**IN THE DISTRICT COURT**
**AT WELLINGTON**

**I TE KŌTI-Ā-ROHE**
**KI TE WHANGANUI-A-TARA**

**[2024] NZACC 005 ACR 267/20**

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| UNDER | THE ACCIDENT COMPENSATION ACT 2001 |
| IN THE MATTER OF | AN APPEAL UNDER SECTION 149 OF THE ACT |
| BETWEEN | JOSEPH JESSUP<br>Appellant |
| AND | ACCIDENT COMPENSATION CORPORATION<br>Respondent |

Hearing: 11 December 2023

Held at: Auckland District Court/Tāmaki Makaurau

Appearances: P Schmidt for the Appellant
S Bisley for the Accident Compensation Corporation ("the Corporation")

Judgment: 8 January 2024

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**RESERVED JUDGMENT OF JUDGE P R SPILLER**
**[Claim for work-related gradual process injury –**
**s 30(3), Accident Compensation Act 2001 ("the Act")]**

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## Introduction

[1] This is an appeal from the decision of a Reviewer dated 30 November 2020. The Reviewer dismissed an application for review of the Corporation's decision dated 12 November 2018 declining cover for a work-place gradual process injury, being chronic organic solvent neurotoxicity as a result of Mr Jessup's work as a spray painter in the air force.

**Background**

[2] Mr Jessup was born in May 1962. He worked for the Royal New Zealand Air Force (RNZAF) between 15 January 1980 and 16 January 1986.

[3] In 1980, Mr Jessup completed trade courses in basic engineering and safety equipment. In 1981-1982, he serviced, packed and maintained parachutes and then survival equipment.

[4] From January to July 1983, Mr Jessup completed an aircraft finishers' painting course, and was posted to the Ohakea Base. From July to October 1983, he was involved in surface finishing of aircraft, and until he left the RNZAF in January 1986, he was mainly involved in other servicing, packing and maintenance of aircraft. Mr Jessup's surface finishing work involved stripping and repainting aircraft. The paints and paint strippers used contained a range of organic solvents, including toluene, xylene, trichloroethylene, and isocyanates. No exposure limits were in place. Personal protective equipment used consisted of "off the shelf" items such as air purifying masks with charcoal cartridges, in-line breathing apparatus when using polyurethane paints, cotton or disposable overalls, rubber gloves, and safety goggles.

[5] On 14 January 1986, when Mr Jessup was discharged from the RNZAF, he completed a medical discharge form and underwent a medical examination. In the discharge form, Mr Jessup answered "yes" only to whether he had experienced backache, spinal injury or disc trouble (low back pain) and also noted that he had difficulty hearing in a noisy crowd. He answered "no" to questions asking whether he had suffered from: troublesome headache, eye trouble, nose, sinus or throat trouble, hoarseness or change of voice, change of weight, loss of appetite, fainting attacks or blackouts, depression or nervous trouble, or any other illness or disability not mentioned. The examining medical officer did not record any neurological, or related, issue.

[6] After Mr Jessup left the RNZAF, he obtained tertiary level qualifications. He was a lecturer at Wintec from 2000 until 2004. During his career, he worked as a

manager at Firestone Tyre Company, worked as an owner/manager of his own tyre shop, and was self-employed as a financial and insurance adviser.

[7] On 11 October 2010, Dr Ng Grace, GP, noted that Mr Jessup presented with a five-day history of altered sensation, numbness and tingling in his left forearm with intermittent pain.

[8] On 26 August 2011, Dr Diane Kimber, GP, reported that Mr Jessup had had severe obstructive sleep apnoea.

[9] On 8 March 2013, Dr Leslie Johansen, GP, referred Mr Jessup to a cardiologist regarding hypertension.

[10] On 13 December 2013, Dr Johansen referred Mr Jessup for an assessment of his six-month history of increasing hoarseness of his voice.

[11] On 18 December 2013, Mr Jessup (now aged 51 years and nearly 28 years after he left the RNZAF) consulted Mr Hans Stegehuis, Ear, Nose and Throat Specialist, about a change in voice (difficulty in being heard and huskiness) over the previous six months. Mr Stegehuis suspected that the change was related to Mr Jessup's hand tremor he had had since childhood.

[12] On 6 March 2014, an MRI was conducted on Mr Jessup. Dr Trunni Bhattacharjya, Radiologist, reported some white matter hyperintensities.[1]

[13] On 11 April 2014, Mr Stegehuis reported further. He noted that Mr Jessup wondered if his time as a spray painter for the Air Force might have been a factor in his present condition. Mr Stegehuis noted:

> The MRI scan of the brain done on 6/3/2014 is normal apart from showing some periventricular and subcortical white matter hyperintensities which have a differential [diagnosis] of small vessel ischaemic changes, vasculitis and demyelination.

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[1] White matter hyperintensities is a common finding in aging population and considered to be a contributor to cognitive decline.

[14] On 8 May 2014, Dr Paul Timmings, Neurologist, examined Mr Jessup about his hoarse voice. Dr Timmings found that:

> It is not clear whether there is a primary neurological disorder affecting his vocal cords. I see that he has undergone MRI scanning of the brain … and I took the opportunity to review those films. They show a number of small deep white matter hyperintensities consistent with small vessel disease, commensurate with his age and history of hypertension. I do not feel they are representative of demyelinating disease or a vasculitic process.

[15] On 26 May 2014, following nerve conduction studies, the Waikato Neurology Group reported:

> Abnormal study. There is electrodiagnostic evidence of a mild, length dependent axonal sensorimotor polyneuropathy. There is no electrodiagnostic evidence of a lower motor neuron disorder of the tongue, nor a disorder of neuromuscular transmission based on a normal 2Hz repetitive nerve stimulation study of a proximal and facial muscle. However it should be noted that the study was performed while the patient was taking pyridostigmine (Mestinon).
>
> The clinical picture raises the possibility of a disorder such as laryngeal dystonia, however this does not easily explain the dysarthria.

[16] On 24 June 2014, Dr Timmings observed that a mild length-dependent neuropathy "is not a rare occurrence in patients who have previously had bariatric surgery", and that there was "no evidence of a neuromuscular conduction disorder".

[17] On 27 May 2015, Dr Timmings considered that no action was needed from a neurological point of view.

[18] On 3 August 2018, Dr Olof Rydin, Mr Jessup's GP, filed a claim for cover for a work-place gradual process injury, being chronic organic solvent neurotoxicity as a result of Mr Jessup's work as a spray painter in the air force.

[19] On 14 August 2018, Mr Jessup completed a claimant questionnaire. He noted symptoms including breathing difficulty, weight gain, high blood pressure, tingling hands and feet, shoulder and joint discomfort, sleep deprivation, urine flow, and lack of concentration. He first noticed asthma in 1986, and a gradual increase in the other symptoms. He noted that depression/anxiety and "temperament" were from 2002, urinary tract from 2008, shoulder discomfort from 2009, sleep deterioration/apnoea from 2011, and lack of concentration and memory/cognitive issues from 2014.

[20] In late 2018, the Corporation referred Mr Jessup to Dr David Prestage, Occupational Physician, for an assessment about Mr Jessup's neurological disorder and asthma.

[21] On 22 October 2018, Dr Prestage reported:

> Mr Jessup said he started having breathing difficulties towards the end of his time with the RNZAF but did not think much of it at the time and did not do anything about it at that stage. Shortly after leaving in 1986, he continued to experience breathing difficulties and was started on Ventolin by his general practitioner. In about 1989 or 1989, he recalled going to Rotorua Hospital and being treated with a nebuliser before being discharged.
>
> Mr Jessup said over the years he has learnt to deal with things and compensate.
>
> In about 2011, Mr Jessup's health problems became more significant. He developed a range of symptoms and consulted a number of health professional. However, he commented that each one tended to assess the problems individually rather than consider his situation in an holistic manner. He said they have all acknowledged something was wrong but they did not know what, and he believes they missed the connection between all his condition which was his exposure to chemicals.
>
> Mr Jessup said cognitive function appears to be getting worse with time. This commenced about 2014 and is the main issue which had led to him lodging a claim. … He was concerned about the perceived deterioration in his cognitive condition. He wondered about a connection with his chemical exposures after reading a newspaper article about an RNZN veteran who won a battle for compensation after successfully claiming his Parkinson's disease was due to a chemical exposure during his military service.

[22] In terms of the neurological disorder, Dr Prestage observed that there had been no formal assessment or investigation of Mr Jessup's reported cognitive symptoms, and said:

> … no formal assessment or investigation of Mr Jessup's reported cognitive symptoms has been undertaken. Until a formal diagnosis has been made, I do not believe it is possible to offer an opinion on whether any condition that may be present is related to Mr Jessup's chemical exposures.
>
> The other important issue is that Mr Jessup's symptoms allegedly commenced in 2014, 28 years after he left the RNZAF. It is implausible that exposures between 1980 and 1986 would lead to symptoms commencing after such an extended period of time. In my opinion, an underlying cause of his cognitive dysfunction should be sought.

[23] Dr Prestage diagnosed that Mr Jessup suffered from respiratory, cognitive and laryngeal symptoms without a specific diagnosis. Dr Prestage stated that Mr Jessup

required formal neuropsychological testing and specialist psychiatric/neurological assessment in order to make a formal diagnosis.

[24] On 24 October 2018, Dr John Monigatti, the Corporation's Lead Occupational Health Advisor, recommended that the Corporation request tests, including cognitive function testing.

[25] On 12 November 2018, Ms Rose Gleeson, Case Coordinator, noted she had contacted Mr Jessup twice to see if he would like her to forward her report (noting the need for specialist assessment) to his GP, to start organising the testing to confirm a diagnosis, but Mr Jessup had not responded.

[26] On 12 November 2018, the Corporation issued a decision declining Mr Jessup cover for his injury, on the basis that there was no medical evidence of a physical injury. The Corporation noted that if Mr Jessup provided further information confirming his injury, the Corporation would reassess his claim.

[27] On 31 January 2019, Mr Jessup saw Dr Jan Schepel, Neurologist. Dr Schepel noted, *inter alia*, that Mr Jessup was always itchy and scratching himself; he complained of severe headaches, usually located frontally; around 2011, his symptoms worsened; he had bariatric surgery in 2012 to reduce weight; his productivity for work had declined over the previous three to four years; he initially believed that this was owing to depression; but, after a one-month trial of an anti-depressant, he stopped the course of treatment because it made him feel worse. Dr Schepel concluded:

> There is no information pointing towards an acute solvent encephalopathy during the time that he worked with the RNZAF. Exposure to high solvent concentrations could lead to unconsciousness, convulsions and death but also low level exposure could lead to permanent neurotoxic effects in the brain.
>
> Chronic solvent encephalopathy is usually defined as permanent neurotoxic effects to the central nervous system ranging from mild cognitive signs to neuropsychiatric dysfunction with loss of work ability.

[28] Dr Schepel thought that chronic solvent encephalopathy was unlikely, observing that:

> Patient's symptoms were increasing, suggesting an ongoing pathomechanism which makes it difficult to link his current symptoms to the exposure to different chemicals between 1980 and 1986.
>
> Most literature suggests that … marked global deterioration in intellect and memory accompanied by neurological signs and neurological findings is at best poorly reversible, but is generally non progressive once exposure had seized.
>
> Patient exposure had seized in 1986 and his current new and progressive symptoms therefore unlikely to be caused directly by organic exposure 32 years ago.

[29] On 7 February 2019, Dr Gil Newburn, Neuropsychiatrist, having examined Mr Jessup, reported:

> Despite Mr Jessup having previously described his symptoms as beginning in around 2011, in fact they dated from the point of his departure from the Air Force when he became aware he needed an afternoon nap.
>
> Those symptoms have then worsened "over the last few years" with an increase in anxiety, socially avoidant behaviour, weight gain and a reduction in language function.

[30] Dr Newburn noted that in his examination of Mr Jessup, he presented material in an open, aware and unembellished manner, and there was no evidence for any over-valued idea, delusional material or hallucination, or that he was other than attempting to achieve well. Dr Newburn found that, on formal cognitive assessment, Mr Jessup's verbal memory was significantly impaired, he did not recall new associations, struggled to complete a task requiring him to hold more than one item in consciousness, there was subtle disturbance in visuospatial memory, and he was very slow in doing a conflicting instruction test.

[31] Dr Newburn diagnosed Mr Jessup under the DSM-V framework as follows:

> 1. Mild neurocognitive disorder, probably consequent on a combination of neurotoxic exposure while working in the armed forces, but aggravated by small vessel cerebrovascular disease and possible malabsorbtion of bariatric surgery.
>
> 2. Adjustment disorder with disturbance of emotions.

This is set against a background of a longstanding intention tremor, of little relevance to the current presentation, respiratory difficulties, and increasing language production difficulties of indeterminate origin. …

Mr Jessup presents with a significant set of symptoms. These had their onset during his service with the Royal New Zealand Air Force, with respiratory issues being most evident initially. However, careful history shows that there was also a degree of fatigue associated with mental activity present at the time that he left the Air Force, and this has existed through to the present. Thus, while there has clearly been an exacerbation of cognitive difficulties in the last ten years, issues have been present long-term.

During his time in the Air Force, he was exposed to a range of substances that are known to be neurotoxic. Clinically, individuals who have sustained neurotoxicity following exposure to these compounds present with attentional difficulties, and increasing fatigue associated with mental effort. Therefore, Mr Jessup's description is of a typical response to exposure.

His level of exposure has been significant. While superficial protective material was provided, in reality there was minimal protection for inhalation, or skin exposure. Indeed, the skin was readily exposed to cleaning agents. Exposure occurring over the majority of a six-year period would be more than sufficient for permanent damage to have occurred.

It is notable that in spite of early symptoms described, Mr Jessup got on with life. He worked, obtained further qualifications, married and had a family. That he did all of this in spite of symptoms mitigates strongly against there being a primary psychological reason for his presentation now. Rather, it is more likely than not that additional factors have added to what was already the consequence of a neurotoxic syndrome arising from his service in the Air Force. …

There is certainly good evidence for cognitive impairment on formal testing. Memory, involving both verbal and visuospatial areas was impaired. His capacity to hold two items in consciousness was disturbed. There was a subtle disturbance in fine motor planning. This was all present without any attempt to maximise his level of impairment. Rather, his function was to clearly succeed where possible, and this was seen in most areas.

Given the above, it is my opinion that Mr Jessup suffers from a mild neurocognitive disorder due to neurotoxic exposure, with exacerbation of symptoms due to other factors, in particular small vessel cerebrovascular disease and the consequences of obstructive sleep apnoea (which may in itself be a secondary consequence of neurotoxicity). However these other factors would be unlikely to lead to his current set of symptoms in the absence of the neurotoxic syndrome, which is therefore a necessary cause of his presentation.

[32] On 15 February 2019, an MRI was conducted on Mr Jessup's brain and cervical spine. Dr Gavin Davis, Radiologist, reported:

> There are established involutional changes with mild generalised cerebral atrophy present, this being out of keeping with the patient's age. In addition the patient has grade 1 deep white matter leukoariaosis.[2]
>
> These two findings occur in solvent encephalopathy in about one third of the patients. These findings however are non-specific and can occur for other reasons such as small vessel ischaemia associated with hypertension or diabetes.

[33] On 11 March 2019, Dr Schepel responded to the Corporation's question "Can you identify the cause of the patient's presenting symptoms and incapacity (if any)?":

> Respiratory symptoms with temporal relationship to solvent exposure.
>
> Sleep apnoea symptoms not optimally treated.
>
> Mood disorder not treated.
>
> Mild polyneuropathy following bariatric surgery 2012.
>
> Diffuse cognitive, general and neurological symptoms likely related to the above mentioned issues.

[34] Dr Schepel also responded to the question whether there was any other "abnormal illness behaviour" in Mr Jessup's condition:

> Patient's pursuit of recognition that all of his symptoms are due to previous solvent exposure and frustration about the lack of 'holistic approach' may interfere with prognosis.

[35] On 22 March 2019, Dr Janice Wong, Respiratory and General Physician, examined Mr Jessup, and reported:

> From the respiratory point of view, I believe Mr Jessup has obstructive airways disease with border-line reversibility. … This is on a background of a previous long period of chemical exposure, including TDI, whilst he was working for the RNZAF. His other health problems include mild neurocognitive disorder, language difficulties, hypertension and gastroesophageal reflux disease.

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[2] Leukoaraiosis refers to white matter changes commonly seen in elderly patients with stroke, hypertension, vascular dementia, mood or gait disturbances.

[36] On 24 June 2019, Dr Jan Prosser, Neuropsychologist, assessed Mr Jessup and reviewed relevant documentation. Included in this documentation was a study of retired French utility workers, with a minimum age of 55 years, assessed with solvent exposure.[3] Retirees at greatest risk for deficits had both high lifetime exposure to solvents and were last exposed 12 to 30 years before testing. Risk was somewhat elevated among those with high lifetime exposure retiring 31 to 50 years prior to testing. The study concluded that the risk of solvent-associated cognitive impairment attenuating with time might not be fully true for those with higher exposure.

[37] Dr Prosser recorded that Mr Jessup was unable to describe when he first noticed his current cognitive symptoms, as they "have gone on for years"; and that his wife had observed a range of mental difficulties in the previous four years, believing them to have become more marked. Mr Jessup reported that, at the end of his time with the RNAF, he had some difficulties with his breathing, and, after leaving, was prescribed Ventolin by his GP. Notes indicated that he presented at a hospital with breathing difficulties which were treated with a nebuliser. He reported that in 2011, his health began to deteriorate.

[38] Dr Prosser noted that Mr Jessup's autobiographical memory was consistent with documents provided to her, he adopted a diligent and interested approach to formal testing, an embedded test of effort did not indicate the possibility of malingering or tendency to exaggeration.

[39] Dr Prosser reported in her summary and impressions:

> Pleasingly, overall most tests Mr Jessup completed were within or above the expected premorbid range of average. Tests focused on visual memory, complex visuospatial perception, and processing speed (rapid processing of visual information and decision making) are in the moderate to severe impaired range. He also has a relative weakness, in selective attention and visual processing speed, short-term visual memory and spatial reasoning ability although they did show some impairment. …

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[3] Sabbath E. L., Gutierrez L. A., et al. "Time may not full attenuate solvent-associated cognitive deficits in highly exposed workers". *Neurology*. 2014; 82 (19): 1716-1723. At the time of Mr Jessup's assessment by Dr Prosser, he was 57 years and had retired from the RNZAF over 33 years before.

> While Mr Jessup is reporting multiple physical complaints, some of which match the criteria for neurotoxicity syndrome, the results of the neuropsychological tests undertaken for the present assessment, do not match the cognitive profile highlighted in the literature for neurotoxicity syndrome. …
>
> Criteria for a diagnosis of solvent induced toxic encephalopathy include long or intense exposure to solvents or both; relevant symptoms such as increased fatigue, memory impairment, difficulties in concentration, and personality changes such as passivity; presence of pathological findings in terms of an objective measure; temporal relationship between exposure and the development of symptoms and sign; and no other obvious cause of the disease. …
>
> Symptoms of neurotoxicity and abnormalities of performance on neuropsychological testing. Type 2 disorder has been subdivided into:
>
> - Type 2A: sustained personality or mood change, fatigue, poor impulse control and poor motivation; and
>
> - Type 2B: impairment in intellectual function (including concentration, memory, learning and psychomotor slowing).
>
> … New Zealand research suggests that the majority of workers diagnosed as Type 2 have symptoms from both Types 2A and 2B. It is suggested that these two types can be combined together to form Type 2.
>
> While Mr Jessup's neurocognitive profile is not completely in line with the literature, it appears there is some moderate to severe impairment with tasks involving visual processing and speed of processing. While these are listed as areas impacted by solvent neurotoxicity, it is unclear, given the other factors described above [sleep apnoea, hypertension and depression with features of anxiety, all associated with cognitive impairment] whether it can be solely attributed to Mr Jessup's exposure to organic solvents some 30+ years ago, especially given their direct link to white matter atrophy, which is linked to hypertension.
>
> … it is often impossible to say with absolute certainty that cognitive and psychological symptoms are the result of neurotoxicity, often due to the complex context the symptoms are embedded in and is difficult to tease apart the organic and psychological causes of depression, fatigue, poor concentration and memory problems in daily life. Therefore, while there is a possible contribution, organic solvent neurotoxicity, is one of a number of possible contributing factors to Mr Jessup's current presentation.

[40] On 14 November 2019, Dr Newburn noted that Mr Jessup had benefitted from prescribed medication, and this further confirmed the hypothesis of neurotoxic brain impairment.

[41] On 21 November 2019, the Corporation's five-member Toxicology Panel (comprising a Professor of Medicine, a Medical Toxicologist, two Occupational

Health/Medical Specialists, and a Clinical Psychologist and Neuropsychologist) reported:

> The Panel agreed that the cognitive deficits reported by Dr Prosser were consistent with solvent neurotoxicity but not diagnostic of it, and that the comorbidities described could give rise to the same profile. The consensus was that Mr Jessup should be offered treatment for his depression and sleep apnoea, in particular. Once these, and his antihypertensive therapy, had been optimised there should be a reassessment of cognitive function to determine what impairment remained and whether a more compelling case for organic solvent neurotoxicity could be made.

[42] On 27 November 2019, following a PET CT scan, Dr B Moon, Radiologist, reported:

> Mild reduction in relative perfusion to the frontotemporal cortex, but the degree of hypoperfusion does not clearly indicate a primary frontotemporal neurodegenerative disorder. Other non-neurodegenerative causes should be considered. No features to suggest Alzheimer's or dementia with Lewy body disease.

[43] On 26 February 2020, Dr David McBride, Occupational Physician, noted, following an examination of Mr Jessup that Mr Jessup could not describe when his cognitive problems began, but described them as being present for years. Dr McBride recorded Mr Jessup's report of symptoms of fatigue going back to his days in the RNZAF, when he had to have afternoon sleeps; the fatigue extended to mental fatigue, has persisted up to the present time, and has gradually worsened. Dr McBride disagreed with Dr Prestage's statement that it was implausible that exposures between 1980 and 1986 would lead to symptoms commencing after such an extended period of time. Dr McBride noted that in 2005 Mr Jessup struggled with teaching, and around 2014 his ability to study, learn and retain information became much more noticeable. Dr McBride observed that "with stoicism typical of service people he battled on".

[44] Dr McBride diagnosed chronic solvent-induced encephalopathy (CSE) (type 2B, impairment in intellectual function), describing it in the following terms:

> Neuropsychological testing showed cognitive deficits affecting attention/concentration, visuospatial skills, and verbal memory. There may be minor neurological signs. If exposure ceases some recovery is likely but full recovery may not occur.

[45] Dr McBride noted that Mr Jessup was exposed to tricholoroethylene (TCE) over a 6-year period. The exposure had to be considered extreme, with inadequate PPE, inadequacies in respirators, and excessive respiratory and skin exposure. He would also have been exposed to toluene, xylene, and methylene chloride. Dr McBride referred to relevant research studies, including the study of retired French utility workers, with a minimum age of 55 years, assessed with solvent exposure.

[46] In summary, Dr McBride commented:

> There is no doubt that solvent exposure was extremely high, due to a combination of inadequate PPE, probable excessive respiratory exposure and definite excessive skin exposure. This is corroborated by 'having to breathe fresh air' during breaks, sleeping heavily, and feeling hungover the next day, which is not normal. This means that cumulative lifetime exposure would have been high for Mr Jessup, it is the product of years of exposure and solvent levels.
>
> This excessive exposure has not been measured, however this was not the responsibility of the claimant. …
>
> The symptoms are of irritability, attentional deficits and poor short-term memory impairment have been developing problem since 2011, but have become recently much worse. There has been discussion about the neuropsychological testing results, and the pattern of these, however there is in fact no 'typical' profile, it differs with the test battery used, and psychologists in general have a preferred battery of tests. Dr Prosser agrees that the symptoms are consistent with solvent induced neurotoxicity.
>
> There have been questions about the additional contributions of age, metabolic disturbance and hypertension. These effects also contribute to the clinical presentation [and] may have occurred to some extent, but if functional reserve is diminished by an initial occupational injury, reserve capacity will have been reduced. Dr Gil Newburn agrees with this approach, the other factors would not have resulted in Mr Jessup being in such a poor state: the neurotoxic exposure is the culprit.

[47] On 9 March 2020, Dr Timmings re-reviewed Mr Jessup. At the outset, Dr Timmings commented that:

> The question posed today is as to whether or not [Mr Jessup's] symptoms might be due to solvent neurotoxicity. It is important to understand that I have carried out a neurological exam, but do not claim expertise in the diagnosis of solvent neurotoxicity syndrome. In that regard, I would defer to the assessment expert committee.

[48] Dr Timmings concluded that Mr Jessup's neurological status was very similar to his previous presentation.

[49] On 8 July 2020, Dr David Black, Occupational Physician, having seen Mr Jessup, provided a report. Dr Black noted that based on Mr Jessup's service records, he had reasonably intensive contact with solvents, mostly through skin contact. Dr Black reported that in his interview with Mr Jessup, there were clear indications of impaired memory, evidenced by tasks imbedded during the consultation. Dr Black added, *inter alia*:

> … In the late 80s [Mr Jessup] also noted the development of symptoms of fatigue requiring an afternoon nap and then the slow onset of anxiety, slowed information processing with symptoms of cognitive overload and difficulty with attentional function. He became impaired in his ability to divide and alternate attention and was distractible with poor organisational ability and impaired memory. …
>
> I note [that] an MRI of the brain and cervical spine undertaken on the 15th February 2019 had found generalised cerebral atrophy and leukoaraiosis. Although there are a number of possible causes for this, given the coincidence of history and his age causality from solvent exposure appears more likely. …
>
> In summary, in my opinion, there is evidence exceeding the balance of probability that Joe Jessup has neurological damage caused by the use of solvents at work. …

[50] On 21 July 2020, Air Commodore A J Woods, Chief of Staff of the New Zealand Defence Force, advised on the products used by safety and surface technicians at the RNZAF Ohakea base between 1980 and 1986. These products included xylene and toluene. Personal Protective Equipment appeared to be "off the shelf" items, and gloves were likely also "off the shelf" and not designed for paint shop use. Aircraft refinishing tasks typically consisted of a solid flow of work during the normal week; group servicing overhaul/repaints of aircraft had an intense 10-14 day period; and pre-deploymment "touch ups" might have overtime. Normal work hours for a usual week might equate to six to seven hours per day, with possible overtime, and there were no known exposure limits.

[51] On 3 October 2020, Professor Des Gorman, Professor of Medicine and Chair of the Corporation's Toxicology Panel, reported:

> 1. The cognitive function testing undertaken by Dr Newburn was inadequate and the findings he reported were not plausible indicators of neurotoxicity. Nor were they validated by the neuropsychological testing conducted by Dr Prosser.
>
> 2. Dr Prosser found Mr Jessup's performance consistent with premorbid levels of functioning except in one area – visuospatial perception, which is a sub-function of perceptual attention. The tests on which Mr Jessup performed poorly were making a copy of a plan, and (to a lesser extent) matching symbols. On other areas of visuospatial function he performed to expectation. In solvent neurotoxicity the Panel would expect deficits in memory, concentration and executive function as well, but these were all normal. One study had shown that visuospatial processing could be impaired in this condition, but never in isolation as here. Mr Jessup's performance was not typical of solvent neurotoxicity and certainly not diagnostic.
>
> 3. Someone with mild solvent neurotoxicity could function well enough in everyday living and basic tasks but not undertake and succeed in tertiary study, as Mr Jessup did. With solvent-related cognitive dysfunction of any significance it would not have been possible.
>
> 4. Cognitive impairment from solvent neurotoxicity either remains the same or improves following cessation of exposure. It does not worsen progressively or commence after delay, as claimed here.
>
> 5. It is highly unusual for anyone who develops neurotoxicity from heavy solvent exposure not to have had acute symptoms of toxicity at the time. There is no contemporaneous evidence that Mr Jessup did. He did not present to the Medical Centre seeking attention. In his RNZAF Discharge Medical on 14th January, 1986 there is a section entitled "Have you since your last examination experienced any .. "followed by a list of symptoms that include, inter alia, "head injury or concussion", "troublesome headache", "fainting attacks or blackouts", "depression or nervous trouble" and, most significantly "any illness or injury not mentioned above". Mr Jessup answered all of them in the negative. The only positive responses he gave were to backache and hearing loss. Apart from a minor noise-induced hearing loss at 6kHz (still H1 standard) the examination findings at that time were entirely normal and he was discharged on the same medical grading as when he had entered.
>
> 6. Mr Jessup has several commodities in the form of sleep apnoea, untreated depression and the emotional fallout from bariatric surgery. Any or all of them could responsible for the range of symptoms he complained of.
>
> The Panel concluded, from the above, that the evidence was strongly against Mr Jessup having developed solvent neurotoxicity whilst serving in the RNZAF. They noted that both Dr Black and Dr McBride had described very heavy solvent exposure at that time is the cause of the symptoms with which Mr Jessup now presents. The two Panel members who served as medical officers in the New Zealand Defence Force during the 1980s can attest that such was not the case and that high standards of occupational health and safety existed and were adhered to in all three Services.

[52] On 27 October 2020, Mr Jessup underwent a microlaryngoscopy and bilateral vocal fold injection laryngoplasty with Restylane and KTP laser ablation of bilateral VF ectasiae.

[53] On 16 November 2020, after a post-surgical consultation with Mr Jessup, Dr Jacqui Allen, Laryngologist. reported the following:

> It is 3 weeks since he underwent laser therapy and bilateral vocal fold injection augmentation. The voice is louder and which is good however it is still quite rough and hoarse and there can also be vocal breaks. This is making the voice still difficult to use but in a different way. Notable findings at the surgery were the ectasias which I feel will be treated successfully by laser. In addition both vocal folds were very stiff and injection was quite difficult. This is unusual and suggests fibrosis within the vocal fold. We do not see this phenomenon typically with any of the usual systemic diseases such as reflux or sleep disorders. This is more typically identified with post-radiotherapy changes or exposures to toxins.

[54] On 20 November 2020, a diagnostic hearing assessment confirmed that Mr Jessup's hearing loss "extended into the high frequency range with absent responses above 12.5kHz on the right and 10kHz on the left".

[55] On 15 July and 2 November 2020, review proceedings of the Corporation's decision of 12 November 2018 were held. Mr Jessup noted that he was exposed to toxins not just when he was working in the paint shop, but if there was a big (painting) task everyone had to help, regardless of where one was assigned.

[56] On 30 November 2020, the Reviewer dismissed Mr Jessup's review, on the basis that the medical evidence did not support that Mr Jessup suffered a personal injury, or that he suffered it due to employment that involved the prescribed level or extent of exposure, or that he was in an occupation, industry or process of a type described by Schedule 2 of the Act.

[57] On 15 December 2020, a Notice of Appeal was lodged.

[58] On 2 February 2021, Dr Allen advised:

> As previously mentioned these types of vocal changes may be evidenced after chemoradiotherapy treatment for head and neck cancer. As Joe has not had chemotherapy these findings would be more in keeping with toxin exposure

> than any of the proposed co-morbidities mentioned in previous correspondence. Chemotherapy is a toxin which can directly affect systemic tissues and is well known to result in high frequency hearing loss particularly with platinum-based drugs and this pattern may also be consistent with toxin exposure which is of a similar nature to chemotherapy. I note that Joe's recent hearing test shows high frequency hearing loss of this nature. Although I cannot be completely categorical as to the cause, the findings at the larynx do not fit with any other particular systemic disorder or primary neurolaryngeal disorder that I regularly treat.

[59] On 8 July 2021, Air Commodore Woods advised further on the working conditions in and around the RNZAF Ohakea Base painting/surface workshops in the 1980s:

> The extraction system in the Paintshop at this time consisted of three large unfiltered extraction fans. These vented directly into the atmosphere, and gave off a strong smell of paint fumes. … Personnel interviewed advised that at that time the extraction system did cause concern, and depending on the wind conditions fumes could be blown back into the SEMS workshop or over into the Motor Transport workshops nearby where non surface finishing personnel were working. …
>
> Personnel from various trades across the base also visited the Paintshop to obtain thinners, Methyl Ethyl Ketone, Toluene, paint and paint remover for use within their work environments when specified in maintenance procedures. …
>
> The Liferaft Bay used Bostic 2402 (Parts A and B) for the repair of liferafts. This product is an isocynate-based two-pot adhesive. They also used Toluene as a degreasing agent prior to applying two-pot adhesive. …
>
> The Parachute Bay … used Trichloroethylene for cleaning and degreasing of Quick Release Fittings, and this product was stored in the Paintshop dangerous goods locker in 20 litre containers.
>
> In summary, the layout of the Base Ohakea Paintshop between 1980 and 1986 was such that S&S and other trades personnel worked adjacent to surface finishing activities, and in some cases shared communal areas and storage areas with Paintshop personnel. Minor surface finishing tasks were also conducted on aircraft located in their respective squadron hangars. Personal accounts from S&S tradesmen from that era tend to suggest that the Paints hop extraction system may have caused personnel outside of the Paintshop to be exposed to fumes. This assertion is supported to some extent by the subsequent modifications to improve the extraction system. It was also common for S&S personnel to transit between the various work areas in order to provide additional manning in periods of high work tempo.

[60] In January 2022, Mr Jessup provided a statement in which he noted, *inter alia*:

> The Ohakea paintshop was … a converted aircraft hangar. It was not purpose built for toxic sprays. … The Air Force recognised that our existing arrangements needed improved and were unsuited to some of the new painting and stripping schemes w were now using. …

> The masks were only partially effective in terms of paint stripper fumes. Even if you were being careful, the fumes would make you dizzy occasionally. We would stop and get fresh air if this happened. The fumes would make you feel tipsy …
>
> I began experiencing occasional breathing difficulty through 1985. In the latter half of 1985 this became more noticeable. By the end of the day, I would feel short of breath. This generally improved overnight but my beathing during the day was occasionally affected. Over this period my ability to exercise and run became noticeably worse and I put on weight. Work had been busy, and I assumed things would improve when I had more time to train.
>
> I left the Air Force at the beginning of 1986. When I went through the medical, I did not mention my occasional night asthma because I did not consider it important. After leaving the Air Force I struggled to breath during exercise. I had occasional symptoms of asthma through 1986. I saw my GP in February 1987 and was diagnosed with asthma. At this stage my asthma was still occasional. I did however have a serious asthma attack towards the end of 1987 and was admitted to Rotorua Hospital with breathing difficulties. I received nebuliser treatment and was prescribed Ventolin. I have continued to use Ventolin from that day onwards.

[61] On 2 February 2022, Dr Allen provided the following opinion on Mr Jessup's fibrosis within the vocal fold:

> All previous investigations have failed to identify a systemic cause or disease that has lead to these vocal changes. The diffuse fibrosis is very unusual and the vocal fold reaction seems to be post inflammatory or reactive in some way. Having excluded all other potential causes, I think there is a high likelihood that exposure to solvent has triggered an inflammatory response within the superficial lamina propria resulting in vocal fold fibrosis.

[62] On 31 January 2022, Mr Terry Austin wrote that he was employed by the RNZAF from 3 June 1980 until 3 June 2000 as a safety and surface worker. He explained that part of his work tasks included paint stripping and applying new paint schemes using various chemicals. The main product during his time from 1984 to 1987 at the Ohakea paintshop was a product which, when mixed, gave off highly toxic fumes. Despite the provision of masks, the day-to-day reality was that inhalation of chemicals was a common occurrence. It was also common when applying paint stripper to get chemical burns on the skin, as the product would get through the protective clothing. In the latter part of 1985, he started to experience a wheezy feeling at the end of some days after repainting aircraft componentry which caused him difficulty breathing.

**Relevant law**

[63] Section 30 of the Accident Compensation Act 2001 ("the Act") defines a work-related gradual process, disease or infection:

> **30 Personal injury caused by work-related gradual process, disease, or infection**
>
> (1) Personal injury caused by a work-related gradual process, disease, or infection means personal injury—
>
> (a) suffered by a person; and
>
> (b) caused by a gradual process, disease, or infection; and
>
> (c) caused in the circumstances described in subsection (2).
>
> (2) The circumstances are—
>
> (a) the person—
>
> (i) performs an employment task that has a particular property or characteristic; or
>
> (ii) is employed in an environment that has a particular property or characteristic; and
>
> (b) the particular property or characteristic—
>
> (i) causes, or contributes to the cause of, the personal injury; and
>
> (ii) is not found to any material extent in the non-employment activities or environment of the person; and
>
> (iii) may or may not be present throughout the whole of the person's employment; and
>
> (c) the risk of suffering the personal injury—
>
> (i) is significantly greater for persons who perform the employment task than for persons who do not perform it; or
>
> (ii) is significantly greater for persons who are employed in that type of environment than for persons who are not.
>
> (3) Personal injury caused by a work-related gradual process, disease, or infection includes personal injury that is—
>
> (a) of a type described in Schedule 2; and
>
> (b) suffered by a person who is or has been in employment—
>
> (i) that involves exposure, or the prescribed level or extent of exposure, to agents, dusts, compounds, substances, radiation, or things (as the case may be) described in that schedule in relation to that type of personal injury; or
>
> (ii) in an occupation, industry, or process described in that schedule in relation to that type of personal injury.

> (3A) To avoid doubt, where a claim is lodged for cover for a work-related gradual process, disease, or infection, section 57 applies to require, among other things, the Corporation to investigate the claim at its own expense.
>
> (4) Personal injury of a type described in subsection (3) does not require an assessment of causation under subsection (1)(b) or (c).

[64] Clause 35 of Schedule 2 (Occupational diseases) of the Act provides for:

> Chronic solvent-induced encephalopathy diagnosed as caused by organic solvents, particularly styrene, toluene, xylene, trichloroethylene, methylene chloride, or white spirit.

[65] Section 60 of the Act (which comes under the heading "Decisions on cover and entitlements) provides:

> The Corporation may decline a claim that a personal injury is a work-related personal injury of a kind described in section 30(3) only if the Corporation establishes that—
>
> (a) the person is not suffering from a personal injury of a kind described in Schedule 2; or
>
> (b) the person's personal injury has a cause other than his or her employment.

[66] In *Priddle*,[4] Venning J stated the judgment of the Court of Appeal:

> [35] When s 30 is considered in context, we conclude that the intent of the legislature was to provide a separate means of cover under s 30(3) for those people suffering from the occupational diseases listed in Schedule 2 without them having to bring themselves within the definition in s 30(1) and, consequently, the circumstances referred to in s 30(2) at all.

[67] In *Hastings*,[5] Cull J stated:

> [34] … *Priddle* does not stand for the proposition that once a claim is made under s 30(3)(b)(i) that workplace exposure may have occurred, there is a *presumption* of cover. …
>
> …
>
> [39] I also find there is some force in Ms Hansen's submission that there is a further consequence if the applicants' position was upheld. It would mean that a mere assertion by a claimant would trigger s 60 of the Act and ACC would be required to disprove all cases, even those which may be speculative. Section

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4 *Estate of Priddle v Accident Compensation Corporation* (2007) 8 NZELC 98,558.

5 *Hastings v Accident Compensation Corporation* [2019] NZHC 761 (Carl Jonathan Hastings David Saul Briscoe as Executors of Estate of McRae).

30(3) of the Act requires there to be both a Schedule 2 disease and workplace exposure before the onus shifts to ACC under s 60. …

[68] In *Monk*,[6] Henare DCJ stated:

[57] Mr Monk must establish on the balance of probabilities, that he has suffered an occupational disease, and the disease is of a type generally accepted by the medical profession as caused by the metals …

[64] The following principles are discerned from the case law regarding the application of s30(3) and s 60 of the Act:

- Section 30(3) stands on its own which means that the three-part test under s30(2) does not apply.
- There must be proof of workplace exposure, potential exposure or risk of exposure is not a basis for cover.
- Where a claimant establishes both a Schedule 2 disease and workplace exposure under s 30(3) that claimant is not required to prove causation or the other requirements under s 30(1).
- Notwithstanding, a claimant must establish the two requirements are satisfied under s 30(3) on the balance of probabilities. 'Some evidence' does not suffice.
- The onus then shifts to the Corporation under s 60 to establish either that the person is not suffering from a Schedule 2 disease or there is another, non-work related, cause for the disease.

[65] The Court accepts that before the presumption operates, and the onus shifts to the Corporation, Mr Monk must establish under s 30(3) that:

- He has suffered a disease which he claims as listed under items 7, 8, 9 and/or 31 of Schedule 2; and
- He suffered the workplace exposure under items 7, 8, 9 and/or 31 of Schedule 2, being to arsenic or its toxic compounds, mercury or its toxic compounds, lead or its toxic compounds and a diagnosis of lung cancer caused by... cadmium.

[69] In *Jessup*,[7] Spiller DCJ stated:

[34] … The Court accepts that the intent of the legislature was to provide a separate means of cover under s 30(3) for those people suffering from an occupational disease listed in Schedule 2 without the need to meet the level of causation requirements required of other work-related gradual process injuries. However, it is well established in case-law that a claimant must prove, on a balance of probabilities, both requirements under section 30(3), that is, a

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6 *Monk v Accident Compensation Corporation* [2021] NZACC 10.

7 *Jessup v Accident Compensation Corporation* [2022] NZACC 218.

> Schedule 2 disease and workplace exposure. A mere assertion, or some evidence, that these two requirements are met, does not suffice.

[70] In *Ambros*,[8] the Court of Appeal envisaged the Court taking, if necessary, a robust and generous view of the evidence as to causation:

> [65] The requirement for a plaintiff to prove causation on the balance of probabilities means that the plaintiff must show that the probability of causation is higher than 50 per cent. However, courts do not usually undertake accurate probabilistic calculations when evaluating whether causation has been proved. They proceed on their general impression of the sufficiency of the lay and scientific evidence to meet the required standard of proof ... The legal method looks to the presumptive inference which a sequence of events inspires in a person of common sense …
>
> …
>
> [67] The different methodology used under the legal method means that a court's assessment of causation can differ from the expert opinion and courts can infer causation in circumstances where the experts cannot. This has allowed the Court to draw robust inferences of causation in some cases of uncertainty -- see para [32] above. However, a court may only draw a valid inference based on facts supported by the evidence and not on the basis of supposition or conjecture … Judges should ground their assessment of causation on their view of what constitutes the normal course of events, which should be based on the whole of the lay, medical, and statistical evidence, and not be limited to expert witness evidence …

[71] In *J*,[9] Kos P stated:

> [52] In *Accident Compensation Corporation v Mitchell* Richardson J observed that the proper approach to construing the Act was that it be given a "generous and unniggardly" construction. We endorsed that approach in *Harrild v Director of Proceedings*. The importance of this principle lies where more than one available interpretation exists. If the Act is unavoidably niggardly or ungenerous, that is that. But if a reasonable choice presents, the more generous path should be taken.

**Discussion**

[72] Personal injury caused by work-related gradual process is governed by section 30 of the Act. Section 30(3) provides for a separate means of cover for those people suffering from an occupational disease listed in Schedule 2, without the need to meet the level of causation requirements required of other work-related gradual process

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8 *Accident Compensation Corporation v Ambros* [2007] NZCA 304, [2008] 1 NZLR 340.

9 *J v Accident Compensation Corporation* [2017] NZCA 441.

injuries.[10] The occupational disease applicable to Mr Jessup is that listed in clause 35 of Schedule 2, being chronic solvent-induced encephalopathy diagnosed as caused by organic solvents, particularly styrene, toluene, xylene, trichloroethylene, methylene chloride, or white spirit. Mr Jessup is required to prove, on a balance of probabilities, both this Schedule 2 disease and his workplace exposure to organic solvents. A mere assertion, or some evidence, that these two requirements are met, does not suffice.[11]

[73] It is accepted by the Corporation that Mr Jessup meets the second requirement for cover under section 30(3), that is, that he was exposed to organic solvents when in employment. In the years January 1983 to January 1986, Mr Jessup was employed in surface finishing of aircraft at an RNZAF base, and the paints and paint strippers used contained a range of organic solvents, including toluene, xylene and trichloroethylene.

[74] The issue in this appeal is whether Mr Jessup has proved on the balance of probabilities that he meets the first requirement for cover under section 30(3), that is, that he suffers from a chronic solvent-induced encephalopathy diagnosed as caused by organic solvents.

[75] In summary, counsel for the Corporation submits as follows. Mr Jessup's symptoms are more likely than not to be something other than solvent-related encephalopathy. His symptom onset was too late; the progressive nature of his symptoms from 2011 is contrary to the expectation for an encephalopathy of that sort; his testing results make that diagnosis unlikely; and there are a number of other possible causes of his symptomology. The Toxicology Panel's reports on Mr Jessup's condition are the most persuasive, and contrary reports are not reliable and depend on the later, less reliable, history provided by Mr Jessup. Further, he has effectively declined to pursue the further testing proposed by both the Toxicology Panel (treatment for depression) and Dr Black (vision testing).

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10 *Priddle*, above n 4, at [35].
11 *Hastings*, above note 5, at [39], *Monk*, above note 6, at [64], *Jessup*, above note 7, at [34].

[76] This Court acknowledges the above submissions. In particular, the Court acknowledges that Mr Jessup's encephalopathy was not diagnosed until a number of years after his workplace exposure ended in January 1986. The Court also acknowledges the medical evidence of the Toxicology Panel and certain other medical specialists. However, the Court points to the following considerations.

[77] First, this Court finds that Mr Jessup's exposure, without stipulated limits, to organic solvents during his RNZAF employment was significant. The Court notes the evidence of Mr Jessup himself, in his statement and consistently reported by him to the medical specialists who examined him. Mr Jessup's evidence is supported by that of Air Commodore Woods, Chief of Staff of the New Zealand Defence Force, who advised on the working conditions in and around the RNZAF Ohakea Base painting/surface workshops in the 1980s. There is also the evidence of Mr Austin, who was employed by the RNZAF from June 1980 until June 2000 as a safety and surface worker, including his time from 1984 to 1987 at the Ohakea paintshop.

[78] Second, this Court finds that Mr Jessup's recall of when he experienced health issues remained, in its essentials, consistent from his first assessment by Dr Prestage in October 2018 to his assessment by Dr Black in July 2020. Mr Jessup repeatedly referred to the emergence of some health conditions towards the end of his time with the RNZAF, and then the emergence of more significant health issues from around 2011. Differences in detail between earlier and later recollections of Mr Jessup do not necessarily undermine the credibility of his later account. This Court notes the comments of medical specialists, including Dr Newburn (in February 2019) and Dr Prosser (in June 2019), that Mr Jessup relayed events in an open and unembellished manner, without a tendency to exaggeration.

[79] Third, this Court notes the following evidence of medical specialists who examined Mr Jessup, which indicates that his condition was, or could be, that of neurocognitive disorder, consequent on his neurotoxic exposure while working in the RNZAF:

(a) Dr Newburn, Neuropsychiatrist, found that Mr Jessup was, during his time in the RNZAF, significantly exposed to a range of substances that

are known to be neurotoxic, and that there was good evidence for cognitive impairment on formal testing. Dr Newburn found that Mr Jessup suffered from a mild neurocognitive disorder due to neurotoxic exposure, with exacerbation of symptoms due to other factors (small vessel cerebrovascular disease and obstructive sleep apnoea). Dr Newburn noted that these other factors would be unlikely to lead to his current set of symptoms in the absence of the neurotoxic syndrome, which was therefore a necessary cause of his presentation. Dr Newburn later noted that Mr Jessup had benefitted from prescribed medication, and that this further confirmed the hypothesis of neurotoxic brain impairment.

(b) Dr Davis, Radiologist, reported that an MRI done on Mr Jessup's brain showed:

(i) established involutional changes with mild generalised cerebral atrophy, out of keeping with the patient's age; and

(ii) grade 1 deep white matter leukoariaosis;

and noted that these two findings occurred in solvent encephalopathy in about one third of patients.

(c) Dr Wong, Respiratory and General Physician, noted that Mr Jessup had a background of a previous long period of chemical exposure, including TDI, whilst he was working for the RNZAF, and that his health problems included mild neurocognitive disorder.

(d) Dr Prosser, Neuropsychologist, reported that some of Mr Jessup's physical complaints matched the criteria for neurotoxicity syndrome, with some moderate to severe impairment with tasks involving visual processing and speed of processing. Dr Prosser accepted that organic solvent neurotoxicity was one of a number of possible contributing factors to Mr Jessup's current presentation.

(e) Dr McBride, Occupational Physician, diagnosed Mr Jessup as has having chronic solvent-induced encephalopathy (CSE) (type 2B, impairment in intellectual function), with his excessive neurotoxic work exposure as the culprit. Dr McBride accepted that the effects of Mr Jessup's age, metabolic disturbance and hypertension also contributed to his clinical presentation but noted that his reserve capacity would have been reduced by an initial occupational injury.

(f) Dr Black, Occupational Physician, reported that there was evidence exceeding the balance of probabilities that Mr Jessup had neurological damage caused by the use of solvents at work.

[80] Fourth, this Court notes that, in contrast to the above medical specialists, the Corporation's Toxicology Panel did not meet with or examine Mr Jessup in person. The Court observes that the Panel agreed that the cognitive deficits reported by Dr Prosser were consistent with solvent neurotoxicity. This Court finds that the Panel's opinions in important respects are not in line with the weight of factual and evidence presented:

(a) "Mr Jessup's performance was not typical of solvent neurotoxicity and certainly not diagnostic": this Court notes the diagnoses of Doctors Newburn, McBride and Black.

(b) "Cognitive impairment from solvent neurotoxicity either remains the same or improves following cessation of exposure. It does not worsen progressively or commence after delay, as claimed here": this Court notes the study of retired French utility workers referred to by Doctors Prosser and McBride. The study concluded that the risk of solvent-associated cognitive impairment attenuating with time might not be fully true for those with higher exposure.

(c) "[V]ery heavy solvent exposure … was not the case and … high standards of occupational health and safety existed and were adhered to in all three Services": this Court notes the evidence of Mr Jessup, Air

Commodore Woods (Chief of Staff of the New Zealand Defence Force), and Mr Austin which indicate to the contrary.

[81] In light of the above considerations, this Court finds Mr Jessup has proved, on a balance of probabilities, that he has a Schedule 2 disease (chronic solvent-induced encephalopathy diagnosed as caused by organic solvents) and that he had workplace exposure to these solvents. He therefore qualifies for cover for a personal injury caused by a work-related gradual process, in terms of section 30(3) of the Act.

[82] Section 60 of the Act provides that the Corporation may decline a claim that a personal injury is a work-related personal injury of a kind described in section 30(3) only if the Corporation establishes that: (a) the person is not suffering from a personal injury of a kind described in Schedule 2; or (b) the person's personal injury has a cause other than his or her employment. This Court finds that the Corporation has not established either alternative:

(a) As noted above, this Court is satisfied, on the evidence presented, that Mr Jessup is suffering from a personal injury of a kind described in Schedule 2.

(b) The Corporation has not established that Mr Jessup's personal injury has a cause other than his employment:

(i) The Corporation's Toxicology Panel goes no further than to propose that any or all of Mr Jessup's several health issues (sleep apnoea, untreated depression and the emotional fallout from bariatric surgery) could be responsible for his range of symptoms.

(ii) Dr Newburn advised that Mr Jessup's other factors (small vessel cerebrovascular disease sleep apnoea) would be unlikely to lead to his current set of symptoms in the absence of the neurotoxic syndrome, and that sleep apnoea may in itself be a secondary consequence of neurotoxicity.

(iii) Dr McBride advised that additional contributions of age, metabolic disturbance and hypertension would not have resulted in Mr Jessup being in such a poor state, and that the neurotoxic exposure is the culprit.

**Conclusion**

[83] This Court acknowledges that Mr Jessup's case is a difficult one, particularly in view of the passage of time between his relevant employment and the subsequent diagnoses of his condition. As Dr Prosser aptly commented, it is impossible to say with absolute certainty that Mr Jessup's cognitive and psychological symptoms are the result of neurotoxicity. However, Mr Jessup is not required to prove causation with absolute certainty, nor even that his workplace exposure is the only cause of his present condition. He is required to prove, on a balance of probabilities, that his workplace exposure was a material cause of his encephalopathy. In deciding on Mr Jessup's appeal, this Court has had regard to the injunction of the Court of Appeal that, where there is a reasonable choice in interpreting the Accident Compensation Act, the more generous and "unniggardly" path should be taken.[12] This Court finds that Mr Jessup has provided sufficient medical evidence from which to draw a robust decision in his favour.[13]

[84] In light of the above considerations, the Court finds that Mr Jessup has established, on a balance of probabilities, that that he suffers from chronic solvent-induced encephalopathy diagnosed as caused by organic solvents. He has met the requirements for cover for a personal injury caused by a work-related gradual process, in terms of section 30(3) of the Act (a Schedule 2 disease and workplace exposure). The Corporation has not established that Mr Jessup is not suffering from a personal injury of a kind described in Schedule 2, or that his personal injury has a cause other than his employment.

[85] Mr Jessup's appeal is allowed, and the decision of the Reviewer dated 30 November 2020 is therefore set aside.

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12 *J*, above note 9, at [52].
13 *Ambros*, above note 8, at [67].

[86] Mr Jessup is entitled to costs. If these cannot be agreed within one month, I shall determine the issue following the filing of memoranda.

P R Spiller
District Court Judge

Solicitors for the Appellant: Schmidt & Peart Law