Anatomy of an occupational hazard: Cabin air contamination in the air transportation industry
Part 4. Cabin air contamination on commercial aircraft

J.M. Lind*

CPCU ARM (USA)

Toxins emanating from the engines and bled into the cabin are critically appraised. It is concluded that the highest risk is due to carbon monoxide, with organophosphates being of secondary importance, and aromatic amines of almost negligible importance. In the absence of reliable measurements of the cabin air concentrations of contaminants, recourse is had to the symptomology of aircrew suffering from ill health.

1. INTRODUCTION

During the years before deregulation imposed its low-fares challenge upon airline executives, air contamination in aircraft cabins was not an issue. I know: I was one of perhaps four workers compensation underwriting officers in a position to know the details of industry-wide airline workers compensation costs. With my additional job description, Director of Safety, it was part of my work to identify unsafe acts, conditions and accident trends, and then offer prevention guidance to our policyholders. Fume events did not occur before deregulation or before competition forced ticket prices down 50% from prederegulation days.

At an 1970s average of 10 MUSD in annual incurred losses per major airline, workers compensation was the first cost to be excised from expense ledgers. Injured employees would learn that if their injuries were much more than short term and incurring low medical costs only, they were on their own. This fraudulent violation of state laws became systemic and remains uncorrected even until today.

Whenever possible, fuel at low cost was purchased at bargain prices in advance to try to level off the usual large price swings in the markets. Without access to before-and-after accounting information we cannot be certain of the depth of postponed or avoided powerplant maintenance costs. Supported by the new engine health management protocols, possibly anticipating the very high cost of maintaining the early high-bypass engines such as the General Electric CF6, the multimillion dollar price tag for overhauls has been generally avoided.

2. IDENTIFYING HEALTH AND SAFETY EXPOSURES FROM AIRCRAFT POWERPLANTS AND CABIN MATERIALS

2.1 Powerplants—main engines and APU exposures

Engines and auxiliary power units (APU) being internal combustion powerplants, they use jet fuel to operate and the same engine oil for bearings and accessory (generators, pumps) gear drive lubrication. Combustion exhaust is discarded overboard. Rarely if ever do jet engine exhaust fumes invade the cabin during flight; their presence in cabins could occur only momentarily during ground operations (taxiing) to, from and at the airport gate.

2.2 Contamination from superheated and burned engine oil

When heated to high temperatures engine oil can burn partially or wholly, leaving combustion products within bearing sumps and anywhere downstream from a bearing sump leak. Products of partial and complete combustion include:

- Visual evidence: thin, moderate and heavy smoke;
- Internal engine deposits: soot, varnish, coke and other carbon and associated material deposits;
- Invisible evidence: fumes derived from superheated compounds in the lubrication formula, carbon monoxide, tricresyl phosphate, oxides of nitrogen, and ultrafine particles [1,2].

When a bearing sump seal located on the spinning central shaft in the turbine section develops a leak, products of combustion can enter the exhaust airstream. If the leak is large enough, smoke may be seen emerging from the back of the engine.[1]

* E-mail: crewadvoc8@yahoo.com

[1] Example: the USAF T-33 trainer—seals in the aft section of the radial flow engine occasionally developed a static leak after engine shutdown. Upon engine start next morning, oil that had been accumulated in the engine exhaust housing sent a cloud of black smoke to the rear.

Journal of Biological Physics and Chemistry **23** (2023) 65–80
Received 30 July 2023; *accepted* 30 September 2023

doi: 10.4024/07LI23A.jbpc.23.01

When a leak escapes from a sump located in the compressor section forward of the bleed valves,[2] black deposits can accumulate in environmental control system (ECS) ducting and thereafter through the air conditioning units (packs) into the cabin. Similar deposits are seen in APU air ducting [4].

2.3 Powerplant degradation—engine and APU systems

During earlier periods of engine maintenance under manufacturer-recommended time between overhaul (TBO), engines were disassembled and cleaned. Time-limited parts were replaced, oil filters cleaned or replaced, areas of wear evaluated, and clearance tolerances checked and corrected as necessary. Essentially, these powerplants were upgraded to "like new" conditions.

Did air-conditioning ducts accumulate soot and other products of combustion in internal engine compartments? Speculating, and recalling that engine oil leaks were a rarity under active TBO maintenance protocols, we defend the idea that duct cleaning was not a significant issue. However, up to the mid-to-late 1980s, when tobacco smoking on aircraft became illegal, cabin air *outflow* assemblies were intolerably polluted.

Under engine health management (EHM) procedures, the TBO protocol was and continues to be postponed or avoided altogether. Engines remain in revenue service until sensors detect anomalies that demand maintenance attention. APU are not usually essential systems. When they fail, temporarily they can be shunted aside by listing them on the aircraft minimum equipment list until the next regularly scheduled maintenance service date. Even so, after the late 1980s and indefinitely later still, gradually the ducting accumulated significant filth. Here is a dialogue from a question-and-answer discussion on airliners.net:

> Q. Traveler—6 Dec 2011: "Travelling on AC034 from YVR to YYZ it was a shock to see this beautiful 777-200LR, which entered the fleet maybe in 2007, had very dirty ceiling air ducts along the aisle on both sides in economy, you could see dust along the vents if you looked up from your seat. Do we know how often these air ducts are cleaned on airlines?
>
> A. Tom—Do you mean the vent grilles (what you could see) or the ducts themselves?
>
> Cleaning the grilles can be done with a vacuum by the cabin-cleaning crew...how often would be up to the airline policy. Cleaning the actual ducts is difficult work and, if done at all, would probably only be done during heavy maintenance checks.
>
> Traveler—Yes, vent grilles. The dust was on them as well as on the ceiling near to them. I hope the grilles get cleaned by the cabin-cleaning crew with their vacuum. The ceilings on the 777 are high, maybe that is why they get left out from being cleaned.
>
> Tom—This is not a scheduled maintenance task; indeed, very few "cleaning" type activities are scheduled by maintenance. If the flight attendants write it up, it will get done, but that's unlikely to happen at any real airline.The vents get dusty. Just like in your home if you have a forced air system. And how often doYOU clean those? Maybe we should send someone over to check!!!
>
> I've seen lav / galley vent system ducts so dirty that it compromised the air extraction quantity out of the lavs to the point of failed smoke extraction testing.Things get messy behind the dado panels where the air flows out of the main cabin too.
>
> The cabin air filters also can be a contributor. I would hate to have to see the air filters that come out of our aircraft when they get changed on C check. Having seen the filters when brand new (when I was in stores we shipped them to the MRO firm) they were very clean. It's no wonder people get sick so often on airplanes.

2.4 Aircraft and aircraft powerplants—machinery by definition

We must recognize that these modern powerplants are the best designed and built pieces of machinery ever produced. Records show them now to be operated in revenue service continuously for 40,000 to 50,000 hours. This is equivalent to roughly12 to16 years of major-maintenance freedom and operationally safe service. Nevertheless customer satisfaction and occupational health issues are ignored. Sound maintenance practices are subordinated to customer demand for low fares and the flow of revenue.

Powerplants, however, are simply equipment, utilized under depreciation and replacement schedules. With continuous use, machinery will deteriorate, degrade and become operationally less efficient over time. Finely engineered tolerances between moving and stationary parts will erode due to vibration, rubbing and wear. In the most dynamic areas of rotational motion, vibration, temperature, pressure and expansion and contraction of parts will take a toll. Should these engines be maintained at high cost with the risk of keeping a somewhat less

[2] See engine schematics in the following Part 5 [3].

efficient tool in the inventory? Or should it simply be utilized to the last, discarded, and replaced with a new version?

A new, clean lubricating system will be an efficient system. Over long hours of uninterrupted use, products of combustion will build up in the bearing sumps and on the spinning central shafts. Soot will circulate to other areas, ultimately restricting flow through the oil filter, which if not changed periodically will become clogged and bypassed altogether. When this happens, dirty oil will pollute the oil reservoir and the greater lubricating system. Air vents and oil scavenger tubes will become restricted. Oil supply jets that spray oil onto bearing surfaces will become clogged or restricted. Operating with an inefficient lubricating system will accelerate dirt and sludge buildup, increase wear and decrease oil seal efficiency. Internal pressures and temperatures will climb. Oil leakage will increase, releasing a flow of toxic components towards the cabin [5].

2.5 Fume events before mid-1980

The period 1978 to 1995 was a nerve-rending time for airline executives. Having never been permitted to compete based upon ticket prices, they had no way of knowing exactly what would be demanded of them after Civil Aeronautics Board (CAB) oversight ended. Airline managers had never had to forage for customers; never worried about profits; didn't know how customer market share would be distributed. Law-makers assumed business-as-usual would continue.

Under CAB rule, the cost of thorough engine and APU maintenance was baked into ticket prices. After the CAB closed, maintenance accounting became an attractive department for cost-cutting. Oil fume "events" rarely happened. Illness symptoms among cabin occupants was not an issue. Accountants had no basis for that kind of worry.

My watch as Assistant VP Workers Compensation served the safety and health needs of our policyholders' employees under the various state workers compensation laws. We, the underwriters, had full and non-delegatable authority over work-related claims compensatability and payments to employees.

Our loss control services from the Hartford Insurance Company constantly monitored operations looking for unsafe acts and conditions. Comprehensive statistical data offered the opportunity to locate accident frequency trends establishing targets for ongoing safety services. No fume incidents came to light. No fume-related illnesses or injuries were ever alleged. No fume-related claims, not even bogus fume claims, were ever filed.

There were about thirty professional ground safety managers, employed by as many airlines as were members of the US National Safety Council Air Transport Section. I, too, was a member and officer. We met three times per year somewhere in the world. Our agendas and working sessions filled a week of discussions of safety problems and standards. Our cabin safety subject matter covered the galley, food and beverage carts, trips and falls, lavatories, emergency egress, etc. Never was the subject of a contaminated aircraft cabin mentioned. These safety professionals could never have overlooked such a hazard, especially since the insurance companies would have been paying the claims. Maybe that is why these safety professionals are no longer employed by US airlines.

2.6 Fume events after 1 January 1995

After deregulation, airline managerial uncertainty influenced a process of anticipatory cost cutting. Cost cutting would become systemic by the millennium's end and into the 21st century. But after 1978 executives presumed to postpone high-expense servicing issues. Statutory workers compensation insurance, highly predictable at an average annual cost per large carrier of 10 MUSD, was cancelled in mid-1980. Increasingly costly engine maintenance of the new high-bypass turbofan engines will have been very worrying to executives as regulator oversight waned during these transition years.

Testimonials from flight attendants and pilots of the time suggest a growing number of fume-related incidents. A significant airline accident in December 1995, American Airlines flight 965 in Columbia, South America, may have been investigated by the US National Transportation Safety Board without full knowledge of potential causes of this catastrophe. Toxic cabin air was not yet a hazard on investigator minds. Indeed, it still is not taken seriously.

Denial of compensation for work-related accidents gradually increased. More and more fume events complicated the flight attendant's daily routine. As my daughter said on the day she telephoned, barely able to speak and feeling ill from her first fume event, "We have been having these fumes and they aren't doing anything about it." She has experienced this entire transition period beginning in 1981. She and others like her have been my aerotoxic "weathervanes".

2.7 Maintenance correlates inversely with cabin air fume events

Considering fume event history over the interval from pre- to post-deregulation, an easy inference suggests the fewer engines brought in for overhaul, the more fume events occur. Under post-1980 airline management, which fatefully coincides with the increasing dominance of low- and high-bypass *turbofan* engines, and with eschewed maintenance under EHM protocols, everything changed. *Turbojet* engines like the Pratt & Whitney JT3 (Fig. 1) (used on the Boeing 707) were overhauled at 5,000 to 8,000 hours at an indicative cost of 250 kUSD. Overhauling a high-bypass turbofan engine such as the General Electric CF6 (Fig. 1) (used on the Boeing 747) is presumed to have cost roughly 1 MUSD during the transition period. Today the cost under EHM probably exceeds 3 MUSD per engine if overhauled at 30,000 or more hours.[3]

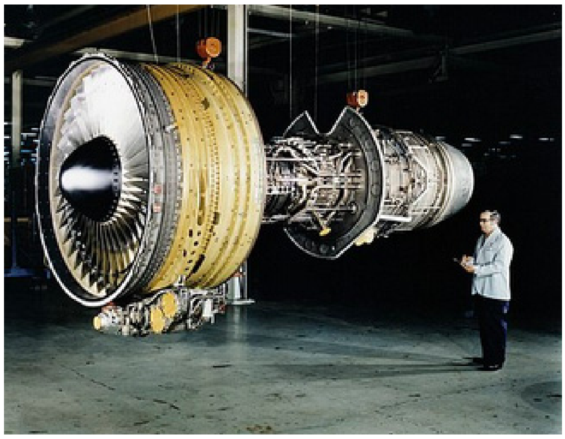

Figure 1. Left, Pratt & Whitney turbojet JT3C (J57) (National Air and Space Museum, Washington DC); right, General Electric CF6 (NASA Glenn Research Center, Cleveland, Ohio).

A JT3 overhauled under TBO protocols was affordably covered by CAB-dictated ticket prices set to cover all operating costs. The CF6 is presumed to have transitioned quickly from TBO to EHM protocols. Major overhaul could now be officially avoided. Specifically, oil seals were not required to be replaced, tolerances can continue unchecked, use of time-sensitive parts can be extended. Let sensor-based monitoring determine when maintenance is needed. As for dirty ducts, gaspers and air vents, ease of housekeeping was not built into the air distribution system. Labour-intensive, time-consuming, costly, and revenue foregone: cleaning ducts is simply avoided.

As an element of systemic cost-cutting, EHM is a primary participant. Over this period in US air transportation history, no proximate cause of cabin air contamination other than low ticket prices and operational cost-cutting is apparent.

3. MOVEMENT OF CONTAMINATION FROM THE POWERPLANTS TO THE CABIN: OBSTACLE—THE COMPRESSOR AIRSTREAM

Primarily concerned with issues internal to the aircraft environment, we have so far discussed only generally machinery and fumes originating in the main engines. Identical contamination can be generated in the auxiliary power units located in the tail section of most passenger aircraft. Cabin contamination produced in the APU will occur in much the same way as contaminants from the engines.

By way of introduction, and to provide a preliminary mental picture of the most likely contaminants that will escape the confines of bearing sumps and other origination points within the aircraft environment, the following list of contaminants is proposed:

Tricresyl phosphate
Nitrogen oxides
Carbon monoxide
Microscopic metal flakes
Particles and ultrafine particles
Composite particles and chemicals.

This list does not include all possibilities. Indeed, some references suggest that more than 125 identifiable contaminants have been found in cabin air. Useful for *research purposes*, they all may be formally addressed someday. Many occur with very low concentrations. For risk assessment and loss prevention purposes, most have received only *a probability of occurrence* assessment and *rejection as inconsequential or impractical to deal with*.[4]

3.1 The airstream through an engine

The flow of air through a turbine engine is a highly engineered, critically manufactured phenomenon. Its

[3] Scott, A. Jet engine makers battle over performance. reuters.com/article/instant-article/idCABRE95F0F220130616 (16 June 2013).
[4] Cf. ref. 6.

primary purpose is to provide a hot source of compressed ambient air from which the oxygen can be mixed with jet fuel, ignited, and violently burnt to produce thrust. The speed of air through an engine is very rapid. It increases from takeoff, through climb to cruise altitude and acceleration to cruise airspeed; that provided by high-bypass turbofan engines is typically 0.82 Mach (660 mph or 1060 kph) at 40,000 feet above sea level (FL400). This is the speed of the air at altitude that tries to enter the air intake of an engine.

Air is ram-forced into the forward engine intake. Generally, the first stage fan, second stage low- and third stage high-pressure compressors start a sequence that heats and pressurizes the air. Incrementally, through the gradually narrowing compressor cross-section, air compression increases, and flow rate decreases somewhat. Decreasing flow rate is caused by increasing flow or fluid (dynamic) pressure. Increasing dynamic pressure decreases static pressure. In this scenario, an open bleed valve should decrease static pressure at the orifice even more and increase fluid pressure into the bleed outlet. If this interpretation is correct, it would create an attractive route for compressor air to follow as it leaves the compressor and begins the trip to the cabin.[5]

Although somewhat slower through the compressor compared to airspeed, the speed remains very rapid. When the relatively static air within the bearing sumps leaks or is forced out, it (and any oil or other contaminants borne by it) must cross this stream if it is ever to invade the passenger cabin and flight deck. This hurdle is rarely discussed. Its apparent function as a barrier to cabin contamination needs a fluid dynamics engineer's interpretation. But we know beyond any doubt from crew member testimonials, crew and passenger visits to emergency rooms, and media reports of smoke in the cabins and on flight decks, that contamination does occur. Black deposits in bleed air ducting confirm passage of the products of combustion across the compressor airstream. They accumulate over years of regular operations, which supports understanding that oil seals are not 100% effective and small leaks through the seals occur at least part of the time on any aircraft.

3.2 The engine compressor—a field investigation

How can oil aerosols, gaseous fumes and ultrafine particles actually cross the compressor airstream? Mick Fowler, a first officer permanently disabled and grounded in 2010, made a luncheon appointment for us with a former Pratt & Whitney turbofan design engineer. In 2018 he was a very alert and spry flight instructor aged 92. We asked him, "How can toxic compounds and fumes from the core of the engine cross the compressor airstream and enter the cabin through the bleed air valves?" Without hesitation he replied, "Spanwise flow in the boundary layer. Contamination can cross the airstream in the boundary layers of the rotors and stators".[6]

Boundary layers occur on all airfoils because of friction, forming when an aerodynamic surface moves through the air.We can imagine the dynamics as the thin friction layer ebbs and flows, sometimes smoothly and sometimes less so. It passes ever outward toward the bleed valves due to centrifugal forces upon the rapidly spinning rotor blades. Contaminated air should slough off the tips of rotors, some of which air will travel at the boundary layer on the inside diameter of the compressor shroud.Some contamination will enter the bleed valves that are located on the surface of the shroud. We can expect that local pressure near the open bleed valves will be relatively lower, attracting quantities of air through the bleed valve and into the ducting on the cabin side.

Regulations limit bleed air extraction on most if not all turbofan engines to approximately 10% [8]. The vast majority of any contamination, i.e. the remaining 90%, must be lost to the rapid flow carrying it to the burner section. At 10% air, including an estimated 10% of contaminants extracted into the cabin, concentrations of contamination produced within the powerplants must be large indeed to cause injuries and illnesses among air travelers. Sensors and air samplers built into the engines and cabin/flightdeck areas may someday prove this theory.

3.3 A speculation

If the volume of contamination is large and persistent enough, the compressor airstream could be flooded by shear force. Then a greater proportion of contamination would enter the cabin. Engine designers probably do not anticipate that smoke and fumes can permeate compressor air. The content of the 10% of air bled off for the cabin must reflect the ratio of contamination over plain outside air. The latter of course enters the system at a much lower density than the contaminated air with which it becomes mixed.

The presence of contamination is mitigated when dispersed throughout the cabin interior. Note, however, that air distribution in most aircraft cabins is zone-controlled. Contamination may be noticed in the sections in which it enters and may leaves the cabin before being noticed in others.

5 From consideration of Bernoulli's equation [7].

6 The boundary layer is a very thin layer of air that attaches to the surface of a wing or airfoil as it travels at high speed through the air (see www1.grc.nasa.gov/beginners-guide-to-aeronautics/boundary-layer/).

4. Beyond the compressor airstream

4.1 Cabin air contaminants

Once within ducting leading from the powerplants toward the environmental control system (ECS), the air thought to be clean, pure and intended for comfortable life support is ready to be cooled and sent onward. The largest portion goes to the passenger cabin, the smallest to the flight deck (reflecting their respective volumes and numbers of human occupants). Passenger cabin and flight deck air do not mix. Each compartment in aircraft will have its own supply duct and routing to outflow valves. Contaminants in the air that enter passenger and flight crew areas do not accumulate over time.[7] The air is expelled regularly at designed intervals. For example, Boeing 737 models exchange air at about 12 times per hour. In other words, every five minutes the air is fully replaced using new air in a continuous flow turnover schedule.

It seems odd that often flight attendants and passengers experience the acrid odour of partially burned oil while the flight deck does not. This anomaly can occur in reverse, or both could experience the odour. More worrisome is the case when both human-occupied areas experience no smoke or odour at all, yet occupants nevertheless experience an insidious onset of illness symptoms. Unaware or ignored these symptoms ultimately can increase toward life-threatening conditions. All crew member training should—it does not at present—include instruction on how to react to these symptoms. When a person's presence of mind is lost due to cognitive impairment, unconsciousness can soon follow. Above all, pilots *must* be taught immediately to put on masks and breathe 100% oxygen.[8]

4.2 An essential toxicological viewpoint

When considering chemical toxins (toxicants), several definitions will add clarity to the specific exposure situation. A standard definition is "Dose plus host makes the poison".[9] In an aeronautical sense this guide can be restated more precisely to fit the exposure experienced on aircraft:

Dose * Host * Repetition * Duration makes the poison,

where * denotes an operation akin to convolution. Elaborating:

Dose: The amount of a chemical toxin that enters the body. The body responds to the actual concentrations in the blood and tissues. Body mass makes a difference among individuals. Given the same nominal dose of a typical *heavier than air* aircraft cabin toxicant to which two individuals are exposed (dose being a function of exposure, which according to the Bunsen–Roscoe law is simply the product of concentration and duration), those of lower stature will receive a larger exposure than those of large stature (for the same body mass). This is critical because dose arising from the source concentration of a toxicant is the most important determining factor of a contaminant's toxicity, for contaminants of roughly equivalent standardized toxicity. From media reports and crew member testimonials, symptoms and recovery experiences after fume events commonly differ among individuals.

Host: Individuals will respond in different ways. As dose increases a response may also increase. Susceptibility depends upon age, general physical health and genetic composition. Immune system health can be expected to favour some over others.[10]

Repetition: Fume events can occur at any time on any aircraft when the ratio of fuel (lubricating oil), air (oxygen), and heat (combustion temperature) are appropriate for initiating the chemical reaction process that produces carbon monoxide. That is not to say that cabin fumes result from all bearing sump fires. Clean and efficient lubrication systems can perform safely when operating with correct tolerances between stationary and moving parts. Products of combustion may be present but from a well maintained sump, most if not quite all will be successfully evacuated through vents and scavenge routes.

Extended engine hours and system cycles[11] without periodic maintenance will degrade these tolerances. With degradation, leaks of oil, smoke, fumes and other particulate matter will gradually become more apparent in cabin air. Ultimately, the leaks will become predictable. However, fume event reporting is not dependable (not least because reporting rules are not enforced). Crew members reporting for duty sometimes find aircraft logbooks that fail to mention previous lubricating and environmental control system problems. Testimonials occasionally tell of these managerial inconsistencies.[12]

---

7 Neglecting adsorption onto surfaces. Textiles in particular offer a large actual area compared with nominal area. On the other hand the greatest quantity of textiles is carried by the human occupants and is *de facto* removed upon disembarkation.

8 Author's personal archives: U.S. Airways Flight 1041, Appendix 2; *ibid.*, Spirit aircraft # N519NK; *ibid.*, Germanwings 19 December 2010; *ibid.*, American Airlines 965 CVR (documentary trailer); *ibid.*, American Airlines—D's story ("Snap Out of It").

9 See [9] and references therein.

10 Toxicology Education Foundation. Basics of dose–response. toxedfoundation.org/basics-of-dose-response (April 2020).

11 One cycle = 1 aircraft takeoff & 1 landing.

12 From my personal archives—one testimonial relates that the official logbook had been replaced by a new, sanitized logbook making no mention of ongoing fume incidents. The incident that followed this illegal falsification cost an entire flight crew their jobs and imposed upon them long years of ongoing health issues.

Crews moving from one aircraft to another in an aging fleet, where some toxic fumes are usually present, may be at risk of accumulating toxins in their blood and tissues.[13] Exposure on one flight is likely to be followed by another before the body has had time to recover. Hence, blood and tissue concentrations may exceed what might be perceived as a safe limit for a completely unexposed body.

Almost all crew members exposed to fumes fail to obtain treatment in the form of 100% oxygen after every flight when excessive fatigue, headache, dizziness or other germane symptoms of unwellness are apparent. Cabin contamination imposes physiological conditions of hypoxia in blood and tissues. Without pure oxygen to flush the body, the contamination tends to remain. This condition of what is in effect extended hypoxia is debilitating and requires apposite treatment to assure recovery.[14] This problem of exposure to contamination imposes upon crew members a dangerous *occupational hazard*. Without warning chemical sensors, a reliable (and enforced) fume incident reporting system, and medically sensitive follow-up, the airline industry will continue to be plagued by these issues.

Duration: Fume events can be *immediate, acute* or *short-term*. In such cases pilots are forced onto aviator's breathing oxygen from masks at their flight deck seats; that is, when the incident occurs with smoke or fumes to warn them. If there are no sight or smell sensations an occurrence is likely to be unnoticed; pilots and flight attendants can then respond only if symptoms of physical impairment (headache, nausea, fatigue, dizziness and cognitive deterioration) are recognized.That crew members will become aware is not a reliable certainty. If a pilot does not respond, or if both pilots do not respond to these insidious symptoms, unconsciousness can overtake them.

Moderate fumes lasting for minutes, hours or longer often come with odours detectable and causing headache, nausea, dizziness, fatigue, confusion and/or tremors. Symptoms like these *must influence a crew member response!* Pilot management options should include: (1) Identify the problem's source; (2) Turn off or isolate the offending system; (3) Evaluate the remaining risk with respect to (a) safety of the flight and (b) passenger and crew health; and (4) Make a decision to (a) isolate the problem and continue to destination, or (b) declare an emergency and divert to the nearest suitable airport.

Long term low level fumes (LTLL)*:* Carbon monoxide (CO) poisoning is often associated with chronic (long-term) presence of fumes. CO is the most dangerous toxin in aircraft and the most debilitating. Known as the great imitator [10] it can induce all of the aforementioned symptoms plus others associated with influenza (but not fever) and Parkinson's disease. CO poisoning can lead to severe long-term cognitive, physical and behavioural symptoms. Cognitive impairment, difficulty concentrating, incontinence, balance issues and mental health problems have been reported. Symptoms can continue even when the source of the gas is removed. Patients affected by CO poisoning that has gone untreated can experience symptoms for months and years after exposure. Some are permanent [11–13].

5. FURTHER EVIDENCE

As official government oversight waned in the 20th century's last decade, stress within the airline community peaked. Survival occupied every corner of every executive's agenda. Mid-year of 2001, Dallas Cowboy quarterback and Hall of Fame football star Roger Staubach joined the American Airlines board of directors. "As I settled into my role as a board member," he said, "I was struck by the sheer volume of difficult issues facing the company. At times it was overwhelming. The company was under constant attack from competitors, it faced skyrocketing fuel prices and complex labor issues, and its balance sheet was in dire need of repair. For years the company teetered on the brink of bankruptcy. The Board's primary focus was saving American Airlines and returning it to health and prosperity" [14,15].

Over the next two decades, airline executives have had no time or willingness to try to face the cabin contamination issue. Outside researchers and investigators, isolated from inside facts, have, in almost every instance, had to be satisfied with carefully considered assumptions and subjective determinations. It is the best we can do. That said, knowledge of the effects of toxic cabin air on crew members and passengers is very close to accurate. The list that follows (Table 1) is offered as an introduction to further information in the appendices of this paper.

[13] Simply because dose rate exceeds the body's rate of detoxification.
[14] Allen, M. Recover from carbon monoxide's long-term effects. cognitivefxusa.com/blog/carbon-monoxide-poisoning-long-term-effects (22 June 2023).

Table 1. Sources of cabin air contamination.

Part 1. Sources outside the aircraft	
Exhaust fumes	A heavy exposure at airports and probable hazard to ground services personnel. The tarmac is a dangerous place. Airports should address it separately.
Oil/fuel/Skydrol	Coolers and heat exchangers for fuel, oil and hydraulic fluid within aircraft systems bring these substances into close proximity with each other. Failure of these units will create problems such that systems will become inoperable until repaired. *Hazard to crew and passengers is unlikely.*
Deicing fluid	A ground-level, seasonal exposure. Vapour and aerosols can enter the cabin by running engines during deicing, hence the operation should be conducted with bleed valves closed. Type 1 deicing fluid is propylene glycol and water, relatively less an irritant than ethylene glycol (antifreeze); some individuals will be sensitive to propylene glycol. *Likelihood of fumes in flight—nil.*
Pesticides	Disinsection spray, usually permethrin. Toxic to susceptible people. Irritating and used indiscriminately. Should be banned. Exposure mostly at arrival before disembarkation. Important but *not an immediate concern* in air overall air contamination.
Solvents	Solvents and cleaning solutions used prior to boarding may still be present at departure. An irritant to some people, but taxiing will usually see them eliminated from the cabin by normal air circulation. *Not considered to be an occupational hazard.*
Viruses & bacteria	A normal hazard in a crowd. Rarely does this inhibit normal conduct on aircraft. Exposure becomes less while in flight due to cabin air partly circulated through HEPA filters.
Fragrances	An irritating and occasionally debilitating exposure for sensitive people. less a bother while airborne. *Not considered in this paper.*
Galley fumes	Usually contained within the galley area. Not a threat to occupant health.
Part 2. From sources internal to the aircraft or the aircraft inflight environment	
CO	(Carbon monoxide.) Source: oxidation of oil hydrocarbons by heat applied in powerplants. Highly toxic gas. Inhalation fatal in large doses, debilitating in moderate and long-term, low level (LTLL) doses if left untreated. Half-life in body: 4–5 hours. Treatment: 100% oxygen. Concentration varies according to oil/air ratio, temperature and combustion interval.
TCP	(Tricresyl phosphate.) Source: engine oil (antifriction, antiwear additive). Neurologically debilitating in large doses as a cholinesterase inhibitor. May act as a phosphorylating agent. Concentration in oil according to safety data sheet: 1– 3% by weight.
NO	(Nitric oxide.) Source: atmospheric nitrogen is oxidized at the high temperatures in the combustion chamber. Toxic in high doses. Common atmospheric pollutant (from motor-car internal combustion engine exhausts). Has medical applications and is essential to warm-blooded animal physiology.
1,4-dihydroxy-9, 10-anthracenedione	Source: engine oil. Skin, eye and respiratory irritant. Concentration in turbine oil < 0.01%. *Disregarded due to its very low concentration.*
HCHO	(Formaldehyde.) Source: may form under heat and pressure in internal combustion engines. Residues may be present in many common textiles and other materials Highly toxic gas. Eye, nose, throat and respiratory irritant. Symptoms of exposure: cough, wheeze, dermatitis. Is a potential carcinogen. Concentration: a few micrograms per cubic metre measured during flight [6]. Not reported during diagnoses of injury, therefore we have no basis from which to estimate exposure.
O_3	(Ozone.) Source: oxygen (O_2) irradiated by ultraviolet light, hence occurs naturally in the stratosphere. Drawn in by engines during high-altitide flights and hence part of the air bled off into the cabin. Can react with oil fumes and aerosols forming a variety of oxidized compounds [16]. Highly reactive gas, used as a sterilizing agent. Respiratory irritant, may cause dryness of mucous membranes [17]. Relatively well-studied; not considered further in this paper. Concentration: greatest when cruising in the stratosphere. Often decomposed to oxygen by in-line converters in the ECS (mandated for flights over 4 hours). Residual concentration is usually very low.

ACKNOWLEDGMENT

The author sincerely thanks the reviewers of this paper for having suggested many valuable improvements.

REFERENCES

1. *Coking*. Houston: Exxon Mobil Corp. (2016).
2. Davidson, G.A. An issue with gas turbines that no one wishes to talk about but is ever present in cabin air. *J. Biol. Phys. Chem.* **21** (2021) 88–102.
3. Lind, J.M. Anatomy of an occupational hazard: Cabin air contamination in the air transportation industry Part 5. Carbon monoxide—how it forms in aircraft engines *J. Biol. Phys. Chem.* **23** (2023) 81–88.
4. Howard, C.V., Johnson, D.W., Morton, J., Michaelis, S.,

Supplee, D. & Burdon, J. Is a cumulative exposure to a background aerosol of nanoparticles part of the causal mechanism of aerotoxic syndrome? *J. Nanomed. Nanosci.* (2018) JNAN-139.

5. Sogut, M.Z., Yalcin, E. & Karakoc, T.H. Assessment of degradation effects for an aircraft engine considering exergy analysis. *Energy* **140** (2017) 1417–1426.
6. Ramsden, J.J. On the selection of aircraft cabin air quality indicators. *J. Biol. Phys. Chem.* **13** (2013) 135–143 (erratum: *ibid.* **14** (2014) 75).
7. Mikhailov, G.K. Daniel Bernoulli, Hydrodynamica (1738). In: *Landmark Writings in Western Mathematics, 1640–1940* (ed. I. Grattan-Guiness), ch. 9 (pp. 131–142). Elsevier (2005).
8. E.g., Type-certificate data sheet no E.067 for CFM56-5 series engines. P. 8, §10. Maximum permissible air bleed extraction.
9. Ramsden, J.J. A nanoscale perspective on chemical toxicology. *J. Biol. Phys. Chem.* **23** (2023) 31–33.
10. Velasquez, C. et al. Carbon monoxide poisoning: The great imitator. *Spartan Med. Res. J.* **2** (2017) 6343.
11. Myers, R.A.M., DeFazio, A. & Kelly, M.P. Chronic carbon monoxide exposure: A clinical syndrome detected by neuropsychological tests. *J. Clin. Psychol.* **54** (1998) 555–587.
12. Wright, J. Chronic and occult carbon monoxide posioning: we don't know what we're missing. *Emerg. Med. J.* **19** (2002) 386–390.
13. Pan, K.-T., Leonardi, G.S. & Croxford, B. Factors contributing to CO uptake and elimination in the body: A critical review. *Intl J. Environ. Res. Public Health* **17** (2020) 528.
14. Petzinger, T. *Hard Landing*. New York: Three Rivers Press (1995, 1996).
15. Kennedy, G. & Maxon, T. *Twelve Years of Turbulence: The inside story of American Airlines' battle for survival*. Brentwood, Tennessee: Permuted Press (2018).
16. Weschler, C.J. et al. Ozone-initiated chemistry in an occupied simulated aircraft cabin. *Environ. Sci. Technol.* **41** (2007) 6177–6184.
17. Beko, G., Allen, J.G., Weschler, C.J., Vallarino, J. & Spengler, J.D. Impact of cabon ozone concentrations on passenger reported symptoms in commercial aircraft. *PLoS One* **10** (2015) e0128454.
18. Di Cera, E., Doyle, M.L., Connelly, P.R., & Gill, S.J. Carbon monoxide binding to human hemoglobin A_0. *Biochemistry* **26** (1987) 6494–6502.
19. ExxonMobile Jet Oil II Safety Data Sheet (October 2022).
20. Aeroshell Turbine Oil 555 Safety Data Sheet (May 2018).
21. Ramsden, J.J. The scientific adequacy of the present state of knowledge concerning neurotoxins in aircraft cabin air. *J. Biol. Phys. Chem.* **11** (2011) 152–164.
22. Mansoor, K. et al. Carbon monoxide: a rare cause of myocardial ischemia (case report). *Marshall J. Med.* **5**(1) (2022) doi: 10.33470/2379-9536.1203.
23. Zucco, G.M. & Doty, R.L. Multiple chemical sensitivity. *Brain Sci.* **12** (2022) 46.
24. Michaelis, S. Contaminated aircraft cabin air. *J. Biol. Phys.Chem.* **11** (2011) 132–145.
25. Inoue, N. Fujishiro, K., Mori, K. & Matsuoka, M. Triorthocresyl phosphate poisoning—A review of human cases. *J. UOEH* **10** (1988) 433–442.
26. Mackenzie Ross, S., Harrison, V., Madeley, L., Davis, K., Abraham-Smith, K., Hughes, T. & Mason, O. Cognitive function following reported exposure to contaminated air on commercial aircraft: methodological considerations for future researchers. *J. Biol. Phys. Chem.* **11** (2011) 180–191.
27. Hyde, B.M. TCP (tricresyl phosphate): Pilot, aircrew and passenger safety and secondary myalgic ncephalomyelitis. *J. Biol. Phys. Chem.* **11** (2011) 172–179.
28. Abou-Donia, M.B., van de Goot, F.R.W. and Mulder, M.F.A. Autoantibody markers of neural degeneration are associated with post-mortem histopathological alterations of a neurologically injured pilot. *J. Biol. Phys. Chem.* **14** (2014) 34–53.
29. Ramsden, J.J. Contaminated aircraft cabin air: aspects of causation and acceptable risk. *J. Biol. Phys. Chem.* **12** (2012) 56–68.
30. Ramsden, J.J. Jet engine oil consumption as a surrogate for measuring chemical contamination in aircraft cabin air. *J. Biol. Phys. Chem.* **13** (2013) 114–118.
31. Ramsden, J.J. An estimate of the chronic exposure to neurotoxins of a neurologically injured pilot. *J. Biol. Phys. Chem.* **16** (2016) 95–102.
32. Henschler, D. Die Trikresylphosphatvergiftung. Experimentelle Klärung von Problemen der Aetiologie und Pathogenese. *Klin. Wochenschr.* **36** (1958) 663–674.
33. Ramsden, J.J. On the proportion of *ortho* isomers in the tricresyl phosphates contained in jet oil. *J. Biol. Phys. Chem.* **13** (2013) 69–72.
34. Marsillach, J. et al., Biomarkers of organophosphorous (OP) exposures in humans. *NeuroToxicology* **32** (2011) 656–660.
35. Furlong, C.E. Exposure to triaryl phosphates: Metabolism and biomarkers of exposure. *J. Biol. Phys. Chem.* **11** (2011) 165–171.
36. Lockridge, O., Norgren, R.B., Johnson, R.C. & Blake, T.A. Naturally occurring genetic variants of human acetylcholinesterase and butyrylcholinesterase and their potential impact on the risk of toxicity from cholinesterase inhibitors. *Chem. Res. Toxicol.* **29** (2016) 1381–1392.
37. Howard, C.V., Michaelis, S. & Watterson, A. The aetiology of "aerotoxic syndrome"—a toxico-pathological viewpoint. *Open Access J. Toxicol.* **1** (5) (2017) doi: 10.19080/OAJT.2017.01.555575.
38. Ramsden, J.J. Adverse synergies between inhaled carbon monoxide and tricresyl phosphate in aircraft cabin air. *J. Biol. Phys. Chem.* **21** (2021) 119–124.
39. Cheng, L. et al. Comparative biotoxicity of N-phenyl-1-naphthylamine and N-phenyl-2-naphthylamine on cyanobacteria *Microcystic aeruginosa*. *Chemosphere* **176** (2017) 183–191.
40. Rehman, H.U. Methemoglobinemia. *Western J. Med.* **175** (2001) 193–196.
41. Strekas, T.C. & Spiro, T.G. Hemoglobin: Resonance Raman spectra. *Biochim. Biophys. Acta* **263** (1972) 830–833.
42. Weinberger, B., Laskin, D.L., Heck, D.E. & Laskin, J.D. The toxicology of inhaled nitric oxide. *Toxicol. Sci.* **59** (2001) 5–16.
43. Masiol, M. & Harrison, R.M. Aircraft engine exhausts and other airport-related contributions to ambient air pollution: A review. *Atmospheric Environ.* **95** (2014) 409–455.
44. Bendtsen, K.M., Bengtsen, E. Saber, A.T. & Vogel, U. A review of health effects associated with exposure to jet engine emissions in and around airports. *Environ. Health* **20** (2010) 10.
45. Johnson, D.W. Lubricants for turbine engines. In: *Recent Progress in some Aircraft Technologies* (ed. R.K. Agarwal), ch. 2. IntechOpen (2016).

APPENDIX A. TOXIC COMPOUNDS FROM SUPERHEATED SYNTHETIC TURBINE ENGINE OIL

A1. INTRODUCTION

Three decades have passed since business management oversight of the US air transportation system terminated. The Deregulation Act of 1978 became the enabler of many new and unintended consequences. This Appendix offers details of just one, the slow, historical accumulation of toxic air in cabins as aircraft and engine operating cycles and time-in-service take unrelenting toll on mechanical systems. Systems, deteriorating ever so slowly, allow unfriendly products of superheated oil to escape confinement in bearing housings. Some of these toxic products find their way into the passenger cabins and flight decks of commercial aircraft. Without official oversight (including dependable incident reporting programmes prompting preventive activities), they continue to threaten the health of aircraft crew members and passengers.

Sources and concentrations of contaminants play primary rôles in the effort to qualify and quantify risks posed by toxic substances. The chemical compound of most concern within the aeroplane is carbon monoxide. Tricresyl phosphate, the wear-reducing additive in oil, is a distant second because of its low concentration. Certain aromatic nitrogen-containing compounds are third in importance.

A2. CARBON MONOXIDE

A2.1 Introduction

Carbon monoxide (CO) in everyday life is undoubtedly the most-studied chemical toxin. Insidious and too easily formed and often deadly, it is both pervasive and persistent across societies. Dubbed the Great Imitator, it masquerades as other illnesses such as influenza and Parkinson's disease. In the lungs CO binds to the iron atom on the haem of haemglobin (in red blood cells), easily outcompeting oxygen (O_2) with a binding strength 230 to 300 times greater [18]. It imposes upon the system the effects of hypoxia. If not interrupted by fresh access to oxygen in the absence of carbon monoxide, the exposed person will lose consciousness and die [10].

CO forms in aircraft when lubricating oil is *partially burned* under conditions of high temperature but limited availability of oxygen within the compressor and turbine bearing sumps (the oil is sprayed directly onto the bearings located along the spinning central shaft of the engines). CO formation requires an appropriate balance of temperature, oil and oxygen concentration.

CO is odourless, tasteless and colourless. Haze, smoke and odours in aircraft cabins and on flight decks are evidence of fire somewhere in the aircraft systems. We have seen that investigators and researchers have settled upon powerplants as the source of most cabin contamination. Combustion of oil compounds does not always produce smoke or odour, however. CO might be present at all times, or any time, or not at all.

Engine bearing housings (sumps) contain the dynamic forces within engines that at times create internal engine fires.[15] These fires are contained within the close confines of the sump and very rarely spread beyond it. Smoke from burning oil compounds and gases including CO are sometimes able to escape the sump. Changing engine performance conditions caused by worn seals, expanding clearance tolerances between moving parts, fluctuating internal pressures, turbulence and pilot throttle technique will facilitate escape of oily components and gases.

A2.1.1 Bearing sump temperatures

Temperatures probably oscillate up and down continuously during engine operation, passing values conducive to CO formation (conditions of incomplete combustion), some falling within the range between the flash points of liquids in the chamber[16] and the highest attaained in the enclosed environment. Combustion exposure time will of course affect the amount of CO produced. "Autoignition" is possible with turbine engine oil vapours; without spark or flame, (auto)ignition occurs at > 320 °C. When temperatures exceed this value and given enough time for combustion to do its work, the entire complement of engine oil in the sump will be consumed.

Usual temperatures within the sump have been measured to reach about 1700 °C. We can imagine the temperature range within which CO formation occurs. From the flash points (very small amounts of CO formation, if any) to autoignition temperature (small to moderate CO formation above it) to possible acute and dangerous levels of CO production, the temperature range conducive to CO production could be quite large. These possibilities could produce toxic conditions in the cabin: from low CO levels to very high levels of exposure to aircraft occupants in the cabin and flight deck [4].

---

[15] See the following Part 5 [3].

[16] "Flash point" is defined as the lowest temperature at which a flammable vapour over a liquid source will ignite when confronted with a spark or flame. The flash point of turbine engine oil exceeds 246 °C [19,20].

A2.1.2 Creation and occurrence of CO in the aircraft cabin—a closer look

Safety and health investigators and researchers are *de facto* not permitted access to airline equipment. They are in effect prohibited from evaluating cabin air quality in its various suspected forms. We do investigate the results of cabin air contamination at the several plausible levels of illness and injury apparent from crew member and passenger experiences. Illness symptoms, medical records and common sense (including corroboration from numerous independent experiences) assure us that CO is likely to exist in small quantities in cabins and on flight decks. Somewhat subjective, but accurate enough for our purposes when denied access to the hard facts, this thesis is powerful enough to place the airline industry and its government regulators on the defensive if they wish to deny our convincing evidence.

For illustrative purposes, the incidents recounted in the following sections occur with haze, smoke, odour or no odour present. If visible and/or odorous evidence during a contamination event[17] is apparent, carbon monoxide assuredly will also be present. If no visible evidence or odour is present, only symptoms of illness in an exposed individual will evince the presence of CO.

A2.2 Acute carbon monoxide exposure events

- 17 July 2015; at cruise. captain unconscious. First officer impaired. Recovered just before flight crew total incapacitation. Flew next day, impaired again. Captain neurologically, cognitively impaired. Died of idiopathic myocardial infarction 50 days later [22].[18]
- 29 July 1995; nausea, burning eyes & airways, lightheaded, numb lips, extreme fatigue. Strong odour throughout cabin during taxi. Flight crew refused flight attendant request to taxi back, isolated faulty air conditioner pack. Next day a cabin crew member became incapacitated, tested positive for CO, emergency evacuation to hospital.
- September 2010; headache, nausea, vomiting, confusion, yellow eyes, tremors, extreme fatigue, semiconscious. Entire cabin crew incapacitated. Deadheading junior flight attendant in uniform stood to provide passenger services in absence of assigned crew. By the time destination was reached, she too was incapacitated to the point of permanent disability. Employer refused to pay her, provide medical or disability assistance. Never again was able to perform duties. Resigned.
- 16 January 2010; entire crew disabled. Neither pilot had odour or visual warning. Nearly incapacitated from cruise altitude to landing. Checklists not used. Neither remembered how they landed. Both pilots grounded, never recovered physically. Both have died: captain by own hand in 2016; first officer contracted cancer in 2023. Aircraft was ultimately scuttled. All but one cabin crew too ill to return. One continues to work through recurring symptoms.
- 27 January 2017; the captain in his own words: "One day prior, the incoming captain had written up 'Odors in the cabin' at gate prior to departure. No maintenance done and flight proceeded to (—) where the captain wrote up 'Odors in the cabin' again upon landing. This is where I approach the aircraft the next morning with an open write-up in the logbook for 'Odors'. I request maintenance and am told it's contract maintenance from (another airline). Our maintenance tells me on the phone that contract maintenance cannot do run-ups so I would have to start the engines so that contract maintenance could identify the problem.

"We could smell the 'dirty sock smell' immediately upon arriving at aircraft. Never did we notice any visible indications. Somewhere during the 2nd run-up the odor turned extremely toxic. We began to choke. Headaches came immediately. The right side of my body started tremoring from shoulder to toe. We cut off the engine and started to evacuate out of the windows.We paused as they brought the jetbridge up very quickly and we left normally.

"At this point the first officer was recouping as was I on the jetbridge when contract maintenance came up from the ramp and looked in to the aircraft. 'We have to air this thing out now', they said. No one moved so I got up and reentered the aircraft and ran to the back, disarmed, and opened both rear doors. Then ran back up to the front and off the aircraft—we think I breathed an extra 5 minutes at a high heart rate during the worst toxicity. This probably caused the worst effects on my health. My first officer is back flying and has no chronic problems."

The captain never flew again. He voluntarily removed himself because he knew he was unable ever again to perform duties of pilot in command. Diagnosed by his medical specialist with Parkinson's disease, probably because of the persistent tremors, we briefed him about the fume event and carbon monoxide effects upon the central nervous system as the probable cause. The doctor altered his treatment accordingly.

[17] "Event" of course implies a discrete occurrence with a definite start and finish. Here we do not explicitly consider the continuous low-level background contamination that has been measured in flight, but asserted to be too low to constitute a health hazard (see ref. 21 and references therein).
[18] avherald.com (4 April 2018).

• 16 February 2017 (Germany to Detroit); prior to take-off, flight attendants recognize seriously acrid odour in the cabin. Report to captain is ignored. He prepares and departs without investigating. (Report suggests pilots use flight deck oxygen for the whole trip.)

Flight attendants soon begin experiencing symptoms: Severe headache, nausea, vomiting, dizziness, loss of equilibrium, tingling in hands & feet, chest tightness and tremors. Passengers begin falling ill. Flight attendant C.M., hypersensitive to toxic fumes, retires to the under-deck crew rest lounge where air circulation is poor. She soon oscillates between consciousness and unconsciousness. Medical personnel on board cannot diagnose her illness. They tried inappropriate options, including administering nitroglycerine, which amplified her condition.

The captain remains unsympathetic while the passenger cabin suffers. C.M. is periodically given oxygen from portable emergency bottles over a four-hour interval.

After the aircraft crosses the north Atlantic Ocean, the captain selects a remote landing strip 41 miles from the Arctic Circle to set the aircraft down. The ground community has no hospital. only a clinic with little oxygen and no hyperbaric treatment facilities. Ill passengers and crew members are taken on buses southward to the nearest hospital. The captain departs to complete the trip to Detroit with the remaining passengers and an unknown complement of cabin crew. Arriving at Detroit, almost everyone on board except the pilots is very ill.

C.M., coming into and out of consciousness during the whole trip, nearly died. Without emergency oxygen she would have died. At the last check, after years of hyperbaric oxygen treatment, her recovery is partial. Destitute and supported by her family, she lives a reclusive life with MCS.[19]

A2.3 Short term, moderate and/or repeated exposure to carbon monoxide

• February 2018; strong odour. Dizzy, sore throat, lightheaded, red eyes. Captain disregarded cabin crew concerns. Complained to captain after landing as they were going to emergency room for assistance. One flight attendant reports blood pressure much above normal for her. Three fume events in 8 years; one entire crew incapacitated. Doctor, aware of airline fumes exposure, said that once sensitized to CO to the degree seen in these flight attendants, the effects never leave the body.

• Flight attendant—various fume events between 2010 and 2017. Headache, dizzy, sinus drip, respiratory difficulty, swollen lymph nodes. Upper respiratory tract haemorrhage during crew rest. Long term, low to moderate repeated exposure caused toxin-induced loss of tolerance and MCS. She has since resigned from her employment.

• 3 January 2018; the flight arrived with crew very ill from cabin fumes. Flight deck fumes were worse than in cabin yet flight attendants apparently more affected than pilots. Our aircraft was grounded by authorities in the foreign country. A second aircraft sent to pick up crew also had a fume event[20] and it, too, was grounded.

• 3 January 2018 (different location from preceding); aft cabin, sudden strong dirty sock smell. Captain returned aircraft to departure airport. Aircraft checked to be OK by line maintenance. Departed a second time, fumes returned, and aircraft returned a second time. Aircraft taken out of service.

One flight attendant is on hyperbaric oxygen treatment. Symptoms of three flight attendants: headache, nausea, vomiting, shortness of breath, confusion, deteriorated motor skills.

A2.4 Long-term, low-level (LTLL) concentration; repetitive occurrences

• 26 March 2011; flight attendant K.R.: "I flew for an airline in the USA for 10 years. Excellent health. Suddenly, one night, 25 March 2011, I collapsed in my hotel room while on an overnight away from home." Her story is long. Here is a summary:

Unexplainable twirling and spinning of the room; indescribable dizziness. Confusion. "What to do? who to contact? knew I needed help but couldn't function, couldn't react." Her cellphone rang, it was her cousin, a registered nurse, asking "What's up?" She instructed her to crawl on the floor from the lavatory to the nightstand to get the house phone, dial 0, and ask for an ambulance.

When first responders entered, "I didn't see their faces, only heard voices." She could say only "help me". In the emergency room (ER), "the staff thought I was drunk. I don't drink alcohol."

"Now, six years later, I've lost my health. Doctors cannot find anything physiologically to explain my condition. I have loss of short-term memory, great fatigue, greatly damaged cognitive skills, blurred vision, PTSD,[21] inability to carry on a conversation, difficulty

---

[19] Multiple chemical sensitivity.

[20] At top of descent when the captain reduced power to begin the landing approach, an acrid, "dirty socks" odour permeated the cabin. Cabin crew symptoms (burning eyes and nose, chest tightness, and disorientation nearly to the point of lost equilibrium) came quickly. Fumes were worse on the flight deck and the captain seemed to be impaired. He asked the lead flight attendant the same question six different times during the descent. She had to tell him to "snap out of it. Your memory is not working".

[21] Post-traumatic stress disorder.

getting words out, dizziness, nausea, great vertigo, inability to count change in purchases, the list is endless." Isolated from the public, she cannot work.*This is a serious condition that needs critical attention for other flight crew that may still be able to fly.* She continues: "My book, *A Flight Attendant's Diary*,[22] is being read by millions. A pilot read it and contacted me because he is now grounded, as I was by doctors, and we are suffering from similar symptoms. They are consistent with carbon monoxide and tricresyl phosphate poisoning. No known way to confirm."

- Flight attendant T.F. relates several fume events since first employed more than ten years ago. One was especially acute, causing upper respiratory tract haemorrhage. Her blood–brain barrier had been compromised by toxic chemicals while working in a state laboratory prior to joining an airline. Her total toxic load is now so great that she has developed toxin-induced loss of tolerance (TILT) and suffers from MCS, a condition not generally recognized by the medical establishment.[23]
- Not unusual are recurring episodes of symptoms that can revisit an exposed crew member without warning. A friend of the present author who was injured on one of these reported cases still experienced tremors and loss of equilibrium seven years after her fume event. Another who was injured in 1992 and still flies actively has mild neurological issues to this day.

Remarkably, it appears that none of these events have been officially investigated. FAA and NTSB are in denial about fume incidents. They do not enforce reporting requirements, nor do they keep formal records of reports they do receive.

A2.5 Conclusion (carbon monoxide)

Where blood oxygen and carboxyhaemoglobin tests are carried out, diagnosis of carbon monoxide poisoning is confirmed. Symptoms tend to be the same as the usual symptoms familiar from virtually all cases of toxic exposure (i.e., headache, nausea, etc.). In cases where diagnosis by appropriate testing was not obtained, the cause of illness among employees in the commercial air transportation environment might be accepted as CO by association, especially since symptoms unique to other potential toxicants (such as TCP and nitrogen oxides) that may also be present in aircraft have not been verifiably identified.

Note that APU can cause fume events. They can contribute to ground and in-flight air contamination emergencies. They should be periodically inspected for degradation and involved in fume event follow-up inspection and maintenance.

Time and time again, we see the maintenance department accept a fume-compromised aircraft that must be examined for cause and corrected before signing off the aircraft to continue revenue-earning services. Time after time, the condition that occurred in flight *cannot be duplicated on the ground*. The creation of carbon monoxide in commercial aircraft at cruising altitude is the subject of the following article [3].

A3. ORGANOPHOSPHATES: ISOMERS OF TRICRESYL PHOSPHATE IN JET ENGINE OIL

A3.1 Introduction and historical background

From the late 1980s to the new millennium research scientists scanned the air transportation environment seeking toxic elements and compounds that could possibly be implicated in the health threat starting to be evidenced among airline flight crew personnel [24]. Appearances suggested that tricresyl phosphate (TCP),[24] the antiwear compound added to synthetic turbine engine lubricating oil, was the primary health hazard for occupants of commercial aircraft.

The reasons for this preoccupation are not hard to understand. TCP is a known neurotoxin. It has a long and well-documented history of human expoure and illness, albeit almost all involving oral ingestion [25]. Such investigations as there were were not inconsistent with the interpretation of TCP-induced neurotoxicity, especially those diagnosing cognitive impairment [26]. The need for more thorough physiological investigation was identified and made precise [27], but its expense, and the general reluctance of aircrew employers to engage with the problem, led to very little being done. The untimely death of a pilot due to a long history of presumed exposure yielded an unprecedented opportunity for a detailed autopsy [28]—albeit less promptly after expiration than would be generally preferable—with the conclusion of injuries consistent with organophosphate-induced neurotoxicity.

A symptom—or set of symptoms constituting a syndrome—can be established on the basis of objective observation as a scientific fact. So much the better if a symptom can be established on the basis of quantitative measurement, but in most of medicine this is an unattained and maybe unattainable ambition.[25] In order to make a

[22] K.A.Russell, *A Flight Attendant's Diary: Cleared for Departure*. (2015).
[23] Not by the World Health Organization (WHO) but has some degree of recognition from the US Centers for Disease Control and Prevention (CDC). See also ref. 23.
[24] Also known as tritolyl phosphate.
[25] Measurement of body temperature, blood pressure, blood oxygen saturation are examples of measurements that can be carried

diagnosis the measurement results must then be correlated with possible causes, and the goal of the researcher is to establish causes—*felix qui potuit rerum cognoscere causas*.Thus, rightly do we honour Pasteur, Robert Koch and others for having discovered them. Bacteria cultured from a biopsy can be identified. The concentration of a toxin and the duration of exposure to it can be measured. Symptoms and possible causes can be associated with each other; in recent decades, establishing causation in medicine has often relied upon Bradford Hill's criteria [29]. The biomedical researcher hopes for what is often loosely called "proof", but is aware that proof in the mathematical sense is unattainable. Moreover, whereas in mathematics a single counterexample to a conjecture suffices to disprove it,[26] and even in physics and chemistry a proposition can usually be dismissed by a counterexample, albeit that the measurement process leading to the result needs to be carefully scrutinized, the complexity of biology means that counterexamples often abound. Indeed, one of the great puzzles of aerotoxicity is the fact that in many cases not all aircrew or passengers exposed to a fume event of presumed uniform severity fall ill, or indeed manifest any symptoms at all.

Safety engineering and risk management have different objectives. They are not scientific disciplines; practitioners seek to identify unsafe acts and conditions to be addressed for loss prevention purposes. Their "proofs of hazard" need to be circumstantially correct. They fall more nearly on the probability of occurrence and legal liability side of an issue rather than the scientific.

To a loss control investigator and a certified risk manager, the lubricant manufacturer-established concentrations (in the lubricant) of the toxic compounds TCP and the aryl amine antioxidants (see §A4) listed in its safety data sheets imply very low exposures in the aviation context and the improbability of adverse health effects. However, one issue hindered the investigation of TCP and the aryl amines, namely, what other compounds are present in a turbine engine lubrication formula? The airline industry had long held the precise formula as proprietary information, necessarily held away from competitors and the general public. Lacking this information, until September 2016 the investigation could go no further toward the identification of unsafe chemical conditions.

It was in that month, on the present writer's birthday (think guardian angel or daemon!), that the formula was released. Realizing that the full engine oil formula would be needed to complete a document, a new search was attempted. "Turbine engine oil formula" was given as the prompt to the search engine. In just a blink of the eye, up popped the comprehensive turbine oil formula complete with compounds, their qualities, quantities, and essential purposes in the oil.[27] It was astounding. The formula had just been posted.

The world of aircraft lubrication opened up on that day. It would take another year of discussions with disabled crew members and a pilot friend who emphasized his positive carboxyhaemoglobin test for me to conclude the investigation process. All, 100%, of the compounds in turbine engine oil are hydrocarbons.[28] Hydrocarbons burn. Smoke is a product of combustion occurring somewhere within the aircraft engines. Where there is smoke there is fire. Where there is fire there is also carbon monoxide. How simple.

A3.2 Tricresyl phosphate (TCP)

TCP, whose presence in oil is acknowledged in the safety data sheets at concentrations typically 3–5% by weight, seemed only capable of providing a small exposure to cabin occupants. Compared to the risks from organophosphate sheep dip exposure, aerial spraying-applied organophosphate insecticides, and the presence of TCP as a fire retardant and/or plasticizer in many common organic polymer materials, the risk from exposure to this low concentration, albeit by inhalation, seems not so compelling. To add to the conundrum, air safety regulators granting powerplant type certifications limit compressor bleed air extraction to roughly 10%.[29] Hence, from the oil leaking across the seals into the compressed air—which might be taken as the principal cause of oil loss during engine operation [30]— only (at most) 10% of the air passing through the compressor should be extracted for purposes other than producing thrust, with 90% passing beyond the engine bleed valves en route to the burner section of the engine.

Flight deck and cabin air sources do not mix: air is sourced separately from the main air conditioning duct to the cabin and to the flight deck. In the B737, 80% of the total goes to the passenger cabin, 20% to the flight deck. In the former, people are mostly at rest and utilize only about 25% of air actually inhaled,[30] the rest of the air is simply

out reasonably objectively. Repetition is highly desirable, not only to average out adventitious error, but also to reveal natural fluctuations, which can be considerable. On the other hand, pain, dizziness and nausea seem to be very difficult to quantify.

26 E.g., consider the conjecture "all odd numbers are prime".

27 See Appendix B, "Synthetic turbine engine lubricating oil formula".

28 That is, all contain the elements C and H. Some also contain the minority elements O, N and P.

29 See, e.g., NOTE 7 of Type Certificate Data Sheet (TCDS) E28NE for CFM56-5 engines (1998), based on Federal Aviation Administration (FAA) regulations. See also ref. 8.

30 Cf. §2.3 in ref. 31.

exhaled unused. This will further diminish the dose of TCP taken up in the lungs to be passed to the bloodstream.

As is well known, TCP has ten isomers; painstaking experiments with pure isomers established that isomers containing the *ortho* substituents are markedly more neurotoxic than the others [32]. This knowledge led to strenuous efforts by the manufacturers to decrease the proportion of *ortho* isomers in the commercial product, which is always a mixture of isomers (it would be far too expensive to purify the mixture).This has created another conundrum: actual measurements in aircraft cabins have shown that the proportion of *ortho* isomers in the cabin air is about 30% [33], far greater than in the TCP supplied to the oil manufacturers. In this context it should be pointed out that the non-*ortho* isomers are not wholly benign: the proteins in the blood that scavenge TCP can be saturated by any isomers and, once that happens, the *ortho* ones are free to cause their neurological damage [34–36].

Of especial interest to emergency situations is the acute acetylcholinesterase inhibition by tricresyl phosphate (this is the mechanism of action of organophosphate nerve gases);[31] inhibition interrupts the breakdown of acetylcholine, which is the main neurotransmitter in the nervous system. Longer-term effects, well known through the numerous cases of oral ingestion by human beings [25], are often labeled "OPIDN" (organophosphate-induced delayed neurotoxicity). Symptoms of TCP poisoning include many of the same symptoms as those found in confirmed cases of CO poisoning: headaches, nausea, dizziness, loss of balance, numbness and neurobehavioural abnormalities such as emotional instability, depression, cognitive dysfunction etc. Additional symptoms *not* associated with CO include excessive salivation, low blood pressure, narrowing of the lung airways and smallened pupils. It is noteworthy that crew members interviewed and reporting on their case have never mentioned salivation, low blood pressure, lung airway problems or small pupils among symptoms experienced. Howard has pointed out that the symptoms associated with aerotoxic syndrome are not those associated with OPIDN [37].

A3.3 Conclusion (TCP)

The amount of TCP inhaled and retained by aircraft cabin occupants must be very small. Ramsden has remarked that "The estimated exposure to tricresyl phosphates of [a] neurologically injured pilot is considerably smaller than current paradigms would suggest is sufficient to cause his neural degeneration and associated problems" [31]. Hence, we consider tricresyl phosphate to have a low probability of causing aerotoxic syndrome (albeit note possible *synergies* with CO [38]).

A4. NITROGENOUS COMPOUNDS

Three compounds in turbine engine oil contain nitrogen: N-phenyl-1-naphthylamine ($C_{16}H_{13}N$);[32] dioctyl diphenylamine ($C_{28}H_{43}N$); and benzotriazole ($C_6H_5N_3$). According to the safety data sheet (SDS) they constitute about 1% w/w of the oil.[33] It seems doubtful whether these compounds contribute to the ill health experienced by some aircrew during and after flights. These compounds are more associated with insidious, longer-term, environmental toxicity (e.g., ref. 39).

We do, however, note that these compounds are similar to others already known to be able to cause methaemoglobinaemia [40], a disease in which the Fe^{2+} in haemoglobin is oxidized to Fe^{3+}, rendering it unable to carry oxygen.[34] Given that the rôle of PAN in the oil is that of an antioxidant this might seem paradoxical but the ultimate outcome would be to exacerbate the mild hypoxia already prevalent in the reduced pressure environment of the aircraft cabin. A corollary is that the administration of hyperbaric oxygen to combat CO intoxication would indeed be ineffective; whereas both carboxyhaemoglobin and methaemoglobin cause conditions of hypoxia and cyanosis (symptomized by pale or bluish tint to skin, fingernails, etc.), they respond in opposite ways to treatment: pure oxygen is the first, best and essential treatment for carbon monoxide poisoning; methylene blue is used to treat methaemoglobinaemia.

In §3 of the main part of this article we mentioned nitrogen oxides as contaninants, and in Table 1 nitric oxide, which is a known toxin [42]. As a by-product of internal combustion (some atmospheric N_2 is oxidized at the high temperatures engendered by hydrocarbon combustion) it is present in jet engine exhaust and, especially during taxiing at a busy airport, it seems unavoidable that some would be drawn into the engines and ~10% bled off into the cabins [43]. It may be a contributor to ill health during the ensuing flight [44]. Treatment is typically the administration of oxygen.

[31] See §2 of ref. 21.
[32] Also known as N-phenyl-α-naphthylamine (PAN).
[33] Safety data sheets do not usually include substances present at concentrations significantly less than 1%. Those that are listed almost invariably contain impurities (which do not adversely affect the attributes for which the listed substances are added to the formulation); they may amount to ~1% of the substance, hence their final concentration in the oil is only about 0.01%, which is usually presumed to be negligible even if the impurity is significantly more toxic than the additive (e.g., β-naphthylamine, a CAT 1 carcinogen [24]).
[34] Cf. ref. 41.

A5. FORMALDEHYDE AND OTHER ALLEGED TOXICANTS IN AIRCRAFT CABINS

From time to time, in the effort to understand what might cause air travelers to become ill on aeroplanes, evidence of many different kinds of contamination is mentioned.[35] Considering that transient toxic elements can be present at any given time, the possibility of adverse effects is recognized. However, identifying and evaluating these unending strings of such unsubstantiated cases would be difficult, extremely time consuming, and unnecessary.

If a way can be found to eliminate or mitigate *carbon monoxide* within the cabin, most if not all other chemical and inorganic particulate contaminants will be eliminated or at least mitigated *pari passu*.[36] Hence the logical target is CO, to which crew members and passengers continue to be exposed (unwittingly), come home ill and fail to have oxygen treatment (because the symptoms are misdiagnosed). Their health deteriorates from delayed reaction caused by untreated carbon monoxide poisoning, and too often they become disabled.

Where is the airline industry? Where is the government oversight? The task of those who perceive the problem is as yet unfinished.

APPENDIX B. SYNTHETIC TURBINE ENGINE LUBRICATING OIL FORMULA[37]

B1. BASE STOCK

High temperature lubricants may be composed from at least six different acids, realizing the high temperature properties of grade 4 oil with a mixture of esters of neopentyl polyols (highly hindered alcohols):
Neopentyl glycol esters e.g., $C_{10}H_{20}O_4$
Pentaerythritol esters e.g., $C(CH_2OH)_4$ ($C_5H_{12}O_4$)
Trimethylolopropane esters e.g., $C_6H_{14}O_3$

The carboxylic acids include:

Valeric acid	$C_5H_{10}O_2$
Isovaleric acid	$C_5H_{10}O_2$
3,3-dimethylbutyric acid	$C_6H_{12}O_2$
Heptanoic acid	$C_7H_{14}O_2$
Octanoic acid	$C_8H_{16}O_2$
Nonanoic acid	$C_9H_{18}O_2$
3,5,5-trimethylhexanoic acid	$C_9H_{18}O_2$

B2. ADDITIVES

B2.1 Antioxidants

These representative antioxidants are unstable. Stabler polymeric antioxidants have been developed to take their place. Their structures are proprietary but the chemistry would likely be similar:

Butylated hydroxyl toluene	$C_{15}H_{24}O$
N-phenyl-l-naphthylamine	$C_{16}H_{13}N$ ($C_{10}H_7NHC_6H_5$)
Dioctyl diphenylamine	$C_{28}H_{43}N$

B2.2 Anticorrosion additives

Carboxylic acid esters	R–COOH
Phosphate esters	OP(OR)(OR')(OR")

The above rust inhibitors create some ash in service; those below have been developed to avoid the ash problem; the exact structures and proportions in the oil formulation are proprietary:
Metal sulfonates—many different forms (organic or inorganic)
Metal carboxylates—many different forms (organic or inorganic)

B2.3 Antifoaming additive

Silicone oils (i.e., any liquid, polymerized siloxane with organic side chains:
Siloxane $(OSiH_2)_n$

B2.4 Metal atom deactivator

Needed because of undesired catalytic properties of some metals:
Benzotriazole $C_6H_5N_3$

B2.5 Boundary lubrication additives

They are required to reduce friction between and wear of the engine components. The triaryl phosphates listed are the compounds of choice for aviation applications. Both are present in their various isomers:

Tricresyl phosphate	$C_{21}H_{21}O_4P$
Butylated triphenyl phosphate	$C_{22}H_{23}O_4P$

---

35 Cf. the many substances mentioned in ref. 6, and those lists are far from exhaustive.
36 Because they all originate within the engines.
37 The following synopsis has been taken from ref. 45, adapted for brevity and in consideration of the flying environment. Chemical formulae for each compound have been added. It is recognized that engine oil formulations are likely to be continually modified, but the essential attributes presumably remain the same.