]*.**

6 In addition, the aircraft's drinking water can be contaminated, because the same bleed air is used to pressurise the potable water tank. ***[Airbus Flight Crew Operating Manual – Water and Waste Vol 1 Tab 18].*** This water is also used to make all forms of hot drinks on board.

7 The manufacture, shipment, use and handling of such dangerous chemicals in most countries attract mandatory labelling and warnings in the form of Material Safety Data Sheets (MSDS). ***[MSDS for Mobil Jet Oil II Vol 2 Tab 15]. Its product label can be seen in Figure 1].***

Figure 1

This warning is displayed on the back of the can. Crew and passengers do not ever see this label; nor it seems, does BA's Chief Medical Officer.

Some thought is required as regards the warning about swallowing. As we shall see later, industry apologists try to say or imply that one has to drink the oil to suffer serious consequences. There can be no doubt that the idea of drinking the oil was not in the minds of those who devised the warning. The industry overlooks two things. Firstly, when oil fumes are inhaled, saliva and the upper-airways mucous are contaminated by it, and of course this is constantly being swallowed. Secondly, it must be recalled that bleed-air pressurises the drinking water of the aircraft. Bleed-air contamination is therefore always likely to have this double effect in respect of both inhalation and swallowing; the two are relatively inseparable. In addition, skin contact is both to the product in liquid form as well as in the form of mists in the bleed-air. As we shall see, even miniscule quantities can have a serious effect, especially in repetitive and cumulative doses. Manufacturers are not usually noted for being overly generous in providing warnings, and one has to assume that such warnings as are provided, will be the minimum required by their lawyers and insurers.

8 Defective and, or, worn seals within the jet engine allow the oil to be sucked into the bleed-air. Some seals are of poor design. ***[AAIB Bulletin No: 2/2004 – Vol 2 Tab 19] [AAIB Bulletin No: 8/2001 – Vol 2 Tab 20] [AAIB Bulletin No: 6/2004 –Vol 2 Tab 21] [Australian Transport Safety Board 200203030 Vol 2 Tab 22] [Pratt & Whitney Report on seal failure Vol 2 Tab 23].*** Some seals require a small quantity of oil to be present on the surface of the seals, from where it is easily liberated into the airstream. Another type of seal depends for its sealing efficiency on air pressure opposing the oil pressure at the seal. This air pressure varies with engine speed. The opposing pressure forces are optimised for a certain engine speed. At other engine speeds oil can leak past the seals. During engine start and shutdown this opposing air-pressure is absent allowing oil-contamination of the surrounding engine structure, from where it is subsequently entrained into the cabin air and ducting. Thus all seals leak, even in normal use. In an engineering paper presented to the National Aeronautics and Space Administration, Raymond Chupp, an industry expert on jet engine seals, and an employee of General Electric a leading jet engine manufacturer, while discussing oil-seals, stated "A zero-

leakage seal is an oxymoron" ***[Chupp et al Excerpt from "Sealing in Turbo-machinery" NASA 2006 Vol 2 Tab 24].*** However, as we have seen, seals also wear in daily use and they fail completely if not replaced timely. The condition of all seals can only be inspected when the engine is disassembled during overhaul. Jet engines operate at very high temperatures, typically between 750° to 950° Celsius. It is accepted that the thermal degradation of the oils and fluids is more toxic than when there is no heating. ***[Van Netten and V Leung Hydraulic Fluids and Jet Engine Oil Pyrolysis and Aircraft air Vol 2 Tab 25].***

9 Jet engines were originally designed for a finite life. However, in practice, and as a measure to assist airlines' economy and mass travel by air, the life of jet engines has been repetitively and progressively extended by the airworthiness authorities, on the basis of a power assurance test that demonstrates that an engine is producing its full rated thrust, and an internal bore scope camera examination showing no structural weaknesses. However, the seals cannot be viewed using the bore scope examination. The state of engine seals is not taken into account at this stage. Rolls-Royce Jet Engine Division, for example, openly boasts on its website that it has an RB211 jet engine – the type which powers the Boeing 757 – and it "is a high time on-wing engine which has completed 40,531 hours without a shop visit" ***[Copy from RR website Vol 2 Tab 26].*** In airline use, this can represent 10 to 15 years life – without an overhaul. There are two ways an airline can tell if the seals need to be replaced. One is when the oil consumption becomes excessive. The other is when an excess of oil products is known to have entered the cabin air. ***[BAe Service Information Leaflet Vol 2 Tab 27].*** From an economical point of view, it will be infinitely less expensive to keep topping up with fresh oil (typically £13 a litre), than to have an engine taken off the wing and sent away for strip-down and rebuild. It is reasonably hypothesised that airlines will only commit to a major engine overhaul when the rising oil consumption risks an in-flight engine failure or mandatory shutdown and diversion. Additionally, airlines will not commit funding to a fleet in the process of being phased out, such as the BA Boeing 767. The

permitted maximum oil loss is 0.63 litre per flight hour per engine. ***[EASA Type Certificate. (area highlighted) Vol 2 Tab 28]***.

10 As we have seen there is abundant evidence that TCP and TBP are routinely present in bleed-air. Airbus 319-321 aircraft are no different from others in this regard. They have standard procedures for encountering it ***[Airbus Operational Engineering Bulletins N°21-00 and N°71-00 Vol 1 Tabs 16 and 17].*** Airbus aircraft operated by British Airways are no different in this respect from Airbus aircraft operated by any other airline. When oil mists contaminate the bleed-air, the ducting, which has thermal and acoustic linings, becomes contaminated. **[*CAA Paper 2004/04 Cabin Air Quality Vol 1 Tab 23*].** Unless the ducting is replaced or cleaned, contamination will remain. It can, therefore, reasonably be assumed that Richard Mark Westgate suffered repetitive and cumulative exposure to the neurotoxic contaminants of aircraft cabin air in the course of his employment. Indeed he has recorded in his case-history to Dr Mulder that he used to see oily puffs of smoke, and smelled oil in the aircraft after engine start ***[Witness statement of Dr Mulder Vol 2 Tab 1] [Case Study Vol 1 Tab 26]***. In any event, in the absence of airline monitoring of contamination levels on all flights, one can only make an estimate of actual exposure, based on studies such as Cranfield. One scientist, Professor Jeremy Ramsden, calculates such an estimate for the accumulation in the last 4 years of the Deceased's flying career (663.5 g TCP and 133 g ToCP) ***[Vol 2 Tab 29].*** Such an exposure is, relatively speaking, massive. It is also a reasonable calculation, given that the air transport industry conspicuously fails to fit equipment which will monitor the exposure level.

11 Due to the genetic variation in the population (DNA coding), some people are more at risk to their health with toxic exposure than others. [***Furlong 2009 Genetic Variability and Sensitivity to OP Vol 2 Tab 30]***. Detoxification of organophosphates, such as TCP, involves the human enzyme, paraoxonase 1. This hydrolyses the TCP, converting it to a metabolite known to science as cresyl saligenin phosphate. This has the scientific nomenclature of CBPD. The CBPD is then expressed from the body. That is what should happen in an ideal world. However we all have genetic switches, polymorphisms and

chromosome combinations, which are inherited. Some people have a good supply of this special enzyme, but others do not. The amount is genetically dictated by a polymorphism of the PON 1 gene. Some people are wholly unable, or have a deficiency, to detoxify such toxins as TCP and TBP, such that, as working crewmembers or even as frequently flying passengers, they are unable to detoxify between flights at a rate equal to, or greater than, the rate of re-exposure. As a result the toxins accumulate until the body is neurologically overwhelmed. The Deceased was, unknowingly, in such a category. ***(DNA Genotype Test Vol 1 Tab 11).***

12 The principal injury that is done is to the peripheral nervous system (PNS) (the nerves in the limbs) and to the central nervous system (CNS) (the brain, the brain-stem, and the spinal cord). As Professor Abou-Donia puts it:

"*There are three neurotoxic actions of organophosphates: first, cholinergic neurotoxicity caused by inhibition of acetylcholinesterase. Second, organophosphorus ester-induced delayed neurotoxicity (OPIDN), which is a central–peripheral degeneration of the central (CNS) and peripheral (PNS) nervous systems, followed by demyelination. The clinical picture for OPIDN is manifested initially by mild sensory disturbances, ataxia, weakness and muscle fatigue and twitching, which may progress to paralysis. Third, organophosphorus ester-induced chronic neurotoxicity (OPICN) is characterized by long-term deficits accompanied by brain neuronal cell death*" ***[Westgate Case study Vol 1 Tab 26].***

13 The Deceased exhibited all of these symptoms for some time before death. ***[Mulder, GP case notes Vol 1 Tab 10] [Westgate Case Study Vol 1 Tab 26]***. His condition was so bad that he needed to have the services of an assistant, Luisa de Angelis ***[Witness Statement of Luisa de Angelis Vol 2 Tab 4]***. The Deceased's heart was seriously compromised by a condition known as lymphocytic myocarditis. One of the causes of such a condition is exposure to toxic substances. ***[Symbiant Report Vol 1 Tab 3].***

14 The pathologists reported that the Deceased had apparently consumed pentobarbital at a potentially lethal dose. The Deceased was suffering great pain and discomfort prior to death, with little or no prospect of alleviation. ***[Witness statement of Dr Mulder Vol 2 Tab 1. Et passim]*** He had been of an athletic and sporting disposition, and enjoyed, and was a champion at, outdoor activities such as paragliding. For such an individual to suffer

affliction like that would have been a burden difficult to carry. It is reasonably assumed that in an attempt to relieve the pain, and in the absence of any cures or treatments, the Deceased self-medicated on well-tried, and traditional substances such as pentobarbital, a well-known barbiturate. Throughout Europe this is a prescription-only medication. After his death a search of his personal belongings, firstly by the police and then by his brother did not reveal any more of the pentobarbital than had apparently been consumed, and, crucially, no proper pharmaceutical container for this medication was found ***[Witness Statement of Police Chief Superintendent Ronald Rijnbeek Vol 2 Tab 3] [Witness Statement of Guy Westgate Vol 2 Tab 2***]. Two reasonable assumptions can therefore be made; firstly there was nothing to provide guidance as to a safe dosage, and secondly, the Deceased may not even have been aware that what he had taken was pentobarbital. This substance can be procured on the black market locally in Amsterdam or obtained via the internet. Many large cities, like Amsterdam, are renowned for back-street apothecaries, who will, for a fee, seek to provide a "miracle cure" ***[Witness Statement of Police Chief Superintendent Ronald Rijnbeek Vol 2 Tab 3]***. For these and other reasons, the Deceased should not be regarded has having deliberately ended his life. There is other evidence pointing to a conclusion that the Deceased's death was unintended: he had been sleeping on a 'pillow-angel', which had been deliberately set to "on", to arouse him in the event of an attack of apnoea; it would have sounded, as in fact it did, as soon as his breathing stopped. He had been sleeping with a mouth guard for the same reason. Richard Westgate never left a note for his family, an omission that they consider unthinkable if his death was intended ***[Witness Statement of Guy Westgate Vol 2 Tab 2]***. The pathologists were able to discover, by hair sample analysis that he had been taking this medication for some time before death ***[Westgate Case-study Vol 1 Tab 26]***. In any event, due to the established damage to the myocardium, to the phrenic nerve and to his nervous systems, death would have been facilitated, more so than in an individual whose heart and nerves were not compromised in this way. ***[Symbiant Report Vol 1 Tab 3] [Westgate Case-study Vol 1 Tab 26].*** Dr Mulder had referred the Deceased to a psychotherapist, who had concluded

that the Deceased had no suicidal tendencies. ***[Witness statement of Dr Marja Visser Vol 1 Tab 29]***

15 In August 2014 a peer-reviewed scientific paper, on the subject of the Deceased, was published as a case study ***[Abou-Donia et al Autoantibody markers of neural degeneration are associated with post-mortem histopathological alterations of a neurologically-injured pilot Vol 1 Tab 26]***. This is the first time that a grounded aircrew member has been able to be followed all the way through from presentation of symptoms, diagnosis and tests to post mortem histopathology findings. The authors were able to demonstrate evidence of extensive injury to the Central Nervous System (CNS – brain and spinal cord), which correlated well with evidence of T-lymphocyte infiltration (severe inflammation) and demyelination of the Peripheral Nervous System (PNS). In turn the injuries to the CNS and the PNS correlate well with *ante mortem* results of neuro-specific autoantibodies, which showed the biomarkers to be greatly elevated as against those of a control group. In turn this correlates well with the diagnosis of neurological injury reached before death. Moreover, the general clinical picture is consistent with (a) what can be expected from OP exposure and (b) that observed time and again in sick and grounded aircrew whose condition is associated with the exposure to the toxic and neurotoxic contamination of aircraft internal air [See Paragraph 25 *infra*] ***[MacKenzie-Ross Cognitive Function Following Exposure to Contaminated Air on Commercial Aircraft: A case series of 27 pilots seen for clinical purposes Vol 2 Tab 31]***.

16 What is even more disturbing, however, is the authors' assertion and proof, in the Westgate Case-Study, that long term low-level exposures **(LTLL)** to ***very*** small doses of organophosphate compounds are more neurotoxic and more efficient in producing organophosphate-induced delayed neuropathy (OPIDN) – a condition from which Richard Westgate suffered - than a large single dose. The authors explain that the ***minimum*** threshold for a ***single*** oral dose of Tri-ortho cresyl Phosphate (T*o*CP) – a constituent of jet engine oil - that produced OPIDN in hens was found to be 250 milligrams (250 thousands of a gram) per kilogram of body weight, whereas, spectacularly, by way of

contrast, 36 daily much smaller doses of half a milligram per kilogram of hen weight caused the same OPIDN. 36 doses of half a milligram each amount only to a total of 18 milligrams (18 thousandths of a gram); in other words, the total minimum threshold ***single*** dose that caused OPIDN was 14 times greater than the total of the repetitive daily smaller divided doses which produced the same effect. ***[Abou-Donia Organophosphorus Ester-induced Chronic Neurotoxicity Vol 3 Tab 1]*** Such proof was also supported by other scientists. ***[Jamal et al Low level Exposures to Organophosphorus Esters May Cause Neurotoxicity Vol 3 Tab 2]***. The observation that LTLL exposure is more damaging than a larger single dose, is extremely important. This is because the air transport industry makes much of the admittedly low levels of these pollutants in the cabin air in order to assert that "cabin air is safe". Airlines do not deny the presence of toxic and neurotoxic components in the airliner air, but they do deny that this contamination is at such a level that could result in harm. However, their position is wholly undermined by the scientific observations of LTLL exposure.

17 Another significant scientific paper was published in September 2014. This demonstrated that glutamate signalling of mouse brain cells was impaired by the administration of ***extremely*** low concentrations of T*o*CP ***[Hauscherr et al Impairment of Glutamate Signaling in Mouse Central Nervous System Neurons In Vitro by Tri-Ortho-Cresyl Phosphate at Noncytotoxic Concentrations Vol 1 Tab 27].*** Glutamate is the most prolific neurotransmitter in the brain. The importance of this paper should not be underestimated. Not only does it show that impairment of brain functioning is observed at *extremely* low dosages of T*o*CP – dosages as low as 100 ***billionths*** of a mole, itself a molecular measure - the paper establishes that neurotransmission is damaged ***even before*** there is any observable brain cell structural damage. The air transport industry while being forced to admit that cabin air contamination is routinely present, maintains that it is at such a low dosage that no possible harm can occur. These two papers, and many others, together with that of Dr Goran Jamal, above, and many others contradict that assertion.

18 An astonishing aspect of the last eighteen months of the Deceased's life, specifically prior to his time in the Netherlands, centers on the inability of the UK's medical profession to propose a diagnosis of his condition and what may have been causing it. ***[Medical GP notes Vol 1 Tab 9].*** For much more than a decade prior to the Deceased's medical crisis, aircrew had been reporting sick, alleging a connection with the toxic and neurotoxic contamination of the aircraft cabin air. A number of specialist UK doctors had confirmed such a connection, for example Dr Jenny Goodman, Dr Sarah Myhill and Dr Charles Forsyth, Doctors Munro and Dr Julu at the Breakspeare Clinic. So too had some grounded crewmembers. ***[For example "Letter from Karen Lysakowska to Captain Warner, Fleet Captain BA Boeing 757 fleet dated 8 May 2006 Vol 3 Tab 3]***. Many grounded crewmembers have brought this to the attention of the CAA, for example, Captain Tristan Lorraine, Captain Julian Soddy and Captain John Hoyte and many others. The airlines and regulators have chosen to disregard such claims and they have never been investigated. British Airways continues to deny that there is any evidence that aircrew are exposed to substances hazardous to health ***[Letter from BA Heath Services to Cannons Law Practice dated 21 March 2013 Vol 1 Tab 31]***. The letter from Dr Mark Popplestone, Consultant Occupation Physician states "***as there is no evidence that pilots and crew on board an aircraft are exposed to any substances hazardous to health, health surveillance is not required and therefore has not been undertaken***". Such a complacent statement cannot be sustained in the light of a plethora of scientific findings to the contrary, some of which is produced in support of this folder of evidence for Richard Westgate's Investigation and Inquest. The industry continues to ignore the injury to crewmembers caused, and overlook, particularly, the genetic variation in the population.

19 When persons enter aircraft, whether as passengers or aircrew, they are not made aware that they may thereby be exposed to toxic breathing air. Similarly when aircrew are hired by airlines, they are not made aware that they may thereby be exposed to toxic breathing air. Neither aircrew nor passengers ever see the warning on the label shown at paragraph 7 of this Statement. Unaware of any risk, they do not make any conscious choice to be

so exposed but more especially they are at a disadvantage when symptoms of neurotoxicity appear. ***[Deceased's GP Records Vol 1 Tab 9].*** In addition, the medical profession are not made aware of the toxicity of aircraft cabin air, and the injury that can be caused. This usually results in misdiagnosis and therefore mistreatment of sufferers, as was the case with the Deceased. He was provided with the wrong medication. He was told that he was imagining his symptoms. On the recommendation of his doctors in England, the Deceased had been admitted to the Nightingale Hospital, a psychiatric hospital. While in the Nightingale, and for several weeks thereafter, the Deceased was under the care of Professor Gordon Turnbull. The Deceased's GP, Dr Glover, had referred his patient to Professor Turnbull because Professor Turnbull is an Aviation Psychiatrist. In spite of quite a lengthy and close relationship between the Deceased and Professor Turnbull, no firm diagnosis was achieved. Professor Turnbull is Consultant Psychiatrist to British Airways and to the CAA. He is a Visiting Professor of Aviation Psychiatry. He is, or ought reasonably to have been, well aware of the allegations made by both specialist doctors and aircrew to both British Airways and the CAA of the alleged association between aircrew neurological injury and cabin air exposure. Yet Professor Turnbull never at any time saw fit to mention it to the Deceased or his GPs.

20 As one of the consequences of the report by the CAA in 2004, and a symposium hosted in London by the British Airline Pilots Association (BALPA) in 2005, the Department For Transport (DfT) commissioned a report, from Cranfield University, on the subject of the contamination of bleed-air. Cranfield reported in 2011. ***[Excerpt From Cranfield Final Report Vol 1 Tab 20]***. The Cranfield Report was welcomed by the air transport industry. This may have been due to a perceived exoneration of the industry in the report's conclusions that detected levels of toxic exposure were very low. The industry's misconception of the report as exonerative, conveniently overlooks the report's actual findings and measurements of toxic and neurotoxic contamination of bleed-air in up to 73% of all flights tested. It is not beyond conjecture or suspicion that the remaining 26% of flights might also have been contaminated. This is because there is an instrument

minimum cut-off level on the measuring devices, below which all that can be said is "not detected". However, in the report these values are accorded a "score" of zero (implying zero contamination), which for all that is known, may not be warranted. Moreover, these toxic measurements were obtained on routine, as opposed to "fume event" flights, and were not conducted "blind" or unannounced. In other words Cranfield *substantiated* allegations that oil seals and hydraulic systems leak in daily use, and that toxic and neurotoxic contamination occurs on a routine basis. One also assumes that the airlines, when they agreed the provision of flight facilities for Cranfield's investigators to be on board with test equipment, the worst individual aircraft for contamination would not have been selected. No longer could it be asserted that aircrew and passengers were only exposed to the risk of toxicity of bleed-air whenever there was a major failure of an engine oil seal.

21 The eventual general admission by the industry that bleed-air was routinely contaminated, albeit at low levels, may have been the prompt for its medical experts to devise causes, other than the neurotoxic contamination, for aircrew symptoms. Two ideas appeared to them to have a chance of bearing fruit – the "nocebo" effect and hyperventilation.

Firstly, the idea of the "nocebo" effect was proffered. This borrowed a notion from double-blind medication trials, where there is often a "placebo" effect. Just as a participant who is taking an inert pill "sees" an improvement, the "nocebo" effect postulates that an aircrew member who knows, or suspects, that he is being exposed to neurotoxic contamination, will "make himself ill" as a result. In short, if he thinks he is being poisoned, he will act like he is being poisoned. This is a disease of psychogenic origin; in other words it is all in the mind – the sick and grounded aircrew are really imagining it. The beauty of this argument is that it is assumed to be difficult to disprove - difficult to disprove, that is, until, in Richard Westgate's case, doctors, scientists and pathologists uncovered, after his death, the very *physical* nature of the Deceased's symptoms. This included neuronal cell-death, demyelination, lymphocytic infiltration and an overweight brain, painfully herniated, painfully striated and painfully pressing and bulging to escape

beyond the skull that contained it. ***[Witness Statement of Dr Mulder Vol 2 Tab 1] [Case study Vol 1 Tab 26]***. In the light of these real *physical* scientific findings it is impossible to maintain a hypothesis of the "nocebo" effect. The "nocebo effect" also conveniently overlooks the fact that, at least at one time, neurologically-injured aircrew were often seen, who were blissfully unaware of any contamination of the bleed-air at all. The "nocebo" theory also fails to explain why only some aircrew are afflicted and others not – the genetic factor. In other words it is difficult to see any possible connection between a genetically derived PON 1 deficiency and a psychogenic aetiology such as needs to be involved in the "nocebo" effect. The use of the term has been extensively criticised by the medical profession itself, not least that when the term was first coined in 1961 by Walter Kennedy, he was careful always to refer to it as "nocebo response", and stressed that it was not an effect in the proper sense) implying it did not have real effect but an imagined effect. However, that notwithstanding, the idea of the "nocebo" effect gained, as we shall see, some traction.

22 The second idea as to a cause of the aircrew symptoms was known as hyperventilation. This postulated, with no proof whatever, that certain aircrew were so concerned about bleed-air contamination, (or even the stresses of their duties) that, to put it simply, they breathed more often than was necessary. Sick and grounded aircrew had stressed themselves, and their bodies, and as a consequence, had over-feasted on super-oxygenation. Of course, despite the fact that hyperventilation is a recognised medical phenomenon, such a preposterous hypothesis for the aircrew symptoms of Aerotoxic Syndrome could not be sustained in the light of the post-mortem findings with regard to the Deceased.

23 In April 2005, after its symposium mentioned above, BALPA presented compelling evidence to the Department for Transport (DfT). ***[Closing Speech by Jim McAuslan, General Secretary of BALPA Vol 3 Tab 4]*** As a result the DfT sought the advice of the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT). COT provided their initial response in 2007. Unfortunately COT interpreted this request for advice in

the narrowest sense thus limiting their activities mainly to questions surrounding so-called "fume events". The terms of reference to COT from DfT were:

"*To undertake an independent scientific review of data submitted by the British Airline Pilots Association (BALPA) due to concerns about the possible effects on aircrew health of oil/hydraulic fluid smoke/fume contamination incidents in commercial aircraft.*"

One result was that COT became fixated on the infrequency of, and the pressing desire to measure contamination in, fume events, to the exclusion of the more obvious question of whether there is daily contamination and whether it can make people sick. COT then embarked on a 4-year examination of the issue, but their initial 2007 advice called for more and better research, ***stressed that aircrew health should be regularly monitored*** (emphasis added) and ended with this advice to HM Government:

"*In view of the plausible nature of evidence linking exposures to short-term health effects, further research in humans should be conducted to determine whether exposure to contaminated cabin air is responsible for the reported ill-health in aircraft crew.*"

It is noteworthy that the DfT appear to have paid almost no regard whatever to any of this preliminary advice. They have never instructed "further research in humans … to determine whether exposure to contaminated cabin air ***is*** responsible for the reported ill-health in aircraft crew". To date no such research has been instructed. The CAA have never been instructed, nor have the CAA seen fit to take it upon themselves, as guardians of the "***occupational health*** and safety of aircrew" ***[HSE – CAA "Memo of Understanding" Vol 1 Tab 30]*** to monitor aircrew health. Then there is no evidence whatever that the CAA, as enforcers, have instructed the airlines to monitor aircrew health. By way of contrast, it is understood that the Royal Air Force does monitor aircrew health every 6 months for neurological and respiratory symptoms that could be associated with the contamination of aircraft air. ***[Personal Communication from Unite the Union].*** The Royal Air

Force aircraft suffer exactly the same contamination of the bleed-air as do the civilian airlines, from transport aircraft to front-line fighters and bombers.

24 Again it can be observed that the fixation of both the DfT and COT with short-term exposure to fume events resulted in the emphasis on only "short-term health-effects". This is because a fume event, being an occasion when the aircraft cabin fills with blue smoke usually for a relatively short period of time, the *immediate* injury is to lungs and airways, from which a recovery can usually be made. There was little or no consideration of the long-term-low-level exposure that causes more serious and irreversible neurological conditions.

25 COT then went on to consider the submission of evidence and finally, at the end of 2013, issued their advice which included the following statements:

"i Contamination of cabin air by components and/or combustion products of engine oils, including triaryl phosphates [Note by author: *triaryl phosphates (TAP) is a generic name for compounds including TCP*], does occur, and peaks of higher exposure have been recorded during episodes that lasted for seconds.

ii Episodes of acute illness, sometimes severely incapacitating, have occurred in temporal relation to perceived episodes of such contamination.

iii There are a number of aircrew with long-term disabling illness, which they attribute to contamination of cabin air by engine oils or their combustion products.

iv The acute illness which has occurred in relation to perceived episodes of contamination might reflect a toxic effect of one or more chemicals, but it could also have occurred through nocebo effects.

v While there is strong scientific evidence that nocebo effects can lead to (sometimes severely disabling) illness from environmental exposures that are perceived as hazardous, there is no simple and reliable way of establishing that nocebo responses are responsible for individual cases of illness. However, they are a plausible alternative explanation ***if toxicity seems unlikely*** *(emphasis added)*. Distinguishing whether acute illness from fume events is likely to arise from toxicity or nocebo responses depends on: assessment of the patterns of symptoms and clinical abnormalities in affected individuals; the levels of relevant chemicals to which they might have been exposed; and what is known about the toxic effects of those chemicals and the levels of exposure at which such toxic effects occur (including the possibility that some individuals might be unusually sensitive).

vi The patterns of illness that have been reported following fume events do not conform with that which would be expected from exposure to triaryl phosphates such as o-TCP (which differs from the pattern of illness that occurs with over-exposure to organophosphate insecticides and nerve agents). Over-exposure to tricresyl phosphates would be expected to cause delayed peripheral neuropathy. Given the short duration of reported fume incidents, in order to cause such toxicity, peak exposures would have to be much higher than those which have been indicated by monitoring to date. For example, the current short-term exposure limit averaged over a 15 minute period) for o-TCP is 300 µg/M^3 whereas the maximum concentration of the compound that was recorded in the cabin air-sampling study was 22.8 µg/M^3. Assuming that a peak of exposure was sustained for a minute, it would need to exceed 4000 µg/M^3 to breech the short-term limit. *(Note added: µg/M^3 means millionths of a gram per cubic metre of air.)*

vii 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***. (*emphasis added*)

viii Decisions to undertake further research will need to balance the likelihood that it will usefully inform further management of the problem against the costs of undertaking the work.

Finally, it should be emphasised that illness can be disabling whether it occurs through toxicity or through nocebo effects, and therefore there is a continuing imperative to minimise the risk of fume incidents that give rise to symptoms."

It is impossible to see how such advice could be mistaken as being comforting to the industry or, for that matter to its regulators or the DfT, who had commissioned the advice. As a piece of scientific detective work it is deficient, because once one had become fixated on fume events as opposed to long-term-low-level exposures, as uniquely applicable to working aircrew, the advice could have been gleaned much cheaper if the DfT had simply purchased and read a standard textbook on toxicology.

26 Bearing in mind the terms of reference of this committee as outlined above – "*To undertake an independent scientific review of data submitted by the British*

Airline Pilots Association (BALPA) due to concerns about the possible effects on aircrew health of oil/hydraulic fluid smoke/fume contamination incidents in commercial aircraft" - it is extremely difficult to see any justification for, or even any meaning behind, paragraph viii (above) of the conclusions of the committee. This, it will be recalled, was in the following terms: "Decisions to undertake further research will need to balance the likelihood that it will usefully inform further ***management of the problem*** against the ***costs*** of undertaking the work." One is left to wonder what was intended to be conveyed by this sentence. Firstly no attempt appears to have been made by the committee to provide the DfT with estimates or costs of "further research". And, secondly, the reference to "management of the problem" seems most odd. One could be forgiven for thinking that this final piece of advice was intended to signal to the air transport industry and its regulator that it would be "cheaper to continue to live with the problem". Could it have been intended to say that a limited number of ruined lives (and families) is a price worth paying?

27 The Common Law of England places a duty of care on employers to provide a safe place of work. **[Donoghue v Stevenson (HL) 1932. But this case, a Scottish Appeal, founded heavily on the minority view of Brett MR (later Lord Esher) in the English case of Heaven v Pender in 1883]** Yet aircrew are expected to work in aircraft that are known to be polluted with neurotoxic substances.

The Health and Safety at Work Act 1974, section 2, requires:

"2 General duties of employers to their employees.

(1) It shall be the duty of every employer to ensure, so far as is reasonably practicable, the health, safety and welfare at work of all his employees.

(2) Without prejudice to the generality of an employer's duty under the preceding subsection, the matters to which that duty extends include in particular—

(a) the provision and maintenance of plant and systems of work that are, so far as is reasonably practicable, safe and without risks to health;

(b) arrangements for ensuring, so far as is reasonably practicable, safety and absence of risks to health in connection with the use, handling, storage and transport of articles and substances;

(c) the provision of such information, instruction, training and supervision as is necessary to ensure, so far as is reasonably practicable, the health and safety at work of his employees;

(d) so far as is reasonably practicable as regards any place of work under the employer's control, the maintenance of it in a condition that is safe and without risks to health and the provision and maintenance of means of access to and egress from it that are safe and without such risks;

(e) the provision and maintenance of a working environment for his employees that is, so far as is reasonably practicable, safe, without risks to health, and adequate as regards facilities and arrangements for their welfare at work.

(3) Except in such cases as may be prescribed, it shall be the duty of every employer to prepare and as often as may be appropriate revise a written statement of his general policy with respect to the health and safety at work of his employees and the organisation and arrangements for the time being in force for carrying out that policy, and to bring the statement and any revision of it to the notice of all of his employees."

The Control of Substances Hazardous to Health Regulations 2002 (COSHH) requires:

6. (1) An employer shall not carry out work which is liable to expose any employees to any substance hazardous to health unless he has—
(a) made a suitable and sufficient assessment of the risk created by that work to the health of those employees and of the steps that need to be taken to meet the requirements of these Regulations; and
(b) implemented the steps referred to in sub-paragraph (a).

7. (1) Every employer shall ensure that the exposure of his employees to substances hazardous to health is either prevented or, where this is not reasonably practicable, adequately controlled;

Regulation 10 requires the employer to monitor exposure, and to maintain monitoring records;

Regulation 11…to carry out health monitoring of employees;

Regulation 12…to provide employees with information, instruction and training on the risk of exposure; and

Regulation 13…to provide an appropriate medical response in the event of exposure (regulation 13).

These regulations must be interpreted against the requirement of Regulation 5, which is in the following terms (editorial emphasis added):

5. (1) Regulations 6 to 13 shall have effect with a view to protecting persons against a *risk to their health, whether immediate or delayed*, arising from exposure to substances hazardous to health". The precise terms of regulation 6 are also noteworthy in respect of the terms of sub-paragraph 3, which dictate that (and again editorial emphasis added): "(3) The risk assessment shall be reviewed *regularly and forthwith* if— (a) there is reason to suspect that the risk assessment is no longer valid; or… (c) the results of any monitoring carried out in accordance with regulation 10 show it to be necessary."

Regulation 13 (appropriate medical response in the event of an actual exposure) is wholly overlooked. Upon the occasion of almost every fume event, which has taken place aboard British Airways jets in flight, the crew have, almost always, been referred to ordinary local A&E units, who are not made aware of what is, or could be, present in the smoke or fumes. So they will not know what they should be testing for. The minimum and most appropriate action for them, in the circumstances, is to draw blood, serum and take a urine sample, and then place them in refrigeration until they can find out. This seldom happens. This sort of response is wholly inadequate. Even local airport-based medical services units (every licensed airport is required to have one in-house) are unaware of what they need to look for in the event of an allegation of exposure to bleed-air [Personal communication under undertaking of anonymity].

Lastly on COSHH it is noteworthy, because of the complex and scientific nature of the bleed-air problem and its adverse health-effects, that employers should seek independent scientific advice, and, as we shall see later, not be reliant on in-house risk assessors or in-house doctors. The pursuit of such a risk assessment as is required in such complex matters requires to be scientific, knowledge-based, inquisitive, positive and pro-active. That is not beyond the capability of a major airline or the UK regulator.

28 The common law, the Health and Safety at Work Act and these regulations apply to airlines and to aircraft. There is no evidence that any airline fulfils these duties. British Airways, as we have seen, are well aware of the leakage of super-heated engine oil mist, hydraulic fluid and other toxic substances into the interior air. They continue to pretend that the interior air is safe. ***(Letter from British Airways (BAHS to Deceased's family's solicitor dated 21 March 2013 Vol 1 Tab 31] [Cabin Crew News issue 42 of 24 October 2003 Vol 3 Tab 5].*** They have been made aware over many years of claims that many of their aircrew become ill from breathing this contamination ***[for example Letter from a pilot to British Airways Vol 3 Tab 3]***. British Airways are aware, or ought reasonably to have been aware, that as a result of genetic variability in the human population, serious harm will result to certain individuals. They have taken no measures to detect, monitor or filter this contamination, and they do not provide any warning. No airline, and certainly not the Deceased's employers, British Airways, provides information, instruction and training to their employees about the risk or the possible symptoms, as required by Regulation 12. On the contrary, they have issued incorrect and misleading information to their aircrew. In the issue of British Airways' Cabin Crew News (issued to all cabin crew) of 24 October 2003, Dr Michael Bagshaw, (who was then Head of Occupational and Aviation Medicine at British Airways and who is now Aeromedical Advisor at Airbus) stated:

"Medical – Fumes in the Cabin

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

effects on the nervous system. However not every organophosphate compound will cause these problems, including those used in jet engine oil."

Obviously, as has been postulated above, nobody had shown the Head of Occupational and Aviation Medicine the warning on the can of Mobil Jet Oil II, shown in paragraph 7 of this Statement (and he has just confirmed that the brand depicted in the photograph is the oil used by British Airways). Certainly this published advice can be taken as a corporate admission of the contamination of bleed-air. The clear message that is taken from the statement is "it's there, but it will not do you any harm". As well as being former Head of Occupational and Aviation Medicine at British Airways for 12 years, and currently Aeromedical Advisor to Airbus and several other airlines, Dr Bagshaw is also Professor of Aviation Medicine at King's College London, and Visiting Professor of Aviation Medicine at Cranfield University. If the statement in Cabin News was intended to be reassuring to cabin staff (*"it's there but it will not do you any harm"*), it is doubtful if it had such an effect, particularly when cabin staff see so many of their colleagues fall sick and grounded allegedly due to the contamination.

29 By Memorandum of Understanding ***[Vol 1 Tab 30]***, the Civil Aviation Authority (CAA) became the enforcement authority for the ***occupational health and safety*** of aircrew (***paragraph 1 and paragraph 5.1.3 of Annex 8***). There is no evidence that the CAA currently fulfils this duty in regard to health risks to aircrew from contaminated bleed-air. In 2004, they produced a report about the health and safety risks, and flight-safety risks, from the neurotoxic contamination of bleed-air. Since then, they have done little or nothing to try to resolve this. On the contrary, it could be said that as regulators they have become complacent and compliant with the industry they should be regulating. ***[Communication from Dr Nigel Dowdall to Sandra Webber, undated but believed to be around the 17 April 2008 Vol 3 Tab 6]***. This communication concerns attempts by Dr Dowdall to counteract efforts of the lobbying groups Global Cabin Air Quality Executive and the Aerotoxic Association, and parliamentary questions by Members of Parliament and Lords of the Upper House. Dr Dowdall is currently the Head

of the Aviation Health Unit (AHU) at the CAA. He was formerly Director of Health Services at British Airways for 6 years. Apparently any apparent conflict of interest in such recruitment must have been undetected. The AHU provides Government with advice on aviation health issues, issues information on aviation health issues "for all stakeholders" (*quotation marks added*) - passengers, flying staff, health professionals, and is responsible for aviation health research. The AHU works alongside the Aviation Health Working Group (AHWG) of the CAA. Sandra Webber was formerly Head of Aviation Regulation at the DfT, then Head of Government Affairs at the CAA and then Head of Consumer Support at the CAA. She has since left the aviation industry completely. What is of public concern about this communication, beyond the intention of "countering the lobbyists" (Dr Dowdall's own words) and the sentence: "At present this issue has not gained momentum, however it is becoming a topic of increasing interest and there is a growing need to act as soon as possible to counter the impact of the lobbying groups", and the phrase "for all of us in the industry", is the obvious admitted collusion with Patrick Spink of British Airways Press Office. The collective apprehension and annoyance of all involved, both at the CAA and at British Airways, that sparked this hurried activity, was a Panorama TV program that was to be screened the following Monday evening entitled " Something in the Air" about contaminated bleed-air on airliners. The industry certainly had cause for concern and apprehension, because the Panorama investigators had taken surface-swabs as well as air samples from inside the cabin of a British Airways Boeing 757 and a BAe 146, and then had the samples analysed by Professor Christiaan van Netten of the University of British Columbia, in Canada. Van Netten, a world-renown aviation toxicologist, confirmed that ***all*** the samples tested positive for TCP, telling the investigators on air: "This proves that oil from the engine gets into the air and this is what you have been breathing when you took your samples." ***[Summary excerpt from BBC Panorama "Something in the Air" Vol 3 Tab 7].***

30 As a result of a request made by the solicitors for the Deceased's family to the CAA seeking statistical numbers of aircrew allegedly affected by the contamination of the aircraft air (HM Coroner had expressed an interest in the

numbers), the CAA replied that they could not provide an answer, as they do not hold medical data in such a form as would allow them to identify the numbers involved ***[Letter from CAA to Cannons Law Practice dated 4 November 2014 Vol 3 Tab 8]***. This is very surprising given that the CAA were able to respond more positively to a similar request (using identical wording as to the criteria of the search) on 4 October 2010. However this puzzle was solved by the terms of a letter from the CAA to Captain John Hoyte, Chairman of the Aerotoxic Association dated 10 June 2013. The background is that Captain Hoyte had sought, under Freedom of Information (FOI) rules, to elicit from the CAA an update of the numbers of aircrew who were grounded through losing their medical certificate by reason of illness which they alleged was due to the contamination of bleed-air. Captain Hoyte had obtained such information by letter from the CAA dated 4 October 2010, under FOI, using the same wording as to the selection criteria, which was in the following terms: "Pilots on the CAA database who have suggested an association between their illness and bleed-air." The answer on that date was 28. In other words the CAA Medical Department, on 4 October 2010, could unequivocally state that 28 pilots claimed to be ill and they and their doctors were alleging a connection with the contamination of bleed-air. When Captain Hoyte required the CAA, again under FOI, to provide an update of the figures in 2013, he was told the figure was "none". Captain Hoyte was astonished. Even he himself "had vanished" off the CAA Medical Database. When he appealed against that information on the basis that he had been given the number in 2010, the appeal was refused by the CAA. The CAA reasoning can be seen as follows.

Captain Hoyte had appealed to the CAA in the following terms:

"I attach a CAA Medical Department Group letter of 4th October 2010 which makes clear reference to there being 28 pilots on a database of pilots 'Who have suggested an association between illness and the cabin environment' to be now told that there are 'no pilots' is not only untrue but does not tell me the actual present number of grounded pilots. Please provide the latest number of pilots by return."

The reply he received from the CAA was as follows:

"The CAA Medical Department has again provided further context to their

initial response to your request.

"The CAA Medical Department has for a number of years maintained a separate record of cases of pilots who reported acute or long-term symptoms, which they considered were linked to exposure to contaminated cabin air. Following a review of the document, it has been determined that this information has no medical validity because: the inclusion of an individual is based entirely on the individual's subjective perception of the cause of their condition; there are no objective or diagnostic medical criteria for inclusion; there is no consistency in the symptoms and signs reported by the individuals or their treating doctors; it is likely that there is incomplete reporting of cases where a pilot experiences brief symptoms at the time of an event. Of the 31 cases currently documented, review of the database has identified 3 pilots who have more than one entry, leaving a total of 28 individuals. 14 of these have since returned to flying, or were never assessed as unfit, leaving 14 who remain unfit. No new cases have been added since June 2012 and we are not aware of any further cases that are currently being managed. As the information includes personal confidential data which will not be used for any purpose by the Medical Department, the document will in due course be deleted in accordance with the requirements of the Data Protection Act. The information will of course still be available in individual medical records.

"Conclusion:
"I have therefore concluded that the original response to your request was accurate, as no pilots have been "grounded as a result of toxic oil fume exposure", the apparent disparity between this statement and the earlier information provided, about a record of 28 such pilots, arising because of the reasons set out above.

Your appeal is therefore not upheld."

Such a conscious change of oversight can hardly be consonant with the duties required of the CAA, both as industry regulator and as enforcer of occupational health and safety of aircrew. They have dumped the only measure they had of the extent of the problem, so that now they are not in a position even to know how many pilots are sick and grounded by any alleged association between their illness and the contamination of bleed-air. However, the most disconcerting aspect of this particular exchange of correspondence is the CAA appeal statement that "no pilots have been "grounded as a result of toxic oil fume exposure" "One logical deduction is that if one gets rid of the list, you can then say there is nobody on it."

31 One must bear in mind that the CAA keeps no medical records whatsoever of cabin crew (CC) health. Until now CC were governed by loose rules that required an initial "medical' by their airline upon recruitment, and then the

airlines' Human Resources or medical department was deemed to "look after" them. The European Aviation Safety Agency (EASA) is in the process of changing that. After a transition period, which started in April 2014, CC will now require to pass an initial medical examination by an Aviation Medical Examiner (AME), and then pass a similar medical examination at intervals of not more than 60 months. Under the new rules, the medical records for CC will be held only by their respective employers, and not by EASA or any of the home countries' regulators such as the CAA. What this means, of course, is that the oversight of the *occupational health* of cabin staff by the CAA (and presumably other regulators in the EASA- block countries) will be blinded and may be wholly ineffectual. It has to be borne in mind that the European Flight Time Limitation Scheme (FTLS) that applies to all pilots – in order to limit fatigue and ensure that pilots are adequately rested before flight – do not apply to cabin staff. As a consequence cabin staff are rostered for more flight time than their flight deck colleagues, and so are exposed to bleed-air for longer accumulations. It could also be thought that the 60 months (5 years) period for renewal of flight attendant medicals is too long. However, while 5 years will be too long a period to pick up timely symptoms of Aerotoxic Syndrome, it will be left to individual airlines to voluntarily impose appropriate screening, or, perhaps, to take their responsibilities under COSHH seriously (like the MOD do with RAF personnel). This concept, by both the EASA and the CAA demonstrates "where their thoughts lie" relative to cabin crew health. While pilots' have the instant capability to allowing aircraft to crash, and so have far more frequent medicals, cabin staff are unlikely to have such an influence, and so do not need to be tested more frequently than every 5 years. What this demonstrates is a regulatory and oversight mind-set solely geared to flight safety only and not ***occupational health and safety*** of all aircrew.

32 Up to and including 2006 the CAA kept statistical data on fume events categorised by aircraft types, some of which types are far worse offenders in this respect than others. After 2006 the CAA appear to have discontinued this practice. 2006 is the last year when the CAA either maintained or published such information in such a form. The last published table appears at Vol 3

Tab 9, and although annual updates were promised after 2006 none have ever been published. There has been no explanation of why this is so, although assumptions can reasonably be made.

33 There is no evidence of any actual health monitoring of aircrew, beyond a database of those pilots who hold current medical certificates. There is no evidence whatever that any health information is gathered at all by the CAA in respect of flight attendants. It could be argued that the CAA duties under the Memo of Understanding are limited to those of an enforcer, but equally there is no evidence whatever that there is any enforcement oversight of airlines and their practices with regard to the health monitoring of aircrew.

34 There is also concern at the risk to flight safety posed by the fact that a pilot or flight attendant will not yet be aware that he or she has cognitive dysfunction, ataxia and all the other deficits inherent in the disease. Indeed the Deceased's own case-history supports this view; at some point in his medical history he must have reached a point, no doubt undetected by his annual medical examination, where it could be said that he was not "free of any condition, mental or physical, which [was] likely to interfere with the safe exercise of the privileges of his licence." The onset of, at least, the chronic illness from long-term-low-level exposure, is obviously not instantaneous, but must advance discretely over a period of years. It can be reasonably postulated that so long as the automatic systems of an aircraft are performing as intended, and the flight is running to plan, "the aircraft looks after itself". On-board flight computers have taken over from pilots to the extent that many regard themselves as flight managers, and they do sometimes struggle to maintain their skill and competence to the required high standard. However there has been more than a hint of serious speculation that certain aviation disasters that could have been averted by a competent crew, could, in fact, be the result of a crewmember or crewmembers who were in fact, and unfortunately, through no fault of theirs, underperforming due to symptoms of cognitive dysfunction and other symptoms caused by the long-term–low-level contamination of bleed-air. One crash upon which there was such speculation was that of the crash of Asiana Flight OZ214 at San Francisco on 6

July 2013. ***[Summary of the Details of the Accident to Flight OZ214 at San Francisco Vol 3 Tab 10]***. The official cause of the crash was "mismanagement of approach and inadequate monitoring of airspeed." That is an astonishing verdict, given the obvious experience and standing of two of the airline's most senior Captains, who were at the controls, one of them a senior training Captain.

35 Another air-crash that attracted the same sort of speculation, was that of Turkish Airlines Flight 1951 that crashed during landing at Amsterdam Schiphol Airport, Netherlands, on 25 February 2009, resulting in the death of nine passengers and crew, including all three pilots. Again the official cause of the disaster was "poorly monitored approach to land in excellent weather conditions". [***Summary of details of Turkish Airlines Flight 1951 at Schiphol 25 February 2009 Vol 3 Tab 11]***

36 With regard to the speculation about these two cases as possibly being associated with pilot dysfunction, it is important to be aware of two considerations. Firstly, the finding in both cases was that an experienced and otherwise competent crew allowed the aircraft to collide with the ground in good visibility. Secondly, on both occasions the aircraft systems had issued the expected aural and tactile warnings. In the case of aural warnings there had been the usual synthetic voice commands of "TOO LOW - PULL UP!!" several times – followed by "POWER – POWER!!". Both were punctuated by the usual warning "gongs" all clearly heard later by investigators on the flight deck sound recorder. The tactile warnings also deployed in the form of vibration from "stick-shakers" on both control columns, again heard clearly on the recorder. Naturally, where experienced aircrew, inexplicably, fly a serviceable aircraft into the ground, this will leave a vacuum to be filled by speculation. In both cases all that was required to save the aircraft and its occupants was for one of the pilots, at any time before 10 seconds to impact, to press the "Go-Around (GA)" button and the aircraft would have, by itself, applied full power and climbed away from the ground automatically. As will be observed, any delay is critical. One theory put forward is that a neurologically-affected pilot will take precious seconds to take in what is

confronting him, and this slower reaction time, even of one or two seconds in taking appropriate action can be critical. Such a matter is mentioned by Dr Mulder in his witness statement ***[Vol 2 Tab 1***]. In view of the seeming reluctance of the air transport and regulators to accept a possible connection between bleed-air contamination and pilot dysfunction and underperformance, such considerations will be over-looked. However if the speculation, even if seemingly wholly misguided, leads to the need for regular health monitoring of pilots, then it will have had some merit.

37 What is not, however, speculation, is the number of times flight deck crew have been acutely affected by debilitating symptoms which they allege are due to the toxic contamination of bleed air, and which have actually presented a clear danger to the safety of the flight concerned. ***[Excerpt pages 30 to 42 BFU Vol 3 Tab 12] [AAIB Bulletin: 7/2007 British Airways Boeing 757 G-CPET Vol 3 Tab 13]***

38 There is anecdotal evidence from grounded pilots, and their families, that what triggers a concern that they are neurologically unfit, is that they fear they will not pass their next simulator testing. Pilots are required to undergo periodic training and testing in a flight simulator. In some airlines this is carried out at six-monthly intervals. In other airlines, for example KLM, pilots undergo simulator sessions every 3 months, alternating sessions between testing of skill and competence in emergency conditions and recurrent training also in emergency conditions. The testing and training standard is strict, with the question for the trainers and examiners being; "are you happy to send your family off on a holiday flight with this pilot?" Against that background, some bleed-air affected pilots, fearing the worst, will self-report their symptoms to their doctor, and thus effectively be medically grounded. Others will fail their recurrent simulator test. They may then be offered extra training, particularly if their "fail" was marginal. The Deceased confided in Dr Mulder that he thought he might fail his next simulator test. ***[Dr Mulder's witness statement Vol 2 Tab 1].*** It may be that such an event as failing a routine skill and competence test could act as a trigger for industry oversight of the bleed-air problem. Airlines are obliged to

maintain an individual Training File for each pilot. Testing is usually graded from A Plus to C Minus. There are minor variations on such schemes within all airlines. Tests involve competence, skill and knowledge. The CAA has regular access to these records on both planned and unannounced inspections. Usually no pilot achieves an A Plus (but it is not unheard of), not even the testers and trainers themselves. Most competent pilots who work at staying knowledgeable, skilled, fit and healthy will always achieve an A or a B, which is the industry standard. When a pilot achieves anything below a B minus, he or she will have a chat with the Chief Training Captain, as to what might be going wrong. Recurrent results in the C's and below, are usually followed by what is known in some airlines as a "Tea and Biscuits Interview" (notwithstanding that there is usually no tea and no biscuits). Airlines will have training records from which statistical data could be drawn. Repeated underperformance at training and testing will necessitate the dismissal of the pilot. The training and testing of pilots requires to be hard and unforgiving - society would not expect it otherwise. Nowadays there are specialised tests that can detect dysfunctional underperformance, but obviously the Deceased's UK doctors were unaware of them, and were unaware of any problem with bleed-air in the first place. Yet another early symptom that can be spotted is operational memory problems. All pilots are required to memorise a complex set of "memory items'. Standard Operations Procedures in all airlines dictate that each pilot, whether Captain or First Officer takes turn about, flight sector by sector, alternating between flying the aircraft - Pilot Flying (PF), and Pilot-Not-Flying (PNF). PF exclusively handles all flight controls and thereby executes the flight plan. PNF looks after the radio, and has routine communications with the cabin staff and passengers, but his/her most important task is to monitor the safe performance of the PF (even when that happens to be the Captain!!) and the flight navigation. When an inflight emergency occurs suddenly and without warning, such as a pressurisation problem, or an engine fire-warning, the PF will require to take precautionary action instantly, while the PNF calls up the check-list – now called a Quick Reference Handbook (QRH). Getting to the correct page for the correct emergency drill, whether it's on paper or on the computer screen, takes time – at times like this it may feel like an eternity - but in reality maybe 5 to 10

seconds or more if it is a complex or unusual warning. In that vital interval, the pilot flying must take the necessary vital actions; this requires to be done straight from memory. A recent example of this was the miraculous outcome of US Airways 1549 which had to ditch in the Hudson River when it suffered a double engine failure from a multiple bird-strike after take-off – the famous 3 minute flight - ***[US Airways 1549 Case Study Vol 3 Tab XX]***. Medical interviews with pilots who have memory deficits show that they will have some degree of difficulty on these, sometimes, complex recall of vital actions. They are usually memorised using mnemonics. Some admit to "going over them" time and again on the way to the airport. On some known cases this phenomenon of poor memory has led a pilot to seek specialised medical help.

39 The Deceased's family submit that the circumstances of the death fall within the powers and duties of Her Majesty's Coroner under section 32 and paragraph 7 of the fifth schedule of the Act. The requirements of that paragraph are:

"*Action to prevent other deaths*

Where—

(a) a Senior Coroner has been conducting an investigation under this Part into a person's death,

(b) anything revealed by the investigation gives rise to a concern that circumstances creating a risk of other deaths will occur, or will continue to exist, in the future, and

(c) in the Coroner's opinion, action should be taken to prevent the occurrence or continuation of such circumstances, or to eliminate or reduce the risk of death created by such circumstances,

the Coroner must report the matter to a person who the Coroner believes may have power to take such action.

A person to whom a Senior Coroner makes a report under this paragraph must give the senior coroner a written response to it.

A copy of a report under this paragraph, and of the response to it, must be sent to the Chief Coroner."

Epilogue

In the course of his Investigation into the circumstances of the untimely death of Senior First Officer Richard Mark Westgate, her Majesty's Senior Coroner posed a simple question: "How many people are we talking about?" He did not need to explain what he was referring to. It was, of course, taken as referring to "Aircrew who were alleging an association between their symptoms and bleed-air contamination." Neither the Deceased's representative nor the members of the scientific and medical team could provide a definitive answer. The Deceased's solicitor knew "about 50", and Dr Mulder knew "maybe 100". Other doctors dealing with such patients could add further to those estimates. Enquiries made of Aerotoxic Association and the GCQAE researchers and helpers could come up with anecdotal figures running, maybe, into "hundreds". A website run by Ms Dee Passon, an aerotoxically-grounded BA Flight Attendant and former nurse, has records of nearly 2,000 aircrew world-wide who devoted "up to an hour" of their time to taking an online health survey with respect to worries about symptoms that could be associated with bleed-air contamination. A few years ago Dee Passon took the results of her survey to British Airways management. They dismissed it as "unscientific". And, no doubt, it probably was, because she had done it at her own expense and using her own very limited skill and knowledge resources with regard to such complexities. However, British Airways said, in the light of the obvious alarm her survey revealed, they said they would undertake their own survey. There is no record anywhere, either anecdotally, or otherwise of any such survey ever having taken place. It is reasonably thought that any crewmembers who were prepared to devote so much of their time to such a "health survey", already had a concern as to the health implications for themselves or a close friend. However, it is noteworthy that the air transport industry and its regulator, the CAA, appear, deliberately, to make no effort to monitor the number of aircrew who are sick and grounded and who, ***and whose doctors***, allege an association between their condition and the neurotoxic contamination of aircraft cabin air. Without that vital information no start can even be made by the industry to rid itself of the serious allegations that have been made and are repeated in this Folder of Evidence.