

BEFORE THE WORKERS' COMPENSATION BOARD
STATE OF OREGON
HEARINGS DIVISION

In the Matter of the Compensation)	WCB Case No. 18-00006H ¹
)	Claim No. 555232469
of)	DOI: 1/21/2017
)	WCD File No. EBX2484
)	
)	WCB Case No. 18-04163
)	Claim No. 555232469
)	DOI: 1/21/2017
)	WCD File No. EBX2484
)	
)	WCB Case No. 19-00865
)	Claim No. 555232469
)	DOI: 1/21/2017
)	WCD File No. EBX2484
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)	WCB Case No. 19-01328
)	Claim No. 555232469
)	DOI: 1/21/2017
)	WCD File No. EBX2484
)	
)	WCB Case No. 19-01791
)	Claim No. 555232469
)	DOI: 1/21/2017
)	WCD File No. EBX2484
)	
ANDREW K. MYERS,)	
Claimant)	OPINION AND ORDER

A hearing was convened in the above-entitled matter on October 22, 2019, in Bend, Oregon, and on March 9, 2020, in Portland, Oregon, before Administrative Law Judge Darren Otto of the Workers' Compensation Board. Claimant was present

¹ The WCD's August 16, 2018 Transfer Order regarding Dr. Ugalde's recommendation for 24 acupuncture visits was mistakenly assigned an H number, 18-00006H (Ex. 98A). Since my task regarding this issue is to find whether or not a causal relationship exists between claimant's compensable injury and the requested medical services under ORS 656.704(3)(b), any appeal of that decision goes directly to the Workers' Compensation Board just like all of the other issues in this case.

and represented by his attorney Glen J. Lasken. The employer, JetBlue Airways, and its processing agent, AIG – Chartis Claims, Inc., were represented by their attorney Matthew M. Fisher.

The employer's request for hearing appealing the May 9, 2019 Order on Reconsideration in WCB Case No. 19-02648 was bifurcated from this proceeding and will be decided in a separate Opinion and Order with different exhibits found in the reconsideration record.

In the current proceeding, exhibits 1 through 149, A², D, E, 9A, 23A, 34A, 60A, 67A, 68A, 80A, 80B, 84A, 84B, 84C, 98A, 104A, 110A, 111A, 112A, 114A, 114B, 116A, 131A, 131B, 137A, 137B, 138A, 139A, 141A, 141B, 141C, and 142A were received into evidence. Claimant subsequently submitted Exhibit 34B, which was also received into evidence without objection.³ The hearing was continued for Dr. Harrison's deposition and written closing arguments. The employer, however, withdrew its request for Dr. Harrison's deposition and the record closed with the receipt of the hearing transcripts into evidence. Those hearing transcripts from

² Mr. Lasken submitted Exhibit A at the March 9, 2020, reconvened hearing. My office numbered that exhibit, pages 1 through 2439. Exhibit A includes medical and scientific research articles regarding airplane fume exposure events from the 1940s to the present.

³ The exhibits were submitted by the parties as follows:

Exs. 1 through 136 with Ex. 11, pages 2-4, being renumbered as 9A, pages 1-3 (submitted by Mr. Fisher on September 4, 2019)
Exs. 137 & 138 (submitted by Mr. Fisher on September 19, 2019)
Exs. 60A & 68A (submitted by Mr. Fisher on October 7, 2019)
Ex. 139 (submitted by Mr. Fisher on October 10, 2019)
Exs. 116A, 137A (previously submitted as Ex. 139), and 140 (submitted by Mr. Lasken on October 14, 2019)
Exs. 80A, 84A, and 92 – which was resubmitted to include pages 5-14, 137A (the same Ex. 137A previously submitted by Mr. Lasken), and 138A (submitted by Mr. Fisher on October 17, 2019)
Ex. 141 – Dr. Pleus' October 21, 2019 report previously marked Ex. 140 (submitted by Mr. Fisher on October 21, 2019)
Ex. 139A (submitted by Mr. Fisher on October 21, 2019)
Exs. 23A, 34A, 67A, 80B (previously 80A), 84B (previously 84A), 84C (previously 84B), 98A, 104A, 110A, 111A, 112A, 114A, 114B, 131A, 131B, 137B (previously 138A), and 142 (previously 141)
Ex. 142 and 139A – Dr. Burton's 10/20/19 report (submitted by Mr. Fisher on January 23, 2020)
Exs. 131B (previously 142A), 141C (previously 143), 143 (previously 144), 144 (previously 145), 145 (previously 146), and 146 (previously 147) (submitted by Mr. Lasken on February 7, 2020)
Ex. 130 – resubmitted with pages 26 & 27 which were inadvertently omitted (submitted by Mr. Fisher on February 27, 2020)
Exs. 148 & 149 (submitted by Mr. Lasken on March 6, 2020)
Ex. 34B (submitted by Mr. Lasken on March 12, 2020)
At the hearing, I received into evidence Exs. A (the research studies), D (Judith Anderson's CV), and E (the airplane diagram). Exs. B (Mr. Lasken's "Myers Research Index/Executive Summary") and C (Mr. Lasken's "Outline of Testimony of Judith Anderson (Murawski)") were not received into evidence.

Since two exhibits were marked Ex. 142, I informed the parties that Dr. Michaelis' October 21, 2019 report would remain Ex. 142 while Dr. Scott's January 10, 2020 deposition transcript would be remarked 142A. On April 29, 2020, the hearing transcripts from October 22, 2019, and March 9, 2020, were submitted and are received into evidence as Exhibit 150.

October 22, 2019 and March 9, 2020 were physically submitted on April 29, 2020, and were received into evidence as Exhibit 150.

On April 28, 2020, claimant filed his initial written closing argument. On July 2, 2020, the employer filed its written response. The hearing concluded on July 14, 2020, upon receipt of claimant's corrected reply.

ISSUES

1. Claimant appeals the employer's February 20, 2019 denial of compensability of his alleged toxic encephalopathy (Ex. 113), its April 2, 2019 denial of compensability of his mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency (Ex. 116), and its February 8, 2019 denial of compensability of his current condition (Ex. 110). The initial procedural issue is whether the scope of the employer's acceptances of claimant's "acute chemical inhalation" and "acute toxic inhalation" included any of the denied conditions (Exs. 35, 107, & 113). Specifically, the questions are whether (a) the employer accepted a mechanism of injury or (b) it accepted a vague or ambiguous condition, requiring reference to the contemporaneous medical records to understand what was actually accepted. If either of those procedural analyses is correct, the issue is whether the employer issued improper "back-up" denials pursuant to ORS 656.262(6)(a).

2. Claimant appeals the employer's February 20, 2019 and April 2, 2019 denials of compensability of his alleged toxic encephalopathy and mild neural cognitive disorder (Exs. 113 & 116). If the scope of the employer's acceptances of "acute chemical inhalation" and "acute toxic inhalation" did not encompass those denied conditions, then the issues are whether claimant's toxic encephalopathy and mild neural cognitive disorder existed and, if so, whether his exposure to toxic chemical fumes at work on January 21, 2017 was a material contributing cause of those conditions pursuant to ORS 656.005(7)(a).

3. Claimant appeals the employer's April 2, 2019 denial of compensability of his vision disorders diagnosed as convergence insufficiency and saccadic eye movement deficiency (Ex. 116).⁴ If the scope of the employer's acceptances of "acute chemical inhalation" and "acute toxic inhalation" did not encompass those denied conditions, then the issues are whether claimant's

⁴ Claimant withdrew his appeal of the employer's April 2, 2019 denial of compensability of his polyneuropathy (Ex. 116). Therefore, that portion of the April 2, 2019 denial will be approved.

convergence insufficiency and saccadic eye movement deficiency existed and, if so, whether his exposure to toxic chemical fumes at work on January 21, 2017, was a material contributing cause of those conditions pursuant to ORS 656.005(7)(a).

4. Claimant appeals the employer's February 8, 2019 denial of compensability of his current condition (Ex. 110). The first issue is whether the "current condition" denial was procedurally proper when (a) it issued before claimant made his new/omitted medical condition claim but after the employer accepted his claim for "acute chemical inhalation and "acute toxic inhalation," and (b) it only stated that claimant's current condition was no longer compensably related to his accepted "acute chemical inhalation" without mentioning his accepted "acute toxic inhalation." If the scope of the employer's acceptances did not encompass claimant's toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency, and the "current condition" denial was otherwise procedurally valid, the issue is whether claimant's exposure to toxic chemical fumes at work on January 21, 2017 was a material contributing cause of his then-current condition pursuant to ORS 656.005(7)(a).

5. Claimant alleges that there is a causal relationship between his January 21, 2017 compensable injury and the 24 acupuncture visits recommended by Dr. Ugalde for his post traumatic headaches. *See* August 16, 2018 Transfer Order (Ex. 98A). The issue is whether the compensable injury was a material contributing cause of claimant's need for the proposed acupuncture treatments pursuant to ORS 656.704(3)(b)(C) and ORS 656.245(1)(a).⁵

6. Claimant seeks a penalty and associated attorney fee pursuant to ORS 656.262(11)(a) for the employer's alleged unreasonable February 8, 2019 denial of compensability of his current condition (Ex. 110). The issues are whether the employer had a legitimate doubt regarding its liability for claimant's current condition and, if not, what are reasonable penalty and attorney fee amounts.

7. Claimant seeks an assessed attorney fee pursuant to ORS 656.386(1) of up to \$200,000, but no less than \$150,000. If one or more of the denials is set aside, the issue is what is a reasonable assessed attorney fee given the extraordinary circumstances of this case.

⁵ At the hearing, claimant withdrew his Request for Hearing in WCB Case No. 18-00006H, which appealed a December 14, 2017 Administrative Order of Dismissal regarding the causal relation between the industrial injury and eight acupuncture visits proposed on June 23, 2017 (Ex. 84A). Therefore, claimant's Request for Hearing regarding that WCB case number will be dismissed.

8. Claimant seeks the award of extraordinary costs and expenses associated with litigation pursuant to ORS 656.386(2)(a) in the total amount of \$38,717.15. The issue is whether claimant's request for extraordinary expenses and costs for records, expert opinions and witness fees was reasonable and appropriate. In particular, the employer contends that claimant is not entitled to reimbursement for (a) Dr. Michaelis' travel expenses because her testimony was not relevant or material to the issues, and (b) claimant's counsel's travel expenses associated with the taking of various doctors' depositions because they do not qualify as recoverable "costs" under the statute.

FINDINGS OF FACT

In the first half of the twentieth century, airplane cabin pressurization was achieved with turbo-compressors, drawing air directly from outside, but they were heavy and costly on fuel consumption (Ex. 141C-1). To save money, aircraft were redesigned in the 1950s to facilitate pressurization and ventilation of the cabin from "engine bleed air" drawn from the compressor stage of the propulsion gas turbine jet engines or auxiliary power units (APUs) on board the plane. *Id.* That redesign, which used unfiltered engine compressor air, is found in all current commercial aircraft except the Boeing 787 model which reverted to the earlier concept of not using bleed air. *Id.*

Quite soon after the introduction of cabin bleed air, the US military realized that there was a problem with oil fumes coming from the engines with crew becoming ill and instructed some pilots to wear oxygen masks during operations. Gas turbine engines operate at high temperatures and the synthetic lubrication oils must contain additives to reduce engine wear, oxidation and corrosion.⁶ The antiwear additives are usually triaryl phosphates, such as Tricresyl Phosphate (TCP), and have neurotoxic properties. The commercially available TCPs consist of a wide range of cresols, phenols and xylenols, not just the 10 TCP isomers, all of which are assumed to have similar reactivity.

With exposure to engine oil contaminants as aerosols and vapours in cabin air, two modes of exposure are recognized. In normal operation, aircraft with bleed air systems have been

⁶ **** Jet engine oil is used to lubricate, protect, and cool engine parts – it reduces friction between moving metal parts and acts as a reservoir for by-products of engine use (e.g. silica). **** (Ex. 141-16).

demonstrated to have a background low level mixture of contaminants present. These are generally at levels difficult or impossible to routinely detect but their presence is incontestable, as shown in many studies. The second form of exposure is when there is a noticeable odour ranging from very short-term transient exposures through to an oil bearing seal failure leading to a more obvious higher dose exposure, usually termed “fume events”. The principal route of exposure is through inhalation.

***.

Id.; see also Judith Anderson & Dr. Michaelis testimony; Exs. 60A & 141A, pages 9-11). The recirculated air is often filtered using a high efficiency particulate air filter (HEPA) which removes microorganisms and other particulate contaminants but does not remove other contaminants such as volatile organic compounds (Judith Anderson & Dr. Michaelis testimony; Exs. 139-155 & 141-4). Apart from noticeable fume events, pilots are chronically exposed to engine vapors that continuously leak through the oil seals in tiny amounts because the use of pressurized air to both seal the jet engine’s bearing chamber and to provide ventilation for the cabin guarantees that fugitive low-level oil emissions will enter the breathing air supply during normal engine operations (Judith Anderson & Dr. Michaelis testimony; Ex. 142, pages 3 & 5).

Airplane cabin fume events are common occurrences (Judith Anderson & Dr. Michaelis testimony; Exs. 126-8, 130-38 & 141-2). Hundreds occur each year, resulting in chemical exposures to tens of thousands of crew and passengers. *Id.* One plane a day is diverted in the United States due to in-flight smoke events (Ex. 139-173). Neurotoxicity is a major flight safety concern, especially where exposures are intense (Exs. 60A-1 & 130-38). Nevertheless, airlines appear to be more concerned about keeping planes in the air than worker safety (Ex. 130, pages 40 & 48). On July 5, 2017, an FAA Inspector, Jack Farenga, stated that he had received over a dozen reports of toxic fume events on JetBlue aircraft, rendering air crews ill from exposure (Ex. 60A-1).⁷ On September 19, 2019, the United States Congress expressed its “deep concern” to JetBlue Airways regarding (1) the significant number of severe fume events over the past few months, which posed a significant health risk to in-flight crewmembers and passengers, (2) a disturbing pattern of fume events onboard JetBlue aircraft, and (3) JetBlue’s attempt to skirt FAA reporting standards and workers’ compensation laws by reclassifying “fume events” or “cabin air safety

⁷ On October 21, 2019, Susan Michaelis, Ph.D., MSc., also confirmed that more than a dozen fume reports had been received by the FAA relating to JetBlue aircraft (Ex. 142-3).

events” as “odour events.” (Ex. 137B).⁸ That last concern raised significant doubt with the United States Congress regarding JetBlue’s intention to faithfully adhere to existing health, safety, and labor laws. *Id.* In light of the prevalence of flight crew members developing neurological problems following toxic gas exposures on planes, JetBlue added “fume events” to its required documentation (Ex. 120-3).

The medical findings and diagnoses of conditions resulting from aircraft fume events, while significant and consistent with exposure to substances in engine fluids and to complex thermally degraded mixtures, have not been well recognized within the aviation industry (Ex. 141A-10). One reason is the airlines have resisted on-board monitoring of cabin air quality (Exs. 139-112 & 141A-10).⁹ Other reasons include a lack of recognized medical protocol, a general reluctance to volunteer information on an issue that is not accepted by the airline industry, a lack of education about aircraft contaminated air, difficulties associated with maintenance investigations for bleed air contamination, and a reluctance by airlines to investigate such events (Ex. 141A-10). Also, there is a clear disincentive to report health effects when a commercial pilot’s license and career depend on good health. *Id.* Thus, there has been a large underreporting of fume events aboard commercial aircraft (Exs. 141A-3 & 142-3).¹⁰ In fact, it is common for pilots who have been exposed to a fume event to continue flying immediately after the event (Ex. 141A-10).

The severity of symptoms experienced by airplane crew members following exposure to cabin fumes depends on (1) the range of contaminants, (2) the intensity, duration and frequency of exposure, (3) the toxicity of compounds, and (4) individual susceptibility (Ex. 130, pages 39-40). The onset of symptoms are often delayed. *Id.* It is well recognized that some individuals will develop disease with chemical concentrations far below accepted standards designed to protect most (but not all) exposed individuals (Ex. 141A-12). Also, the use of those standards do not

⁸ Industrial Hygienist Anderson testified that the difference between an odor event and a fume event was that an odor event involved crew members only reporting an odor in the cabin whereas a fume event indicated the presence of an odor associated with symptoms consistent with toxic exposure.

⁹ Dr. Harrison stated that toxic fume events were “difficult to assess because of the challenges in conducting and implementing the proper studies that largely were the result of roadblocks placed by airlines and the manufacturers.” (Ex. 139, pages 47-48). In fact, Dr. Harrison had a device ready to roll out which would have measured the levels of tricresyl phosphates in the cabin air during airline fume events, but he was “not able to get permission from the airlines to do that.” *Id.* On March 4, 2020, Dr. Harrison emphasized that “the airline industry has pushed back against any effort to have constant monitoring of air quality in their airplanes, perhaps because of cost, perhaps because they don’t want to have proof available as to what is really happening.” (Ex. 149-4).

¹⁰ In 2009, a retired pilot in the UK proposed a significant reason for the under-reporting of fume events, stating, “**** [P]ilots have the best job in the world and do not want any conflict with management. Pilots are self-centered on their own career; they will not fill out questionnaires truly [sic] even if it is supposed to be anonymous. Consequently the number of entries in any tech-log needs to be factored by 100.” (Ex. 139-175). Many other reasons for underreporting fume events have been identified (Ex. 139, pages 177-178). Dr. Michaelis stated that less than 3.66% of fume events were reported as required (Ex. 141-3).

apply to the public flying at altitude or exposed to a complex pyrolyzed chemical mixture. *Id.* It is common for crew members' responses to chemical exposures to vary widely (Ex. 141-10 & 141A-11). Individual susceptibility to damage by organophosphate (OP) exposure is highly variable because (1) some people have constitutionally lower levels of liver enzymes that detoxify OPs and (2) individuals with chronic exposure to toxic chemicals may build up a higher concentration of toxins in their system. *Id.* Chemical hypersensitivity occurs in two to three percent of exposed individuals (Ex. 130-47). The more exposed people are to chemicals over time, the more adverse the effects become (Exs. 141-10 & 141A-11). Thus, "acute-on-chronic" toxic inhalation injuries may occur when individuals are cumulatively pre-exposed for hundreds or thousand of hours and become more vulnerable to harm from a subsequent high dose fume event (Ex. 141B-2).

The primary explanation for fume events in airplanes is that jet engine oil has been pyrolyzed (heated) at extremely high temperatures and leaked through oil seals before entering the ventilation systems where the fumes are pumped into the cabin environment (Ex. 139-65). When jet engine oil is pyrolyzed, it also produces a complex mixture of new fugitive chemical emissions in addition to Ultrafine Particles (UFPs), which are the same size as Nano-particles, namely 1 – 100 nm (Exs. 141-7 & 141C, pages 4-5). Particles that small become much more reactive, even for materials that are chemically inert in bulk, because they (1) induce inflammation, largely irrespective of what they are made of, (2) are preferentially deposited to the deepest alveolar regions of the lungs, where gas exchanges between air and blood are conducted, and (3) can act like Trojan Horses as they cross the Blood Brain Barrier (BBB), which has evolved to keep unwanted chemicals at bay, thereby avoiding the metabolic defense mechanisms of the BBB while the toxins adhere to the surface of the Nano-particles (Ex. 141C-5).

While the majority of consumed jet engine oil goes out via the vent system, there is low level oil leakage past the seals into the ventilation system because (1) the seals are not an absolute design, (2) leakage occurs during engine power changes, air supply changes, low internal pressure (start up, spool up, taxiing, top of descent, and descent), thermal and mechanical changes in engine structures, and under moisture conditions, (3) oil loss can occur during certain abnormal operations, such as oil overfilling, seal wear, component degradation or a failure condition, and (4) UFPs act like gases and cross oil seals along with other vapor phase molecules derived from the engine lubrication oil (Exs. 126-21, 139-361, 141-5, & 141C-6).

Mobile Jet Oil II was used in the jet flown by claimant on January 21, 2017 (Claimant & Judith Anderson testimony). That oil was composed of a synthetic base

stock of esters and fatty acids with a complex mixture of 250 to 400 chemical compounds and four additive ingredients that improved specific performance characteristics such as oxidation and wear tendencies (Judith Anderson testimony; Ex. 141-16). Those additive ingredients included Tri-cresyl phosphate (TCP) at concentrations from 2-5%, n-phenyl-1-naphthylamine (PAN) at 1%, 9,10-anthracenedioine, 1,4-dihydroxy- at <0.1%, and alkylated diphenyl amines at 1-<5%. *Id.* Mobile Jet Oil II included a large number of toxic components, including TCP (Ex. 139, pages 181-182). The three cresyl groups in a given molecule of TCP can attach to the phosphate in different configurations, called isomers (Ex. 139-380). There are ten different isomers of TCP, the most toxic of which are the ortho-isomers (Judith Anderson testimony; Ex. 139, pages 182 & 380). Of those, the three mono-ortho isomers (MOCP) and two di-ortho isomers (DOCP) are five to ten times more neurotoxic than the tri-ortho (TOCP) isomer (Ex. 139, pages 182 & 381; *see also* Judith Anderson testimony; Ex. 141-30). There are also four meta/para isomers (Ex. 139-380). The ortho isomer makes up about .3% of the TCP and the vast majority (99.97%) of the ortho isomers are MOCP and DOCP, while there is very little TOCP. *Id.*¹¹ Over time, the ortho content in jet engine oil has been reduced from 1% to .2% (Judith Anderson testimony).

Even without a fume event, TCP has been found in 25 to 100 percent of *ad hoc* air samples during normal engine operations (Ex. 141A, pages 1-2). TCP has neurotoxic properties, but the widespread belief that only ortho isomers of TCP are dangerous is invalid (Ex. 141A-10). The 99.7% of non-ortho isomers of TCP and TAP can cause nerve demyelination and inhibit various enzymes, including those linked to cognition. *Id.* All forms of TCP have significant neurotoxic effects (Judith Anderson testimony). Thus, chronic exposure to toxic chemicals in airplane cabins is caused by the oil vapors released through oil leaking continuously over the seals during engine power changes (Ex. 141A-9). When the jet engine oil is heated to high temperatures, its decomposition also results in the formation of a range of additional hazardous compounds through pyrolysis, including ultrafine particles, additional TCP isomers, carbon dioxide, carbon monoxide, aromatics, alkanes, amines, cresyl- and more volatile butyl-phosphate esters, as well as irritant carboxylic acids, aldehydes and ketones (Ex. 139, pages 184-186 & Ex. 142-7).^{12 13 127} oil pyrolysis products are related to oils heated to high temperatures (Ex. 142-7).

¹¹ Little is known about the relative amounts of the remaining meta and para isomers in jet engine oil and most research has been done on the effects of TOCP because of two highly publicized TOCP mass poisonings resulting from adulteration of (1) a popular alcoholic drink called "Ginger Jake" in 1929 and (2) a large batch of cooking oil in 1959 (Judith Anderson testimony; Ex. 139-380). TOCP is also the only TCP isomer for which an OSHA exposure limit exists (Ex. 139-380).

¹² "Pyrolysis is the chemical decomposition of a condensed substance by heating." (Ex. 139-184).

¹³ Dr. Michaelis explained the process of pyrolyzation of jet engine oil as follows.

Airplane crew members exposed to fume events frequently experience symptoms soon afterward, even if those symptoms are sometimes diffuse and nonspecific (Ex. 130, pages 38-39). There have been approximately 15,000 documented cases of flight crew members developing neurological problems following toxic gas exposure (Ex. 120-3). The association between airplane chemical exposure fume events and the onset of symptoms was so strong that the term "Aerotoxic Syndrome" was coined in 1999 (Ex. 130, pages 45 & 56). Although not yet accepted as a medical term, the syndrome is well documented in the scientific literature (Dr. Michaelis testimony). The spectrum of neurological signs and symptoms associated with "Aerotoxic Syndrome" constitute a group of non-localizing functional deficits which are consistent with a diffuse toxic encephalopathy caused by the continual presence of nanoparticle aerosols mixed with a complex mixture of organophosphates that target the brain (Ex. 141C-6). Crew members' symptoms from single or short-term fume exposure may include blurred or tunnel vision, disorientation, memory impairment, shaking and tremors, nausea/vomiting, paresthesia, loss of balance and vertigo, seizures, loss of consciousness, headache, lightheadedness, dizziness, confusion and feeling intoxicated, breathing difficulties including shortness of breath, tightness in the chest, and respiratory failure, along with increased heart rate and palpitations, nystagmus, and irritation of the eyes, nose and upper airways (Ex. 130-39). Symptoms from long-term low level exposure or residual symptoms from short-term exposures have also included memory impairment, forgetfulness, lack of coordination, nausea/vomiting, diarrhea, respiratory problems, chest pain, severe headaches, dizziness and feeling intoxicated, weakness and fatigue (leading to chronic fatigue), exhaustion, increased heart rate and palpitations, numbness in the fingers, lips and limbs, hot flashes, joint pain, muscle weakness and pain, salivation, irritation of the eyes, nose and upper airways, skin itching and rashes, skin blisters on uncovered body parts, signs of immunosuppression, hair loss, and chemical sensitivity (Exs. 130-39 & 139-373).

The oil contained within the engine lubrication system will be exposed to the bulk oil temperatures of above 200 [degrees] C for the majority of time while in the oil reservoir or circulating throughout the lubricated areas. However the oil will be exposed to very high temperatures for a very short period of time when in contact with metal parts in the lubricated areas. These temperatures may be up to 500 [degrees] C in the compressor area and up to 1700 [degrees] C in the turbine area, as the oil circulates the entire system. Temperatures are estimated to be much higher again for nanoseconds as the synthetic ester-based oils under shear can form an aerosol of nano droplets due to the pressures and shear stresses in the bearings. This would occur on a recurring basis. Oil escaping past seals in the compressor area may then be exposed to high temperatures up to around 500 [degrees] C as identified above.

(Ex. 142-6).

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Chemicals can effectively enter the body via inhalation, skin, and ingestion, with inhalation being the most effective because of the large surface area of the lung and the fact that inhaled chemicals escape first-pass metabolism (Ex. 90-2). Nerve system damage may result from a single large chemical exposure that causes neurological deficits. *Id.* Nerve system damage can also result from repeated low-level chemical exposures that cause small neural injuries which accumulate and result in neurological deficits over time (Ex. 90-2 & Ex. 126, pages 8-10). Nerve system damage is more likely when there is exposure to multiple chemicals because those chemicals compete with each other for the body's defense mechanism with subsequent increased delivery of each chemical to the neurotoxicity target (Exs. 90-2 & 126-9). Common neurocognitive complaints following toxic exposure events include confusion, fatigue, headache, difficulty with concentration, information processing speed, memory and learning, along with depression, anxiety, irritability, and restlessness (Ex. 86-25; *see also* Ex. 132-2). Those cognitive deficits may present after a brief latency period (Ex. 86-26).

In the present case, claimant was 54 years old at the time of hearing (Ex. 25). He had childhood asthma, which resolved when he was twelve or thirteen years old (Claimant testimony). Later in life, he developed occasional sinus problems with sinus headaches over the forehead, nose and cheeks from being around lots of sick airline passengers, especially in the winter. *Id.* He sought preventive care from doctors for those sinus problems to avoid rupturing his eardrums while climbing and descending on airplanes (Claimant testimony; *see also* Exs. 1, 2, 3, 4, 6 & 9). Claimant was very physically active before the industrial injury (Claimant, Captain Mark Schussler & Captain James Richards testimony; Ex. 122, pages 27-28). During the 1980s, he competed in a number of triathlons and was training for another triathlon in 2016 (Claimant testimony; Ex. 122, pages 27-28). He liked to swim, ride his bike, and run. *Id.* He also kayaked and hiked. *Id.* He enjoyed sports and physically pushed himself. *Id.* In addition, he was a qualified marksman who usually obtained perfect scores in firearm competitions. *Id.*

In 2002, claimant began working for the employer in its commercial airline business and, three years later, he became a captain (Claimant testimony). Claimant came from a family of airplane pilots, loved his job and, as a result of his seniority by 2017, was able to set his own schedule and destinations (Claimant, Captain Mark Schussler & Captain James Richards testimony). During the winter months, he frequently flew to the Caribbean Islands to enjoy the sun and sea (Claimant testimony). He intended to continue flying until the legally allowed age of 65. *Id.* At the time of injury, he was making \$248,000 per year plus significant health and retirement benefits. *Id.* Most pilots commuted to work and claimant was no different.

Id. He and his wife lived in Bend, Oregon, and he was headquartered in New York City where he shared an apartment with other pilots. *Id.* Claimant spent about half his time in Bend and the other half on the road. *Id.*

Before 2017, claimant was training to compete in a triathlon (Claimant testimony). From the fall of 2015 through the spring of 2016, he experienced bad headaches. *Id.* He sought treatment for that condition and the headaches resolved when he changed his diet and stopped exercising so much (Claimant testimony; Exs. 14 through 17). He did not have any headaches between April 2016 and January 2017 (Claimant testimony; Exs. 19 & 20). He also did not have any memory problems or difficulties speaking during that period of time (Ex. 31-1).

On November 17, 2016, claimant underwent his bi-yearly physical examination in order to maintain his flying status (Exs. 21 & 79). At that time, the Aviation Medical Examiner, Jerry Bass, M.D., issued a Medical Certificate to fly with no significant medical history and no abnormal physical findings. *Id.* Dr. Bass examined claimant every six months for at least ten years (Ex. 79-4). Throughout that ten-year period, Dr. Bass had no doubt that claimant was in good health and able to perform his duties as an airline captain. *Id.* Before the industrial injury, claimant was “an affable and happy pilot” who was extremely pleased to be working (Ex. 116A-2). He was also a generous and cheerful man with no history of malingering or secondary gain. *Id.*

On January 21, 2017, claimant was exposed to toxic chemical fumes on board an Airbus A320 jet airplane at work (Claimant testimony; Exs. 23, 24, 29, 31, 32, 33, 67A, 113 & 141-5). The day before, the flight attendants and customers on the airplane that claimant was scheduled to fly reported a strong foul odor in the cabin while descending for landing (Exs. 23, 23A & 67AA). Therefore, a fume event was written up for maintenance to resolve. *Id.* The next day before take-off, claimant and his First Officer, Dirk Murray, attempted to isolate the source of the fumes by performing three engine runs (Claimant testimony; Exs. 23, 24 & 67A). During the first run, claimant smelled the foul odor throughout the plane, but the First Officer, who remained in the cockpit, did not. *Id.* During the second engine run, claimant and the First Officer both smelled a very apparent, choking, burning odor like dirty socks or an oily smell. *Id.* It was the worst toxic fume event claimant ever experienced as a pilot (Ex. 23-1). Both claimant and the First Officer immediately developed headaches and were coughing and had to leave the airplane to get fresh air (Ex. 23-1). The First Officer also had throat irritation in the form of dryness and hoarseness (Ex. 67A). Claimant fell down on the jetway and a mechanic ran up the stairs to the

plane and said, "Oh my God. There's a haze in here." (Claimant testimony).¹⁴ Claimant and the First Officer re-entered the plane for the third engine run and the odor was again very apparent to both of them (Exs. 23 & 67A). The First Officer was still coughing and he had a headache focused on the front of the head and pain was developing behind his right eye as if someone was poking him in the eye. *Id.* Claimant was coughing, his eyes stung and watered, the right side of his body shook with tremors, and he had mild shortness of breath, headache, and congestion (Claimant testimony; Exs. 30 & 33). He also had some ulnar aspect numbness in his forearm and hand that resolved after a couple of days (Ex. 33-1).

Claimant shut the engines down after the third run and instantly opened the flight deck windows to get fresh air (Claimant testimony; Exs. 23 & 67A). As the jet bridge was reattached, one of the in-flight crew members saw haze in the main cabin. *Id.* Claimant contacted maintenance control and reported what happened. *Id.* Maintenance agreed that burning oil probably leaked through an engine seal into the ventilation system (Ex. 23-1). A February 22, 2017 Engineering Disposition Report confirmed that the airplane APU was cracked and leaking oil with oil contamination into the ducting (Ex. 34A; *see also* Judith Anderson & Dr. Michaelis testimony; Ex. 139-63).¹⁵ That report of oil leakage in the engine and ventilation systems was very typical for fume event chemical exposure cases and confirmed a pathway for jet engine oil pyrolysis products to enter the cabin air (Ex. 139, pages 64-65).¹⁶

Shortly after claimant advised maintenance that the plane was unable to fly due to continuing cabin fumes, the flight was canceled and claimant and the First Officer were flown to San Francisco (Exs. 23 & 67A). Upon arrival in San Francisco, the First Officer did not consult a physician, but he was still experiencing a headache and respiratory symptoms, including a dry and sore throat (Exs. 23 & 67A). The headache continued to improve throughout the day and evening (Ex. 67A). While the headache resolved by the following day, the First Officer continued to have the soreness and dry throat for days after the event. *Id.*

¹⁴ The presence of haze suggested a significant exposure occurred (Ex. 130-69).

¹⁵ On September 5, 2017, JetBlue's Operations manager Jeffrey Martin wrote an e-mail to fellow JetBlue pilots to inform them that claimant's "prolonged exposure and attempts to ventilate the fumes from the cockpit and cabin are significantly different from the recent odor and fume events experienced by some Crewmembers during various phases of flight" and, during claimant's January 21, 2017 toxic exposure, "very strong fumes were detected with possible associated haze in the cockpit and throughout the cabin." (Ex. 68A). In light of those multiple fume events, Mr. Martin stated, "With safety as our top priority and out of an abundance of caution, we are reviewing and amending engine run up protocols for the continued safety of our Pilots and Tech Ops Crewmembers. ***" *Id.*

¹⁶ In the majority of in-service contamination events, the source of the fumes is the lubricating oil from the APU (Ex. 142-5).

In contrast to the First Officer, claimant experienced more significant symptoms immediately after the fume event (Claimant testimony). When claimant arrived in San Francisco, he tried to go for a run but couldn't do it because he had trouble breathing. *Id.* He returned to his starting point but was confused and couldn't remember which hotel he was staying at. *Id.* He walked around until he saw another JetBlue crew member and went into the right hotel. *Id.* Claimant returned to his room, called Crew Services, and said he did not know if he would be well enough to fly the next day. *Id.* He talked to a doctor and described the fume event, his symptoms and confusion. *Id.* The doctor told claimant to get as much fresh air and water as possible and he should be fine in the morning. *Id.* The next day, claimant felt much better. *Id.* He still had a headache but no other symptoms. *Id.* During the flight from San Francisco to New York, First Officer Dirk Murray flew the plane and claimant operated the radios. *Id.* In the middle of the flight, claimant's headache became severe again. *Id.* That was the last flight he worked due to the severity of symptoms that worsened over the ensuing weeks and months. *Id.*

On January 25, 2017, claimant sought treatment from Sean Suttle, PAC, for shortness of breath, headache, coughing, constipation, nausea and a feeling of foginess (Ex. 26). Mr. Suttle diagnosed a cough with exposure to chemical inhalation and toxic inhalation. *Id.* Chest x-rays showed no acute cardiopulmonary processes (Ex. 27). Mr. Suttle recommended claimant continue with rest and fluids and follow up if his symptoms worsened (Ex. 26).

By February 2017, claimant was continuing to experience tremors severe enough that he could not hold a glass of water and developed stuttering that made it hard for him to be intelligible (Claimant testimony). There were also times he did not know where he was and he required a cane to prevent himself from falling. *Id.* The severity of those symptoms varied over time. *Id.* On February 2, 2017, claimant sought emergency room treatment for shortness of breath, persistent low grade headache, upper extremity tremors, confusion, and aphasia (Ex. 29). Brian Rapacz, M.D., diagnosed an acute altered mental status with vague neurocognitive symptoms and toxic exposure. *Id.* He referred claimant to his attending physician. *Id.*

On February 10, 2017, claimant went to the emergency room again with complaints of mental confusion and headaches along with memory and word finding difficulties (Ex. 30). An MRI scan of the brain was interpreted as showing (1) no acute intracranial abnormality with no evidence of ischemic infarction and (2) mild bilateral cerebellar tonsillar ectopia, slightly worse on the right than left (Ex. 30-4). MRI scans, however, are not sensitive for detecting toxic exposure to tricresyl phosphate, one ingredient used in jet airplane oil (Ex. 33-2). The emergency room

physician, Dr. Siebe, concluded that claimant had nonspecific neurologic symptoms after exposure to burning jet fuel and toxic smoke inhalation and diagnosed (1) nonspecific neurologic symptoms, (2) acute headache, (3) exposure to jet fuel smoke, and (4) reported exposure to TCP (Ex. 30, pages 4-5).

On February 14, 2017, claimant sought treatment from Larsen Farris, PA-C, for worsening symptoms of fatigue, headaches, a resting upper extremity tremor, a feeling like his whole body was shivering inside, memory difficulties and aphasia with delayed speech and word finding difficulty (Ex. 31-1). Mr. Farris diagnosed (1) exposure to chemical inhalation, (2) tremor, (3) aphasia, and (4) cognitive deficits (Ex. 31-3). He believed that claimant's symptoms were most likely related to exposure to TCP from jet engine oil and recommended evaluations by a neurologist and neuropsychologist. *Id.* Specifically, Mr. Farris stated, "His current symptoms are due to his OJI on 1/22/17 when he was exposed for a prolonged period of time in an enclosed space to jet engine oil smoke and neurotoxins most likely carbon dioxide as well as tricresyl phosphate. His current symptoms are consistent with TCP poisoning symptoms as stated on MDS sheet." *Id.*

On February 21, 2017, claimant sought treatment from his longtime treating physician David Schloesser, M.D., for mental processing, speech and reading difficulties, fatigue, daily headaches, mild photophobia and phonophobia, an internal sensation of tremors, and nausea (Ex. 33-2; *see also* Exs. 32 & 116A-2).¹⁷ The examination demonstrated diminished sensation into the feet (Ex. 33-2). As a result, nerve conduction testing was performed which demonstrated evidence for sensorimotor polyneuropathy affecting the lower and upper extremities (Exs. 33 & 34). Dr. Schloesser diagnosed migraine headaches and polyneuropathy due to toxic agents (Ex. 33-2). He prescribed medications and recommended neuropsychological testing. *Id.*

On February 23, 2017, the employer accepted claimant's "acute chemical inhalation" as a disabling industrial injury (Ex. 35). On February 6, 2019, the employer amended that Notice of Acceptance to include claimant's "acute toxic inhalation" as well (Exs. 107 & 113).

On March 3, 2017, claimant began treating with Viviane Ugalde, M.D., for cognitive deficiencies that involved speaking, reading, writing, and recalling information (Ex. 39-1). Claimant also had continued coughing, hoarseness, trouble

¹⁷ On April 2, 2019, Dr. Schloesser confirmed that he had treated claimant prior to the industrial injury when he was an "affable and happy pilot." (Ex. 116-2).

swallowing, fatigue, visual changes, and daily body tremors, worse on the right side. *Id.* Dr. Ugalde diagnosed (1) toxic encephalopathy, (2) toxic peripheral neuropathy, (3) exposure to chemical inhalation, (4) tremor, (5) wheezing on expiration, (6) vision impairment, (7) vestibular disequilibrium, and (8) daily persistent headache (Ex. 39-4). Toxic encephalopathy means that the brain tissue has been damaged by toxic exposure (Ex. 118-3). Dr. Ugalde ordered a neuropsychological evaluation, referred him for acupuncture treatments to reduce his headaches, and indicated she would be contacting medical experts in the field of chemical exposure to discuss the case (Ex. 39-5). She remained claimant's primary care physician for his acute toxic inhalation and acute chemical inhalation conditions until claim closure on February 6, 2019 (Exs. 39, 41, 46, 47, 48, 63, 66, 67, 68, 70, 75, 78, 80, 81, 87, 89, 93, 98, 99, 101, 118, and 124).

On March 15, 2017, a CT scan of claimant's chest showed no evidence of pneumonitis or other acute lung injury (Ex. 40). No significant air trapping or excessive airway collapse was seen on expiratory imaging. *Id.*

On March 28, 2017, claimant returned to Dr. Schloesser with low energy, forgetfulness, and difficulties with mental processing and reading retention (Ex. 42-1). Dr. Schloesser diagnosed peripheral neuropathy, although claimant's reflexes were slightly improved on examination. *Id.* He recommended a PET scan. *Id.*

On April 3, 2017, claimant underwent neuropsychological testing with Psychologist Leah Schock, Ph.D., who concluded that the January 21, 2017 fume event resulted in a mild neurocognitive disorder which was consistent with the types of cognitive changes described in the post-toxic exposure literature (Ex. 43-8). Claimant's test findings were consistent and he showed no evidence of malingering or faking his symptoms (Dr. Schock testimony). Dr. Schock did not believe that claimant was suffering from a conversion disorder, somatic disorder, other psychological disturbances, or unconscious issues. *Id.* She recommended speech and language therapy, continued psychological support, and repeat neuropsychological testing to assess changes in claimant's cognition over time (Ex. 43-9).

On April 10, 2017, claimant sought treatment for breathing difficulties from Jean Verheyden, M.D., who performed a flexible nasolaryngoscopy of the larynx (Ex. 44-2). During that test, claimant exhibited abnormal vocal cord movement. *Id.* Dr. Verheyden believed that condition was due to an underlying neurologic or pulmonary issue and recommended the issue be monitored long-term. *Id.*

On May 26, 2017, claimant sought treatment for visual problems including photophobia, spatial confusion, and blurred vision from Optometrist Kirsten Scott O.D., who diagnosed convergence insufficiency and presbyopia (Ex. 51).¹⁸ She recommended SV reading glasses with blue lenses to calm his visual symptoms. *Id.* Dr. Scott continued to treat claimant for his vision problems through at least March 6, 2019 (Ex. 117-1). Ultimately, she concluded that the January 21, 2017 fume event was the major contributing cause of visual problems including convergence insufficiency, which meant that his eyes were not pulling together properly, and (2) saccadic eye movement deficiency, which meant that his eyes were not tracking well together (Ex. 117-2). She also believed that the combined effect of those two conditions left claimant with impaired vision which required medical care. *Id.* Her expert medical opinion was based on a number of factors including (1) claimant's vision conditions were consistent with the effects of a brain injury, (2) toxic encephalopathy involves damage to the brain tissue which can readily cause claimant's types of visual problems, (3) the testing she performed, consisting of a Brain Health IQ Test, showed objective evidence of damage resulting in the diagnosed conditions, and (4) a very strong temporal relation since claimant had been flying airplanes for many years and had achieved marksman status in firearms training before the fume event and he had impaired vision that precluded him from working as an airplane pilot after the fume event (Ex. 117, pages 3-4). On September 18, 2019, Dr. Scott concluded that, although claimant's visual performance deficits were consistent with the effects of a brain injury, as an optometrist, she was not qualified to determine whether claimant's visual performance deficits were caused by exposure to a harmful level of toxic chemicals (Ex. 138-1). Dr. Scott deferred to the specialists to answer that question (Exs. 138-2 & 140-5). On October 7, 2019, Dr. Scott decided to offer her opinion regarding causation and concluded that claimant's vision diagnoses were due in major part to the toxic fume exposure he endured on January 21, 2017 (Ex. 140-4). In arriving at that opinion, Dr. Scott relied on Dr. Ugalde's assessment that claimant had toxic encephalopathy due to the January 21, 2017 chemical inhalation fume event (Ex. 142A-22)

On May 31, 2017, claimant sought treatment for voice, breathing, and swallowing problems from Speech Pathologist Linda Bryans, M.A., C.C.C., because his voice was hoarse, effortful, weak, and reduced in loudness (Ex. 53). Ms. Bryans diagnosed dysphonia, dyspnea, and laryngeal hyperfunction. *Id.* She provided voice therapy to improve efficiency of breathing and reduce laryngeal hyperfunction. *Id.*

¹⁸ According to Dr. Scott, "Saccadic eye movements are the movements we use when we're reading, so there's a quick jumping eye movement that we use to track across a page." (Ex. 142A-9).

She did not believe that claimant's dyspnea was characteristic of paradoxical vocal fold motion and might have a functional component, but it was difficult to make that determination without a complete pulmonary work-up, which she recommended. *Id.*

On May 31, 2017, Joshua Schindler, M.D., performed a complete pulmonary work-up (Ex. 52). Based on that evaluation, he did not believe that claimant was suffering from classic vocal cord dysfunction (Ex. 52-5). Instead, Dr. Schindler believed that claimant had a pattern of breathing most consistent with diminished lung compliance and need for Valsalva on exhale to prevent collapse of the alveoli. *Id.* He recommended more extensive cardiopulmonary workup and additional speech therapy with Ms. Bryans. *Id.* A June 14, 2017 esophagram was interpreted as showing mild oropharyngeal discoordination (Ex. 57).

On June 15, 2017, the insurer-arranged medical examiner and Medical Toxicologist Brent Burton, M.D., performed a file review on behalf of the employer (Ex. 58). After reviewing the medical evidence, he believed it was impossible that TCP or TOCP or any other substance in the jet engine oil caused any of claimant's symptoms because claimant was supposedly not exposed to a sufficient quantity of those toxins (Ex. 58, pages 7-8). Dr. Burton also believed that claimant did not exhibit any evidence of an organic illness or injury and there were no reported cases of toxicity stemming from an inhalational exposure to TOCP (Ex. 58-7). Therefore, Dr. Burton concluded that claimant was suffering from a psychogenic disorder (Ex. 58-8). Dr. Burton's belief that "aero toxic syndrome" was a myth was not supported by the scientific literature and his belief that claimant did not exhibit any immediate signs of injury was incorrect (Exs. 92-3). On October 20, 2019, Dr. Burton reiterated his opinion that claimant experienced a perceptual event and did not suffer a physical injury or disease as a result of the chemical exposure (Ex. 139A-5). That opinion, however, was limited to whether or not there was significant exposure to TCP and/or TOCP (Ex. 139A-7).

On August 9, 2017, the insurer-arranged medical examiner and Neurologist Lynne Bell, M.D., performed a records review on behalf of the employer (Ex. 65). Based on her review of the medical records, she found no objective evidence of any neurological disorder, including polyneuropathy, peripheral neuropathy, or acute toxic encephalopathy (Ex. 65, pages 16-19). Dr. Bell concluded that claimant's worsening symptoms since the fume event raised the possibility of a psychogenic source in the absence of verifiable objective neurological deficits (Ex. 65-19).

On September 29, 2017, claimant was examined by the insurer-arranged medical examiner and Neurologist Patrick Radecki, M.D., who performed nerve

conduction studies and found no electrical evidence of a diffuse peripheral neuropathy on examination and inconsistent physical findings in the medical record relative to peripheral neuropathy (Ex. 69-9).

On October 30, 2017, and November 8, 2017, claimant underwent another round of neuropsychological testing by Dr. Schock, who continued to diagnose a mild neurocognitive disorder without improvement (Ex. 74). In comparison to the April 3, 2017 neuropsychological evaluation, claimant demonstrated ongoing generalized cognitive dysfunction with primary deficits in language expression, complex attention, and working memory. *Id.* He displayed consistent difficulties on tasks requiring complex attention, motor coordination and working memory (Ex. 84-1). The neurocognitive testing included validity measures which showed no evidence of malingering or somatization. *Id.* Claimant participated fully in the testing without evidence of suboptimal effort. *Id.* Subsequently, Dr. Shock reiterated her conclusion that claimant exhibited clear objective evidence of neurological dysfunction and he was not faking or making up his symptoms (Ex. 115-2). She believed that claimant's exposure to toxic fumes on January 21, 2017 was the major contributing cause of his neurocognitive disorder (Ex. 115-3). That expert medical opinion was based on a number of factors, including (1) repeated objective evidence of neurocognitive impairment during testing, (2) no evidence of malingering or somatization, (3) the mechanism injury was consistent with the diagnosis because his general pattern of results was consistent with what is seen in the toxic exposure population, (4) a strong temporal relationship suggested a sudden change in his condition immediately following the toxic exposure, (5) a complete absence of any other toxic exposure to account for the development of his condition, (6) a lack of family history of similar cognitive disorder, and (7) no evidence claimant had any system-wide problems (Ex. 115, pages 3-4; *see also* Exs. 74 & 84). During her June 25, 2019 deposition, Dr. Schock testified that claimant's April 4, 2017 test results showing cognitive confusion and cognitive impairment were consistent with the confusion he experienced immediately after the fume event when he became lost while trying to find his hotel in San Francisco (Ex. 130-11).

Claimant's treating physician, Dr. Ugalde, agreed that claimant's abnormal neurocognitive testing with Dr. Schock in April 2017, and again in October/November 2017, represented objective evidence of impairment (Ex. 81-6). Other objective measures of impairment included ongoing vestibular signs and symptoms including sustained nystagmus with large movement higher frequency extraocular lateral movements, positive vestibular ocular reflex, and loss of balance with pivot and quick changes in gait. *Id.* Objective evidence of impairment also included claimant's headaches, which were severe enough to classify as migraines, vocal cord

adduction with expiration documented by ENT, which claimant felt as difficulty breathing, decreased pinprick sensation in a stocking glove distribution, which had improved over time and was consistent with improving reflexes and nerve conduction studies, along with fatigue, total body tremors, and mood disturbance. *Id.*

On December 20, 2017, claimant underwent his bi-yearly examination with FAA Medical Examiner Dr. Bass, who refused to issue a Medical Certificate and stated that claimant's "tragic incident related to toxic fume inhalation in January 2017 has rendered him incapable of functioning in this capacity [as an airline captain]." (Ex. 79-4). Dr. Bass observed a marked change in claimant's condition after the fume event regarding his cognitive abilities, neurological findings, and visual difficulties (Ex. 120-2). In fact, Dr. Bass found claimant's whole post-injury demeanor grievously impaired and believed that he was absolutely not faking or exaggerating his condition (Ex. 122-29).

On January 4, 2018, the insurer-arranged medical examiner Timothy Craven, M.D., performed a treatment review at the employer's request and concluded, "There is objective support that [claimant] has developed a physical problem and neurocognitive problems since the [fume] exposure. Some of his current symptoms are possibly explained on a psychological basis but there could be a long term effect from the exposure to TCP." (Ex. 80A-4). On January 25, 2018, Dr. Craven, added an addendum to his earlier report (Ex. 84A). Assuming there was no confirmation of toxic encephalopathy in flight crews from exposure to TCP in the medical literature, Dr. Craven concluded that claimant's exposure to toxic fumes at work on January 21, 2017, caused his symptoms during the first few days, but it was very unlikely to have caused his long-term persistent neurological problems and cognitive impairment (Ex. 84A-2). Dr. Craven recommended a psychological consultation. *Id.* Dr. Burton subsequently agreed with Dr. Craven's assessment regarding causation (Ex. 92, pages 3-4).

On January 4, 2018, Dr. Ugalde disagreed with the conclusions of the IME physicians, Drs. Burton and Bell, noting that claimant did have immediate symptoms following the fume event and Dr. Schloesser's clinical examination was consistent with the NCS findings of peripheral neuropathy (Ex. 80-1). Dr. Ugalde explained that claimant's peripheral nerve function subsequently improved and he had consistent symptoms of toxic encephalopathy with documented neurocognitive changes on neuropsychological testing. *Id.* Dr. Ugalde also found the opinions of Drs. Burton and Bell invalid because they did not have serial assessments of claimant. *Id.* Based on all of the evidence, Dr. Ugalde continued to believe that

claimant's toxic encephalopathy was caused by the January 21, 2017 exposure to fumes at work (Ex. 80, pages 1-2).

On January 9, 2018, Dr. Schloesser believed that Dr. Bell's conclusions regarding causation based on a records review were "clearly misguided." (Ex. 83-1). Dr. Schloesser emphasized that claimant "is simply unable to work as a result of his injury, which is very unfortunate, as he was very happy as a pilot and had intended to continue flying for many years. [Claimant] does not have a history of malingering or secondary gain agenda. He has simply been injured by direct exposure to an organophosphate through the workplace. ***" (Ex. 83-2).

On February 1, 2018, Brett Wyrick, D.O., denied claimant's application for airman medical certification due to his toxic encephalopathy requiring the use of disqualifying medication (Ex. 85). Therefore, it was unlawful for claimant to fly a plane. *Id.*

On February 14, 2018, claimant alleged that the employer did not accept a diagnosis or condition and asked the employer to accept his toxic encephalopathy as a compensable component of the January 21, 2017 industrial injury (Ex. 111A).

On February 26, 2018, Matthew Bentz, M.D., interpreted a PET scan of claimant's brain, stating, "Mildly and symmetrically decreased uptake within the posterior fossa is of uncertain etiology. A case report of organic tin poisoning (Korean Journal of Occupational and Environmental Medicine, 21 (2009), pp. 289-292) leading to similar findings raises the possibility of toxic encephalopathy." (Ex. 88).

On March 2, 2018, claimant was examined by the insurer-arranged medical examiner and Clinical Neuropsychologist Tracy Kreiling, Psy.D., who diagnosed a major neurocognitive disorder due to toxic inhalation (Ex. 86, pages 24-25). During that examination, claimant had a slight resting tremor, headache, nausea, confusion, and sensitivity to light (Ex. 86-23). On standardized neuropsychological testing, claimant demonstrated significant cognitive decline in sustained attention and aspects of visual memory. *Id.* Dr. Kreiling did not believe that claimant's cognitive deficits were better explained by a mental disorder (Ex. 86-24). Instead, Dr. Kreiling believed that claimant's symptoms, neuropsychological measures, and the stability of his performance from repeated neuropsychological evaluations over time were caused by his toxic exposure. *Id.* Dr. Kreiling found no evidence of malingering or symptom magnification during the evaluation (Ex. 86, pages 26-27). If anything, he felt that claimant was possibly underreporting his symptoms. *Id.* Dr. Schock's earlier

neurocognitive tests were consistent with Dr. Kreiling's test findings and conclusions (Ex. 130, pages 24-25).

On March 9, 2018, Toxicologist Mohamed Abou-Donia, Ph.D.,¹⁹ performed an autoantibodies test in serum involving a sample of claimant's blood to determine if he had any nerve damage (Ex. 90-1). The specially developed test sought to establish the level of serum-derived autoantibodies circulating in the blood which could indicate nerve damage. *Id.* Based on the test results, Dr. Abou-Donia believed that claimant's elevated levels of serum autoantibodies against certain neuronal proteins were highly significant (Ex. 90, pages 1-2). He also concluded that the presence of circulating autoantibodies against neuronal and glial proteins at higher levels confirmed claimant's chemical-induced nervous system injury and resulting neurological deficits (Ex. 90-5).

On March 9, 2018, a SPECT scan of claimant's brain was interpreted as normal (Ex. 91).

On March 20, 2018, the IME physician, Dr. Burton, believed that TOCP was the only component of TCP that represented a potential toxic hazard, but the concentration of TOCP was so low in jet engine oil that it was not possible to suffer any adverse effects without first experiencing overwhelming toxicity from inhalation and/or ingestion of the oil (Ex. 92-2). Dr. Burton also believed that "aerotoxic syndrome" was a myth and any symptoms experienced by airplane crew members were due to underlying medical conditions or factors inherently related to flying, such as low humidity, altitude, or personal hygiene (Ex. 92-3). However, he continued to agree with Dr. Craven, who concluded that claimant's exposure to toxic fumes initially resulted in a myriad of chemically-induced symptoms (Ex. 92-4).

On April 10, 2018, claimant returned to Dr. Schloesser with daily headaches, photophobia, phonophobia, low-level nausea, and difficulties with memory, balance, and coordination (Ex. 94-1). Dr. Schloesser concluded that claimant's significant injury with cognitive problems, headaches, tremors, reduced energy, and peripheral neuropathy had all been documented both by examination and history as well as with objective testing including nerve conduction, a PET scan, laboratory analysis, and significant neurocognitive impairment (Ex. 94, pages 1-2; *see also* Ex. 102). Subsequently, Dr. Schloesser concluded that claimant's January 21, 2017 exposure to toxic fumes was the major contributing cause of his polyneuropathy, headaches,

¹⁹ Dr. Abou-Donia was a Professor of Pharmacology and Cancer Biology and Professor of Neurobiology at Duke University Medical Center (Ex. 90-1).

and cognitive problems (Ex. 116A-1). That opinion was based on a number of factors, including: (1) Organophosphate toxicity was well established in detail in the medical literature, (2) Dr. Schloesser treated claimant before the industrial injury and there was a significant clinical change following the fume event, (3) claimant had no pre-existing problems immediately preceding the fume event, (4) the nerve conduction study he performed, which showed evidence of peripheral neuropathy, was not flawed, (5) there was other objective evidence of physical injury including a PET scan and neuropsychological testing, (6) claimant was an affable and happy pilot without any history suggesting a tendency toward malingering or secondary gain prior to the fume event, and (7) claimant had no injuries after the fume event which would explain his persistent symptoms (Ex. 116, pages 1-2).

On June 19, 2018, claimant sought treatment from The Headache Center Director and Neurologist, Robert Kaniecki, M.D.,²⁰ who diagnosed chronic post-traumatic headaches (Ex. 97-3). Dr. Kaniecki stated,

*** [Claimant's] headaches may be classified as either "secondary to toxin exposure" or "post-traumatic" in nature ***. The injury most likely involved aerosolized tricresyl phosphate, an organophosphate compound, with subsequent neurotoxicity. A toxic encephalopathy, including cognitive and headache complaints, as well as neuropathy, are among the most common neurologic complications from exposure to these compounds. I now have experience with over two dozen cases of headaches and neurological compromise from similar "fume events" experienced with a number of airlines, more commonly to date with flight attendants.²¹

Id. Given the series of toxic chemical exposures claimant experienced on January 21, 2017, and his subsequent medical history, Dr. Kaniecki believed that claimant

²⁰ Dr. Kaniecki was a very highly regarded neurologist in the medical community (Judith Anderson testimony).

²¹ During his September 23, 2019 deposition, Dr. Kaniecki was asked how many patients he was treating for symptoms following airplane fume event and where those patients had come from (Ex. 138A, pages 20-21). He responded:

A variety of sources and a variety of locations and airlines. Believe me, this is not a job I have sought out. The – it's been coast to coast. I have those from Alaska Air, from the west coast and JetBlue, and on the east coast, my first was Southwest, from Chicago, was just last week.

Now I have over five dozen flight attendants and half a dozen pilots from a variety of sources, and sometimes it's a referring physician who knows – sent for headache management, post-traumatic headache.

Id. Dr. Kaniecki added, "These flight attendants and pilots are exposed to a lot of different chemicals." (Ex. 138A-22).

had been more severely affected than most by the fume event. *Id.* During his September 23, 2019 deposition, Dr. Kaniecki confirmed his opinion that claimant's toxic fume event was the major contributing cause of his toxic encephalopathy and post-traumatic headaches (Ex. 138A, pages 37 & 45).

On August 16, 2018, claimant's request for payment of 24 acupuncture visits to treat his headaches was transferred by the WCD to the WCB Hearings Division to determine whether a causal relation existed between the compensable injury and those requested medical services (Ex. 98A).

On October 16, 2018, Dr. Ugalde diagnosed (1) toxic encephalopathy, (2) cognitive deficits, (3) chronic headaches, (4) dyspnea, (5) migraine, (6) paradoxical vocal cord movement on respiration, and (7) vestibular disequilibrium (Ex. 101-7). In addition, she believed that claimant's persistent cognitive, neurologic and pulmonary complaints were medically stationary with residual impairment and he was precluded from returning to his regular job as an airline pilot (Ex. 101, pages 1 & 7). At that time, claimant continued to have persistent daily headaches, frequent vertigo resulting in falls several times a week, visual problems that included seeing double and difficulty focusing to read, breathing difficulties which limited his physical activity, cognitive deficits, and significant fatigue. *Id.* During a typical day, claimant did exercises for physical strengthening, cognition, speech, vision, and vestibular problems (Ex. 101-1). He also took about a dozen medications to help alleviate those problems while continuing with speech therapy, physical therapy, occupational therapy, and mental health counseling (Ex. 101, pages 2-3). Dr. Ugalde concluded that claimant's head injury resulted in Rancho Los Amigos Level VIII impairment with class II head and brain impairment (Ex. 101-7). Subsequently, Dr. Ugalde concluded that the January 21, 2017 fume event was the major contributing cause of claimant's toxic encephalopathy (Ex. 118, pages 2-4). That expert medical opinion was based on a number of factors including (1) the mechanism of injury was consistent with the diagnosis based, in part, on the medical literature and reports from various experts, (2) exposure to TCP and other chemicals in jet fuel oil can cause neurological problems and have toxic effects, (3) a strong temporal relationship, and (4) experts in toxic exposure agreed that toxic fume exposures caused cognitive impairment (Ex. 118, pages 4-6).

On February 6, 2019, claimant's compensable industrial injury claim for acute toxic inhalation and acute chemical inhalation was closed without a permanent disability award on the grounds that his impairment was not due to the accepted conditions of "acute chemical inhalation" and "acute toxic inhalation." (Ex. 108).

On February 8, 2019, the employer denied compensability of claimant's current condition on the grounds that his accepted "acute chemical inhalation" was no longer materially contributing to any disability or need for treatment (Ex. 110). Specifically, the "current condition" denial stated, in relevant part,

Your claim was previously accepted for disabling acute chemical inhalation. A Notice of Closure on your disabling claim issued February 6, 2019. A preponderance of medical evidence indicates your accepted acute chemical inhalation resolved and is no longer materially contributing to any disability or need for medical treatment. We therefore deny that your current condition, disability, or need for medical treatment are compensably related to the accepted acute chemical inhalation.

Id. That "current condition" denial did not assert that claimant's other accepted claim for "acute toxic inhalation" had resolved and was no longer a material contributing cause of any disability or need for medical treatment. *Id.*

On February 20, 2019, the employer denied compensability of claimant's toxic encephalopathy on the grounds that the condition did not exist and, if it did, the industrial injury was not a material contributing cause of that condition (Ex. 113). On March 21, 2019, claimant asked the employer to accept new or omitted medical condition claims including (1) mild neural cognitive disorder, (2) polyneuropathy, (3) convergence insufficiency and (4) saccadic eye movement deficiency (Ex. 114B). On April 2, 2019, the employer denied compensability of all four of those new or omitted medical conditions (Ex. 116). Subsequently, claimant withdrew his claim for peripheral neuropathy on the grounds that it was an early symptom of toxic encephalopathy and did not need to be accepted as a separate condition (Ex. 131B).

On April 22, 2019, claimant was examined by Robert Harrison, M.D., one of the foremost toxicology experts in assessing airline toxic fume events (Ex. 121).²² Dr. Harrison diagnosed (1) toxic effect of fumes, (2) toxic encephalopathy, (3) vascular headache, and (4) polyneuropathy due to toxic exposure (Ex. 121-1). Dr. Harrison believed that claimant's chemical exposure at work on January 21, 2017, caused chronic neurological damage including neuropathy, headache, tremor, slight gait disturbance and cognitive impairment. *Id.* Benjamin Schanker, M.D., who worked with Dr. Harrison, also believed that claimant's exposure to toxic fumes on

²² Dr. Kaniecki testified that two of the foremost toxicology experts that his clinic relied on in assessing fume event conditions were Dr. Abou-Donia on the east coast and Dr. Harrison on the west coast (Ex. 138A-45).

January 21 2017, most likely aerosolized tricresyl phosphate, caused his neurologic symptoms (Ex. 121-6).

On May 9, 2019, an Order on Reconsideration rescinded the February 6, 2019 Notice of Closure on the grounds that there were insufficient findings to establish the extent of claimant's permanent disability resulting from the accepted industrial injury claim (Ex. 123).

On May 24, 2019, Dr. Ugalde was asked to make permanent impairment findings regarding claimant's accepted acute chemical inhalation and acute toxic inhalation industrial injury claim (Ex. 124). She deferred to Dr. Scott regarding claimant's visual impairment and to Dr. Verheyden regarding claimant's respiratory impairment (Ex. 124, pages 1-2). However, regarding claimant's permanent neurological impairment, including issues with speech, swallowing and neuropsychological issues, Dr. Ugalde concluded that claimant was at a "Rancho IX," in that claimant had difficulties with multitasking and novel situation-problem solving. *Id.* Dr. Ugalde also believed that claimant had neurological dysfunction with headaches, migraines, vertigo and tinnitus. *Id.*

On May 31, 2019, Dr. Abou-Donia performed a file review on claimant's behalf and concluded that claimant met the criteria for major neurocognitive disorder due to toxic inhalation caused by many years of low level exposure to toxins which caused small increments of nervous system injury (Ex. 126, pages 3 & 14). While Dr. Abou-Donia believed that some of claimant's symptoms resolved because his peripheral nerves regenerated, his central nervous system injury did not regenerate (Ex. 126, pages 5-6). Dr. Abou-Donia also disagreed with Dr. Burton's opinions regarding causation on the grounds that he did not consider claimant's low level exposure to chemical fumes before the industrial injury (Ex. 126-26). Along with Dr. Harrison, Dr. Abou-Donia was one of the foremost toxicology experts in assessing airline toxic fume events (Ex. 138A-45).

On July 25, 2019, claimant was examined by the insurer-arranged medical examiner and Neurologist Lynne Bell, M.D., Ph.D., who incorrectly believed that claimant never exhibited any objective physical examination abnormalities following the January 21, 2017 fume event (Ex. 134, pages 35-36). Dr. Bell also incorrectly believed that claimant's clinical presentation in the medical records was "consistent with a 'functional neurological presentation,' *i.e.*, a psychogenic condition in which there is no objective pathology at the level of the central or peripheral nervous system." (Ex. 134-36). Her assessment included claimant's mild

neural cognitive disorder, which she also erroneously believed was functional in nature (Ex. 134-39).

On July 30, 2019, a Notice of Closure awarded claimant no permanent disability benefits for his accepted “acute toxic inhalation” and “acute chemical inhalation” claim (Ex. 136). On September 12, 2019, an Order on Reconsideration rescinded that Notice of Closure (Ex. 137A).

On October 21, 2019, Susan Michaelis, Ph.D.,²³ MSc., ATPL, performed a file review on claimant’s behalf and authored a report in which she concluded that claimant’s symptoms were consistent with exposure to oil contaminants according to her own expert opinion and the medical literature on the subject (Ex. 141-12). At the hearing, Dr. Michaelis also testified that claimant’s diagnoses of toxic encephalopathy, neurocognitive disorder, and visual dysfunction were consistent with other crew members who experienced symptoms following airline fume events. She concluded that claimant’s toxic fume inhalation very probably caused his diagnosed conditions (Dr. Michaelis testimony).

On October 21, 2019, Toxicologist Richard Pleus, Ph.D., M.S., authored a lengthy file review report for the employer, incorrectly concluding that there was no evidence claimant was exposed to toxic chemical fumes on January 21, 2017, and his work environment was not a material cause of any physical conditions (Ex. 141-7). His report focused primarily on an evaluation of TCP and its isomers (Ex. 141-6). In support of his opinion, Dr. Pleus relied on (1) the hen TCP ingestion studies in concluding that claimant was not exposed to a sufficient dose of TCP to cause physical problems (Ex. 141, pages 6 & 32-34), (2) his understanding that no studies supported the existence of short-term or long-term neurological health effects from fume events and no studies suggested that exposure to oil compounds other than TCP caused symptoms (Ex. 141, pages 12 & 48), (3) TCP did not cause cognitive effects or coughing or breathing problems (Ex. 141-6), (4) claimant’s nonspecific symptoms did not correspond with objective findings (Ex. 141, pages 6, 49 & 59), and (5) OSHA standards for TCP were much higher than the dose that would cause physical injury (Ex. 141, pages 20 & 42). Dr. Pleus also did not agree with the opinions of Drs. Abou-Donia or Harrison that the January 21, 2017 fume event was a material cause of claimant’s neurocognitive conditions primarily because neither

²³ Dr. Michaelis worked as an airline pilot from 1986 to 1997 (Susan Michaelis testimony). She was medically retired when she developed chronic symptoms after repeatedly inhaling oil fumes from aircraft. *Id.* Those symptoms included headaches, voice problems, throat irritation, nausea, concentration problems, fatigue, and sensitivity to chemicals. *Id.* All of those symptoms resolved between flights. *Id.* Subsequently, she obtained a Masters of Science and became a qualified air accident investigator. *Id.* During that period of study, she focused on how oil leaked from jet engines. *Id.* In 2010, Dr. Michaelis obtained her Ph.D. in safety science in the workplace regarding aircraft contamination. *Id.*

doctor identified a dose of TCP that was sufficient to cause claimant's physical injuries (Ex. 141, pages 50-57 & 59-64). In fact, Dr. Pleus could not conclude that claimant was exposed to any toxic fumes at work (Ex. 141, pages 51 & 62).

At the hearing, Industrial Hygienist Judith Anderson testified on claimant's behalf. She worked in the Air Safety, Health, and Security Department of the Association of Flight Attendants representing cabin crews and had a specialty in chemical exposure hazards with a focus on exposure to engine oil fumes (Judith Anderson testimony; Ex. D). During her twenty years of work with that organization, Ms. Anderson had received thousands of calls from cabin crew members complaining of symptoms from airplane fume events with hundreds requiring medical treatment (Judith Anderson testimony). Typically, crew members complained of acute symptoms during the fume exposure that included headaches, light-headedness, confusion, stomach cramps, and respiratory issues including difficulty breathing and coughing. *Id.* Post-exposure symptoms occurring one or two weeks later involved neurological symptoms such as difficulties with memory, speech, multi-tasking, cognition and balance, along with tingling in the hands and feet, respiratory symptoms, and visual disturbances including problems with tracking, photophobia and tunnel vision. *Id.* Ms. Anderson believed that claimant's confirmed fume event was consistent with his physical impairment afterward. Her expert scientific opinion was based on her knowledge of the neurotoxic effects of pyrolyzed jet engine oil along with the expert opinions of Drs. Abou-Donia, Kaniecki, and Harrison. *Id.*

CONCLUSIONS OF LAW AND OPINIONS

1. Scope of acceptance (Exhibits 35, 107 & 113)

From the outset of this claim, the employer's acceptances of claimant's "acute chemical inhalation" and "acute toxic inhalation" caused confusion regarding the actual scope of its acceptance. Eventually, that procedural uncertainty led claimant's counsel to file a new or omitted medical condition claim on February 14, 2019, which stated, in relevant part,

In this regard, I note that you initially accepted this claim for "disabling acute chemical inhalation" and in your acceptance at closure, you describe the acceptance of claimant's condition as "acute toxic inhalation". In both instances, this would describe what happened to the claimant, but neither describes a diagnosis or a condition. For this reason, we are making this expansion

request, and you can anticipate further such requests after we determine what conditions have developed from this toxic exposure.

(Ex. 111A-1). In response to that new or omitted condition claim, the employer denied compensability of claimant's previously diagnosed conditions of toxic encephalopathy, mild neural cognitive disorder, polyneuropathy, convergence insufficiency, and saccadic eye movement deficiency, as well as his current condition (Exs. 110, 113, & 116).

On April 29, 2020, after all of the witnesses had testified at the hearing and the documentary evidence was complete, I sent a letter to both attorneys outlining all of the exhibits the Hearings Division had received into evidence and advising the parties,

Please let me know if I missed anything in the evidentiary record. Otherwise, I look forward to your closing arguments. Another issue which I hope both of you will address (along with the compensability, causal relation to medical services, penalty, attorney fee, and costs issues of course) is the scope of the employer's acceptance and the practical meaning of the accepted conditions of "acute toxic inhalation" and "acute chemical inhalation" (Ex. 113). I'm unsure what conditions and/or symptoms were actually encompassed by the accepted conditions. If that requires Mr. Lasken to submit a supplemental opening argument please do so.

(April 29, 2020 e-mail to Mr. Lasken and Mr. Fisher).²⁴ I made this request because I could not determine whether the current condition denial or other compensability denials should be set aside or approved without knowing what the employer had already accepted. Also, I could not reasonably determine whether there was sufficient information to close the claim without knowing what conditions were encompassed by the accepted claim. After reading the record, researching the issues, and listening to the arguments of the parties, I continue to believe that understanding

²⁴ Correspondence between the ALJ and the parties was not normally done via e-mail. During this period, however, Governor Brown had declared a state of emergency due to the COVID-19 pandemic and many lawyers and their staff were working remotely from home in an attempt to "flatten the curve" of the infection rate. Thus, e-mail communications, instead of standard letters, were deemed a better way to reach all of the parties in a timely manner.

the scope of the employer's acceptance is a necessary prerequisite and threshold issue to determining the compensability and premature claim closure issues.²⁵

The employer did not discuss the scope of acceptance issue in its 92-page July 2, 2020 Employer's Closing Argument or the 37-page Addendum A to that argument. Instead, the employer confined its arguments to the issues of compensability, attorney fees, costs, penalties, medical services, and premature claim closure. In his written reply, claimant's counsel addressed the absence of the employer's arguments regarding the scope of acceptance issue, stating,

During the exchange of communication concerning the briefing schedule this Court asked for some clarification as to what condition was actually accepted in the two Notice of Acceptances issued by the employer.

Employer chose to not address this concern in their Respondent's Argument, perhaps because there is no answer they can offer. Neither the Initial Notice of Acceptance nor the Notice of Acceptance at the time of closure purports to accept any CONDITION. Acute chemical inhalation is not a condition. That describes what happened. Similarly, acute toxic inhalation doesn't represent a condition either. By the time that NOA at closure was issued, there was ample evidence as to the conditions being treated.

(July 14, 2020 Claimant's Reply Closing Argument, page 8, capital letters in original). In addition, claimant notes that the problems with the employer's acceptances could not be explained away by asserting that there was very little medical evidence when they issued since the Notice of Acceptance at Closure (which expanded the initial acceptance to include "acute toxic inhalation") occurred two years after the industrial injury when "the medical record was replete with specific diagnoses and conditions which the employer could have chosen to accept." *Id.* Claimant argues, "Instead, they issued an acceptance which is really a meaningless piece of paper. This Judge has no idea what they accepted and neither do I, because they didn't accept any condition." (July 14, 2020 Claimant's Reply Closing Argument, pages 8-9).

²⁵ Claimant's theory of compensability is the starting point, but the entire record must be examined to determine the appropriate standard of review. *Glenn D. Kramer*, 64 Van Natta 2245 (2012) citing *Daniel Suing*, 56 Van Natta 2600, 2601 (2004) (citing *Dibrito v. SAIF*, 319 Or 244, 248 (1994)); *Daniel S. Field*, 47 Van Natta 1457, 1458 (1995) ("it is our obligation as a fact finder to apply the appropriate legal standards to determine the compensability of a worker's claim.")

As previously stated, the employer initially accepted claimant's workers' compensation claim for "acute chemical inhalation" as a disabling industrial injury on February 23, 2017 (Ex. 35). On February 6, 2019, the Updated Notice of Acceptance at Closure modified the earlier Notice of Acceptance when it stated, "Your claim is classified as disabling and is accepted for acute toxic inhalation." (Ex. 107). On February 20, 2019, the employer explained the discrepancy between the two acceptances, stating, "Your claim was previously accepted for disabling acute chemical inhalation. The Updated Notice of Acceptance at Closure also described this as 'acute toxic inhalation.' Medical records used these terms interchangeably. However, to the extent these are separate conditions, we consider them both to have been accepted." (Ex. 113-1). Since both acceptances involved terms typically used to describe a mechanism of injury and not a distinct medical condition involving the body, it is important to determine what exactly the employer accepted in terms of specific conditions, symptoms and treatment before proceeding to the compensability issues.

a. Accepting a mechanism of injury

ORS 656.262(6)(b)(A) provides that a Notice of Acceptance shall, among other things, "[s]pecify what conditions are compensable." It is imperative that a condition be accepted because there is no entitlement to medical services in the absence of a compensable injury as defined by ORS 656.005(7)(a). An employer is not responsible for medical services which are not caused in material part by the compensable injury. ORS 656.245(1)(a); *See also Stuart P. Luxenberg*, 65 Van Natta 65 (2013). Acceptance of a claim encompasses only those *conditions* specifically or officially accepted in writing. *Johnson v. Spectra Physics*, 303 Or 49 (1987). Whether an acceptance occurs is a question of fact. *SAIF v. Tull*, 113 Or App 449 (1992). It need not meet any particular degree of specificity. *Lawrence H. Eberly*, 42 Van Natta 1965, 1966 (1990); *see also Danny Ward*, 45 Van Natta 99 (1993). Acceptance is an act through which the insurer acknowledges responsibility for the claim and obligates itself to provide the benefits due under the law. *Gene C. Dalton*, 43 Van Natta 1191 (1991).

A carrier is only required to accept a "condition," not a mechanism of injury. *See Royal S. Buell*, 50 Van Natta 702, *aff'd without opinion*, 157 Or App 723 (1998).²⁶ A "condition" is defined as "the physical status of the body as a whole ***

²⁶ In *Buell, supra*, claimant contended that "crush injury" was a diagnosis or condition that the insurer should be required to accept. In support of his argument, claimant cited several cases where "crush injury" was determined to be an accepted "condition." *See Steve W. Hooten*, 49 Van Natta 1370 (1997); *Vickie L. Wing*, 49 Van Natta 1468 (1997); *Chris W. Poe*, 49 Van Natta 1367 (1997); OPINION AND ORDER, Page 31 of 91

or of one of its parts.” *Young v. Hermiston Good Samaritan*, 223 Or App 99, 105 (2008). The distinction between a “condition” and a “mechanism of injury” is an issue of fact that is determined on a case-by-case basis. The medical record must be examined to distinguish between a “condition” and a “mechanism of injury.” *Buell*, 50 Van Natta at 702 (“crush injury” described a mechanism of injury, not a distinct medical condition); *Justin T. Jones*, 68 Van Natta 754, 756 (2016) (where doctor used the term “crush injury” to describe the history of the injury, but listed finger laceration and contusion as the diagnoses, the Board did not consider “crush injury” to be a distinct medical condition); *Manu R. Kamanda*, 65 Van Natta 1571 (2013) (“bite,” as opposed to accepted contusion, was not found to be a “condition” because it did not constitute “the physical status of the body as a whole *** or of one of its parts.”); *but see Allen Bakken*, 70 Van Natta 206, 208 (2018) (doctor’s description of elevated levels of benzene and xylene, which she termed “solvent toxicity,” constituted a condition, *i.e.*, a physical status of claimant’s body, where there was no medical evidence that “solvent toxicity” itself was not a “condition.”); *Jeremy Schaffer*, 65 Van Natta 2191, 2193-94 (2013) (specific medical evidence established that a “crush injury” was not only a mechanism of injury, but was also an appropriate medical diagnosis describing the claimant’s specific condition); *Jeffrey S. Lyski*, 54 Van Natta 1875, 1876-77 (2002) (“condition” was established where “electrocution” was diagnosed and a trauma specialist expressly opined that the diagnosis was a medical condition).²⁷

In this case, claimant’s medical providers began diagnosing symptoms or distinct medical conditions along with a description of the mechanism of injury almost immediately after the toxic chemical fume event. On January 25, 2017, claimant sought treatment from Physician Assistant Sean Suttle, PAC, whose assessment was “**exposure to chemical inhalation**” and cough with a “**chief complaint [of] toxic inhalation.**” (Ex. 26-1; bold added). Mr. Suttle explained, “[Patient] exposed to fumes while working on airplane at work *** - immediately had cough, tremors, headache, congestion, mild [shortness of breath] and some confusion ***.” *Id.* On February 2, 2017, claimant was examined by Brian Rapacz, M.D., whose impression was “acute altered mental status” and “toxic exposure.” (Ex. 29-3). Dr. Rapacz wrote, “The patient states that he was exposed to toxic fumes

Bessie B. Mitts, 49 Van Natta 799 (1997); *Michael T. Alioth*, 49 Van Natta 688 (1997); and *Richard L. Henley*, 49 Van Natta 621 (1997). While the cases the claimant cited did concern crush injuries, the Board concluded in *Buell, supra*, that none of them contained a determination on the merits that a “crush injury” was a distinct medical condition or diagnosis. Even if they had, the Board emphasized that its decision in *Buell* was based on the medical evidence in that case, citing *See Ellen G. Johnson*, 49 Van Natta 1360, 1363 n. 1 (1997).

²⁷ The Board in *Lyski, supra* at 1877, stated, “In sum, we find that several doctors have described ‘electrocution’ or ‘electrical injury’ as a medical diagnosis. Additionally, a trauma specialist has expressly opined that the diagnosis of ‘electrocution’ is a medical condition. Based on the record as a whole, we conclude that ‘electrocution’ is a condition.”

*** [and] complains of a persistent low grade headache, upper extremity tremors, confusion, and aphasia ***.” (Ex. 29-1). On February 10, 2017, Cory Siebe, M.D., stated, “Basically this is a 52-year-old male who presents today with nonspecific neurologic symptoms after **exposure to burning jet fuel, toxic smoke inhalation** (Ex. 30-4; bold added). On February 14, 2017, claimant was examined by Physician Assistant Korena Larsen Farris, PAC, whose assessment included (1) **exposure to chemical inhalation**, (2) tremor, (3) aphasia, and (4) cognitive deficits (Ex. 31-3; bold added). She also stated that his “symptoms are most likely related to **exposure to tricresyl phosphate** from jet engine oil.” *Id.* (bold added). On February 21, 2017, claimant saw David Schloesser, M.D., whose impression was migraine headache and polyneuropathy “due to other toxic agents.” (Exs. 33 & 34; *see also* Ex. 42). Dr. Schloesser stated, “[Claimant] is a 52-year-old gentleman with **exposure to tricresyl phosphate** on 01/21/17.” *Id.* (bold added). On March 3, 2017, claimant began treating with Dr. Ugalde, whose assessment included (1) toxic encephalopathy, (2) toxic peripheral neuropathy, (3) **exposure to chemical inhalation**, (4) tremor, (5) wheezing on expiration, (6) vision impairment, (7) vestibular disequilibrium, and (8) new daily persistent headache (Ex. 39-4; bold added). She explained, “[Claimant] was exposed for a minimum of 45-50 minutes to toxic fumes [which] subsequently caused confusion, memory loss, massive headaches, coughing, teary eyes, tremors, concentration problems, difficulty processing information, difficulty finding words and expressing himself, extreme fatigue, slurring words, stuttering and an internal shaking sensation with some hand tremors.” (Ex. 39-1). On March 17, 2017, Dr. Ugalde stated, “Incident occurred on 01/21/17 **from toxic inhalation** OJI with persistent cognitive and pulmonary complaints.” (Ex. 41-1; bold added). At that time, Dr. Ugalde added cognitive deficits and aphasia to her assessment of claimant’s conditions (Ex. 41-3). On April 14, 2017, Dr. Ugalde stated, “[Claimant] has growing concerns about the **injuries related to his chemical inhalation.**” (Ex. 46-1; bold added). On April 3, 2017, Dr. Schock diagnosed a **mild neurocognitive disorder related to acute toxic inhalation** (Ex. 43-8; bold added). On April 10, 2017, claimant was examined by Dr. Verheyden, M.D., whose assessment included “[t]oxic effect of fumes.” (Ex. 44-2). On May 26, 2017, Optometrist Dr. Scott diagnosed the visual condition of mild convergence insufficiency and subsequently diagnosed saccadic eye movement deficiency, which she believed were caused by toxic encephalopathy (Exs. 51 & 117-2).

On February 23, 2017, the employer accepted “acute chemical inhalation” and, on February 6, 2019, amended that Notice of Acceptance to include “acute toxic inhalation.” (Exs. 35, 107 & 113). Before the initial acceptance was modified, the IME physicians, Drs. Burton, Bell, and Radecki all concluded that claimant did not

have a work-related distinct medical condition (Exs. 58, 65, 69 & 92). On the other hand, on December 20, 2017, Dr. Bass denied claimant's request for an Airman Medical Certificate that would have enabled him to pilot an airplane because of the following diagnoses: (1) Migraines, (2) aphasia, (3) toxic encephalopathy, (4) tremor, (5) cognitive deficits, (6) peripheral nervous system and central nervous system damage, (7) vestibular disequilibrium, (8) vision impairment from tracking issues and depth perception due to neurological damage, and (9) variable extrathoracic obstruction and paradoxical vocal cord movement (Ex. 79-3). Dr. Bass also stated,

I have examined Captain Myers at 6 month intervals over at least ten years & during which time there was no doubt as to his good health and fitness to perform his duties as an airline captain. **His tragic incident related to toxic fume inhalation** in January, 2017 has rendered him incapable of functioning in this capacity.

(Ex. 79-4; bold added). On January 25, 2018, Dr. Craven provided a Providence MCO Medical Treatment Review and concluded that claimant's symptoms in the first few days, but not his subsequent neurological and cognitive problems, were caused by the workplace airborne exposure (Ex. 84B-2). In addition, on March 2, 2018, the IME physician and Clinical Neuropsychologist, Dr. Kreiling, diagnosed a **major neurocognitive disorder due to toxic inhalation** (Ex. 86-24; bold added). On March 9, 2018, Toxicologist Dr. Abou-Donia diagnosed a chemical-induced nervous system injury (Ex. 90-5). On April 10, 2018, Dr. Schloesser diagnosed a significant injury with cognitive problems, headaches, tremors, reduced energy, and peripheral neuropathy from organophosphate toxicity (Ex. 94-1; *see also* Ex. 102). On June 19, 2018, Neurologist Dr. Kaniecki diagnosed chronic post-traumatic headaches secondary to toxic exposure, concluding, "A toxic encephalopathy, including cognitive and headache complaints, as well as neuropathy, are among the most common neurologic complications from exposure to these compounds." (Ex. 97-3). On October 16, 2018, Dr. Ugalde continued to diagnose (1) toxic encephalopathy, (2) cognitive deficits, (3) chronic headaches, (4) dyspnea, (5) migraine, (6) paradoxical vocal cord movement on respiration, and (7) vestibular disequilibrium (Ex. 101-7). She concluded, "Ongoing cognitive deficits related to on-the-job exposure to toxins resulting in a toxic encephalopathy. Ongoing issues are causally related to *** this exposure." *Id.*

Before the initial Notice of Acceptance was amended on February 6, 2019, the following medical and scientific experts did not offer their opinions regarding the meaning of the accepted conditions or what conditions claimant was suffering

from as a result of the toxic fume event: Dr. Harrison (Ex. 121, 132, 139 & 149), Judith Anderson (Judith Anderson testimony; Ex. A), Dr. Pleus (Dr. Pleus testimony; Ex. 141), and Dr. Michaelis (Dr. Michaelis testimony; Ex. 142). Therefore, their opinions and any other expert medical evidence generated after February 6, 2019 did not constitute “contemporaneous medical evidence” and were not relevant for purposes of determining the scope of the employer’s acceptance other than to understand the meaning of the term “toxic.”

The employer accepted claimant’s “acute chemical inhalation” as a disabling industrial injury shortly after he first sought medical treatment on January 25, 2017 (Ex. 26-1). At that time, Physician Assistant Suttle’s assessment was that claimant had “[e]xposure to chemical inhalation” with a chief complaint of “[t]oxic inhalation.” *Id.* He believed that claimant was “exposed to fumes” and immediately had a variety of symptoms (Ex. 26-1). Mr. Suttle did not state that either “chemical inhalation” or “toxic inhalation” constituted a distinct medical condition. In fact, at no time before February 6, 2019, when the employer added “acute toxic inhalation” to the claim, did any of the medical experts conclude that claimant’s “acute chemical inhalation” and “acute toxic inhalation” were distinct medical conditions. In other words, none of the doctors specifically stated that either of the accepted conditions represented a “physical status of the body as a whole *** or of one of its parts.” *See Young, supra.* This was especially true for the IME physicians, Drs. Radecki, Bell, and Burton, who all believed that claimant’s exposure to toxic fumes did not result in any physical conditions. While the medical providers at times listed “toxic inhalation” or “chemical inhalation” under their assessments, they were diagnosing and treating symptoms or conditions related to or caused by that mechanism of injury. When they included those terms in their assessments, they were describing the toxic chemical fume event as the mechanism of injury that resulted in the symptoms and distinct medical conditions they were treating. Those phrases included: “Patient presents with toxic inhalation” (Ex. 26-1), “Exposure to chemical inhalation” (Exs. 31-3, 39-4 & 41-3), “neurologic symptoms after exposure to burning jet fuel, toxic smoke inhalation,” (Ex. 30-4), “Incident occurred on 01/21/17 from toxic inhalation OJI with persistent cognitive and pulmonary complaints” (Ex. 41-1), “Mild Neurocognitive Disorder related to acute toxic inhalation,” (Ex. 43-8), “injuries related to his chemical inhalation” (Ex. 46-1), “[h]is tragic incident related to toxic fume inhalation in January, 2017 has rendered him incapable of functioning [as an airline captain]” (Ex. 79-4), and “[claimant] meets criteria for major neurocognitive disorder due to toxic inhalation.” (Ex. 86-24).

An employer would not be accepting a distinct medical condition if it accepted “walking on broken glass” (instead of “lacerated feet”), “touching a hot flame”

(instead of “burned hand”), “listening to loud noise” (instead of “hearing loss”), or “staring into the sun” (instead of “blindness”). All of those actions might result in the physical injuries described in parentheses, but they do not describe the physical status of the body or one of its parts. “Status” is defined as “state or condition with respect to circumstances.” *Merriam-Webster.com*. “Inhalation” does not describe the status, state, or condition of the body; it is defined as “the act or an instance of inhaling.” *Id.* “Inhaling” is defined as “to draw in by breathing.” *Id.* Thus, the employer’s acceptance of toxic or chemical “inhalation” described the fume event, or mechanism of injury, that occurred on January 21, 2017, when claimant breathed toxic chemical fumes. “Inhalation,” or the act of breathing, did not describe “the physical status of the body as a whole *** or of one of its parts.” *See Young, supra*. The same conclusions were drawn by the Board in *Buell*, 50 Van Natta at 702, and *Jones*, 68 Van Natta at 756, where the evidence established that a “crush injury” described a mechanism of injury, not a distinct medical condition, as well as in *Kamanda*, 65 Van Natta at 1571, where the evidence established that a “bite” was not a distinct medical condition either.

The medical evidence in the present case was unlike the facts in *Schaffer*, 65 Van Natta at 2193-94, where specific medical evidence established that a “crush injury” was an appropriate medical diagnosis describing the claimant’s specific condition, and unlike the facts in *Lyski*, 54 Van Natta at 1876-77, where “electrocution” was diagnosed and expressly found to be a medical condition. Here, in contrast, the evidence established that “inhalation” was an action taken by claimant, not a status, state, or condition of the body, and there was no specific medical evidence establishing that “acute chemical inhalation” or “acute toxic inhalation” constituted both mechanisms of injury and diagnoses describing claimant’s specific medical conditions. Since the employer’s acceptance described the mechanism of injury, *i.e.*, the manner and circumstances surrounding claimant’s injury, and did not describe a distinct medical condition, *i.e.*, the physical or mental damage done to the body as a result of that injury, the contemporaneous medical evidence must be examined to determine what conditions were actually accepted.

The opinions of the IME physicians, Drs. Radecki, Bell, and Burton, are not relevant to this procedural question because the employer accepted a disabling industrial injury due to the toxic chemical inhalation and those IME physicians did not believe that any workplace injury occurred. ORS 656.005(7)(c) provides, “A ‘disabling compensable injury’ is an injury that entitles the worker to compensation for disability or death. An injury is not disabling if no temporary benefits are due and payable, unless there is a reasonable expectation that permanent disability will result from the injury.” Inasmuch as the IME physicians did not believe that claimant

suffered a physical injury that entitled him to compensation, their opinions regarding the scope of the accepted conditions were legally incorrect and are disregarded.

Considering the treating and examining physicians who offered their assessments and opinions before the February 6, 2019 Modified Notice of Acceptance, all of them except Dr. Craven believed that claimant's toxic chemical inhalation fume event caused his toxic encephalopathy, mild neural cognitive disorder, and vision disorders diagnosed as convergence insufficiency and saccadic eye movement deficiency. Dr. Craven did not have the advantage of examining claimant on more than one occasion, he did not have access to all of claimant's medical records, he did not have expertise in toxic fume events, toxicology, brain injuries or vision disorders, and he did not persuasively rebut the diagnoses and opinions of Drs. Ugalde, Schloesser, Schock, Kreiling, Siebe, Scott, Abou-Donia, Kaniecki, and Bass. Based on the overwhelming weight of the contemporaneous medical evidence from those physicians who recognized that the fume event resulted in a variety of physical injuries (which was consistent with the employer's acceptance of a disabling industrial injury), I conclude that the scope of the employer's acceptance included the four distinct medical conditions of toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency. Assuming that the employer accepted a disabling compensable injury, there was no persuasive or relevant contrary contemporaneous medical evidence.

Once an employer accepts a condition, that acceptance can be properly withdrawn only under the narrowest of circumstances which do not exist here. ORS 656.262(6)(a) provides, in relevant part:

Written notice of acceptance or denial of the claim shall be furnished to the claimant by the insurer or self-insured employer within 60 days after the employer has notice or knowledge of the claim. Once the claim is accepted, the insurer or self-insured employer shall not revoke acceptance except as provided in this section. The insurer or self-insured employer may revoke acceptance and issue a denial at any time when the denial is for fraud, misrepresentation or other illegal activity by the worker. If the worker requests a hearing on any revocation of acceptance and denial alleging fraud, misrepresentation or other illegal activity, the insurer or self-insured employer has the burden of proving, by a preponderance of the evidence, such fraud, misrepresentation or other illegal activity. Upon such

proof, the worker then has the burden of proving, by a preponderance of the evidence, the compensability of the claim. If the insurer or self-insured employer accepts a claim in good faith, in a case not involving fraud, misrepresentation or other illegal activity by the worker, and later obtains evidence that the claim is not compensable or evidence that the insurer or self-insured employer is not responsible for the claim, the insurer or self-insured employer may revoke the claim acceptance and issue a formal notice of claim denial, if such revocation of acceptance and denial is issued no later than two years after the date of the initial acceptance. If the worker requests a hearing on such revocation of acceptance and denial, the insurer or self-insured employer must prove, by a preponderance of the evidence, that the claim is not compensable or that the insurer or self-insured employer is not responsible for the claim. Notwithstanding any other provision of this chapter, if a denial of a previously accepted claim is set aside by an Administrative Law Judge, the Workers' Compensation Board or the court, temporary total disability benefits are payable from the date any such benefits were terminated under the denial. ***

In *Jude S. Hardesty*, 67 Van Natta 991, 992 (2015), the Board held,

Where a carrier attempts to deny a previously accepted condition, such a denial constitutes an impermissible "back-up" denial of that condition under ORS 656.262(6)(a). *Paula M. Sinclair*, 59 Van Natta 1759, 1762 (2007); see *Bauman v. SAIF*, 295 Or 788, 794 (1983) (a carrier may not accept a condition and later assert a position that contradicts the express language of its acceptance). However, there is no impermissible "back-up" denial where the denied condition was not the same as the previously accepted compensable condition. *Sinclair*, 59 Van Natta at 1762. Furthermore, a carrier may not prospectively deny its future responsibility for payment of benefits relating to a previously accepted claim. *Evanite Fiber Corp. v. Striplin*, 99 Or App 353, 357 (1989); *Barbara J. Ferguson*, 63 Van Natta 2253, 2258-59 (2011); *William J. Splichal*, 55 Van Natta 732, 733 (2003).

In this case, the employer attempted to deny the same conditions it had previously accepted as a result of accepting a mechanism of injury. Consequently, the employer's February 20, 2019 denial of toxic encephalopathy and April 2, 2019 denial of mild neural cognitive disorder, convergence insufficiency and saccadic eye movement deficiency are set aside as improper "back-up" denials of those conditions under ORS 656.262(6)(a).

b. Accepting a vague or ambiguous condition

Even if the employer's acceptances did not solely describe claimant's mechanism of injury but also constituted acceptances of medical conditions, I would still find that the acceptance of "acute toxic inhalation" encompassed the distinct medical conditions of toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency. One of the claims processing problems inherent in accepting a vague or ambiguous condition couched in terms of a mechanism of injury, as opposed to accepting a distinct medical condition involving a body part, is that the acceptance may amount to nothing more than an "empty shell" that does not reasonably apprise the parties or the medical providers of the symptoms, conditions or treatment for which the employer is responsible. On the other hand, a vague or ambiguous acceptance may amount to an acceptance of all symptoms, conditions and treatment rendered at the time.

As previously stated, the scope of an acceptance is a question of fact. *Columbia Forest Products v. Woolner*, 177 Or App 639, 643 (2001). When a carrier accepts a specific condition, it is not necessary to resort to contemporaneous medical records to determine what condition was accepted. See *Jerry W. Gabbard*, 54 Van Natta 1022 (2002); *Kim D. Wood*, 48 Van Natta 482, 484 (1996), *aff'd without opinion*, 144 Or App 496 (1996) (because there was a specific acceptance of a "left knee strain," it was not necessary to examine the contemporaneous medical evidence to determine what condition was accepted). If the specific acceptance is ambiguous or vague, however, the contemporaneous medical evidence is examined to determine what was accepted. *Gilbert v. Cavenham Forest Indus. Div.*, 179 Or App 341, 344 (2002); *Judy A. Cooper*, 62 Van Natta 884, 885 (2010) (where SAIF issued an acceptance for "back-lower" that did not identify the condition accepted, the Board looked to the contemporaneous medical evidence to determine what condition SAIF accepted); *Jack L. Kruger*, 52 Van Natta 627, 628 (2000); *Fred L. Dobbs*, 50 Van Natta 2293, 2295 (1998), *aff'd SAIF v. Dobbs*, 172 Or App 446, *adhered to on recon*, 173 Or App 599 (2001) (the Board looked to the contemporaneous medical records to determine what condition was accepted where the carrier did not identify the

specific condition accepted); see also *Mary Marrs-Johnston*, 49 Van Natta 1757 (1997); *Timothy Hasty*, 46 Van Natta 1209 (1994).

In this case, if the employer had accepted claimant's headaches, respiratory irritation, neural cognitive disorder, toxic encephalopathy, or the specific vision disorders diagnosed by Dr. Scott, there would be little doubt what treatment and benefits resulted from those accepted conditions. By accepting "acute toxic inhalation" and "acute chemical inhalation," however, the employer instead appeared to accept the fume event itself, *i.e.*, claimant inhaled toxic chemicals in the workplace. To the extent that this mechanism of injury doubled as a medical condition, it was vague. The FAA Medical Examiner, Dr. Bass, defined "toxicity" as "tissue damage in an appreciable, significant manner." (Ex. 122-11). He also believed that the term "acute chemical inhalation" could be entirely different than "acute toxic inhalation." (Ex. 122-13). The IME physician and toxicologist, Dr. Burton, agreed that those two terms were different and defined "toxicity" as the "absorbed dose of the substance." (Ex. 139A-2). Dr. Burton emphasized, "It's the dose that makes the poison." *Id.* "Toxic" is defined generally as "containing or being poisonous material especially when capable of causing death or serious debilitation." *Merriam-Webster.com*. Thus, even if the employer accepted a medical condition, the definition of "toxic" meant that the employer accepted claimant's inhalation of toxic chemicals which resulted in an absorbed dose of the substance sufficient to cause tissue damage in an appreciable, significant manner. In simpler terms, the employer accepted claimant's significant physical injuries caused by absorption of toxic chemicals through inhalation. The difference between chemical inhalation and toxic inhalation was that the former might not result in significant physical injuries while the latter did. If the employer accepted a medical condition, the "acute toxic inhalation" definition involving significant physical injury made procedural sense because, if the toxic chemical inhalation did not result in significant physical injury, the employer accepted nothing and the acceptance would have amounted to a meaningless exercise. The more precise definition of significant physical injuries caused by toxic chemical inhalation encompassed by the accepted condition was consistent with the employer's acceptance of a "disabling compensable injury" and the definition of a that phrase under ORS 656.005(7)(c).

In *William W. Hoffnagle*, 66 Van Natta 1522, 1530 (2014), the employer accepted claimant's "lower back injury" and then denied compensability of his lumbar strain as well as his L4-5 and L5-S1 disc conditions with left leg radiculitis symptoms and sciatica. The Board held that the employer's denial of those new/omitted medical condition claims constituted an impermissible "back-up" denial because its acceptance of claimant's "lower back injury" was vague, instead

of specific, and the contemporaneous medical evidence established the existence of the denied conditions. *Id.*²⁸

The present case is similar to the Board's decision in *Hoffnagle, supra*. Here, the employer's acceptance of claimant's significant physical injuries due to toxic chemical inhalation was so vague and ambiguous that it encompassed all significant physical injuries diagnosed by claimant's treating and examining physicians in the contemporaneous medical record (Exs. 110, 113, & 116). The overwhelming weight of that contemporaneous medical evidence (which acknowledged the existence of a disabling workplace injury) established that claimant's "acute toxic chemical inhalation" resulted in significant physical injury to the brain diagnosed as toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency and saccadic eye movement deficiency. Under this alternative procedural reasoning regarding the scope of acceptance issue where the employer actually accepted a medical condition involving significant physical injury caused by the inhalation of toxic chemicals, the February 20, 2019 and April 2, 2019 denials still constituted improper "back-up" denials pursuant to ORS 656.262(6)(a) because the acceptance of significant physical injuries resulting from a toxic chemical inhalation encompassed the four denied conditions. Therefore, the employer's denials of compensability of those specific conditions are set aside.

Assuming, *arguendo*, that these two procedural analyses of the scope of the employer's acceptances and its "back-up" denials of claimant's toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency and saccadic eye movement deficiency are legally incorrect, and the employer's acceptances of "acute toxic inhalation" and "acute chemical inhalation" actually constituted acceptances of two distinct medical conditions which were "empty shells" that did not encompass any of claimant's post-fume event symptoms, diagnoses, or need for treatment, I proceed to address the compensability issues on the merits.

²⁸ Although the case of *Georgia Pacific v. Piwowar*, 305 Or 494 (1988) concerned a "back-up" denial in the context of a carrier accepting an injured worker's *symptoms* where the acceptance encompassed the causes of the symptoms, similar principles apply in the present case where the carrier accepted vague significant physical injuries caused by toxic chemical inhalation. In *Piwowar*, 305 Or at 501-02, the carrier accepted a claim for a "sore back." Medical evidence showed that a preexisting disease (ankylosing spondylitis) caused the sore back, and the carrier denied compensability of that condition. *Id.* at 497. The supreme court concluded that, because the carrier had accepted a claim for a symptom of the underlying disease, and not a separate condition, its denial of the preexisting condition constituted a "backup" denial. *Id.* at 501-02. Similarly, in *Janet R. Christensen*, 50 Van Natta 396 (1998), the carrier accepted the claimant's "low back pain r/o HNP." The "low back pain" was caused in part by spinal stenosis and degenerative disc disease and, therefore, the carrier's acceptance included those conditions. *Id.*

2. Compensability of toxic encephalopathy and mild neural cognitive disorder (Exs. 113 & 116)

Claimant contends that his exposure to toxic fumes at work on January 21, 2017, was a material contributing cause of his toxic encephalopathy and mild neural cognitive disorder. Therefore, claimant asks that the employer's denials of those conditions be set aside. The employer asserts that the more persuasive expert medical evidence established that claimant did not have either condition and those portions of its denials should be approved.

To establish compensability of his claimed new/omitted medical conditions of toxic encephalopathy and mild neural cognitive disorder, claimant must prove that those conditions existed and that his January 21, 2017 work injury was a material contributing cause of his need for treatment or disability for the claimed conditions. See ORS 656.005(7)(a); ORS 656.266(1); *Betty J. King*, 58 Van Natta 977, 977 (2006); *Maureen Y. Graves*, 57 Van Natta 2380, 2381 (2005). Claimant must prove both legal and medical causation by a preponderance of the evidence. *Harris v. Farmer's Co-op Creamery*, 53 Or App 618 (1981); *Carolyn F. Weigel*, 53 Van Natta 1200 (2001), *aff'd without opinion*, 184 Or App 761 (2002). Legal causation is established by showing that claimant engaged in potentially causative work activities; whether those work activities caused claimant's condition is a question of medical causation. *Darla Litten*, 55 Van Natta 925, 926 (2003).²⁹

In *Seeley v. Sisters of Providence*, 179 Or App 723, 729-30 (2002), the court concluded that a claimant's burden of production under ORS 656.266³⁰ may be satisfied as long as there is *some* affirmative evidence that permits a reasonable trier of fact to *infer* a causal link between the condition and his work exposure, but that does not do so by disproving other causes for the disease; *i.e.*, by deductive reasoning alone. See *Martin v. City of Portland*, 178 Or App 505, 510 (2001); *McTaggart v. Time Warner Cable*, 170 Or App 491, 503-04 (2000), *rev den*, 331 Or 633 (2001). In *Seeley*, 179 Or App at 726, the Court found claimant's Hepatitis C claim compensable even though the needle stick injury which initially led her to file the claim had not resulted in the transmission of that disease. Nevertheless, claimant

²⁹ Claimant's alleged brain injury developed as a result of a discrete event, *i.e.*, acute chemical or toxic inhalation. Therefore, the claim is properly analyzed as an industrial injury, as opposed to an occupational disease. *Dynea USA v. Fairbanks*, 241 Or App 311, 318 (2010) citing *Smirnoff v. S.A.I.F.*, 188 Or App 438, 449 (2003).

³⁰ ORS 656.266(1) provides, "The burden of proving that an injury or occupational disease is compensable and of proving the nature and extent of any disability resulting therefrom is upon the worker. The worker cannot carry the burden of proving that an injury or occupational disease is compensable merely by disproving other possible explanations of how the injury or disease occurred."

prevailed because she had no history of risk factors outside of work that would have made it likely that she contracted the disease off the job, she had stuck herself with needles and other sharp objects numerous times at work over the years, there was statistical evidence linking claimant's condition to work, and the persuasive expert medical evidence established that claimant's work exposure was the most likely source of her Hepatitis C.³¹ *Id.* Similarly, claimant contends in this case that he should prevail against the employer's denials of his toxic encephalopathy and mild neural cognitive disorder because (1) he had no history of off-work risk factors making it more likely for his brain damage to have occurred off-the-job, (2) he had been exposed to chronic low levels of toxic chemical fumes at work over the years making him more susceptible to brain damage, (3) there was scientific and medical research linking claimant's condition to the toxic chemical fume event, and (4) the persuasive expert medical evidence established that his work exposure to toxic chemical fumes was the most likely cause of his toxic encephalopathy and mild neural cognitive disorder.

Based on the disagreement between experts, the causation issue presents a complex medical question that must be resolved by expert medical opinion. *Barnett v. SAIF*, 122 Or App 279, 282 (1993); *Matthew C. Aufmuth*, 62 Van Natta 1823, 1825 (2010). Absent persuasive reasons to the contrary, greater weight is generally given to the opinion of an attending physician. *Weiland v. SAIF*, 63 Or App 810, 814 (1983); *Gary S. Knight*, 63 Van Natta 1206, 1207 (2011). More weight is given to those medical opinions that are well reasoned and based on complete information. *Somers v. SAIF*, 77 Or App 259, 263 (1986).

³¹ In *Seeley*, 170 Or App at 728-29, the Court reasoned that ORS 656.266 did not prevent claimant from establishing compensability of her claim, stating,

We agree with claimant that the Board interpreted ORS 656.266 too broadly. That statute provides that a claimant cannot carry his or her burden of proof "merely by disproving other possible explanations of how the injury or disease occurred." ORS 656.266 (1). As we explained in *McTaggart v. Time Warner Cable*, 170 Ore. App. 491, 503-04, 15 P3d 1154 (2000), *rev den* 331 Or 633 (2001):

"All that the legislature did [in ORS 656.266] was to prohibit claimants from doing *no more than* disproving other explanations. It did not prevent them from disproving other possible explanations as part of carrying their burden of proof; thus, a claimant may still exclude alternative explanations to assist in proving the claim." (Emphasis in original.)

Conversely, we explained in *Bronco Cleaners v. Velazquez*, 141 Ore. App. 295, 298, 917 P.2d 539 (1996), that a claimant will satisfy ORS 656.266 as long as he or she provides "some affirmative evidence that the condition is caused by the claimant's work exposure." Read together, *McTaggart* and *Bronco Cleaners* make clear that, as long as a claimant provides some evidence that his or her condition was caused by the work exposure as well as disproving other causes, ORS 656.266 provides no bar to holding a claim compensable. See *Martin v. City of Portland*, 178 Ore. App. 505, 510, 37 P3d 209 (2001); *McTaggart*, 170 Ore. App. at 503-04; *Bronco Cleaners*, 141 Ore. App. at 298-99.

In order to establish compensability of his alleged toxic encephalopathy and mild neural cognitive disorder, claimant must show that he had symptoms and a need for treatment following the fume event, there was objective evidence of a physical injury to the brain, the toxic chemical fumes which he inhaled on the date of injury were capable of causing injury to the brain, and the expert medical evidence established by a preponderance of the evidence that the fume event was a material contributing cause of the claimed conditions.

a. Symptoms and treatment after the fume event

Before the January 21, 2017 industrial injury, claimant “always was working out, he’d done – he was a triathlete, done marathons. In the months before he had the [fume] event, he had bicycled across the state of North Carolina and run a four-mile race.” (Ex. 122-27). At the hearing, claimant’s long-time friends and colleagues, Captain Schussler and Captain Richards, both testified that, before the fume event, claimant was a happy, vivacious, “intense workout guy” who loved his job and exercised all the time. On a scale of one to ten in terms of physical fitness, he was an “eleven.” (Ex. 122-27). Captain Schussler also testified that claimant would never fake an injury and it was completely implausible that he would give up his career for a life of feigned disability. He described how claimant was young and active before the fume event, but after the event, he walked with a cane and his skin was yellow. In Captain Schussler’s opinion, claimant could not possibly be faking those huge changes. Captain Richards was hired along with claimant in 2002, they went running together, shared an apartment in New York, and became good friends. Like Captain Schussler, Captain Richards testified that claimant loved his job and was absolutely incapable of faking a mental injury. The dramatic changes Captain Richards saw in claimant after the fume event were very upsetting to him. The employer did not present any contrary evidence. Thus, I conclude that claimant loved his job and was in extraordinary physical and mental shape with outstanding vision before the January 21, 2017 fume event. Claimant began exhibiting symptoms of respiratory distress, headache, and confusion immediately after his exposure to toxic fumes during the three engine run-ups on the airplane and his cognitive symptoms continued to worsen over the next several months.

Almost three years after the fume event, Drs. Ugalde, Schloesser, Schock, and Kreiling all concluded that claimant’s post-fume event symptoms were reliable, and he was not malingering or exhibiting any evidence of secondary gain (Exs. 80, 83, 84, 86-26, 94, 115-2 & 116A-2). If anything, Dr. Kreiling felt that claimant was underreporting his symptoms (Ex. 86-26). Dr. Bass, who examined claimant every six months for ten years before the fume event and once afterward, testified that

claimant was “absolutely not” faking or exaggerating his injury (Ex. 122-29). Dr. Kaniecki emphasized that individuals could not fake a PET scan, saccadic eye movement deficiencies, or convergence insufficiency, and there was no sign of symptom magnification or embellishment on the three neuropsychological tests claimant took (Ex 138A, pages 33-36). Dr. Schock testified that she had conducted several thousand neurological evaluations during her twenty year career as a Licensed Psychologist and had observed malingering and faking on many occasions because the examinations were designed to flesh that out (Dr. Schock testimony). In her opinion, claimant exhibited no evidence of malingering or faking. *Id.* She also did not believe that claimant exhibited vague shifting symptoms because his symptoms were consistent across her two evaluations and the third neuropsychological evaluation done by Dr. Kreiling. *Id.* Drs. Abou-Donia and Kreiling both concluded that claimant’s symptoms were a textbook, classic example of toxic fume exposure that he could not have made up (Exs. 86-25 & 137-22). Dr. Bass also concluded that claimant exhibited a substantial change in his demeanor after the fume event and many other pilots had similar neurological problems following their own toxic fume exposures (Ex. 120-3). Dr. Kaniecki believed that claimant’s ability to operate the radio on the plane flight to New York the day after the fume event was consistent with a toxic exposure because toxic effects took time to express themselves (Ex. 138A-29). Dr. Harrison agreed that claimant’s symptoms after the fume event were consistent with the progression seen in other fume event patients (Ex. 132-6).

Based on my own observations of claimant’s attitude, appearance and demeanor at the hearing, I found him to be a credible witness. His memory difficulties and mental fatigue resulted in some inability to recall details at times, but he was straightforward while acknowledging those deficiencies, and his testimony was otherwise trustworthy. In light of my own assessment of claimant’s credibility at the hearing, along with the unrebutted observations and opinions of Captains Schussler and Richards, and the persuasive expert medical opinions of the physicians who examined claimant closest in time to the fume event, I conclude that claimant’s reporting of his complaints and version of events was reliable. *Julie C. Schaber*, 72 Van Natta 303, 308-09 (2020) citing *Darrin Diegel*, 64 Van Natta 265, 265-67 (2012) (the substance of the record, including historical medical records, was found sufficient to establish the claimant’s credibility); *Robert L. Cross*, 72 Van Natta 108, 109 (2020) (the claimant was found credible based on the Board’s review of the record). That is not to say, however, that claimant’s significant disability and need for treatment were caused by the fume event, as he believed they were. That is a matter for the medical and scientific experts to decide.

b. Objective evidence of brain injury

Claimant's attending physician, Dr. Ugalde, examined claimant on many occasions and found ample objective evidence of impairment such as: Ongoing vestibular signs and symptoms including sustained nystagmus with large movement higher frequency extraocular lateral movements, positive vestibular ocular reflex, and loss of balance with pivot and quick changes in gait, headaches which were severe enough to classify as migraines, difficulty breathing caused by vocal cord adduction with expiration documented by ENT, decreased pinprick sensation in a stocking glove distribution which had improved over time and was consistent with improving reflexes and nerve conduction studies, along with fatigue, total body tremors, and mood disturbance (Ex. 81-6).

Claimant's other treating physician, Dr. Schloesser, who treated him before and after the industrial injury, concluded that claimant's significant injury with cognitive problems, headaches, tremors, reduced energy, and peripheral neuropathy had all been documented both by examination and history, as well as with objective testing including nerve conduction studies, a PET scan, laboratory analysis, and significant neurocognitive impairment (Ex. 94, pages 1-2; *see also* Ex. 102). Dr. Schloesser addressed the IME physicians' criticism of his February 21, 2017 nerve conduction study, stating, "As to the concerns of an IME regarding skin temperature with nerve conduction testing, all of my patients are seen in a warmed room, and I always check for normal temperature to touch. There was no evidence for altered result on the basis of skin temperature or the patient would have been warmed. ***" (Ex. 116A-2). Dr. Abou-Donia agreed with Dr. Schloesser that the first nerve conduction study was performed correctly, the results of that test were consistent with claimant's symptoms, and Dr. Abou-Donia was surprised the IME physicians attacked the validity of Dr. Schloesser's nerve condition study without knowing the facts (Ex. 137, pages 34-35 & 38).

Dr. Bass noted that claimant exhibited diminished deep tendon reflexes during his December 17, 2017 examination, approximately eleven months after the toxic fume event (Ex. 122-23). Dr. Abou-Donia believed that claimant's positive Babinski and Romberg tests confirmed the presence of peripheral neuropathy (Ex. 137-26). Dr. Abou-Donia's autoantibodies test measured how much brain damage had occurred from the toxic chemical exposure because organophosphates kill nerve cells in the brain and antibodies respond to the proteins in the blood (Ex. 137-53). Dr. Kaniecki agreed with Dr. Abou-Donia that claimant's positive autoantibodies test suggested brain damage due to toxic exposure (Ex. 138A-34).

Dr. Kaniecki explained that PET scans documented dysfunction in the nervous system, they could not be faked, claimant's PET scan results were consistent with his symptoms, and that test supported the conclusion that he was exposed to toxic chemicals (Ex. 138A, pages 32-33). Although Dr. Kaniecki believed that claimant's PET scan results meant he was exposed to toxins, he did not know the specific level or type of toxic exposure (Ex. 138A-40). He also stated that PET scans and neuropsychological tests were some of the most important bases for assessing toxic encephalopathy (Ex. 138A-34). Dr. Schock concluded that claimant's neurocognitive testing was objective evidence of neurological dysfunction (Ex. 115-2). Dr. Kaniecki agreed with Dr. Schock and found no signs of symptoms magnification or embellishment on the neuropsychological tests (Ex. 138A-35). While claimant's MRI scans and CT scans were not sensitive enough to show toxic brain damage, they did rule out the presence of a stroke or multiple sclerosis (Ex. 131, pages 7 & 20).

On April 3, 2019, Dr. Scott concluded that claimant exhibited both objective and clinical evidence of vision damage and it was impossible that claimant "could have had this condition prior to his toxic exposure and still be a marksman and airline pilot." (Ex. 117, pages 3-4). Dr. Craven also supported the existence of objective evidence of physical injury resulting from the fume event. On January 4, 2018, Dr. Craven stated, "From review of his medical records, it appears he had an acute exposure to tricresyl phosphate at work while working in the cockpit of an airplane. The exposure may have been at a high level in the air in airplane. There is objective support that he has developed physical problems and neurocognitive problems since the exposure. ***" (Ex. 80A-4). On January 25, 2018, Dr. Craven authored an addendum report based on additional medical records and, while his thinking changed regarding the cause of claimant's persistent neurological problems, he still concluded that the toxic chemical exposure "likely caused his symptoms in the first few days after the exposure ***." (Ex. 84B-2).

c. Toxins capable of causing brain injury

The opinions of Drs. Burton and Pleus that claimant did not suffer any physical injury from his exposure to toxic fumes rested significantly on their over-focus on the amount of TCP present during the January 21, 2017 fume event. Both doctors downplayed the fact that there were many other toxic chemicals contained in the jet engine oil which multiplied dramatically when the oil was pyrolyzed at extremely high temperatures and that combined chemical exposure increased the toxicity of all the chemicals introduced into claimant's body (*See* Ex. 137-16). Dr. Harrison persuasively explained that the toxicity of jet engine oil increased when it

was volatilized at high temperatures (Ex. 139-55). In fact, the UFPs, or Nano-particles themselves, could be extremely toxic by themselves (Ex. 141C-5). Dr. Harrison concluded that claimant was exposed to the toxic products of jet engine oil, including the various isomers of TCP and Tributyl Phosphate, along with toxic by-products pyrolyzed at high temperatures, which were absorbed into the body through the lungs and crossed over into the blood-brain barrier, resulting in neurological symptoms (Ex. 139, pages 11-12 & 17-18). Dr. Harrison believed that “over 10 or 12 different isomers” were created during pyrolyzation (Ex. 139-51).

Dr. Abou-Donia agreed with Dr. Harrison’s opinion regarding the various toxic fume compounds from burning jet engine oil and explained that the amount and duration of the toxic chemical exposure in the environment was more important than the actual concentration of TCP in the oil (Ex. 137, pages 20-21). Dr. Abou-Donia also stated that, beside the significant exposure during the acute toxic fume event on January 21, 2017, claimant had been exposed to low levels of toxic fumes for 25 years. *Id.* Inhalation was also far worse than ingestion in accentuating the effects of a toxic exposure (Ex. 137-58). During his deposition, Dr. Abou-Donia stated,

There’s definitely [a] difference because when we drink something or eat something, it goes through the blood – through the portal vein to the liver. And the liver – these chemicals are metabolized and broken down so the amount that goes to the blood is much less than what we [would have] inhaled. Inhalation – it takes 10 seconds from the time we inhale something for it to go to the brain. This is number one. Number two, inhalation is – we inhale 15 times a minute, and every time we inhale half a liter. So inhalation is a very, very efficient way to introduce chemicals not only in the body but in the brain.

Id. In addition, Dr. Abou-Donia testified that pyrolyzed chemicals were much more toxic than if they were not heated to high temperatures (Ex. 137-60).

The medical literature established that TCP has neurotoxic properties, but the widespread belief that only ortho isomers of TCP are dangerous is invalid (Ex. 141A-10). Instead, the 99.7% of non-ortho isomers of TCP and TAP can cause nerve demyelination and inhibit various enzymes, including those linked to cognition. *Id.* Therefore, Dr. Burton’s belief that TCP non-ortho isomers “are substances that do not have the capability of causing neurologic injury or disease in any dose” was incorrect (Ex. 139A-3).

d. Expert medical opinions

i. Claimant's treating physicians

Claimant's treating physician, Dr. Ugalde, was Board Certified in brain injury medicine and focused her practice on patients with brain injuries (Ex. 131-20). She examined claimant on many occasions after the January 21, 2017 fume event. Dr. Ugalde did not know the exact amount of toxic chemicals claimant was exposed to but believed that he suffered a sufficient level of toxic exposure to cause his brain injury (Ex. 131-22). She relied on the opinions of Drs. Abou-Donia, Harrison, Kaniecki, and Burton to understand the amount of the exposure. *Id.* Dr. Ugalde persuasively rebutted Dr. Burton's opinion that it was impossible for chemicals in jet engine oil to have caused any of claimant's problems (Ex. 118-5). She explained her opinion briefly by stating, "[Claimant] was healthy and cognitively function[ing] at a high level as a pilot. He had exposure to fumes. He now has a cognitive deficit objectively measured by neurocognitive testing. There is no other explanation for his cognitive impairment. Experts in toxic exposure concur with this assessment." (Ex. 118-6). That expert medical conclusion was based on a number of factors, including: (1) The mechanism of injury was consistent with the diagnosis based, in part, on the medical literature and reports from various experts, (2) exposure to TCP and other chemicals in jet fuel oil can cause neurological problems and have toxic effects, (3) there was a strong temporal relationship, and (4) experts in toxic exposure agreed that toxic fume exposures caused cognitive impairment (Ex. 118, pages 4-6). Since Dr. Ugalde persuasively addressed and rebutted alternative theories for claimant's symptoms, her expert medical opinions are entitled to great weight. *Julie L. Schaber*, 72 Van Natta 303, 310, fnt 3 (2020), citing *Thomas C. Foley*, 66 Van Natta 1269, 1272-73 (2014) (relying on the opinion of the treating surgeon, which rebutted contrary theories and was based on observations).

Claimant's other treating physician, Dr. Schloesser, concluded that the January 21, 2017 exposure to toxic fumes and tricresyl phosphate was the major contributing cause of claimant's polyneuropathy, headaches, and cognitive problems (Ex. 116A-1). Dr. Schloesser's expert medical opinion was based on a number of factors, including: (1) Organophosphate toxicity was well established in detail in the medical literature, (2) he treated claimant before and after the industrial injury and there was a significant clinical change following the fume event, (3) claimant had no pre-existing problems immediately before the fume event, (4) the nerve conduction study he performed, which showed evidence of peripheral neuropathy, was not flawed, (5) there was other objective evidence of physical injury including

a PET scan and neuropsychological testing, (6) claimant was an affable and happy pilot without any history suggesting a tendency toward malingering or secondary gain prior to the fume event, and (7) claimant had no injuries after the fume event which would explain his persistent symptoms (Ex. 116, pages 1-2).

The FAA Medical Examiner, Dr. Bass,³² examined claimant twice a year for ten years before the January 21, 2017 fume event and again in December 2018 (Ex. 122, pages 26-27). Although Dr. Bass did not have any special expertise in toxicology, he was in a unique position to compare claimant's physical and mental status before and after the fume event (Ex. 122, pages 7 & 19). During his deposition, he stated, "*** The whole picture of Captain Myers was he was a wreck when he walked in[to the December 2018 examination]. *** I've known him since 2008. [After the fume event], [h]e was walking with a cane, he had trouble talking, trouble with memory, and had the – had the diagnosis of toxic encephalopathy ***." (Ex. 122-19).

ii. Claimant's consulting medical, toxicology and fume event experts

Dr. Harrison had experience and expertise in cases involving occupational exposure to organophosphates including 75 to 100 airline personnel who had been victims of fume events (Exs. 80A-4 & 132-2). Dr. Harrison was also very familiar with the medical research and, although he was not a Board Certified Toxicologist, he had specialized training in toxicology and was one of the foremost experts in toxic exposure cases, having treated more than five thousand individuals who were exposed to chemicals in the workplace (Exs. 132-2, 138A-45 & 139-7). Based on his expertise, examination of claimant, and knowledge of the case, Dr. Harrison disagreed with Dr. Burton's opinions regarding causation, in part, because Dr. Burton's reliance on OSHA standards was wrong (Ex. 132, pages 4-5). Dr. Harrison explained that OSHA standards were based on outdated science, they did not take into account the effect of exposure to multiple toxic agents in addition to tricresyl phosphate nor did they take into account the pernicious effect of gradual minimal exposure over the course of many years (*Id.*; *see also* Ex. 139-37).³³ Instead, Dr.

³² Dr. Bass was a Naval Aviator in his youth who graduated from OHSU medical school in 1963 (Ex. 122-25). He spent thirty years doing cardiac anesthesia and running the intensive care unit at Good Samaritan Hospital before he began conducting pilot examinations for the FAA in March 2001 (Ex. 122, pages 25-26).

³³ OSHA regulations regarding TCP were based on science done more than fifty years ago in studies that measured the amount of those TCP compounds that a hen would have to ingest before becoming paralyzed (Ex. 139-53). It was likely that neurological damage was happening to those hens prior to paralysis (Ex. 139, pages 53-54). Industrial Hygienist Anderson also testified that the OSHA standards had not been updated since 1968 and did not apply to the flight deck environment because of the complex mixture of toxic compounds in pyrolyzed jet engine oil. Dr. Michaelis testified that OSHA regulations only focused on TOCP levels, did

Harrison believed that claimant's toxic exposure at work was the major contributing cause of all diagnosed conditions (Ex. 132-4). His opinion regarding causation was based on a number of factors, including: (1) Science and research made it "clear that exposure to tricresyl phosphate and other such additives can rapidly cause neurological damage and the types of problems that [claimant] is suffering from, [meaning] there is a medical plausibility between the exposure and the result," (2) there was a complete absence of any other rational explanation for claimant's symptom complex, (3) claimant had no exposure to any comparable chemicals off the job, (4) there was an abundance of objective evidence of a brain injury due to toxic exposure including the PET scan, the autoimmune antibodies test, and other diagnostic tests, and (5) a strong temporal relationship existed between claimant's toxic exposure and the diagnosed conditions (Ex. 132-5). Dr. Harrison was not disturbed by the delay in claimant's cognitive symptoms either because, in his experience, "[i]t can take days and even weeks for the effect of chemicals to become evident." (Ex. 132-6). Therefore, Dr. Harrison felt that claimant's attempt to do his job the following day was entirely consistent with the progression of symptoms he had observed in other patients (Ex. 132-6 & 139-13). It was more important to Dr. Harrison that claimant had "immediate and significant symptomatology while he was being exposed in the cockpit." (Ex. 132-6).

Dr. Abou-Donia was one of the foremost toxicologists in the country who spent his career studying the effects of toxic agents on the nervous system (Judith Anderson testimony; Exs. 133-5 & 138A-45). Although claimant's counsel ultimately chose not to rely on his occupational disease theory of the case, Dr. Abou-Donia's expertise in toxic exposure cases and the autoantibodies test he developed offered valuable information. He persuasively explained that more damage is done when an acute chemical exposure follows years of low level exposures and when there are exposures with multiple toxic chemicals because of competition for detoxifying enzymes (Ex. 126, pages 8-9). On May 31, 2019, he disagreed with Dr. Burton's opinion because the IME physician did not consider claimant's low level exposure to chemical fumes before the industrial injury (Ex. 126-26). In other words, claimant's low level exposure to chemical fumes throughout his career as an airplane pilot increased his susceptibility to brain damage, which Dr. Burton did not address. *Id.* Through his own autoantibodies testing on pilots and flight attendants with symptoms following fume events, Dr. Abou-Donia found 34 subjects who developed brain and neurological damage consistent with being poisoned by organophosphates, rebutting Dr. Burton's mistaken belief that no one had ever suffered brain damage

not consider all isomers, and were therefore quite inapplicable to measuring the level of toxins present during claimant's fume event.

from a toxic fume event (Ex. 129-1).³⁴ On July 12, 2019, Dr. Abou-Donia concluded that, although it was entirely possible that claimant could have ended up with a brain injury without the acute fume event, given long-term exposure to organophosphates in trace amounts, the January 21, 2017 fume event was the major contributing cause of his toxic encephalopathy because he was exposed to a high level of poison at that time (Ex. 133-6 & Ex. 137, pages 64 through 69). Dr. Abou-Donia again rebutted Dr. Burton's conclusion that claimant needed a much higher exposure to liquid TCP by explaining that the toxic effects were most prevalent when the chemical was in aerosol form, making the compounds small enough to pass through the bodies' various defense mechanisms and attack the nervous system (Ex. 133-7). Dr. Abou-Donia explained that Dr. Burton did not take into account the effect of multiple toxic chemicals working in unison and claimant was likely exposed to more than just tricresyl phosphate during the fume event. *Id.* Dr. Harrison agreed with Dr. Abou-Donia's opinion, stating, "*** [I]n general for occupational exposures, that there is synergy. That one plus one is equal to three or four when there are multiple chemical exposures. And I would not have any reason to doubt that it occurs in the case of the TCP isomers." (Ex. 139-39). Moreover, The FAA guidelines were outdated and more current science revealed that much lower toxic fume exposure levels resulted in severe neurological effects (Ex. 133-7). Dr. Ugalde also believed that claimant's positive autoimmune antibodies test was consistent with neurological damage (Ex. 131-20).

Claimant's susceptibility to the debilitating effects of a toxic fume exposure was particularly important to a number of physicians. Dr. Schock examined claimant on three occasions while conducting two serial neurocognitive tests. She explained that an individual's sensitivity to chemicals and the cumulative effect of chemical exposure over time may override a lesser dose to cause injury (Ex. 130-20). Dr. Harrison also explained "that there is a very wide range of affects that individuals can have from comparable exposures. Some individuals simply have less adequate defense mechanisms to fend off the effects of those poisons. As such, some individuals have had very little affect, some have had a more moderate affect and a very few have had significant affects. [Claimant] has had one of the more serious reactions that [he had] seen." (Ex. 132-3). Dr. Bass offered the same conclusion regarding the issue of susceptibility, stating that there was "a wide degree of variability on the effect that these poisons have on different individuals, many show little or no effect and an unfortunate few are severely affected. [Claimant] is one of

³⁴ At the hearing, Industrial Hygienist Anderson explained Dr. Abou-Donia's biomarker development, stating that, when someone experienced damage to the central nervous system, the blood-brain barrier (which kept toxins out of the brain) was compromised and, when the brain proteins from that damage were in general circulation, antibodies responded to their presence, which indicated damage to the central nervous system.

those individuals who apparently has some susceptibility and has been severely affected by the toxic effects of these organo phosphates.” (Ex. 133-6; *see also* Ex. 122-17). Dr. Kaniecki agreed with those assessments, stating that individuals exposed to some of the most minor fume events ended up with the worst problems and there was no accepted safe level of TCP exposure because of different susceptibilities (Ex. 138A, pages 9-10). Because of that wide range of individual variability to fume events, Dr. Kaniecki was not surprised that the First Officer’s symptoms were not nearly as bad as the symptoms claimant experienced following the fume event (Ex. 138A-29). Dr. Kaniecki believed that the January 21, 2017 fume event was the major contributing cause of claimant’s toxic encephalopathy (Ex. 138A-37).

Dr. Harrison explained that claimant’s response to the fume event was much worse than the First Officer for two reasons. He stated,

*** One is with any toxic exposure, as I often say, if somebody sneezes not everybody catches a cold. So when there’s a toxic exposure, there’s a variability in response, ranging from none to mild to severe and persistent. There’s a biological difference in the body’s response.

The second is that as I’ve already said, Captain Myers had a previous history of migraine headaches. So it’s plausible that the toxic chemical exposure triggered his headaches. That he was a – that he was more susceptible.

(Ex. 139-57).

Dr. Michaelis echoed Dr. Harrison’s understanding of some individuals being more susceptible to fume events, especially airline employees with a long history of chronic exposure to small amounts of toxins, stating,

Our research as well as considerable previous literature related to fume events via the aircraft air supply identify variability between individual crew. *** This is well explained in our recent papers. *** We have reported that this is to be expected with the symptomatology of OP exposure being ‘rather non specific’, with a ‘diffuse pattern of neurological symptoms.’ *** The acute on chronic pattern described above “could explain the apparent differential vulnerability between aircrew and passengers.” ***

Individual susceptibility to damage by OPs is well described and suggested to be highly variable, with not all crew affected the same by fume events. ***

(Ex. 142-10; citations omitted). Dr. Michaelis explained that fume events were common occurrences in the airline industry and pilots were chronically exposed to small amounts of toxic vapors (Ex. 142-3). She also believed that claimant's symptoms were consistent with exposure to oil contaminants and "a clear and consistent acute and chronic pattern of adverse effects, including central and peripheral nervous system effects, neurobehavioural, gastrointestinal, respiratory, cardiovascular, general irritant, skin and sensitizing effects." (Ex. 142, pages 8 & 12).

Industrial Hygienist Judith Anderson was qualified to render opinions on toxicology and believed that claimant's symptoms were consistent with his airline toxic fume exposure (Judith Anderson testimony). She also believed that claimant's symptoms were consistent with the symptoms experienced by thousands of other crew members following other airline fume events. *Id.* Even though Ms. Anderson conceded that it was impossible to calculate the actual amount of toxins claimant inhaled on January 21, 2017, she was still able to render an opinion regarding the causal relationship because claimant was exposed to 300 chemical compounds, he developed acute and chronic symptoms following the fume event, and those symptoms were consistent with the thousands of other crew members who experienced symptoms after fume events. *Id.*

Drs. Shock and Kreiling, who performed neurocognitive tests, both believed there was objective evidence of cognitive deficits that were consistent with a toxic chemical exposure (Ex. 43-8, Ex. 74-5, Ex. 86, pages 23-25, & Ex. 130, pages 25 through 29). Also, neither of those two clinical psychologists who examined and tested claimant, found any evidence that he was malingering or otherwise faking his symptoms (Exs. 84 & 86-26). Their opinions persuasively established the existence of claimant's mild neurocognitive disorder supported by objective findings. ORS 656.005(19); see *Ana Barajas-Valencia*, 72 Van Natta 297, 298 (2020), citing *SAIF v. Lewis*, 335 Or 92 (2002) (where a physician bases a medical opinion on a patient's symptoms, the physician need not personally reproduce, measure, or observe those symptoms and may rely on, among other things, self-reports of symptoms, so long as those symptoms are capable of being verified); *Vicki L. Williamson*, 62 Van Natta 341, 345 (2010) (finding physician's opinion based on clinical history and test results more persuasive than opinions that relied on test results only).

Dr. Ugalde agreed with Drs. Schock and Kreiling that claimant's repeat neuropsychological test results were consistent with cognitive deficits from the industrial injury (Ex. 131-20). She also agreed with the clinical psychologists that claimant showed no evidence of malingering during neuropsychological testing or during the many times Dr. Ugalde treated claimant (Ex. 131-21). Dr. Ugalde's assessment of the validity of claimant's complaints is given great weight since she examined claimant every month for a couple of years and was intimately familiar with his physical and mental presentation. *Id.* Similarly, the FAA Medical Examiner, Dr. Bass, examined claimant every six months for ten years (Ex. 122, pages 28-29). During his deposition, Dr. Bass testified that, when he examined claimant in December 2017, almost a year after the compensable fume event, claimant walked with an unsteady gait, his speech was slurred, and it was pretty difficult for him to talk (Ex. 122, pages 28-29). Dr. Bass was convinced that claimant's "whole demeanor was grievously impaired" and he was "absolutely not" faking or exaggerating his injury. *Id.* Like Dr. Ugalde, Dr. Bass' opinion regarding the validity of claimant's examination findings and symptoms after the fume event is entitled to great weight given his long-term history of examining claimant twice a year for ten years and his observations of claimant before and after the toxic fume event. *See Julie L. Schaber*, 72 Van Natta 303, 310 (2020) (the Board considered claimant's treating physician to be in an advantageous position to assess her conditions and their relationship to the work injury because he was most familiar with her complaints over an extensive period); *William J. Friend*, 69 Van Natta 119, 128 n 4 (2017); *Barbara A. Courtain*, 66 Van Natta 862, 865 (2014) (more weight accorded to physician who had the advantage of examining the condition shortly after the work injury); *Kevin G. Gagnon*, 64 Van Natta 1498, 1500 (2012) (longitudinal history with the claimant rendered physician's opinion more persuasive); *Anthony A. Miner*, 62 Van Natta 2538, 2540 (2010) (physician who treated the claimant soon after the work injury was in a better position to evaluate the claimant's injury-related conditions than the physician who examined the claimant three months later).

iii. The employer's IME physicians

On March 2, 2018, claimant was examined by the insurer-arranged medical examiner and Clinical Neuropsychologist Tracy Kreiling, Psy.D., who diagnosed a major neurocognitive disorder due to toxic inhalation (Ex. 86, pages 24-25). Dr. Kreiling did not believe that claimant's cognitive deficits were better explained by a mental disorder (Ex. 86-24). Instead, she believed that claimant's symptoms, neuropsychological measures, and the stability of his performance from repeated neuropsychological evaluations over time were caused by his toxic chemical exposure. *Id.* Dr. Kreiling found no evidence of malingering or symptom

magnification during the evaluation (Ex. 86, pages 26-27). If anything, she felt that claimant was possibly underreporting his symptoms. *Id.* Dr. Schock's earlier neurocognitive tests were consistent with Dr. Kreiling's test findings and conclusions (Ex. 130, pages 24-25).

Dr. Burton, a toxicologist, did not examine claimant but performed a file review on the employer's behalf (Ex. 58). His belief that claimant's symptoms were psychological in nature and Dr. Bell's opinion that people exposed to toxic fume events experienced "mass hysteria" were not supported by the scientific literature regarding airplane toxic fume events or the more persuasive expert medical opinions of claimant's treating and consulting physicians (Exs. 58-8 & 134-41). A 2009 Expert Panel on Aircraft Air Quality determined that, based on the available evidence, "malinger [and] primary psychiatric illness *** lacked plausibility" in explaining symptoms experienced in toxic fume events (Ex. 139-121). Similarly, a textbook on fume events stated that malingering and primary psychiatric illness were "[t]heories of causation considered unlikely and/or lacking plausibility" regarding symptoms attributed to "Aerotoxic Syndrome." (Ex. 139-264). During his deposition, Dr. Harrison testified, "I've read nothing in the medical or the scientific literature to suggest that this is mass psychogenic illness. And it – it is very unlikely that over time and place, to many different individuals, that they would all just by coincidence have the same set of signs and symptoms. So that's – that's one piece of evidence that this is likely linked to a toxic chemical exposure." (Ex. 139, pages 13-14). His own clinical experience confirmed the literature in that regard. Specifically, Dr. Harrison stated, "*** I've diagnosed, evaluated or treated probably about 100 cabin crew, including flight attendants and pilots, over the last 25 years. And with few exceptions, their symptoms and signs are of neurotoxic injury. And I have, in almost all cases, ruled out other causes, including considering what is often on insurance carrier or defense medical exams, considered to be psychological illness." (Ex. 139, pages 14-15). Dr. Harrison summed up his position regarding the "mass hysteria" theory of toxic fume events by stating, "*** I believe from my practice, experience and review of the literature that there definitely is a physical injury [from toxic fume events]. That this cannot be explained by Somatoform Disorder or psychological issues." (Ex. 139-45). Specifically with regard to claimant, Dr. Harrison offered the following conclusion, "*** If you ask me for my medical legal opinion in this context of Captain Myers, I would say in his case the exposure to that – to those chemicals on January 21st, 2017, did cause an injury and that I am very certain of that." (Ex. 139-49).

Although Dr. Burton and Bell relied on animal studies that showed very large amounts of TOCP had to be ingested to cause physical injury, they also seemed to

understand that TOCP-ingestion studies were irrelevant to human toxic inhalation cases (Ex. 134-40).³⁵ Those ingestion studies were irrelevant because, as Dr. Harrison stated, “Probably there’s a direct higher-dose effect by inhalation, immediate passage into the blood and then into the – into the brain, which is why in human populations like flight attendants and pilots, they will say that they have acute effects within minutes to a few hours. So it’s not a slow onset by ingestion; it’s an immediate toxicity.” (Ex. 139, pages 54-55). Dr. Harrison’s opinion that animal ingestion studies were not relevant in determining the cause of toxic inhalation cases was consistent with the scientific literature.³⁶ Industrial Hygienist Judith Anderson explained at hearing that the EPA understood inhaling toxins was more toxic than ingestion because the body processed them differently. Even Dr. Pleus conceded, “However, extrapolation from dermal exposure to inhalation exposure, for example, is not a common procedure as the mechanisms of absorption differ considerably.” (Ex. 141-44).³⁷

Like Dr. Burton, Dr. Bell also over-focused on claimant’s exposure to TOCP only, disregarded the effect of many other toxic chemical compounds to which he was exposed, and incorrectly believed that there was no objective evidence of brain injury from the fume event. Dr. Bell’s belief that Dr. Schloesser’s NCS was flawed was incorrect, as were her beliefs that the neuropsychological tests showed no objective evidence of organic brain damage (*Compare* Ex. 134, pages 38-39, with Exs. 43-8, 74-5, 86-22, & 116A-2). Dr. Bell’s dismissal of Dr. Abou-Donia’s positive autoimmune antibodies test and the PET scan were also not persuasively explained. One of Dr. Bell’s main objections to the presence of objective findings of a toxic brain injury was that claimant “developed new symptoms and new disabilities as the months and years have progressed” and a “true toxic encephalopathy would have maximal clinical symptoms at the beginning, experience gradual improvement

³⁵ On July 25, 2019, Dr. Bell stated, “*** The bulk of the medical literature cited by Dr. Abou-Donia] is of animal studies in which large doses are given orally or dermally, resulting in a wide range of neurological deficits, including peripheral and central nervous system pathology. The more important question is the relevance of any of these case reports or animal studies to [claimant’s] case – which, as discussed by Dr. Burton – is nil.” (Ex. 134-40).

³⁶ The *Bleed-Air Contaminant Exposure Management Guide* stated,

The majority of published research on the toxicity of engine oils has assessed symptoms of peripheral neuropathy among laboratory animals that either ingest the oil or absorb it through the skin. However, aircraft occupants are primarily exposed via inhalation with the potential for limited dermal exposure. There is no evidence that ground-based dermal/oral research data can be applied to inhalation exposures that are often incurred in a reduced oxygen environment. Inhalation toxicity testing in a controlled laboratory setting, with post-mortem brain analysis of exposed animals may be necessary to confirm the observations of chronic neurotoxicity among exposed aircraft occupants.

(Ex. 139-381).

³⁷ A rationale for basing regulatory guidelines on animal ingestion studies instead of occupational inhalation data was that there was not enough information regarding the effects of airborne levels of TOCP on human health to use in establishing a threshold limit value (TLV) (Ex. 141-45).

with time, or remain stable.” (Ex. 134-40). That opinion regarding the supposed “normal” progression of a toxic chemical exposure was not supported by the scientific literature which frequently documented immediate short-term respiratory and cognitive symptoms followed weeks or months later by the development of long-term neurocognitive symptoms (Ex. 130-39). Dr. Bell’s failure to consider the scientific literature amplified Dr. Schock’s testimony that Dr. Bell exhibited some bias by using certain data to support her viewpoint while leaving out other information that did not support her arguments. Dr. Abou-Donia persuasively explained that claimant exhibited a typical manifestation of initial signs of peripheral neuropathy followed by the more slowly progressing signs of central nervous system injury to the brain and spinal cord (*See* Ex. 126-10). Dr. Harrison agreed that delays in symptoms following toxic chemical exposures were very common (Ex. 139-56).

In contrast to the long-term doctor/patient relationships claimant developed with Drs. Ugalde, Schloesser, and Bass, (and to a lesser extent Drs. Scott and Schock), the IME physician, Dr. Bell, saw claimant on one occasion only and neither Dr. Burton nor Dr. Pleus examined claimant at all. Thus, Dr. Bell’s conclusion that claimant’s “clinical presentation is not reliable” was based on extremely limited examination data and was not nearly as persuasive as the opinions of claimant’s treating and consulting physicians who possessed a much broader and deeper knowledge base regarding claimant’s actual condition (Ex. 134-39). Dr. Burton’s conclusion that a “face to face exam would have no relevance in determining if [claimant] experienced a toxic exposure, or if his reported findings are consistent with a toxic exposure” defies logic and the more persuasive expert medical opinions of claimant’s long-time treating and examining physicians in a case where the clinical findings over time were so important in determining causation and the extent of disability (Ex. 139A-5). In fact, Dr. Ugalde found the opinions of Drs. Burton and Bell invalid precisely because they did not have serial assessments of claimant (Ex. 80-1). The consistency of claimant’s symptoms over a long period of time and the consistency of his responses to three different neurocognitive tests were essential elements in determining the cause of his conditions. Thus, Drs. Bell, Burton and Pleus were at a distinct disadvantage in forming valid opinions regarding causation due to their lack of clinical familiarity with claimant.

In addition, Dr. Burton’s opinions were internally inconsistent. On the one hand, he did not believe that claimant was exposed to sufficient levels of toxic fumes to cause any physical problems. On the other hand, he agreed with Dr. Craven’s assessment that claimant’s exposure to toxic fumes at work on January 21, 2017, caused his symptoms during the first few days afterward (Ex. 84A-2 & Ex. 92, pages 3-4). Dr. Burton’s criticism of Dr. Schloesser’s nerve conduction study was not well-

founded either. On April 2, 2019, Dr. Schloesser rebutted the IME's erroneous assumption, stating,

As to the concerns of an IME regarding skin temperature with nerve conduction testing, all of my patients are seen in a warmed room, and I always check for normal temperature to touch. There was no evidence for altered result on the basis of skin temperature or the patient would have been warmed. The patient also had a PET scan performed on 03/09/2018 which demonstrated reduced metabolism particularly in the posterior fossa but also to my review some reduction in the bitemporal regions. Furthermore, the patient had neuropsychological testing demonstrating problems to which I will refer you for more information.

(Ex. 116A-2).

Dr. Burton did not address the different levels of susceptibility that individuals have to chemical exposures. Dr. Bass, among others, explained that "even small concentrations [of toxic chemicals], depending on the patient and his ability to respond to that insult, could be variable." (Ex. 122-17). Given the great weight of medical and scientific evidence supporting compensability of the claim, Dr. Burton's personal bias against toxic exposure injuries became apparent when he stated that it was an "absurdity" to conclude that claimant "could have been exposed to TCP in any form that could result in toxicity." (Ex. 139A-2). Even if Dr. Burton had a reasonable disagreement with the many physicians and scientific articles that found a causal relationship between TCP-related toxic fume events and neurocognitive injuries, there was nothing "absurd" about that opposing point of view. Knowing that airlines routinely prevented the installation of cabin air quality monitoring equipment and knowing that the animal TOCP ingestion studies were not relevant to human fume event inhalation cases, Dr. Burton's outrage that any medical provider could assess potential human toxic exposure without performing an adequate exposure assessment appeared disingenuous (Ex. 139A-4). Dr. Burton's dismissal of Dr. Abou-Donia's autoantibodies test as "junk science" was also overwrought and not supported by the more persuasive expert medical opinions (Ex. 139A-6). His sarcastic assertion that Dr. Abou-Donia's conclusions amounted to saying, "I don't know or understand what's going on so it must be related to my ill-conceived theory," revealed personal animus toward one of the preeminent experts in the field of airline toxic chemical exposure (Ex. 139A-6). Dr. Burton's attempt to create plausible deniability regarding causation by relying on the airline industry's

refusal to allow air quality monitoring equipment on planes was not persuasive either. Despite the lack of specific fume event air quality measurements resulting from the airline industry's ongoing pattern of obstruction, it was well known in the scientific literature and with claimant's treating and consulting physicians that many jet engine oil components and their pyrolyzed compounds were toxic and a significant number of airline crew members beside claimant suffered both short-term respiratory and long-term cognitive symptoms following specific fume events (Harrison, Abou-Donia, Michaelis & Anderson testimony). It was not necessary to know the exact amount of TOCP inhaled by a crew member to understand that a causal relationship could be established between the inhalation of burning toxic chemical jet engine oil fumes and physical injuries. As claimant's counsel explained, "*** The assertion of [Drs. Burton and Pleus] that causation can never be established without exact dose information creates an impossible and legally unnecessary burden of proof." (Claimant's Reply Argument, page 4).

Dr. Burton's opinions were not persuasive because they were inconsistent, they did not account for claimant's low level exposures to toxic fumes for many years, and they did not account for the many different toxic chemicals in jet engine oil which multiplied during pyrolysis. Dr. Harrison pointed out Dr. Burton's failure to take into account the difference in volatility between inert and highly heated substances (Ex. 139-55). Also, Dr. Burton did not adequately consider the toxic effect of UFPs (Nano-particles) or claimant's personal susceptibility to lower levels of chemical exposure. Industrial Hygienist Anderson testified that increased nano-particles in the airplane's contaminated air supply facilitated the transport of neurotoxic particles from the pyrolyzed oil. Dr. Burton's opinion, however, was single-mindedly focused on claimant's exposure to TCP and TOCP without considering many other relevant factors or toxic chemical compounds. Moreover, Dr. Burton's reliance on animal studies which involved the ingestion of TCP was misplaced because inhalation is much more damaging than ingestion. It was clear that the ingestion studies underestimated the risks of chemical inhalation (Ex. 137-58).

There was ample evidence in the scientific literature and from claimant's treating and consulting medical providers that exposure to toxic chemical fumes was associated with neurological and cognitive dysfunction. Thus, Dr. Burton's opinion that "Aerotoxic Syndrome" was "a myth" was incorrect. In fact, his belief that claimant's post-exposure symptoms were psychological in nature was consistently dismissed in the scientific literature and by all of claimant's treating and consulting physicians as a likely cause of post-exposure cognitive symptoms. Therefore, Dr. Burton's belief that individuals exhibiting neurocognitive symptoms following fume

events could be explained solely in terms of psychological conditions was more likely the myth than a chemical poisoning explanation for symptoms associated with “Aerotoxic Syndrome.” The failure of Drs. Burton and Bell to acknowledge the existence of multiple layers of objective medical evidence of injury was a critical flaw in their analysis and leads me to reject their opinions that the medical records lacked objective findings of an injury. *Mack R. Neal*, 72 Van Natta 314, 318 (2020), citing *Cornelio Garcia*, 67 Van Natta 893, 897 (2015) (where a physician’s opinion disputed the existence of the claimed condition, but did not explain the presence of objective findings, opinion was found unpersuasive).

The IME physician, Dr. Pleus, was another toxicologist who authored a lengthy report on behalf of the employer, but he did not have the benefit of examining claimant, let alone treating him on a long-term basis. Instead, his opinions regarding causation were based entirely on a review of claimant’s medical file, leaving him at a distinct disadvantage compared to claimant’s examining and consulting physicians in determining causation (Ex. 141-5). Dr. Pleus testified at the hearing that he had been an expert witness in other airline toxic fume cases, but he always testified on behalf of the airlines (Ex. 150-220). In fact, he acknowledged never rendering an opinion that any airline personnel, pilot or flight attendant had ever been adversely affected by a fume event (Ex. 150-221). Industrial Hygienist Anderson was familiar with Dr. Pleus and testified that he was hired by many airlines to discredit crew members’ complaints about fume events. In the face of daunting medical and scientific evidence to the contrary, Dr. Pleus’ inability to conceive of an airline toxic chemical fume event that could cause physical injury suggested professional bias.

While Dr. Pleus’ education and credentials were impressive, his lengthy analysis of the case was riddled with flaws, both large and small. First, Dr. Pleus found no evidence that claimant was exposed to toxic chemical fumes on January 21, 2017, when the employer conceded that fact by accepting the claim for “acute toxic inhalation” and “acute chemical inhalation” as a disabling industrial injury (Ex. 113 & Ex. 141, pages 48-49, 51 & 62).³⁸ Contrary to a preponderance of the legal and medical evidence, Dr. Pleus believed it was pure speculation that claimant was not just “exposed to only one dose, however, he was exposed to low-level phosphates for 25 years.” (Ex. 141-52). Also, Dr. Pleus believed there was no scientific literature that suggested toxic exposures on airplanes caused symptoms or evidence that claimant’s exposure was sufficient to cause any adverse effects (Ex.

³⁸ Although Dr. Pleus used this explanation to criticize Dr. Abou-Donia’s autoantibodies test, the rationale can be applied equally to Dr. Pleus’ view of the fume event itself. He stated, “Since exposure is not quantified, any reported neurological effects cannot be related to exposures.” (Ex. 141-56).

141, pages 48 & 58). There were, however, so many instances of airline fume event-associated symptoms and so many scientific studies regarding that association that the term “Aerotoxic Syndrome” was coined in 2000. According to the scientific literature, there have been approximately 15,000 documented cases of flight crew members developing neurological problems following toxic gas exposure (Ex. 120-3). Also, in this particular case, the medical evidence overwhelmingly established that claimant experienced significant respiratory symptoms, headaches, and cognitive difficulties as a result of the toxic exposure. For that reason, claimant’s initial treating physicians immediately diagnosed numerous fume event-related conditions. The First Officer’s severe headache and respiratory difficulties during and after the fume event were consistent with claimant’s physical responses, albeit less severe. Revealing a small breach in his otherwise unwavering defense of the airline industry, Dr. Pleus conceded it was “possible that [claimant] was exposed to low concentrations of combustion by-products for a short time, which might explain his initial reactions.” (Ex. 141-6).

Second, Dr. Pleus focused his attention on the effects of TCP and its isomers and offered only conclusory opinions regarding the effects of other pyrolyzed chemical compounds (Ex. 141, pages 6, 25, 42, 46, & 48). The more persuasive medical and scientific evidence, however, established that the chemical fumes claimant inhaled contained many toxic compounds that were created during the oil pyrolyzation process. Even Dr. Bass, who lacked specific expertise in toxicology but was a long-time ICU physician, explained that the toxic organophosphates in jet engine oil can be broken down further by heat when they pass through the engine, are split into toxic nanoparticles, and contaminate the cabin air that way as well (Ex. 122-15).

Third, Dr. Pleus relied heavily on hen TCP-ingestion studies when the evidence established that those ingestion studies were not relevant in evaluating toxic chemical inhalation cases (Ex. 134-40, Ex. 139, pages 54-55 & 381, and Ex. 141-33). Even Dr. Pleus conceded that he could not extrapolate any relevant information from such studies (Ex. 141-44).

Fourth, Dr. Pleus believed that claimant’s symptoms were nonspecific to exposure to identified jet engine oil components and did not correspond to objective findings from medical testing (Ex. 141-6). On the contrary, claimant’s initial nerve conduction study, consistent clinical findings over time, the PET scan of his brain, the autoantibodies test, as well as the three neurocognitive tests conducted by Drs. Schock and Dr. Kreiling, all constituted objective evidence of a neurological injury. While claimant experienced a broad range of symptoms, they were consistent with

many other airline fume event cases documented in the scientific literature and seen repeatedly by Drs. Harrison and Abou-Donia in their medical careers.

Fifth, while Dr. Pleus acknowledged the existence of variable responses to chemical exposures, he did not adequately address claimant's particular susceptibility, especially given his 25-year history of flying and chronic low level exposure to jet engine oil fumes. Instead, Dr. Pleus summarily dismissed that idea at hearing, testifying that claimant did not have any pre-existing susceptibilities.

Finally, Dr. Pleus was convinced that organophosphate-induced delayed neuropathy (OPIDN) was the only condition that could be caused by a toxic fume event when both the central nervous system and peripheral nervous system were effected by fume events which resulted in a wide range of symptoms and lesser conditions (Dr. Michaelis testimony).

Dr. Pleus over-focused on the effects of TCP in hen ingestion studies, the cause of OPIDN, and an exact measurement of TCP exposure during claimant's fume event. He also erroneously believed there was no research regarding the adverse effects of nanoparticles and he lacked clinical familiarity with claimant because he did not conduct an examination. All of those shortcomings, in addition to the problems described above, prevented him from completely and correctly analyzing the causal relationship between claimant's toxic fume inhalation event and his subsequent symptoms. As Dr. Pleus conceded at hearing, the persuasive value of a report is lessened if critical pieces of data or medical information are omitted.

Dr. Harrison summed up his disagreement with the IME's over-focus on obtaining an exact measurement of claimant's toxic exposure by agreeing with the following paragraph on March 4, 2020:

*** [I]t would certainly be better if we knew exactly what Captain Myers was exposed to. However, the fact that we do not have exact measurements does not prevent you from providing a medical opinion to a reasonable and medical probability. From a medical standpoint, Captain Myers has a neurocognitive disorder, toxic encephalopathy, and visual disturbance. These medical conditions were likely caused in major part by his toxic exposure. This conclusion is based upon the established fact that the toxic exposure did take place, the established medical fact that Captain Myers does have these conditions, and the science and research that establishes that the ortho component of

tr cresyl-phosphate is not the only potential harmful component. The other isomers including the meta and para isomers can be harmful, this harm is increased when the isomers are combined and the harm is further increased when the oil is pyrolyzed. Therefore, even though you don't know the exact composition of the fumes that Captain Myers was exposed to, you are nevertheless able to render a medical opinion to a reasonable and medical probability based upon these facts and this science.

(Ex. 149-5).

Dr. Harrison persuasively rebutted Dr. Pleus' assumption of claimant's maximum toxic chemical exposure, which was based on air quality measurements that had been taken on planes with no fume events and after the fact on planes where fume events had taken place. *Id.* Specifically, Dr. Harrison concluded that air samples taken on planes *following* fume events did not indicate the quality and composition of the fumes *during* the fume event itself, every fume event was different regarding the maximum toxic chemical exposure, and combining a few isolated measurements taken on different planes at different times under different conditions did not correlate with claimant's toxic exposure or prove what happened during his fume event. *Id.* In Dr. Harrison's opinion, it was impossible to make an accurate quantitative assessment of the exact exposure or the maximum exposure, and the guesswork that led to Dr. Pleus' conclusions regarding the cause of claimant's conditions was not sound science (Ex. 149, pages 5-6). In light of the wealth of information available in this case, Dr. Harrison's qualitative assessment was that the January 21, 2017 toxic fume event, which did in fact take place, was the major contributing cause of claimant's physiological and neurological diagnoses (Ex. 149-6).

e. Conclusions

The employer's assertion that claimant's theory of the case was premised on *post hoc ergo propter hoc* ("after this therefore because of this") was not persuasive (Employer's Closing Argument, page 52). Claimant's counsel responded to that argument by stating,

The employer proudly trots out some Latin "*post hoc ergo propter hoc*" to suggest that our claim must fail because we are simply relying on the fact that B followed A and therefore A must have caused B. This ignores the third piece of the bookend

argument, the medical and science causation. This argument would have merit if we offered no medical evidence and offered no scientific evidence, and our case simply consisted of our assertion that our guy is messed up and since this happened after the fume event it must have been caused by the fume event. This argument ignores hundreds of pages of medical evidence and testimony, and thousands of pages of scientific evidence and testimony that we provided regarding causation. ***

(Claimant's Reply Argument, page 32).

Although claimant certainly emphasized the fact that he was a happy, athletic, hard-working pilot and expert marksman before the industrial injury and was mentally and physically debilitated afterward, he presented a mountain of persuasive medical evidence that was not based solely on a temporal relationship between the toxic fume event and his conditions. For example, Dr. Schloesser based his expert medical opinions on a number of factors, including: (1) Organophosphate toxicity was well established in detail in the medical literature, (2) he treated claimant before the industrial injury and there was a significant clinical change following the fume event, (3) claimant had no pre-existing problems immediately before the fume event, (4) the nerve conduction study he performed, which showed evidence of peripheral neuropathy, was not flawed, (5) there was other objective evidence of physical injury including a PET scan and neuro-psychological testing, (6) claimant was an affable and happy pilot without any history suggesting a tendency toward malingering or secondary gain prior to the fume event, and (7) claimant had no injuries after the fume event which would explain his persistent symptoms (Ex. 116, pages 1-2). Claimant's other treating and examining physicians also based their opinions on claimant's pre- and post-injury presentations, his clinical and objective examination findings, the mechanism of injury, and the scientific research in the area of airplane toxic fume exposure. This was not a case where claimant failed to carry his burden of proof because the expert medical opinions were based solely on a temporal relationship.

Claimant was exposed to a wide variety of toxic chemicals during the fume event on January 21, 2017. Claimant's counsel aptly described it as a "primordial soup of toxins." (Claimant's Reply Argument, page 24). The more persuasive expert medical and scientific evidence, as well as the legal posture, established that claimant inhaled toxic chemical fumes in the workplace which resulted in significant physical injuries and the development of short- and long-term symptoms caused by toxic encephalopathy and a mild neural cognitive disorder. The opinions of

claimant's treating physicians were also consistent with the Bradford Hill causation criteria. Despite the lack of data regarding a specific dose-response relation as a result of an absence of on-board air quality monitoring equipment, the Bradford Hill causation criteria were met in eight of nine categories, thereby establishing a causal relationship between fume event chemical exposure and symptoms (Ex. 141A-11; *see also* Ex. 126-18).³⁹ Based on the evidence as a whole, I conclude that the file reviews of Drs. Pleus and Burton, as well as the one-time examination of Dr. Bell, were flawed because they were based on incomplete or inaccurate information. The opinions of those IME physicians were not nearly as persuasive as the opinions of claimant's treating physicians, Drs. Ugalde and Schloesser, his consulting physicians, Drs. Kaniecki, Bass, Schock, Abou-Donia and Harrison, the IME physician, Dr. Kaniecki, or the other consulting experts, Dr. Michaelis and Ms. Anderson. Dr. Harrison addressed the bottom line on causation, stating,

And so what is the more probable chain of causation here? Is it more probable that a neurotoxic exposure leads to neurotoxic effects or is it more probable that neurotoxic effects just happened to occur within exactly the same timeframe that I would expect after a toxic chemical exposure from idiopathic causes? Number one is much more likely than number two. And that's the methodology that I write about, that I teach that's standard in occupational medicine. That's why people come to see me rather than to their primary care provider, who doesn't know much about toxic chemical exposures.

(Ex. 139, pages 20-30). Based on the more persuasive expert medical evidence, the extensive scientific literature regarding the effects of toxic fume events, and Dr. Harrison's bottom line in the context of all the evidence, claimant has established that his January 21, 2017 toxic chemical fume event at work was a material contributing cause of his toxic encephalopathy and mild neural cognitive disorder. Therefore, even if the employer's February 20, 2019 and April 2, 2019 denials of compensability of those two conditions were procedurally proper, they would be set aside on the merits.

³⁹ The "Hill Criteria" are a set of nine questions that scientists can ask themselves to assist in the assessment of a cause and effect relationship (Ex. 141-56). They are: (1) the demonstration of a strong association between the causative agent and the outcome, (2) consistency of the findings across research sites and methodologies, (3) the demonstration of specificity of the causative agent in terms of the outcomes it produces, (4) the demonstration of the appropriate temporal sequence, so that the causative agent occurs prior to the outcome (*i.e.*, temporality), (5) the demonstration of a biological gradient, in which more of the causative agent leads to a poorer outcome, (6) the demonstration of a biologic rationale, such that it makes sense that the causative agent causes the outcome, (7) coherence of the findings, such that the causative argument is in agreement with what we already know, (8) experimental evidence, and (9) evidence from analogous conditions (*i.e.* analogy). *Id.*

3. Compensability of visual conditions including convergence insufficiency and saccadic eye movement deficiency (Ex. 116)

Claimant contends that the industrial injury was either a material contributing cause of his visual dysfunction as a direct sequelae or the industrial injury was the major contributing cause of his visual dysfunction as a compensable consequence of his toxic encephalopathy and neural cognitive disorder. The employer asserts that claimant does not have any visual dysfunction and, if he does, the industrial injury is neither a material cause nor the major cause of those conditions.

A preponderance of the procedural and substantive evidence established that claimant's January 21, 2017 toxic chemical fume event resulted in compensable toxic encephalopathy and a mild neural cognitive disorder. See "Scope of Acceptance" and "Compensability" sections above. Therefore, claimant need only prove that those compensable conditions were either a direct and material cause of his vision diagnoses or the indirect and major contributing cause of those consequential conditions. ORS 656.005(7)(a)(A) provides that no injury or disease is compensable as a consequence of a compensable injury unless the compensable injury is the major contributing cause of the consequential condition. See *Albany General Hospital v. Gasperino*, 113 Or App 411 (1992) (holding that, when a condition or need for treatment is caused by the compensable condition, as opposed to the industrial accident, the major contributing cause standard is applied). Thus, in this case, if a consequential condition analysis is appropriate, claimant must prove by a preponderance of the evidence that his toxic encephalopathy and/or mild neural cognitive disorder were the major cause of his saccadic eye movement deficiency and convergence insufficiency. *Charlotte M. Ashford*, 49 Van Natta 2172 (1997).

Based on claimant's objective evidence of visual impairment and the temporal relationship with the toxic fume event, claimant's treating optometrist, Dr. Scott, stated on April 3, 2019, "It is therefore frankly impossible that [claimant] could have had this condition prior to his toxic exposure and still be a marksman and airline pilot. Rather, the before and after picture supports a very strong temporal relationship, in that this condition developed following the toxic exposure. ***" (Ex. 117-4). Thus, Dr. Scott concluded that claimant's need for treatment, including diagnoses of convergence insufficiency and saccadic eye movement deficiency, were caused in major part by the January 21, 2017 work incident (Exs. 117 & 138-1). On September 18, 2019, Dr. Scott also concluded that, although claimant's visual performance deficits were consistent with the effects of a brain injury, as an optometrist she was not qualified to determine whether claimant's visual performance deficits were caused by exposure to a harmful level of toxic chemicals

(Ex. 138-1). Dr. Scott deferred to the specialists to answer that question (Exs. 138-2, 140-5 & 142A, pages 23 & 39). However, on October 7, 2019, Dr. Scott decided to offer her opinion regarding causation and concluded that claimant's vision diagnoses were due in major part to the toxic fume exposure he endured on January 21, 2017 (Ex. 140-4). During her deposition, Dr. Scott explained that she did not know what kinds of toxic chemicals claimant inhaled on January 21, 2017 (Ex. 142A, pages 18, 31 & 35). Nevertheless, she continued to conclude that claimant's toxic exposure caused his vision diagnoses (Ex. 142A-21). She explained, "*** I know that those conditions [saccadic eye movement deficiency and convergence insufficiency] develop after brain injury. So with his diagnosis from Dr. Ugalde of the toxic encephalopathy, I made that jump, yes." (Ex. 142A, pages 21-22). She further explained that claimant could not have worked before the fume event as a pilot with the brain injury-related vision problems he had, those vision problems were not related to pre-existing migraines, she knew there was a toxic exposure event, and there was brain damage afterward (Ex. 142A, pages 33-35). Relying in part on Dr. Ugalde's conclusion that claimant's chemical exposure caused toxic encephalopathy, Dr. Scott made the only link she could make, *i.e.*, the fume event caused claimant's visual dysfunction (Ex. 142A-35). It was reasonable for Dr. Scott to rely on the expertise of other physicians to determine if claimant's toxic chemical exposure resulted in toxic encephalopathy. It was also reasonable for Dr. Scott to arrive at her own conclusions regarding the cause of claimant's vision dysfunction based on those assumptions.

Dr. Harrison explained that saccadic eye movement, neuropsychological impairment, and difficulties with balance were frequent manifestations of the type of toxic poisoning claimant experienced during the airplane engine run-ups (Ex. 132-3). He subsequently concluded that, based on his examination of claimant along with his experience and expertise treating airline personnel following toxic fume inhalation, claimant's saccadic eye movement deficiency and convergence insufficiency with respect to his vision were real, objectively verifiable conditions that were likely caused by toxic exposure (Ex. 149-2).

Dr. Kaniecki concluded that saccadic eye movement deficiencies and convergence insufficiency were often seen with traumatic brain injuries and toxic exposure events (Ex. 138A-35). He also indicated that patients could not fake those conditions. *Id.*

Dr. Michaelis testified that claimant's vision dysfunction following the fume event was consistent with many other crew members who also experienced vision problems after fume events. Based on her expertise and review of the medical

evidence, Dr. Michaelis concluded that claimant's toxic fume inhalation very probably caused those diagnosed vision conditions (Michaelis testimony).

Dr. Bell did not offer an opinion regarding the cause of claimant's visual dysfunction. She stated, "I am not an ophthalmologist and therefore, I am not able to address the proposed diagnosis of convergence insufficiency and 'saccadic eye movement'. These conditions should be evaluated by an IME ophthalmologist." (Ex. 134-39). To the extent that Drs. Burton and Pleus did not believe that claimant's toxic encephalopathy and mild neural cognitive disorder were compensable, their opinions regarding the vision diagnoses were based on inaccurate information and were not persuasive.

Although Dr. Scott conceded that she did not have the expertise to determine whether claimant's toxic exposure resulted in toxic encephalopathy, she relied on Dr. Ugalde's expert medical opinion to make that connection. Thus, assuming that the fume event resulted in a compensable toxic encephalopathy condition, Dr. Scott concluded that claimant's toxic fume-induced brain damage was the major cause of his vision disorders diagnosed as convergence insufficiency and saccadic eye movement deficiency. Dr. Scott's opinion was thorough and well-reasoned. It was also consistent with the expert opinions of Drs. Harrison, Kaniecki and Michaelis. Based on that evidence, claimant has established that his convergence insufficiency and saccadic eye movement deficiency were compensable under either a direct injury or consequential condition theory. Therefore, even if the employer's April 2, 2019 denial of compensability of those two vision disorders was procedurally proper, it would be set aside on the merits.

4. "Current condition" denial (Ex. 110)

Claimant contends that his compensable injury remains a material contributing cause of his current condition and the employer's "current condition" denial should be set aside. The employer asserts that the more persuasive expert medical evidence established that the compensable injury was no longer a material contributing cause of claimant's current condition and its denial should be approved.

On February 23, 2017, the employer accepted claimant's "acute chemical inhalation" and, on February 6, 2019, it accepted "acute toxic inhalation," both as disabling industrial injuries (Exs. 35 & 107). Those acceptances encompassed claimant's toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency (*See* "Scope of Acceptance" section above). Even if the employer did not accept claimant's new/omitted medical

condition claims, the persuasive expert medical evidence established that the conditions diagnosed as toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency were compensably related to the January 21, 2017 toxic chemical inhalation event. Therefore, the employer's February 8, 2019 "current condition" denial will be analyzed in that context. Before addressing the merits of the "current condition" denial, it is also incumbent upon me to determine whether that denial was procedurally proper.

The chronology of the claim processing events occurred as follows:

1. February 23, 2017 acceptance of "acute chemical inhalation" (Ex. 35),
2. February 6, 2019 acceptance of "acute toxic inhalation" (Ex. 107),
3. February 6, 2019 Notice of Closure (Ex. 108),
4. February 8, 2019 denial of current condition (110),
5. February 14, 2019 claimant's expansion request to accept toxic encephalopathy as a new or omitted medical condition (Ex. 111A),
6. February 20, 2019 denial of toxic encephalopathy (Ex. 113),
7. March 21, 2019 claimant's expansion request to accept mild neural cognitive disorder, polyneuropathy, convergence insufficiency, and saccadic eye movement deficiency (Ex. 114B),
8. April 2, 2019 denial of mild neural cognitive disorder, polyneuropathy, convergence insufficiency, and saccadic eye movement deficiency (Ex. 116).

Throughout that period, claimant was disabled and continued to seek medical treatment for a myriad of symptoms. The claim was closed on February 6, 2019, and the employer denied claimant's "current condition" two days later, on February 8, 2019, but before claimant filed his new or omitted medical condition claims (Exs. 108, 110, 111A & 114B). Since the employer denied compensability of claimant's "current condition" after claim closure, it was not required to accept a combined condition before issuing the "current condition" denial. *See Jeffrey T. Wagner, 71 Van Natta 790, 791 (2019)* (the Board held that, since the employer did not accept the claimed combined conditions or issue a pre-closure denial of the combined conditions, its denial was procedurally proper); *compare Croman Corp. v. Serrano, 163 Or App 136 (1999)* (before a carrier may issue a pre-closure denial under ORS 656.262(6)(c) and ORS 656.262(7)(b), it must have first accepted the combined condition). That conclusion, however, does not end the inquiry regarding the procedural propriety of the employer's "current condition" denial.

- a. “Current condition” denial issued before new or omitted medical condition claim made

The February 8, 2019 “current condition” denial stated, in relevant part,

Your claim was previously accepted for disabling acute chemical inhalation. A Notice of Closure on your disabling claim issued February 6, 2019. A preponderance of medical evidence indicates your accepted acute chemical inhalation resolved and is no longer materially contributing to any disability or need for medical treatment. We therefore deny that current condition, disability, or need for medical treatment are compensably related to the accepted acute chemical inhalation.

(Ex. 110).

In order to determine whether the employer’s denial was procedurally proper, I first must decide what the employer’s denial purported to deny. *Barbara J. Ferguson*, 63 Van Natta 2253, 2257 (2011), citing *Cervantes v. Liberty Northwest Ins. Corp.*, 205 Or App 316, 322 (2006) (“[I]t is highly unlikely that an insurer would issue a denial with the purpose of denying nothing.”). The employer’s denial in *Ferguson*, *supra* at 2256, contained the following statements: “(1) claimant’s accepted lumbar strain ‘is no longer a material contributing cause of [her] ongoing symptoms, or need for treatment’; (2) the accepted lumbar strain ‘is medically stationary with no permanent impairment’; and (3) ‘[t]herefore, [the employer denied claimant’s] current condition.’” Based on those facts, the Board held that the employer improperly denied claimant’s current condition in the absence of a claim being made for a new or omitted medical condition. *Ferguson*, *supra* at 2257. The Board explained,

*** By its terms, the employer has not denied a medical service, but a “condition.” [citation omitted] As set forth above, because claimant has not filed a new/omitted medical condition claim, any denial of such an unclaimed “condition” would be premature and invalid. *Altamirano*, 133 Or App at 19-20; *Lauri A. Chambers*, 63 Van Natta 1322, 1325 n 4 (2011); *Charles L. Kachel, Sr.*, 56 Van Natta 3842, 3847 (2004); *Tony Cervantes, Jr.*, 56 Van Natta 2054, 2056, *rev’d on other grounds*, 205 Or App 316 (2006); *Guillermo Ruvalcaba*, 51 Van Natta 313, 315

(1999). Yet, that is the most reasonable interpretation of the employer's denial – *i.e.*, a denial of an unidentified *unclaimed* “current condition” that is “separate and severable” from the accepted lumbar strain. (*See Ex. 50-1*). Because such a denial of an unclaimed condition is prohibited, we affirm the ALJ's order setting aside the employer's denial.

Ferguson, supra at 2257.

In the present case, the employer's February 8, 2019 denial stated, in relevant part, “We therefore *deny that your current condition*, disability, or need for medical treatment are compensably related to the accepted acute chemical inhalation.” (Ex. 110; emphasis added). According to the express language of its denial, the employer denied compensability of claimant's “current condition” which it believed was “separate and severable” from the accepted “acute chemical inhalation” claim.⁴⁰ At that time, claimant had not made any other claims for new or omitted medical conditions. Instead, his expansion requests were made *after* the “current condition” denial issued (Exs. 111A & 114B).⁴¹ Therefore, when the denial issued, there were no outstanding claims for conditions. In accordance with the Board's decision in *Ferguson, supra*, the employer's denial of claimant's “current condition” in this case was procedurally improper because claimant had not made any claims for new or omitted medical conditions at that time and its denial of an unclaimed “condition” was premature and invalid.⁴² Consequently, the employer's “current condition” denial is set aside as procedurally improper.

⁴⁰ The employer did not state that claimant's “current condition” was unrelated to his accepted “acute toxic inhalation,” which was a separate condition from his “acute chemical inhalation” and resulted in significant physical injury.

⁴¹ Under ORS 656.267(1), a worker must request acceptance of a new or omitted medical condition from the carrier. The statute does not allow anyone other than the “worker” to file a new or omitted medical condition claim. *Andria D. Costello*, 55 Van Natta 498 (2003), *aff'd without opinion*, 193 Or App 484 (2004) (no legislative intent to allow physicians to file a new or omitted medical condition claim on behalf of workers). In contrast, a physician's report may constitute an *initial* claim, which triggers a carrier's claim processing obligations. *Paris Jennings*, 68 Van Natta 322, 324 (2016).

⁴² In *Penny I. Cooper*, 64 Van Natta 1644, 1648 (2012), the Board explained the circumstances required for a “current condition” medical services denial to be procedurally permissible, stating,

Thus, although a “current condition” medical services denial is permissible, it must nevertheless be issued in response to a “current claimed need for treatment.” *See [Altamirano v. Woodburn Nursery*, 133 Or App 16, 19-20 (1995)]. In other words, there must be a medical services “claim” for the employer to deny; otherwise, the denial is a nullity. *See id.*; *Barbara J. Ferguson*, 63 Van Natta 2253, 2255 (2011); *compare William E. Hamilton*, 41 Van Natta 2195, 2198 (1989) (medical services denial issued in the absence of a medical services claim set aside as a nullity), *with Rodney Danielson*, 60 Van Natta 1978, 1981-82 (2008) (the employer's receipt of an 827 Form, in conjunction with the accompanying chart notes and medical bills, sufficient to establish a “claim” for medical services; therefore, the employer's denial of “current medical treatment” constituted a valid medical services denial that needed to be addressed on the merits).

See also Altamirano v. Woodburn Nursery, 133 Or App 16, 19-20 (1995); *Boise Cascade Corp. v. Hasslen*, 108 Or App. 605 (1991); *Green Thumb, Inc. v. Basl*, 106 Or App. 98 (1991). However, these cases are distinguishable from the facts of the current

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Even if claimant had made his new/omitted medical condition claim *before* the employer's "current condition" denial issued, the denial would still not be procedurally proper because the employer had already accepted the denied conditions of toxic encephalopathy, mild neural cognitive disorder, saccadic eye movement deficiency, and convergence insufficiency. In short, the employer's acceptance of a mechanism of injury or a vague/ambiguous condition resulted in the actual acceptance of claimant's toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency. Even if the employer accepted a medical condition, the definition of "acute toxic inhalation" included significant injuries resulting from the toxic chemical inhalation event which encompassed the denied conditions (*See* "Scope of Acceptance" section above). Therefore, claimant's expansion request to include those previously accepted conditions was redundant and the new/omitted medical condition denial was void.

b. Denial referred only to "acute chemical inhalation," not "acute toxic inhalation," as the compensable injury

The employer's "current condition" denial only indicated that claimant's "current condition" was no longer compensably related to his "acute chemical inhalation," omitting any reference to his accepted "acute toxic inhalation" claim (Ex. 110). Those were two different conditions and the "acute toxic inhalation" claim encompassed claimant's significant physical injuries while the "acute chemical inhalation" claim constituted a less severe form of the injury (*See* "Scope of Acceptance" section above). Since the employer's "current condition" denial did not include claimant's "acute toxic inhalation" and that term was different than "acute chemical inhalation" in that "acute toxic inhalation" resulted in significant physical damage that included his toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency according to the contemporaneous medical evidence, the "current condition" denial did not terminate the employer's responsibility for claimant's ongoing disability and need for treatment regarding those conditions. Inasmuch as the employer's "current condition" denial purported to deny claimant's "otherwise compensable condition" but failed to do so by omitting his "acute toxic inhalation," it missed the mark and would again be set aside as procedurally improper.

matter because the employer's denial went beyond a "current condition" medical services only denial (which would have only required a request for medical services) to deny claimant's "current condition" which required claimant to first make a claim for a new or omitted medical condition.

c. Compensability of current condition

Assuming, *arguendo*, that the employer's February 8, 2019 "current condition" denial was procedurally proper, I would still set it aside on the merits because there was no persuasive expert medical evidence establishing that the compensable conditions resolved over time.

On April 8, 2019, Dr. Ugalde agreed with the following statement regarding claimant's then-current condition,

*** [Claimant] does in fact have significant permanent impairment as a starting point. Second, you would not expect for his condition to miraculously change or improve and have no indication that that has happened. Rather, you expect that these effects would essentially be permanent effects. There has been no change in his condition that justifies denying his claim by contending that his condition has stopped being work related. His current condition is still due to the toxic exposure.

(Ex. 118-5; underline in original). During her deposition, Dr. Ugalde also concluded that claimant's condition did not change in 2019 (Ex. 131-22). Claimant's other treating and consulting physicians expressed similar sentiments. To the extent the IME physicians, Drs. Burton, Bell, and Pleus, believed that the fume event did not result in any physical injuries, their opinions were legally and factually incorrect and are not relevant in determining whether claimant's compensable conditions resolved in the context of the "current condition" denial.

5. Causal relationship between compensable condition and acupuncture treatments – August 16, 2018 Transfer Order (Ex. 98A)

Claimant contends that his request for 24 acupuncture visits was materially related to his compensable injury and I should find that a causal relationship between the two was established here. The employer asserts that no persuasive expert medical evidence was presented to establish that causal connection and claimant's request should be denied.

ORS 656.704(3)(b)(C) provides, "Any dispute that requires a determination of whether a sufficient causal relationship exists between medical services and an accepted claim to establish compensability is a matter concerning a claim. A carrier

must cause to be provided medical services for “ordinary” conditions “caused in material part” by a compensable injury. ORS 656.245(1)(a); *SAIF v. Sprague*, 346 Or 661, 672 (2009). The phrase “in material part” means a “fact of consequence.” *Mize v. Comcast Corp.-AT&T Broadband*, 208 Or App 563, 569-70 (2006).

Dr. Ugalde referred claimant for acupuncture treatments to reduce his headaches (Ex. 39-5). Dr. Kaniecki treated claimant’s headaches and concluded that those headaches were due to claimant’s toxic fume exposure on January 21, 2017 (Ex. 97-3 & 138A-45). During his September 23, 2019 deposition, Dr. Kaniecki confirmed his opinion that claimant’s fume event was the major contributing cause of his toxic encephalopathy and post-traumatic headaches (Ex. 138A, pages 37 & 45). For the same reasons that claimant’s denied conditions were found compensable on the merits in the sections above, the persuasive expert medical evidence established that the industrial injury was the major contributing cause of claimant’s headaches and need for acupuncture treatments. In addition, the employer had previously accepted claimant’s headaches as a sequela of the accepted toxic encephalopathy claim (*See* “Scope of Acceptance” section above). For all those reasons, claimant has established the requisite causal connection between the industrial injury and the requested medical treatment.

The Hearings Division does not have jurisdiction to award an assessed attorney fee under ORS 656.385 because such proceedings are before the Director.⁴³ *Antonio L. Martinez*, 58 Van Natta 1814, 1822, *aff’d SAIF v. Martinez*, 219 Or App 182 (2008). In addition, claimant is not entitled to an attorney fee under ORS 656.386(1) *at this time* for the efforts of his attorney in establishing the requisite causal connection between his compensable injury and the requested acupuncture visits. In *AIG Claim Services v. Cole*, 200 Or App 170, 178-79 (2006), the court concluded that a fee under ORS 656.386(1) is awarded only when a claimant “prevails finally” over a denied claim. A claimant does not “prevail finally” until both aspects of a challenge to a medical services claim (the causal relationship under ORS 656.704(3)(b)(C) and whether the medical services are medically appropriate under ORS 656.704(3)(b)(B)) have been decided in favor of claimant. Because this proceeding pertains only to the causal relationship under ORS 656.704(3)(b)(C), claimant has not yet “prevailed” on the medical services claim and, therefore, he is not entitled to an attorney fee under ORS 656.386(1) *at this time*. *Antonio L. Martinez*, 58 Van Natta at 1822, *aff’d SAIF v. Martinez*, 219 Or App 182 (2008).

⁴³ ORS 656.385(1) provides that the Administrative Law Judge shall require the insurer to pay a reasonable attorney fee to claimant’s attorney when claimant prevails in a dispute of compensation benefits or medical services pursuant to ORS 656.245.

Furthermore, the award of an assessed attorney fee under ORS 656.382(2) requires a determination that the “compensation awarded to a claimant should not be disallowed or reduced[.]” *Antonio L. Martinez*, 58 Van Natta at 1822-23, *aff’d SAIF v. Martinez*, 219 Or App 182 (2008). Because this order does not determine whether claimant has “prevailed,” I cannot determine whether his compensation will be disallowed or reduced. Claimant is, therefore, not entitled to an assessed fee for his attorney’s services *at this time* under ORS 656.382(2) either. *Antonio L. Martinez*, 58 Van Natta at 1822, *aff’d SAIF v. Martinez*, 219 Or App 182 (2008).

In *Steven R. Cummings*, 57 Van Natta 2223 (2005), the Board observed that the court had conditionally granted the claimant’s counsel a fee for services on judicial review in the event that he prevailed on remand; because the claimant prevailed on his denied claim after remand, it held that he was entitled to the attorney fee award conditionally granted by the court. *Id.* at 2230. In light of *Cummings*, as well as other Board precedent, the Board in *Antonio L. Martinez*, 58 Van Natta at 1822, *aff’d SAIF v. Martinez*, 219 Or App 182 (2008), concluded there was legal authority for a “contingent” attorney fee. *See David Converse*, 50 Van Natta 2067 (1998) (court remanded on merits and granted the claimant a specified attorney fee for services rendered on judicial review, conditioned on the claimant prevailing on remand; Board found claim compensable on remand and awarded attorney fees for services at hearing and on review, in addition to the specified “conditional” attorney fee awarded by the court); *Gene H. Gosda*, 50 Van Natta 2279 (1998) (same).

Consistent with the aforementioned Board and Court decisions awarding contingent assessed attorney fees under these circumstances, an award of a contingent attorney fee is appropriate in this case. Therefore, although claimant has not yet “finally prevailed” within the meaning of ORS 656.386(1), in the event that he ultimately prevails, *i.e.*, if both aspects of the challenge to the medical services claim for acupuncture visits are decided in favor of claimant, he is entitled to a reasonable assessed attorney fee in the amount of \$3000. *Juan H. Zapata*, 69 Van Natta 638, 647 (2017).

6. Penalties for alleged unreasonable “current condition” denial (Ex. 110)

Claimant contends that the employer’s February 8, 2019 “current condition” denial was unreasonable and he is entitled to a 25 percent penalty along with a reasonable penalty-associated assessed attorney fee. The employer asserts that it had a legitimate doubt regarding its liability for claimant’s current condition and no penalty or associated attorney fee is warranted.

Claimant is entitled to a penalty if the carrier “unreasonably delays or unreasonably refuses to pay compensation, or unreasonably delays acceptance or denial of a claim.” ORS 656.262(11)(a). The standard for determining an unreasonable resistance to the payment of compensation is whether, from a legal standpoint, the carrier had a legitimate doubt as to its liability. *International Paper Co. v. Huntley*, 106 Or App 107 (1991). If so, the refusal to pay is not unreasonable. “Unreasonableness” and “legitimate doubt” are to be considered in the light of all the evidence available at the time of the denial. *Brown v. Argonaut Insurance Company*, 93 Or App 588 (1988); *Frank K. Nicholas, Jr.*, 49 Van Natta 80 (1997).

The employer’s “current condition” denial was procedurally improper because (1) the denial issued before claimant filed his new/omitted medical condition claims, (2) the employer’s prior acceptance of claimant’s “acute toxic inhalation” encompassed his toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency either because “acute toxic inhalation” was either a mechanism of injury or a vague/ambiguous medical condition, and (3) the denial referred only to claimant’s “acute chemical inhalation” and did not state that his “acute toxic inhalation” claim was not compensably related to his current condition. The law regarding the first procedural error was well settled and not in dispute. *See Barbara J. Ferguson*, 63 Van Natta 2253, 2257 (2011). On the merits, the opinions of the employer’s IME physicians provided the employer with a legitimate regarding its liability for claimant’s initial injury claim, but there was no evidence from any physician in this record that claimant’s compensable injury, assuming he had one, had resolved. For all those reasons, the employer’s “current condition” denial was unreasonable. I conclude that claimant is entitled to a 25 penalty along with a penalty-associated attorney fee. The penalty shall be based on all compensation due to claimant at the time of hearing. A reasonable attorney fee is determined to be \$2000.

7. Assessed attorney fees

Claimant’s counsel contends that the time, effort and risk involved in representing claimant in this workers’ compensation case “justifies an assessed attorney fee in excess of \$100,000 and probably less than \$200,000” and, based on the factors contained in OAR 438-015-0010, he is at least entitled to an extraordinary assessed attorney fee in the amount of \$150,000 for his efforts in overturning the employer’s compensability denials (Claimant’s Closing Argument, page 80, and Claimant’s Reply Argument, page 41). The employer acknowledged the complexity of this matter and the amount of time and effort that went into litigation on both sides (Employer’s Closing Argument, page 88). Nevertheless, it asserts that claimant’s

request is excessive and, based on a rate of \$275 an hour, \$101,200 is more appropriate given the circumstances. *Id.*⁴⁴

Claimant's attorney is entitled to an assessed fee for services at hearing and on review. ORS 656.386(1).⁴⁵ OAR 438-015-0010(4) further provides:

In any case where an Administrative Law Judge or the Board is required to determine a reasonable attorney fee, the following factors shall be considered:

- (a) The time devoted to the case for legal services;
- (b) The complexity of the issue(s) involved;
- (c) The value of the interest involved;
- (d) The skill of the attorneys;
- (e) The nature of the proceedings;
- (f) The benefit secured for the represented party;
- (g) The necessity of allowing the broadest access to attorneys by injured workers;
- (h) The fees earned by attorneys representing the insurer/self-insured employer, as compiled in the Director's annual report under ORS 656.388(7) of attorney salaries and other costs of legal services incurred by insurers/self-insured employers pursuant to ORS Chapter 656;
- (i) The risk in a particular case that an attorney's efforts may go uncompensated and;
- (j) The contingent nature of the practice of workers' compensation law; and
- (k) The assertion of frivolous issues or defenses.

a. Time devoted to the case

Claimant's counsel submitted a weekly estimate of the time he spent on this case and represented that he spent over 368 hours (Claimant's Closing Argument, Addendum D, pages 1 through 4). He later supplemented that total by an additional

⁴⁴ OAR 438-015-0033(1) now provides, "In accordance with ORS 656.262(14)(a), a reasonable hourly rate for an attorney's actual time spent during a personal or telephonic interview or deposition conducted under that statute is [\$]350. ***." Previously, attorneys were compensated at an hourly rate of \$275.

⁴⁵ Claimant's counsel is entitled to an assessed attorney fee for the efforts of his attorney in obtaining rescission of the "current condition" denial on procedural grounds under ORS 656.386(1); *Cervantes v. Liberty Northwest Ins. Corp.*, 205 Or App 316 (2006).

24 hours to approximately 392 hours (Claimant Reply Argument, page 41). Mr. Lasken explained that he spent so much time preparing for litigation because (1) he is a sole practitioner in Central Oregon without a lot of resources and management, (2) he was faced with the challenge of learning about this emerging science of airplane fume events which required him to read hundreds of research articles going back eighty years in order to effectively prepare his witnesses and his arguments in a way that was both accurate and understandable, (3) his client had a complicated assortment of conditions with an attending physician, several different specialists, and seventeen experts, which resulted in many conferences, reports, depositions and/or trial testimony in this area of emerging science, and (4) he responded to the employer's IME opinions by engaging in 27 conferences, drafting nine letters, and preparing for seven depositions and live testimony for three other witnesses (Claimant's Closing Argument, pages 76 through 80). Mr. Lasken noted, "The vigorous defense involved the cross-examination of virtually every single one of our witnesses, and the time spent traveling to Portland, Pittsburgh and San Francisco added substantially to the hours spent." (Claimant's Closing Argument, page 77). As Mr. Lasken points out, it is well established that time spent traveling to out-of-town hearings and depositions counts toward the attorney time spent on a case. *Carmen O. Macias*, 53 Van Natta 689 (2001). Mr. Lasken also indicated that he did not include the time he spent in the twenty conferences and forty phone calls with his client or the clerical work he did involving making copies or filing.

If claimant were paid an assessed fee based solely on the 392 hours spent on the case multiplied by \$400 per hour (a commonly used benchmark, according to Mr. Lasken) and a reasonable hourly rate in my view, then claimant would be entitled to an attorney fee in the amount of \$156,800. The assessed fee, however, is not determined simply by multiplying the time Mr. Lasken spent on the case by his usual hourly rate. *Catherine Cutter*, 71 Van Natta 432 (2019); *Philip Case II*, 71 Van Natta 911 (2019). Thus, the other rule-based factors must be applied as well.

b. The complexity of the issues involved

Given the size of the evidentiary record with thousands of pages of documents, the number of witnesses, the emerging science, and the multiple issues involved, Mr. Lasken states, "It is hard to imagine a more complex case." (Claimant's Closing Argument, page 77). As the ALJ, the amount of time it took me to read and understand this record, the complexity of the parties' arguments, and length of this Opinion and Order are consistent with claimant's contention that this is one of the most complicated cases any of us involved will ever see in this forum.

c. The value of the interest involved

Mr. Lasken states,

Once again this is not a back strain. This is a career ending, brain damage injury to somebody who had an extremely high salary and valuable career. Short of a death case, it is hard to imagine that there could be a higher value of interest involved than in the present matter. Everything is at stake for Captain Myers: his claim is denied, his financial and medical benefits are cut off, and all of his actual conditions are denied. This deserves at least one arrow UP and perhaps two.

(Claimant's Closing Argument, page 78).

Claimant was 54 years old at the time of hearing. His high income as a long-time airline pilot guaranteed a maximum temporary disability rate. His severe disability resulting from the toxic chemical fume exposure may preclude him from returning to work in any capacity. If he does return to work, he may require vocational rehabilitation efforts. His medical treatment remained extensive with multiple attending and consulting physicians regarding cognitive, vision, speech, gait, and other issues. His permanent disability award could be substantial. For all those reasons, the value of the interest involved was extremely high.

d. The skill of the attorneys

Mr. Lasken and Mr. Fisher are both excellent attorneys who have been practicing workers' compensation law for many years. Their expertise and intelligence allowed them both to identify arguments and issues and develop a complex record that less experienced attorneys would not have been able to do. They both zealously represented the interests of their clients which often included complicated legal strategies and thorough questioning of witnesses. The sheer volume of exhibits and dense layers of medical and scientific information required both attorneys to work at a very high level for a long period of time.

e. The nature of the proceedings

This case involved more than half a dozen complex issues, thousands of pages of exhibits, two days of hearing, and many expert medical opinions whose complicated reports were fleshed out through depositions and live testimony. It is

one of the most complicated cases I have been involved with during my 34 years of work as an attorney and administrative law judge in Oregon.

f. The benefit secured for the represented party

A number of physicians in this record have indicated that claimant is unable to return to work as a result of his industrial injury. Also, as a result of his high paying airline pilot job, he was at the maximum time loss rate. At the age of 54, claimant most likely has a long life ahead of him. He also is in need of continued medical treatment for a variety of conditions caused by the toxic chemical fume inhalation event. The workers' compensation benefits secured by setting aside the denials of compensability of his toxic encephalopathy, mild neural cognitive disorder, convergence insufficiency, saccadic eye movement deficiency, and current condition were substantial in terms of vocational rehabilitation, temporary disability, permanent disability, and medical services.

g. The necessity of allowing the broadest access to attorneys by injured workers

Undervaluing the time and effort expended by claimant's counsel in this case would undermine the necessity of allowing broad access to attorneys by injured workers.

h. The fees earned by the defense attorney

Neither party submitted evidence regarding this factor.

i. The risk in a particular case that an attorney's efforts may go uncompensated

Mr. Lasken stated,

There has never been a case in Oregon, or perhaps even in America, establishing that toxic airline fumes can cause neurological and neurocognitive disability. This is clearly a case of first impression. Given the force differential of the two sides, this is quite a mountain to climb. The risk of not being compensated in this case is extreme. I believe that this deserves at least one and perhaps two arrows UP.

(Claimant's Closing Argument, page 78).

Mr. Lasken is correct. The employer aggressively defended its denials of compensability of claimant's conditions and the science in this area is evolving, despite the longstanding efforts of the airline industry in general to block air quality monitoring of cabins and cockpits. This was a very tough case to take on. Mr. Lasken was required to pull out all the stops to prevail, but even with the foremost experts on airline toxic exposure events in his corner, the risk of losing on the main compensability questions was very high.

j. The contingent nature of the practice of workers' compensation law

If claimant lost this case, his attorney would not get paid for approximately 400 hours of his time preparing and participating in the litigation of an extraordinarily complex matter. The risk of losing was substantial given the employer's aggressive defense, the airline's longstanding position of not permitting air quality monitoring equipment in airplanes, and the extensive IME reports submitted into evidence.

k. The assertion of frivolous issues or defenses

Neither party presented any frivolous issues or defenses.

l. Assembling the factors

Neither Mr. Fisher nor I have any reason to question Mr. Lasken's representation that he spent at least 392 hours on Captain Myers' workers' compensation case. I have easily spent half that amount of time shepherding the case through litigation, reading the exhibits, and writing this Opinion and Order. Mr. Lasken could reasonably have spent 392 hours over the last year and a half traveling to hearings and depositions, researching the science behind airline fume events, discussing the case with claimant and the medical experts, preparing for and participating in conferences and depositions, presenting witnesses at hearing, reviewing the exhibits, and drafting concurrence letters and extensive written closing arguments. Together, all of those activities took an enormous amount of time over an extended period. Mr. Lasken's Attorney Fee Summary of Hours, contained in Addendum D of Claimant's Closing Argument, documents his hours with great detail but leaves out the twenty conferences and forty phone calls with his client. He also neglects to mention the clerical work he did by making copies or filing. A thorough review of Mr. Lasken's documentation of hours reveals nothing

unwarranted or unreasonable. The size and complexity of the case, combined with the interest to claimant and the risk of losing, justified Mr. Lasken spending that much time to ensure there was sufficient evidence and argument to set aside the employer's denials. Mr. Fisher presented an all-out, formidable defense and the claim had a high likelihood of failure with extremely high stakes for Captain Myers.

After considering the factors set forth in OAR 438-015-0010(4) and applying them to this case, I find that a reasonable fee for claimant's attorney's services at hearing regarding the compensability issue is \$175,000 (one hundred and seventy-five thousand dollars) payable by the employer. In reaching this conclusion, I have particularly considered the extraordinary amount of time devoted to the issues as outlined above, a reasonable hourly rate of \$400, the great complexity of the medical and legal issues, the extremely high value of the interest involved, and the very high risk that counsel would go uncompensated. I have also taken into account the time that claimant's counsel spent on the penalty, medical services, and premature claim closure issues, which generated additional separate attorney fee awards.

8. Reasonable costs and expenses

Claimant seeks reimbursement from the employer for extraordinary costs associated with litigation in excess of \$1500. The employer objects on the basis that they are excessive in several key respects.

ORS 656.386(2)(a) provides:

(a) If a claimant finally prevails against a denial * * * [the] Administrative Law Judge may order payment of the claimant's reasonable expenses and costs for records, expert opinions and witness fees.

(b) The * * * Administrative Law Judge shall determine the reasonableness of witness fees, expenses and costs for the purposes of paragraph (a) of this section.

(c) Payments for witness fees, expenses and costs ordered under this subsection shall be made by the insurer or self-insured employer and are in addition to compensation payable to the claimant.

(d) Payments for witness fees, expenses and costs ordered under this subsection may not exceed \$1,500 unless the claimant

demonstrates extraordinary circumstances justifying payment of a greater amount.⁴⁶

OAR 438-015-0005(8) defines “expenses and costs” reimbursable under ORS 656.386(2) as “reasonable expenses and costs incurred by the claimant for things and services reasonably necessary to pursue a matter, but do not include attorney fees.” Pursuant to the rule, “examples” of “expenses and costs” include, but are not limited to, “costs of records, expert witness opinions, witness fees and mileage paid to execute a subpoena and costs associated with travel.”

In *SAIF v. Siegrist*, 297 Or App 284 (2019), the Court identified two factors that could make a case extraordinary: extraordinary complexity and the need to obtain opinions from more expensive out-of-region expert witnesses. On remand in *Kevin J. Siegrist*, 72 Van Natta 491 (2020), the Board declined to award claimant’s request for extraordinary costs in the amount of \$1550 instead of the statutory maximum of \$1500. The Board stated,

Under ORS 656.386(2), if a claimant finally prevails against a denial, the claimant may be awarded reasonable expenses and costs for records, expert opinions, and witness fees. ORS 656.386(2)(d) limits the award to \$1,500 unless the claimant demonstrates extraordinary circumstances justifying payment of a greater amount. *See also* OAR 438-015-0019(2). “Extraordinary circumstances” means circumstances that are not usual, regular, common, or customary for workers’ compensation matters. *See Siegrist*, 297 Or App at 293.

Siegrist, 72 Van Natta at 493-94. The Board in *Siegrist*, 72 Van Natta at 494, acknowledged that the complexity of a claim may reach a level establishing extraordinary circumstances justifying a request for extraordinary litigation costs and expenses. On the other hand, the Board articulated a number of reasons why a request for extraordinary costs and expenses might be denied. *Id.* First, litigation of disputed occupational disease claims, even with higher burdens of proof and multiple expert opinions, was not uncommon in workers’ compensation cases. *Id.*

⁴⁶ OAR 438-015-0019(2) states that, in the absence of the parties’ stipulation, an ALJ may award reasonable expenses and costs for the above described charges, which the claimant may subsequently claim by submitting a cost bill to the carrier in the manner prescribed by OAR 438-015-0019(3) (*i.e.*, within 30 days after the order finding that the claimant finally prevails against a denied claim becomes final). *See Barbara Lee*, 60 Van Natta 159 (2008).

Second, obtaining expert medical reports in situations where the attending physician already supported compensability of the claim might not be extraordinary. *Id.* Third, bolstering a claim with expert opinions was relatively customary and expected. *Id.* at 495. Fourth, It was not unusual to go beyond the arena of an attending physician to obtain expert medical opinions and it was common to secure consulting or expert opinions to assess and diagnose complicated medical conditions. *Id.* Fifth, rebuttal reports were regularly secured by both parties during preparation of a workers' compensation matter. *Id.* Sixth, costs and expenses which did not greatly exceed the \$1500 statutory maximum indicated circumstances that were not contemplated by the Oregon legislature to be extraordinary. *Id.* Seventh, a carrier's decision to obtain a specialist's opinion did not establish extraordinary circumstances because carriers often obtained opinions from specialists. *Id.* at 496. Finally, the Board concluded that a worker's lack of private health insurance also did not constitute extraordinary circumstances if the evidence established it was uncommon for workers to lack private health insurance. *Id.*

There is little doubt that the medical and legal posture of this case made it extraordinarily complex. The evidentiary record was enormous. The scientific explanations regarding airplane toxic fume exposures were highly technical and frequently not in agreement. The expert medical opinions were lengthy and, at times, diametrically opposed. In addition, the case has been in litigation for three years and part of the record included more than seventy years and thousands of pages of research articles. More importantly, claimant established that the scientists doing the research and the doctors treating airline toxic fume exposure patients who had the most expertise in this fairly recent area of scientific litigation were not located in Oregon and were necessary to bring to the hearing to testify in order to rebut the employer's medical experts who authored lengthy, detailed reports and testified at the hearing as well. Some of claimant's most important and essential witnesses were located in San Francisco (Dr. Harrison), Pittsburgh (Dr. Kaniecki), North Carolina (Dr. Abou-Donia), and London, England (Dr. Michaelis). Extraordinary circumstances justified claimant's request for extraordinary litigation costs and expenses because the medical questions were highly complex and there was a legitimate need to obtain opinions from more expensive out-of-region expert witnesses. *See SAIF v. Siecrest*, 297 Or App 284 (2019).

The employer specifically objected to the costs associated with Dr. Michaelis' travel from London, England to Oregon to testify as a witness. Mr. Fisher asserted that Dr. Michaelis' testimony was "entirely irrelevant" because the employer conceded that claimant was exposed to toxic chemical fumes by accepting the claim (Employer's Closing Argument, page 89). Claimant persuasively rebutted the

employer's objection by noting that it vigorously litigated the nature and extent of the fume event that took place while attempting to minimize the significance of the exposure. As discussed at length, it was also unclear what conditions the employer actually accepted as a result of accepting a mechanism of injury or a vague/ambiguous condition. Everything about this claim was on the table for litigation. Dr. Michaelis testified that claimant was exposed to high levels of toxic chemical fumes and explained how that exposure occurred in technical terms. Her testimony was relevant and material to the claim because it helped establish that the toxic fume exposure was significant enough to result in claimant's toxic encephalopathy and associated conditions. Mr. Lasken added, "Dr. Michaelis is involved in cutting edge research and has published numerous papers in the last fifteen years directly on this topic. Given a choice of conjuring up somebody who is marginally qualified and bringing in somebody who is extremely qualified, and given the contingent nature of this litigation, excuse me for going all out to win this case." (Claimant's Reply Argument, pages 39-40). Claimant is correct that it was reasonable and necessary to bring Dr. Michaelis to Oregon to testify at the hearing. Thus, the costs associated with her travel are reimbursable.

The employer also objected to claimant's request for travel reimbursement costs associated with Mr. Lasken's expenses while attending the out-of-state depositions of Drs. Kaniecki and Harrison. In support of its position, the employer cited *Shirley A. Smith*, 63 Van Natta 2354 (2011), where the Board held,

[A] "cost associated with travel" must be associated with one of the three items listed in ORS 656.386(2)(a), *i.e.*, it must be "for records, expert opinions and witness fees." *See Harrison v. Taylor Lumber & Treating, Inc.*, 111 Or App 325, 328 (1992) (an agency's rule may not alter, amend, enlarge, or limit the terms of an applicable statute); *Daniel S. Frazer*, 63 Van Natta 1098, 1099 (2011) ("expenses and costs" reimbursable under ORS 656.386(2) must be "for records, expert opinions and witness fees").

In *Shirley A. Smith*, *supra* at 2354-55, claimant sought reimbursement for mileage costs incurred by her attorney to attend a deposition. The Board declined claimant's request, concluding that her attorney's mileage to and from the deposition and hearing was not a reimbursable "cost associated with travel" under the statute and rule because it was not "for records, expert opinions and witness fees." *Id.* Based on the Board's decision in *Shirley A. Smith*, *supra*, claimant's counsel in the present case is not entitled to the reimbursement of costs associated with his travel to the

depositions of Drs. Kaniecki and Harrison, which amounted to \$1,264.43 (Claimant's Closing Argument, Employer's Closing Argument, page 89).⁴⁷

Mr. Lasken's Cost Ledger, which was attached to Claimant's Closing Argument, requested costs in the amount of \$38,717.15. The fees for medical records and CD of the hearing (\$53.50, \$25.00, \$35.00, 60.39, and \$5.00) totaling \$178.89 were reasonable and are granted. Also, the fees for "supplies, copying, mailing" (46.05, \$25.00, \$19.05, \$287.48, \$55.50, and \$831.40) totaling \$1264.48 were reasonable and are granted. In addition, the following modest fees incurred were also reasonable and are granted:

- \$200 for Dr. Ugalde's 4/8/19 concurrence letter
- \$187.50 for Dr. Schock's 3/19/19 and 4/2/19 record review and phone conference
- \$250 for Dr. Schock's 6/17/19 and 6/34/19 review of Dr. Abou-Donia's report and phone conference
- \$100 for Dr. Scott's 9/26/19 and 10/7/19 phone conferences
- \$450 for Dr. Kaniecki's 5/1/19 phone conference
- \$175 for Dr. Ugalde's 9/17/19 conference
- \$231 for Dr. Schloesser's 3/21/19 conference
- \$750 for Dr. Harrison's 9/24/19 deposition prep meeting

Finally, the larger cost fees included the following:

- \$3,262.48 for Dr. Michaelis' 12/19/19 report
- \$9,937.50 for Dr. Abou-Donia's 6/21/19 expert consultation/depo prep
- \$5,975.55 for Dr. Michaelis' 10/29/19 RT plane ticket from London to US
- \$186.59 for Dr. Michaelis' RT plane ticket from Seattle to Portland
- \$216.60 for Judith Anderson's 12/23/19 RT plane ticket from Seattle to Portland
- \$158.18 for Judith Anderson's 12/30/19 lodging for hearing
- \$339.12 for Dr. Michaelis' 12/30/19 lodging for hearing
- \$3000 for Dr. Schock's 10/14/19 and 10/22/19 record review, pretrial meeting and court appearance
- \$195 for Judith Anderson's 3/9/20 childcare while attending trial
- \$9,494.83 for Dr. Michaelis' 3/9/20 preparation, travel, conference, trial, and return travel

⁴⁷ Claimant's counsel's 8/14/19 airfare of \$887, plus 8/31/19 lodging of \$197.33, plus 9/24/19 lodging of \$100.10, plus 9/23/19 parking of \$7, plus 9/24/19 parking of \$28, plus 9/25/19 parking of \$45, equaled a total of \$1264.43 (Claimant's Closing Argument, Addendum C, Cost Ledger, page 2).

- \$900 for Dr. Harrison's 2/7/20 depo bill

Beside the employer's objection to the relevancy of Dr. Michaelis' testimony, which I have overruled, and claimant's counsel's costs for travel to the depositions, which I have sustained, the employer did not contest the reasonableness of specific cost requests nor did it present evidence to cast doubt on the reasonableness of those costs. Pursuant to this Opinion and Order, claimant has finally prevailed against multiple denied claims under ORS 656.386(1). In addition, claimant has satisfied the Court's requirements for the assessment of extraordinary costs under ORS 656.386(2). Thus, it is appropriate to award extraordinary expenses and costs to claimant for records, expert opinions, and witness fees in the amount of \$37,452.72 (\$38,717.15 minus the \$1,264.43 costs of claimant's counsel to travel to the depositions of Drs. Kaniecki and Harrison).

ORDER

IT IS HEREBY ORDERED that JetBlue Airways and AIG – Chartis Claims' February 20, 2019 denial of compensability of claimant's toxic encephalopathy is set aside (Ex. 113). The claim is remanded to the employer for acceptance and processing according to Oregon workers' compensation laws.

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims' April 2, 2019 denial of compensability of claimant's mild neural cognitive disorder is set aside (Ex. 116). The claim is remanded to the employer for acceptance and processing according to Oregon workers' compensation laws.

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims' April 2, 2019 denial of compensability of claimant's vision disorders, including convergence insufficiency and saccadic eye movement deficiency, is set aside (Ex. 116). The claim is remanded to the employer for acceptance and processing according to Oregon workers' compensation laws.

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims' February 8, 2019 denial of compensability of claimant's "current condition" is set aside (Ex. 110). The claim is remanded to the employer for acceptance and processing according to Oregon workers' compensation laws.

IT IS FURTHER ORDERED that a causal connection was established between claimant's compensable injury and the 24 acupuncture visits recommended by Dr. Ugalde to treat the post traumatic headaches associated with his toxic encephalopathy.

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims are assessed a contingent assessed attorney fee pursuant to ORS 656.386 in the amount of \$3000 (three thousand dollars) in the event that claimant “finally prevails” over the employer's refusal to authorize payment of the 24 acupuncture visits recommended by Dr. Ugalde.

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims shall pay a 25 percent penalty pursuant to ORS 656.262(11)(a) for its unreasonable February 8, 2019 “current condition” denial. That penalty shall be based on all compensation due to claimant at the time of the hearing and shall be paid directly to claimant. The employer and its processing agent are also assessed a reasonable penalty-associated attorney fee in the amount of \$2,000 (two thousand dollars) to be paid directly to claimant's attorney.

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims are assessed a reasonable attorney fee pursuant to ORS 656.386 in the amount of \$175,000.00 (one hundred and seventy-five thousand dollars) for the efforts of claimant's counsel in setting aside the employer's February 20, 2019 denial of compensability of claimant's toxic encephalopathy, its April 2, 2019 denial of compensability of claimant's mild neural cognitive disorder, convergence insufficiency, and saccadic eye movement deficiency, and its February 8, 2019 denial of compensability of claimant's current condition. The assessed attorney fee shall be paid directly to claimant's attorney.

IT IS FURTHER ORDERED that claimant's request for hearing regarding WCB Case No. 18-00006H is dismissed because the claim was withdrawn.

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims' April 2, 2019 denial of compensability of claimant's alleged polyneuropathy is approved because claimant withdrew his appeal of that portion of the employer's denial.

IT IS FURTHER ORDERED that claimant's Request for Hearing in WCB Case No. 18-00006H is dismissed because claimant withdrew that Request for Hearing. The medical service claim involved a December 14, 2017 Administrative Order of Dismissal regarding eight acupuncture visits proposed on June 23, 2017. *See Ex. 84A.*

IT IS FURTHER ORDERED that JetBlue Airways and AIG – Chartis Claims shall pay all extraordinary and reasonable expenses and costs for records, expert opinions, and witness fees pursuant to ORS 656.386(2) and OAR 438-015-0019(2) in the amount of \$37,452.72 (claimant's total cost request of \$38,717.15 minus \$1264.43, which represented unreimbursable costs associated with claimant's counsel's travel for the depositions of Drs. Kaniecki and Harrison). Those costs shall be paid directly to claimant's attorney.

Notice to all parties: If you are dissatisfied with this Order, you may request Board review. A request for review must be submitted within thirty (30) days after the mailing date on this Order. You must timely submit your request for review by any of the following methods:

- (1) Mail: Workers' Compensation Board
2601 25th St SE, Suite 150
Salem, OR 97302-1280
- (2) E-mail: request.wcb@oregon.gov
- (3) Fax: 503-373-1600
- (4) In-person: Workers' Compensation Board office in Salem, Portland, Eugene, or Medford
- (5) Website portal: For attorneys, self-insured employers and insurers that are registered users

You must also provide a copy of your request to all other parties to this proceeding within the same 30-day period. All other parties will have the remainder of the 30-day period, and in no case less than 10 days, to request Board review. The 10-day minimum is provided even if it extends the time allowed to request Board review beyond 30 days.

Failure to provide a timely request for review to the Board and provide copies to all other parties within the time allowed will result in the loss of your right to appeal this Order and the Board will be unable to review the Administrative Law Judge's decision.

Entered at Portland, Oregon, on JUL 31 2020 , with copies mailed to:

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Workers' Compensation Board



Darren Otto
Administrative Law Judge