

**IN THE DISTRICT COURT
AT WELLINGTON**

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

[2022] NZACC 218

ACR 52/22

UNDER THE ACCIDENT COMPENSATION ACT
2001

IN THE MATTER OF AN APPEAL UNDER SECTION 149 OF
THE ACT

BETWEEN JOSEPH JESSUP
Appellant

AND ACCIDENT COMPENSATION
CORPORATION
Respondent

Hearing: 17 November 2022
Held at: Hamilton/Kirikirioa

Appearances: P Schmidt for the Appellant
S Bisley for the respondent

Judgment: 25 November 2022

RESERVED JUDGMENT OF JUDGE P R SPILLER
[Work-related gradual process injury - s 30,
Accident Compensation Act 2001 (“the Act”)]

Introduction

[1] This is an appeal from the decision of a Reviewer dated 16 March 2022. The Reviewer dismissed an application for review of the Corporation’s decision dated 12 March 2020 declining Mr Jessup cover for asthma as a work-related gradual process injury.

Background

[2] Mr Jessup was born in 1963. He was a safety and surface worker for the Royal New Zealand Air Force (RNZAF) between 1980 and 1986.

[3] During his time at the RNZAF, Mr Jessup was involved in aircraft maintenance, specifically, the stripping and repainting of aircraft. The paints and paint strippers used contained 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.

[4] 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 “no” to questions asking whether he had suffered from: any hoarseness or change of voice; persistent cough; chest pain or discomfort; or undue shortness of breath. The examining medical officer did not record any issue with asthma or breathing.

[5] Around a year after leaving the RNZAF, Mr Jessup was diagnosed with asthma.

[6] On 18 December 2013, Mr Jessup consulted Mr Hans Stegehuis, Ear, Nose and Throat Specialist about a change in voice over the last six months. Mr Stegehuis suspected that the change was related to Mr Jessup’s hand tremor he had had since childhood.

[7] On 8 May 2014, Mr Jessup consulted Dr Paul Timmings, Neurologist, about a hoarse voice. Dr Timmings found that it was not clear whether there was a primary neurological disorder affecting Mr Jessup’s vocal cords. The MRI scan showed a condition consistent with small vessel disease, commensurate with his age and history of hypertension, not representative of demyelinating disease or a vasculitic process.

[8] On 3 August 2018, Mr Jessup, through his GP, Dr Olof Rydin, lodged a claim for cover for neurotoxicity said to have been caused by spray-painting while in the air force. Dr Rydin also completed a medical practitioner questionnaire. He wrote that he was unable to assess whether Mr Jessup's symptoms were due to solvent toxicity as he had never encountered this problem before. He believed that Mr Jessup's claim required specialist assessment.

[9] On 22 October 2018, Dr David Prestage, Consultant Occupational Physician, recorded that Mr Jessup advised that he started having breathing difficulties towards the end of his time with the RNZAF, he was started on Ventolin (an asthma treatment) "shortly" after leaving, and that he was treated with a nebuliser at a hospital in 1988 or 1989. Dr Prestage advised:

Isocyanates are a known cause of occupational asthma due to sensitisation which leads to symptoms on minimal further exposures. This potentially occurred with Mr Jessup but the difficulty is that no formal diagnosis was ever made and treatment with bronchodilators has had inconsistent benefits. The available information does not suggest that reversible bronchoconstriction has ever been demonstrated.

One of the basic tenets in diagnosing a work-related health condition is that a specific diagnosis should be made before linking the condition with a chemical known to cause that condition. This has not occurred in Mr Jessup's case and in my opinion it is impossible to give a definitive opinion on whether his respiratory symptoms have been caused by chemical exposures during his time with the RNZAF ...

Mr Jessup suffers from respiratory, cognitive and laryngeal symptoms without a specific diagnosis ...

Mr Jessup was exposed to a variety of chemicals between 1980 and 1986 but apart from his respiratory symptoms, it is impossible to link his symptoms to these exposures. Occupational asthma may be present but needs to formally be diagnosed.

[10] On 15 February 2019, Mr Jessup underwent an MRI of his brain and cervical spine. Dr Gavin Davis, Radiologist, recorded the findings of generalised cerebral atrophy and leukoaraiosis, with mid-cervical spondylosis.

[11] On 22 March 2019, Dr Janice Wong, Respiratory and General Physician, recorded:

Mr Jessup was emotional and upset, in part because when asked about his solvent exposure and respiratory symptoms while in the Air Force he said that he “cannot recall this very well as it was some 30 years ago”. In particular, Mr Jessup could not recall whether he had respiratory symptoms during his RNZAF service.

In 1986, the year he left the RNZAF, he developed shortness of breath and a wheeze. This was treated with Ventolin initially, and then with other inhalers, but none made a difference to his respiratory symptoms ...

From the respiratory point of view, I believe Mr Jessup has obstructive airways disease with borderline reversibility. He was previously diagnosed obstructive sleep apnoea. 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 issues include mild neurocognitive disorder, language difficulties, hypertension and gastroesophageal reflux disease.

[12] On 31 May 2019, following Dr Wong’s second meeting with Mr Jessup, she recorded that Mr Jessup was not satisfied with her identification of a possible diagnosis of interstitial lung disease as the cause of his respiratory difficulties. He had not performed the tests for isocyanate exposure, refused to perform a hypertonic saline challenge, and declined to follow her treatment recommendations.

[13] On 26 June 2019, Mr Jessup underwent a comprehensive neuropsychological assessment with Dr Prosser.

[14] On 31 May 2019, Dr Andrew Veale, Respiratory Physician, confirmed that a pulmonary function test result on Mr Jessup was consistent with asthma.

[15] On 9 January 2020, Mr Jessup claimed cover for occupational asthma as a work-related gradual process injury. The claim was lodged on the basis of an accident date of 16 January 1986 and an accident description of having worked in the military for six years with paints and solvents, including isocyanates.

[16] On 26 February 2020, Mr Jessup consulted with Dr David McBride, Occupational Medicine Specialist, who reported as follows:

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. Current practice is that occupational surveillance is carried out by monitoring the levels of solvent vapour in the personal breathing zone of the worker, backed up by biological monitoring to detect the levels of solvent metabolites in the urine. Had this been done, I have no doubt that excessive levels would have been detected.

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 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.

I note that he also has a claim for occupational asthma. To this end, he cannot undergo saline or methacholine challenge because of poor lung function, the test would simply not be safe. I also do not believe that he should undergo immunological testing until the sensitivity, specificity and predictive value of the testing with has been explored and the implications explained to him. The reason that I say this is the period of time since exposure: the immune system may have modulated with time. It is also only one part of the diagnostic regime and the assessment should be carried out by a respiratory physician with experience in the diagnosis of occupational asthma.

Note also that toluene diisocyanate (TDI) has been held responsible for reactive airways dysfunction syndrome or RADS, in which case there will be no immune response. There is simply not enough data on the long-term pathophysiological consequence of TDI to refute the claim. Occupational Asthma due to isocyanate exposure is also a Schedule 2 occupational disease.

[17] On 8 March 2020, Dr John Monigatti, Occupational Physician, reported:

Having asthma is one thing; having occupational asthma is another. Mr Jessup maintains that his asthma is attributable to exposure to chemicals, including isocyanates, during his time in the RNZAF. Isocyanates are a well-recognised cause of occupational asthma. Typically, there is a period of sensitisation to the allergen during repeated exposures over a period of days, weeks or even months, during which the body develops antibodies to the isocyanate molecules. Once the person has been sensitised, further exposures trigger an allergic bronchial reaction manifesting as wheezing, breathing difficulty and cough (i.e. an asthmatic attack), which usually occurs within minutes of inhaling the allergen, but can be delayed for several hours. The asthmatic

attack subsides after cessation of exposure but recurs the next time the person is exposed, and this can happen even months or years after the last contact and in response to very low concentrations. When we see this pattern occurring in the workplace the diagnosis is occupational asthma.

The problem with Mr Jessup's claim is that he had no symptoms suggestive of asthma during his time in the NZRAF. He told Dr Prestage he thought he might have done, and Dr Wong that he could not remember. In his RNZAF discharge medical on 14 January 1986 ... to the question "Have you since your last examination experienced any ...?" he answered "no" to "persistent cough", "chest pain or discomfort" and "undue shortness of breath". All branches of the Armed Forces were well aware of isocyanate asthma at that time and the need to protect exposed personnel. ...

... Dr Wong gave him the opportunity for antibody testing to isocyanates but Mr Jessup decided he was being set up to fail ... A positive test would argue quite strongly for isocyanate asthma, as Dr Wong pointed out, whereas a negative test would mean nothing. ...

... Now that Mr Jessup has confirmed that he was indeed asymptomatic during his Air Force service, I agree that [isocyanate antibodies] are not indicated.

[18] On 12 March 2020, the Corporation declined cover for asthma as a work-related gradual process injury, on the basis that his work environment did not cause his condition and/or did not put him to a significantly greater risk of developing his medical condition.

[19] On 15 June 2020, Dr Ron Hayudini, Respiratory, Sleep and General Medicine Physician, confirmed that Mr Jessup had (among other conditions) moderate airflow obstruction likely due to underlying asthma.

[20] On 3 October 2020, a Toxicology Panel (comprising six medical practitioners) reported as follows:

2. ... 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 be 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 as 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.

[21] On 22 February 2022, review proceedings were held. In his evidence at review, Mr Jessup said that he experienced asthma symptoms while employed by RNZAF as early as his first spray painting course (that is, in around 1980), that, “over the years” in the RNZAF, he found his fitness tests harder to pass, and that “sleeping problems from actual lack of breath” were “prevalent”. He said that he did not discuss those problems during his medical examination at discharge because at that stage he had not “had any problems for a few months”, which he attributed to not having had exposure to paints during that time.

[22] In January 2022, Mr Jessup provided a statement in which he noted:

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 breathing 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 breathe 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.

[23] 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. 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. However, these symptoms would usually fade away overnight. In early December 1985, his breathing issues worsened so he sought treatment in the new year. He was given an inhaler which he continued to use until 1990. He was also removed from the paint shop and told not to work with isocyanate-based products. When his ACC claim was processed, isocyanate poisoning was identified as the cause of his asthma.

[24] On 22 February 2022, review proceedings were held. On 16 March 2022, the Reviewer dismissed the review, on the basis that there was no reliable objective evidence to establish that Mr Jessup was suffering from occupational asthma.

Relevant law

[25] 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).

[26] Clause 37 of Schedule 2 (Occupational diseases) of the Act provides for occupational asthma diagnosed as caused by recognised sensitising agents inherent in the work process such as, but not limited to, isocyanates, certain wood dusts, flour dusts, animal proteins, enzymes, and latex.

[27] 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.

[28] In *Priddle*,¹ 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.

[29] In *Hastings*,² Justice Cull 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 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 ...

[30] In *Monk*,³ Judge Henare 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).

¹ *Estate of Priddle v Accident Compensation Corporation* (2007) 8 NZELC 98,558.

² *Hastings v Accident Compensation Corporation* [2019] NZHC 761.

³ *Monk v Accident Compensation Corporation* [2021] NZACC 10.

- 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.

Discussion

[31] The issue in this case is whether Mr Jessup has established on a balance of probabilities that that his condition is occupational asthma diagnosed as caused by isocyanate exposure. The key legal issue is what criteria trigger the operation of section 30(4) of the Act.

[32] Section 30(3) of the Act provides that personal injury caused by a work-related gradual process 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 that involves exposure, or the prescribed level or extent of exposure, to things described in the Schedule in relation to that type of personal injury. Clause 37 of Schedule 2 provides for occupational asthma diagnosed as caused by recognised sensitising agents inherent in the work process, such as isocyanates. Section 30(4) provides that personal injury of a type described in subsection (3) does not require an assessment of causation under section 30(1)(b)-(c) of the Act.⁴ Section 60 provides that the Corporation may decline a claim for a work-related personal injury, of a kind described in section 30(3), only if the Corporation establishes that the person is not

⁴ These subsections require that: (b) the particular property or characteristic causes, or contributes to the cause of, the personal injury; and is not found to any material extent in the non-employment activities or environment of the person; and may or may not be present throughout the whole of the person's employment; and (c) the risk of suffering the personal injury is significantly greater for persons who perform the employment task than for persons who do not perform it; or is significantly greater for persons who are employed in that type of environment than for persons who are not.

suffering from a personal injury of a kind described in Schedule 2, or the person's personal injury has a cause other than his or her employment.

[33] Mr Schmidt, for Mr Jessup, submits as follows. Section 30(4) of the Act is triggered by a claim being filed for cover for a Schedule 2 disease by the claimant's GP, as occurred with Mr Jessup. In filing the claim, the GP must diagnose a Schedule 2 disease and record that the claimant was exposed to a substance listed in Schedule 2 through his or her work. The filing of the claim in this manner creates a presumption of cover. It is both illogical and pointless to assert that claimants can only enjoy the benefit of the evidential presumption provided by s 30(4) after they have proven that work caused their disease. Such an approach makes Schedule 2, the s 30(4) presumption and section 60 pointless. Mr Schmidt also points, in support of Mr Jessup's claim, to the report of Dr McBride, Occupational Medicine Specialist.

[34] This Court acknowledges the above submissions. 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.⁵ 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 Schedule 2 disease and workplace exposure. A mere assertion, or some evidence, that these two requirements are met, does not suffice.⁶

[35] It is accepted that Mr Jessup meets the second requirement, that is, that he had workplace exposure to isocyanates. Mr Jessup must now prove on the balance of probabilities that he meets the first requirement, that is, that he suffers from a Schedule 2 disease, being occupational asthma diagnosed as caused by isocyanates. It is not enough simply to point to the fact that he has asthma and has been exposed to isocyanates. As to whether Mr Jessup has met the required level of proof, the Court points to the following considerations.

⁵ *Priddle*, above n 1, at [35].

⁶ *Hastings*, above n 2, at [39], and *Monk*, above n 3, at [64].

[36] First, the evidence available indicates that Mr Jessup's asthma developed after his workplace exposure ended in January 1986:

- 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 "no" to questions asking whether he had suffered from: any hoarseness or change of voice; persistent cough; chest pain or discomfort; or undue shortness of breath. The examining medical officer did not record any issue with asthma or breathing.
- Dr Wong, Respiratory and General Physician, recorded after interviewing Mr Jessup in March 2019, that, when she asked Mr Jessup about chemical exposure and respiratory symptoms during his time in the Air Force, he said that he could not recall this very well or whether he had any respiratory symptoms during his time there. He said that, the year he left the RNZAF, he developed shortness of breath and a wheeze with a dry cough.

[37] The Court notes that in contrast to Mr Jessup's situation, the clear evidence of Mr Terry Austin, who was also employed by the RAAF doing similar work to Mr Jessup, was that Mr Austin's breathing symptoms emerged, worsened and were addressed while he was still in this employment.

[38] Second, as late as December 2013 and May 2014, medical specialists consulted by Mr Jessup did not diagnose asthma. In December 2013, Mr Stegehuis, Ear, Nose and Throat Specialist, suspected that the recent change in Mr Jessup's voice was related to his hand tremor he had had since childhood. In May 2014, Dr Timmings, Neurologist, found that it was not clear whether there was a primary neurological disorder affecting Mr Jessup's vocal cords.

[39] Third, in October 2018, Dr Prestage, Consultant Occupational Physician, noted, after seeing Mr Jessup, that a specific diagnosis should be made before linking a work-related health condition with a chemical known to cause that condition, and that this has not occurred in Mr Jessup's case. In Dr Prestage's

opinion, it was impossible to give a definitive opinion on whether Mr Jessup's respiratory symptoms had been caused by chemical exposures during his time with the RNZAF.

[40] Fourth, in March 2019, Dr Wong provided Mr Jessup with a blood test request form to assess his exposure to isocyanates, but, in May 2019, she reported that he had not performed the tests for isocyanate exposure.

[41] In March 2020, Dr Monigatti, Occupational Physician, reported that when occupational asthma (as opposed to asthma *per se*) occurred in the workplace, further exposures triggered an allergic bronchial reaction manifesting as wheezing, breathing difficulty and cough, which usually occurred within minutes of inhaling the allergen. The asthmatic attack would subside after cessation of exposure but recurred the next time the person was exposed. The problem with Mr Jessup's claim was that he had no symptoms suggestive of asthma during his time in the NZRAF.

Conclusion

[42] In light of the above considerations, this Court finds that Mr Jessup has not proved on the balance of probabilities that he suffers from a Schedule 2 disease, being occupational asthma diagnosed as caused by isocyanates. He has therefore not established on a balance of probabilities that he is entitled to cover for occupational asthma as a work-related gradual process injury.

[43] The appeal is therefore dismissed, and the review decision of 16 March 2022 is confirmed.

[44] I make no order as to costs.



P R Spiller
District Court Judge

Solicitors: Schmidt & Peart Law for the appellant