

Cabin Air Quality: A review of current aviation medical understanding

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Concerns continue to be expressed about cabin air quality and perceived effects on health; this is a review of the current aviation medical understanding.

Cabin Pressurisation

Atmospheric pressure reduces with altitude, but normal people can tolerate a reduction in oxygen partial pressure up to around 10,000ft; above this, oxygen partial pressure reduces rapidly and impairs brain function. To provide a buffer for those not fully fit, the maximum certified cabin altitude in normal operation is 8,000ft.

During flight, air is derived from the compression stage of the jet engine or, in the case of the B787, from electrically driven compressors. This bleed air is conditioned and filtered, with an exchange of 10-15 times per hour with outside air and 20-30 times per hour including outside and filtered recirculated air.

Human Toxicology

Chemicals foreign to the human body must be absorbed from the surrounding environment and transported to the target site in the body for a toxic effect to occur. Routes of entry include ingestion (swallowing), skin absorption and inhalation (breathing) with sufficient concentration for the chemical to cross the many cell membranes. In the case of inhalation, the absorption of the chemical will depend on the percentage partial pressure it exerts within the total pressure in the lung alveoli as well as its solubility. The human senses, particularly smell, are generally effective in detecting potentially hazardous substances at a level well below that which causes harm (the major exception being carbon monoxide). For most volatile organic compounds, the normal detection level is around 1,000 times less than the level which is likely to harm health.

For organophosphates, exposure to sufficient doses of the ortho isomer may cause adverse effects on the nervous system, including impairment of neuromuscular and peripheral nerve synapse function (but not brain cognitive function). The majority of cases recorded in the medical literature since 1943 have been associated with swallowing contaminated food or drink, and reports of occupational intoxication are rare with no cases due to inhalation. There are legal exposure limits for hazardous substances at work, the Indicative Occupational Exposure Limit Values (IOELVs); for ToCP, the workplace limit is $0.1\text{mg}/\text{m}^3$ for 8 hours with an emergency short term limit of $0.3\text{mg}/\text{m}^3$ for 15 minutes. From knowledge of aviation respiratory physiology, it can be shown that these values remain valid up to a cabin altitude of 8,000ft (Ernsting J, Ward J, Rutherford OM. Cardiovascular and respiratory physiology. In Rainford DJ, Gradwell DP (eds). Ernsting's Aviation Medicine 4ed (2006) ISBN-10 0 340 81319 9: 13-40).

Physiology of Breathing

The total pressure in the lung alveolus is the sum of the partial pressures of all the gases in the mixture, and the transfer of any gas across the alveolar membrane depends on the properties of the membrane and the partial pressure exerted by that gas within the mixture.

Oxygen and carbon dioxide are exchanged in the alveoli; the partial pressure of oxygen is higher in the air than in the blood so it combines with haemoglobin to be carried to the tissues, whereas carbon dioxide is at a higher partial pressure in the blood so is given up to the alveolar air. It is important to note that it is partial pressure (related to concentration) which drives the exchange. There is water vapour in the alveoli as well as oxygen, carbon dioxide and nitrogen, and while the partial pressures of the atmospheric gases fall with increasing altitude, the water vapour pressure remains constant as a result of metabolism.

Alveolar absorption depends on Dalton's Law of partial pressures, as well as Fick's Law, and the partial pressure of bleed air contaminants would therefore be a very small proportion of the total alveolar gas pressure, reducing rapidly.

Organophosphates and Jet Engine Oil

Jet engine oils contain synthetic hydrocarbons and additives, including the organophosphate tricresyl phosphate (TCP). Engine lubricating oil contains around 3% TCP which acts as an anti-wear additive, alongside flame retardant properties. Small differences in the molecular structure alter the chemical properties and any associated health effects; the para and meta isomers are not toxic to humans, while absorption of sufficient doses of the ortho isomer by ingestion (swallowing) may cause adverse effects.

Of the 3% concentration of TCP in engine oil, the ortho isomers (ToCP) consist of less than 0.2% of the total TCP. Thus the overall concentration of ToCP within the engine oil is less than 0.006% of the total constituents.

Taking the RB211 engine as an example, the maximum engine oil possible in the bleed air is 0.4kg. Of this, 3% is TCP of which around 0.1% is ToCP. In the worst case scenario of the total discharge of an engine's lubricant into the engine bleed system, 0.4kg of oil would pass into the cabin ventilation system. This would give a peak cabin atmosphere ToCP level of 0.025 mg/m³, reducing rapidly as a result of normal cabin ventilation. The peak level would be a quarter of the statutory 8hr workplace limit of 0.1 mg/m³, and less than a tenth of the 15min emergency workplace limit of 0.3 mg/m³ (The United Kingdom Parliament - Select Committee on Science and Technology – Fifth Report (04-10-2006): 4.39).

Of the published levels of ToCP detected in cabin air, most are less than 0.005 mg/m³.

Another way of expressing gas concentration is as parts per billion (ppb), and for TCP 1 ppb is approximately 0.007 mg/m³. [To assist visualisation, in terms of time 1 ppb would be analogous to expressing 1 second in 32 years.]

It would be highly unlikely, if not impossible, for such small concentrations of contaminant to cross the alveolar membrane so as to cause organophosphate poisoning through inhalation. It is important to note in this regard that there are no published peer reviewed reports of

acute organophosphate poisoning with analytical confirmation of the diagnosis after cabin air fume exposures.

Studies of Cabin Air

In-flight studies in Canada (1998), USA (1997, 2000) and UK (2004) failed to detect TCP, with concentrations of all oil compounds well below the human toxicological threshold on which IOELVs are based.

The DfT Aviation Health Working Group commissioned Cranfield University to carry out cabin air monitoring. The initial ground investigation in a BAe146 found low levels of tri-n-butyl phosphate (TBP) and TCP in air samples, together with other organic compounds. An in-flight fume event was observed in a Boeing 757 when slightly elevated levels of TBP and TCP were measured, all significantly below the relevant workplace exposure limits.

The Institute of Occupational Medicine published a study of contaminant residues on cabin surfaces in 2012. The residues were similar to those in control ground vehicles, consistent with findings from the University of British Columbia in 2009, which noted TCP is found in wipe samples taken in buildings and other public places.

In February 2012, BRE UK facilitated a workshop at Hunton Park of international aviation, health and toxicology experts to review evidence associated with cabin air fume events. It concluded that there are no published peer reviewed reports of acute organophosphate poisoning with analytical confirmation of the diagnosis after cabin air fume exposures. There is no evidence to support a causative association between cabin air fume exposure and short or long term nerve damage.

It was noted that there is similarity between the reported symptoms of some crew members after fume events, particularly when emergency oxygen masks have been used, and the classical symptoms of hyperventilation.

The Australian Government Civil Aviation Safety Authority independent Expert Panel on Aircraft Air Quality in 2012 reached similar conclusions.

A German study in 2013 of 332 crew members who had reported fume/odour during their last flight, failed to detect metabolites of TCP in urine samples. The authors concluded that health complaints could not be linked to TCP exposure in cabin air.

Aerotoxic Syndrome (sic)

A syndrome is a symptom complex, consistent and common to a given condition. Sufferers of the 'aerotoxic syndrome' describe a wide range of inconsistent symptoms and signs with much individual variability.

The evidence was independently reviewed by the Aerospace Medical Association, the US National Academy of Sciences and the Australian CASA Expert Panel. All concluded there is

insufficient consistency to establish a medical syndrome and the 'aerotoxic syndrome' is not recognised in aviation medicine.

Irritability

Individuals vary in their response to sensory stimuli, including smells. Genetic differences are thought to cause some people to experience sensitivity to some chemicals with a range of irritant symptoms affecting well-being. This might explain some cases of reported ill-health following cabin air smells.

Hyperventilation

This is a normal human response to any form of stress or anxiety, and to resistance to breathing as when using an oxygen mask. Resulting nerve sensitivity and brain effects can cause alarming symptoms which increase anxiety. Obviously not every case of 'aerotoxic syndrome' is caused by hyperventilation, but it offers a plausible explanation for some well-publicised events. Research has raised concerns about the prevalence of unrecognised hyperventilation amongst airline pilots and the potential risk to flight safety.

Summary

What is known:

- The maximum theoretical peak concentration of ToCP from engine oil contaminating the cabin air is one quarter of the long-established validated workplace safety limit
- Most pressurised aircraft occupants do not report symptoms despite having the same exposure as those who do
- 'Aerotoxic syndrome' does not fulfil the definition of a medical syndrome
- Some symptoms in some cases of 'aerotoxic syndrome' can be explained by hyperventilation
- Other cases may be explained by individual chemical sensitivity to smells.

What is unknown:

- Whether there are substances present in cabin air which cause harm to health
- Why there is such individual variability in perception, response, symptoms and signs
- Why some crew in a limited geographical area report a wide variety of symptoms attributed to contaminated cabin air, yet others world-wide do not
- Why there are no reports from individual passengers on pressurised aircraft
- With such small numbers, it is difficult to establish a causative association.

Conclusion

There has been an increase in reported incidents of in-flight smoke/fume events since 1999, with a small number of crew members reporting adverse health effects which they associate with the events.

The source of oil contamination of engine bleed air was identified in early versions of the BAe 146 and the Boeing 757 and suitable modifications were implemented. A range of chronic health effects continue to be reported by some crew members.

The toxic effects of organophosphates are specific and are due to impairment of neurotransmission in the peripheral nerves, giving rise to muscular weakness and paralysis. In terms of medical toxicology, it is impossible to explain the wide range of symptoms and signs reported by some crew members as a unified result of TCP exposure.

Symptoms reported by some crew members who have been exposed to fumes in the cabin, particularly when emergency oxygen masks are used, are the same as those seen in acute or chronic hyperventilation. Obviously not every case of 'aerotoxic syndrome' is caused by hyperventilation, but it offers a plausible explanation for some reported events.

In some cases, the symptoms may be due to irritation associated with enhanced chemical sensitivity to certain volatile organic compounds.

The reported symptoms are wide-ranging with insufficient consistency to justify the establishment of a medical syndrome. It has been noted that many of the acute symptoms are normal symptoms experienced by most people frequently; some 70% of the population experience one or more of them on any given day.

Individuals can vary in their response to potential toxic insult because of age, health status, previous exposure or genetic differences.

In addition, it can be difficult to disentangle the physical, psychological and emotional components of well-being, and there is no doubt that different people will respond in different ways on different occasions.

It is not understood why most occupants of pressurised aircraft do not report symptoms despite having the same exposure as those who do. There have been no reports from individual airline passengers, even on flights where crew members have reported fumes or smells.

Finally, so far as scientific evidence has been able to establish to date, the amounts of organophosphates to which aircraft occupants could be exposed, even over multiple long-term exposures, are insufficient to produce neurotoxicity.

Investigations of aircraft cabin air world-wide have failed to detect levels of TCP above well-established and validated occupational exposure limit values. The partial pressure in the alveolar gas mixture of any TCP contamination of the cabin air is so low that it is unlikely to cross the alveolar membrane and be absorbed into the bloodstream.

Genetic or particular susceptibility to a particular adverse effect of certain chemicals on the part of an individual does not alter the need for there to have been a sufficient chemical exposure to cause the injury or damage. For the reasons set out above, the possible exposure levels to ToCP on aircraft are so low relative to what is required to create a toxic effect through inhalation that a toxic injury is simply not medically feasible with current understanding.

In seeking to explain the cause of reported symptoms and signs, aviation medical professionals throughout the world continue to monitor the scientific evidence and remain receptive to objective peer-reviewed evidence.

References

A detailed and referenced review paper is available on the web site of the Guild of Air Pilots and Air Navigators: <https://www.gapan.org/aviation-matters/guild-policy-and-comment/discussion-papers/>