

**IN THE DISTRICT COURT
AT WELLINGTON**

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

[2023] NZACC 176

ACR 74/22

UNDER	THE ACCIDENT COMPENSATION ACT 2001
IN THE MATTER OF	AN APPEAL UNDER SECTION 149 OF THE ACCIDENT COMPENSATION ACT
BETWEEN	STUART LEE Appellant
AND	ACCIDENT COMPENSATION CORPORATION Respondent

Hearing: 19 September 2023
Heard at: Auckland/Tāmaki Makaurau

Appearances: Mr P Schmidt for the Appellant
Ms F Becroft for the Respondent

Judgment: 26 October 2023

**RESERVED JUDGMENT OF JUDGE C J MCGUIRE
[Work Related Gradual Process Injury s 30(3), Schedule 2 Clause 35
Accident Compensation Act 2001]**

[1] At issue is a decision by the Accident Compensation Corporation dated 6 December 2021 declining cover for a work related gradual process injury.

[2] The appellant's case is that his Parkinson's Disease was caused by exposure to trichloroethane, an industrial solvent, in his work with New Zealand Post between 1979 and 1986 and that he is entitled to automatic cover under Schedule 2 of the Accident Compensation Act 2001.

[3] The respondent's position is that there is insufficient evidence of a causal link between the appellant's Parkinson's Disease and a workplace exposure.

Background

[4] On 30 June 2021, an ACC injury claim form was filed for poisoning due to a chemical substance. The description of injury was:

Worked for the Post Office from 1979 to 1986, was regularly exposed to trichloroethane and is concerned that his Parkinson's Disease was caused by the exposure.

[5] The accident date listed was 1 March 1979, and his occupation was listed as "electrician". The respondent arranged for the appellant to complete a Work Related Exposure to Substance client questionnaire on 11 July 2021. In it he indicated that he had been diagnosed with Parkinson's Disease in 2009 and that he had been exposed to trichloroethane during his work in the New Zealand Post Office workshops, cleaning parts with no gloves or mask.

[6] The claim was declined by ACC on 26 July 2021 as a medical questionnaire had not been returned to ACC.

[7] Further information was subsequently provided, including a number of literature studies relied on by the appellant which supported a link between trichloroethane and Parkinson's Disease, together with a number of treatment notes.

[8] Dr Wallis, neurologist, provided a report to the appellant's GP on 5 March 2009. In his report he said:

His neurological symptoms started about 12 months ago with intermittent trembling of the right hand. This has become gradually more prominent but remained intermittent. It is aggravated by stress and fatigue.

...

As you know, his mother developed a tremor in her mid-60s and now carries a diagnosis of Parkinson's Disease.

...

Impression

He has the earliest inroads of idiopathic Parkinson's Disease. Although previously we never considered this condition particularly familial in nature, there is now evidence that there are some forms of this disorder that are definitely familial and can be identified with DNA markers. Perhaps his condition falls into that category, but investigations for that do not necessarily change the patient's management.

...

One could certainly hold off on treatment, as he appears to have very little in the way of functional disability. He, however, has a different view and would like to take a trial of drug therapy, and I think this is quite reasonable.

He will start Sinemet 100/25 tabs ...

[9] He was seen by neurologist, Dr Burgen, from 2011 onwards. In his report of 17 March 2011, Dr Burgen said:

I saw Stuart today regarding this Parkinson's Disease. This was diagnosed back in 2009. However he reports that he has gradually had symptoms since about 2006. Initially he developed a tremor of the right hand. This has gradually become more prominent and more persistent, and at times is now also affects the right foot.

...

He saw Dr Wallis almost exactly two years ago. He confirmed a diagnosis of early Parkinson's Disease at that time. Dr Wallis suggested treatment, but Stuart elected not to commence treatment then.

There is a family history of Parkinson's Disease. Stuart's mother was diagnosed between 10 and 15 years ago with Parkinson's Disease.

...

I agree that the features here are of Parkinson's Disease. There are several genes which have now been identified as causing Parkinson's Disease in some families, though since there is no preventative treatment, there is little benefit in making a genetic diagnosis.

[10] In his next report, dated 6 June 2013, Dr Burgen reported:

Stuart is generally well, though he notices that the benefit from each dose of Sinemet wears off after a couple of hours. After this period, the tremor becomes more apparent. The tremor only affects the right hand.

He is managing to work as an electrician, though he reports that he has slowed up. There is some loss of dexterity.

...

I did not notice any tremor at rest ...

[11] Dr Burgen saw him again on 12 January 2015 and noted:

In the clinic he looked well. He did move his hands quite frequently, shifting from an arms folded position to putting his hands on his knees and vice versa, but there was no other evidence of dyskinesia or restlessness.

[12] After a further consultation on 16 February 2016, Dr Burgen reported:

Stuart initially reported that he is “not too bad”. However, it became clear that he does feel despondent at times because of the manifestations of his Parkinson’s Disease. It is now interfering with his work as an electrician.

...

In the clinic today there was a more prominent tremor of the right hand than I recall seeing previously.

[13] The appellant saw Dr Mark Simpson, neurologist, on 5 May 2016 to discuss his potential involvement in the NT Cell trial for Parkinson’s Disease. In his report, Dr Simpson summarised the appellant’s diagnostic history and said:

Stuart doesn’t have any other significant medical problems. His mother was diagnosed with Parkinson’s Disease in her mid-50’s. His father is now taking Sinemet.

...

On examinations, Stuart gave a clear account of his symptoms. He did have rest tremor in the right hand. This was associated with mild Parkinsonism in that there was rigidity and bradykinesia. There was subtle tremor in the left hand.

[14] The appellant had further consultations with Dr Simpson on 20 March 2017.

[15] On 8 March 2021, clinical neuropsychologist, Petina Newton, carried out a neuropsychological assessment. The report noted that the appellant mentioned exposure to trichloroethane which he believes increases the chance of developing Parkinson’s Disease. The report also mentions that the appellant’s mother was diagnosed with Parkinson’s Disease and that his father also had some Parkinsonism.

[16] The file was reviewed by Dr Monigatti, principal clinical advisor and occupational medicine specialist on 4 December 2021. He noted that the appellant had

been diagnosed with idiopathic Parkinson's Disease in 2009 and that there was a family history. More recently however the appellant had associated his condition with trichloroethane. Dr Monigatti said:

Solvents have long been suspected as potential causative agents in Parkinson's Disease because some of them can induce symptoms akin to Parkinsonism. There is evidence that mitochondrial dysfunction being a potential mechanism for the development and progression of Parkinson's Disease and that specific agents linked to Parkinsonism are generally mitochondrial toxins.

...

A systemic review by Lock et al in 2013 found no consistent evidence from the toxicological epidemiological perspective that any specific solvent or class of solvents was a cause of Parkinson's Disease. The authors noted that most TCE animal studies positive for damage to the substantia nigra and striatum had used doses that were 37 to 74 times higher than those which typically occur with human occupational exposures. They recommended further toxicological research and addressed mechanisms of nigra damage from trichloroethane and its metabolites, with the exposure routes in doses relevant to human exposures.

In the current state of knowledge, there is no sound evidence for a causative association between trichloroethane and Parkinson's Disease, or any increase in incidence or prevalence of this condition to point to a significantly greater risk in Mr Lee's occupational group or comparable groups.

[17] On 6 December 2021, the Corporation issued a decision declining the claim again, on the basis that the information provided confirmed that the appellant's work environment did not cause and/or put the appellant at significantly greater risk of developing Parkinson's Disease.

[18] On 9 December 2021, the appellant applied for a review of the Corporation's decision. In a decision dated 13 April 2022, the reviewer dismissed the application, relying on the evidence from Dr Monigatti.

[19] A notice of appeal was filed by Mr Schmidt for the appellant on 29 April 2022. The notice of appeal noted that cover was sought for chronic solvent induced encephalopathy, in the form of Parkinson's Disease, as a Schedule 2 occupational disease.

[20] The appellant filed a brief of evidence dated 11 June 2023, which in large part described his exposure to trichloroethane following his employment with New

Zealand Post Office as an apprentice electrician, a role commencing on 21 February 1979.

[21] In the course of repairing and servicing electrical machinery, he used trichloroethane daily. He said:

11. A cleaning area with a bench was set up in the workshop. A green plastic bin used for cleaning parts sat on the bench. The trichloroethane was poured from a can into the green bin, which we would use our hands and a brush to clean the parts. We did not use gloves and occasionally the skin on my hands would become dry and irritated. Occasionally I would get cracks on the skin, which would be treated with cream. The cleaning area was not equipped with any ventilation and we did not use masks.
12. The cleaned parts would dry off quickly and the product evaporated. The mixture had a very distinctive chemical smell of solvent. On occasion, staff in adjacent work areas would complain about the smell and we would stop the cleaning for a while. Sometimes the smell would be too much and I would have to leave the area and return later. Sometimes I would get itchy eyes and headaches. The smell with the trichloroethane solvent would stick to your clothes, which had to be washed when I got home.
13. After I left the Post Office, I continued to work with solvents in my work as a electrician and data technician, but not to the same extent.

[22] The appellant, through his lawyer Mr Schmidt, had his case reviewed by Dr McBride, occupational medicine specialist. Dr McBride reported on 9 February 2023 and concluded:

Considered together, the toxicological, genetic and epidemiological papers provide good evidence that TCE (trichloroethane) likely does contribute to the onset of Parkinson's Disease and the genetic evidence says that some individuals will be more at risk than others. In this case, there has been heavily TCE exposure, a family history of Parkinson's-like Disease and very early onset of illness (46). This is unlikely to be genetics acting alone. For those reasons, I believe it likely that TCE exposure has contributed to the early onset of Parkinson's in Mr Lee. I note that he did not have material exposure to solvents outside of his work and that the peer reviewed literature does indicate a significantly increased risk of developing Parkinson's Disease in genetically vulnerable individuals exposed to TCE for a significant period, ie. those with a family history of Parkinson's Disease.

[23] In response to Dr McBride's report, Ms Becroft posed a number of questions to Dr Monigatti.

[24] Dr Monigatti reported on 10 April 2023. He commented on the exposure levels to trichloroethane that the appellant indicates in his brief of evidence. He agrees that the neurological advice in this case appears to be that Mr Lee suffers from an idiopathic condition. He discusses the research literature and concludes:

Collectively, these data present an interesting contrast for occupational solvent exposure and neurodegenerative disease, however, small cohort size, recall bias and the long time period over which idiopathic Parkinson's Disease develops, suggest that further study is warranted.

[25] Dr Monigatti says that a family history implies the genetic cause or contribution to developing a disease and that not all family members who developed Parkinson's Disease were exposed to trichloroethane.

[26] As to the early onset of Parkinson's Disease in respect of the appellant, Dr Monigatti notes that with early onset forms of the disease, the appellant's family history may be a factor and there is no reason to implicate trichloroethane instead.

[27] Finally, Dr Monigatti disagrees that the cause of Parkinson's Disease is either workplace exposure or genetics. Dr Monigatti says that most cases of Parkinson's Disease are called "sporadic" or "idiopathic", meaning the causes is not known.

Appellant's Submissions

[28] In his submissions, Mr Schmidt records that the appellant became aware of the link between exposure to trichloroethane and Parkinson's Disease through an article in the New Zealand Herald on 31 June 2021. Following this, his GP filed a claim for Parkinson's Disease caused by workplace exposure to trichloroethane. The claim form notes that Mr Lee worked for the Post Office from 1979 to 1986 and was regularly exposed to trichloroethane.

[29] He refers to the appellant's evidence of his exposure to trichloroethane over the seven years he worked for New Zealand Post Office.

[30] He refers to the reports of Dr McBride and Dr Monigatti and he reminds the Court that the standard of proof is on the balance of probabilities, not to scientific certainty.

[31] He says the question of whether an occupational physician thinks that causation is likely, depends on the view taken of the science. He notes that in the ordinary course, there is at first suspicion as to what gives rise to a disease like Parkinson's and then the science evolves. He says there has been a long held suspicion amongst doctors that Parkinson's Disease could be accelerated by solvents. He notes that with Parkinson's Disease, the myelin sheath starts to decay, leading to a breakdown in efficiency of the nervous system that affects movement and cognition.

[32] He poses the question as to whether Parkinson's Disease is a form of encephalopathy caused by solvents. He notes that Schedule 2 of the Accident Compensation Act, which lists occupational diseases, includes:

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

[33] Mr Schmidt also refers to two scientific studies, Goldman & Ors, Solvent Exposures and Parkinson's Disease Risk in Twins – Ann Neurol 2012 June; 71(6): 776-784; and Neilsen SS & Ors – Solvent Exposed Occupations and Risk of Parkinson's Disease in Finland – Clinical Parkinsonism and Related Disorders, 4(2021)100092.

[34] Mr Schmidt submits that Dr Monigatti acknowledges the worth of these studies linking solvent exposure to risk of Parkinson's Disease.

[35] He submits that further studies would be of assistance, but that cover needs to be determined on the basis of the existing science.

[36] He notes that in the Goldman study, there is confirmation of a significant association between trichloroethane exposure and Parkinson's Disease risk in a population based study.

[37] The study also notes that in all the reports linking trichloroethane exposure to Parkinson's Disease, there is a very long time lag (10-40 years) between exposure and clinical disease.

[38] He submits that we have got to a point where science says early exposure to solvents can cause Parkinson's Disease. In this regard, he says that Dr Monigatti does not really disagree, but just wants more certainty.

[39] Mr Schmidt refers to Schedule 2, Occupational Diseases, under the Accident Compensation Act 2001.

[40] Section 30(3) sets out a category of personal injury caused by work related gradual process disease or infection, including 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 involved exposure to substances described in that Schedule in relation to that personal injury.

[41] Mr Schmidt submits that in our case, to use the wording commonly used in Schedule 2, it is generally accepted by the medical profession that exposure to solvents, in this case trichloroethane, may cause Parkinson's Disease and that in our case, on the balance of probabilities, that has occurred.

[42] He notes that the Court of Appeal decision in *Estate of Priddle*¹ excludes the need to prove causation if the occupational disease is listed in Schedule 2.

[43] He also refers to the judgment of Cull J in *Hastings*² where the Court confirmed that causation does not arise, given Schedule 2, that case involving malignant mesothelioma arising from exposure to asbestos.

[44] Mr Schmidt submits that the literature accepts that with Parkinson's Disease causation is multi-faceted. There may be vulnerability, plus an increased risk if the person is using solvents.

¹ *Estate of Priddle v Accident Compensation Corporation* CA 223/05, 19 October 2006.

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

Respondent's Submissions

[45] Ms Becroft notes that the starting point is that generally, diseases do not attract cover. Also, often the causes of diseases are unknown.

[46] She refers to s 30(2), which includes a requirement that the risk of suffering personal injury is significantly greater for persons who perform the employment task than for persons who do not perform it.

[47] She notes that the Act in s 30(3) sets out an alternative approach to cover for work related gradual process disease or infection, where the personal injury is of a type described in Schedule 2 and is suffered by a person who has been in employment that involves exposure to substances described in the schedule in relation to that type of personal injury.

[48] She describes Schedule 2 as providing a shortcut to cover in appropriate cases.

[49] She also notes that this approach is demonstrated by the words used in most of the clauses in Schedule 2.

[50] She submits that the occupational diseases listed in Schedule 2 are there because there is an established body of science confirming their link to occupational exposure.

[51] She submits that Parkinson's Disease is an idiopathic disease of the brain; a puzzle that medical science is yet to unravel, and is not the type of condition covered by Schedule 2.

[52] She refers to the Ministry of Business Innovation and Employment discussion document dated 17 March 2022 relating to the proposed review framework for the list of occupational diseases in the Accident Compensation Act 2001.

[53] She notes that the document says at paragraph 8, in the portion entitled "An Overview of Work Related Gradual Process Injuries", that:

In order for an occupational disease to be included in Schedule 2, there must be strong scientific evidence of a causal link to render any other cause

unlikely. This would typically require a very high work related risk demonstrated over multiple clinical studies.

[54] She notes that Parkinson's Disease is not listed in Schedule 2 as an "Occupational disease", however she acknowledges that ACC accepts that Parkinson's Disease falls under the umbrella of "Encephalopathy".

[55] However, she notes the contrasting language used in Schedule 2 for different Occupational diseases. There are those conditions described as "generally accepted by the medical profession as caused by...". And there are those Occupational diseases described as "diagnosed as caused by..." The latter wording is used in paragraph 35 of Schedule 2 referring to chronic solvent induced encephalopathy. Therefore, she submits that as the wording of the Schedule is that this Occupational disease must be diagnosed as caused by organic solvents, there is a requirement for causation to be proven

[56] Ms Becroft refers to *Monk*³ and submits that it is not enough just to have a diagnosis, but that it needs to be established that the exposure caused the disease.

[57] She submits that regardless of whether the claim falls for consideration under s 30(2) or Schedule 2, this is an evidential issue with a focus on causation.

[58] She submits that the first Schedule 2 Question is:

Does Mr Lee have a chronic solvent induced encephalopathy diagnosed as caused by trichloroethane?

[59] Secondly:

Is there a particular characteristic of Mr Lee's work that has caused his Parkinson's Disease? Is the risk of suffering Parkinson's Disease significantly greater for persons undertaking the same employment tasks than those who do not?

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

[60] In this case, she refers to the evidence of Drs Burgen, Wallis, and Monigatti, who all agree that the appellant's Parkinson's Disease is idiopathic and the result of a familial link.

[61] She notes that Dr Monigatti says that there is no disease classification system in the world that places Parkinson's Disease under the umbrella of chronic solvent neurotoxicity. Dr Monigatti also says that Parkinson's Disease is not recognised as a solvent induced condition. Dr Monigatti also says that exposure significant enough to cause encephalopathy would likely also have significant acute effects not reported by Mr Lee.

[62] In this regard, she refers to Dr Monigatti's report of 10 April 2023, where he says:

Mr Lee's cognitive function testing was consistent with pre-morbid estimates across all domains, in particular his concentration, memory and learning capacity, which rules out both type 2A and type 2B neurotoxicity.

[63] She notes that Dr McBride, acknowledges that the appellant's condition was not necessarily caused by exposure to trichloroethane.

[64] She also notes that Dr McBride acknowledges that the appellant did not have material exposure to solvents outside of his work and that the appellant, over the exposure period, was in the Post Office workshop for only half the year.

[65] She submits that Mr Lee has a confirmed family history of Parkinson's Disease and there is no reason therefore to link Parkinson's Disease in his case to the trichloroethane exposure.

[66] She therefore submits that the appellant does not meet the requirements of either Schedule 2 (clause 35) or s 30(2), which sets the criteria for otherwise establishing personal injury caused by work related gradual process disease or infection.

Appellant's Reply

[67] Mr Schmidt submits that the thrust of ACC's submissions is that Dr McBride's views are those of an outlier. However, the studies produced to the Court refute that.

Mr Schmidt refers to the increased risk shown in the studies for those who are electronic repairers and electricians.

[68] He submits that the Corporation is looking for a higher standard of proof than what is required by Schedule 2.

[69] He submits that Schedule 2 is intended to give relatively generous cover, otherwise he questions the point of it.

[70] He submits that the appellant should enjoy that cover unless the respondent is able to identify a more likely cause.

Decision

[71] The appellant appeals against the decision of the Accident Compensation Corporation dated 6 December 2021 declining cover for a work related gradual process injury, namely Parkinson's Disease.

[72] On 21 February 1979, the appellant, then aged 17, commenced working for the Post Office. His work was that of an electrician. For the first year of his apprenticeship, he was in the Post Office workshops. After that he cycled through the workshop on a six monthly rotation and during his time in the workshop he had significant solvent exposure related to the tasks he performed in repairing and servicing electrical machinery.

[73] This involved the use of trichloroethane on a daily basis, whilst he was working in the workshop, with nothing in the way of protective clothing or gloves used. This work concluded on 25 July 1986.

[74] In March 2009, he consulted Mr Wallis, neurologist, who diagnosed idiopathic Parkinson's Disease. Dr Wallis, in his report of 5 March 2009, noted that the appellant's neurological symptoms started about 12 months earlier, with an intermittent trembling of the right hand. Dr Wallis diagnosed idiopathic Parkinson's Disease. The appellant was aged 47 when the symptoms first appeared in about March 2008.

[75] An ACC injury claim form was lodged on behalf of the appellant on 30 June 2021, giving the accident date as 1 March 1979, namely, immediately following his commencement of employment with the Post Office. In his claim form is the statement:

Worked for the Post Office from 1979 to 1986, was regularly exposed to trichloroethane and is concerned that his Parkinson's Disease was caused by the exposure.

[76] Both the appellant's parents suffered from Parkinson's Disease. In the report of neurologist, Dr Simpson, dated 9 May 2016, he records the following:

His mother was diagnosed with Parkinson's Disease in her mid-50's. His father is now taking Sinemet.

[77] Dr Simpson further reported on 10 March 2021 that the appellant's father:

... also had some Parkinsonism (ie. expressionless face, quiet voice).

[78] After the claim for cover was lodged, the respondent sought advice from Dr Monigatti, principal clinical advisor and lead occupational health advisor to ACC.

[79] In his report of 4 December 2021, Dr Monigatti referred to a systemic review by Lock et al in 2013 that found no consistent evidence from the toxicological epidemiological perspective that any specific solvent or class of solvents was the cause of Parkinson's Disease.

[80] Following that advice on 6 December 2021, ACC issued its decision declining cover.

[81] On behalf of the appellant, the opinion of Dr McBride, occupational medicine specialist, was obtained. In his report of 9 February 2023. In his report, Dr McBride referred to a case control study of trichloroethane and Parkinson's Disease carried out in 189 discordant twin pairs. The study concluded that the odds of trichloroethane exposure was six times more likely in those with Parkinson's Disease. Dr McBride also referred to a population based case control study from Finland, in which electronic and telecommunications workers had increased odds of exposure to solvents

and the risk of Parkinson's Disease had an odds ratio of 1.63, in other words, a 63 per cent higher chance of Parkinson's Disease, following exposure to solvents.

[82] Dr McBride concluded by saying:

I note that he did not have material exposure to solvents outside of his work and that the peer reviewed literature does indicate a significantly increased risk of developing Parkinson's Disease in genetically vulnerable individuals exposed to trichloroethane for a significant period, ie. those with a family history of Parkinson's Disease.

[83] In a report of 10 April 2023, Dr Monigatti replied to a number of questions following Dr McBride's report. Dr Monigatti said:

No disease classification system in the world places Parkinson's Disease under the umbrella of chronic solvent neurotoxicity and the two conditions do not overlap to any appreciable extent. Parkinson's Disease cannot be construed as a Schedule 2 condition despite Dr McBride's implication to the contrary. For ACC cover purposes, the three part test in s 30 applies.

[84] Dr Monigatti comments on other scientific papers referred to by Dr McBride.

[85] He concludes:

Collectively, these data represent an interesting contrast for occupational solvent exposure and neurodegenerative disease, however, a small cohort size, recall bias, and the long time period over which idiopathic Parkinson's Disease develops, suggest that a further study is warranted.

[86] As to the weight that Dr McBride places on the early onset of the appellant's Parkinson's Disease, Dr Monigatti says:

Early onset forms of Parkinson's Disease are often, but not always inherited, and some forms have been linked to specific alterations in genes. With his family history, Mr Lee is likely to be one of these. There is no reason to implicate TCE exposure instead.

[87] It is noted that in the same study from Finland, which yielded an odds ratio of 1.63, those employed in the role of cooker/furniture worker had an odds ratio of 1.73 and smelting/metallurgic/foundry workers had an odds ratio of 1.5.

[88] The ultimate question to be answered in this case is whether the appellant has brought himself within the cover provisions of the Act.

[89] The focus in this case is on s 30(3) and Schedule 2, which lists occupational diseases. In this case, it is acknowledged by ACC that Parkinson's Disease comes within the purview of encephalopathy, which for our purposes, given the appellant's case that it was solvent induced, is listed at item 35 in Schedule 2, Occupational Diseases.

[90] In this case, there is no argument against the proposition that over the period in question, from 1979 to 1986, the appellant was exposed to trichloroethane.

[91] It appears that Schedule 2 deals with a number of diseases which have been accepted by the medical profession as having been caused by listed exposures to toxic substances. In those cases, cover will effectively be automatic unless the Corporation, under s 60(b) can establish that the person's personal injury had a cause other than his or her employment.

[92] However, as is understandable in this context, there are many occupational diseases listed in Schedule 2, which include the qualification that they are "diagnosed as caused by various substances".

[93] In our case, clause 35 reads:

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

[94] The ultimate issue in this case therefore is "has the appellant established on the balance of probabilities that his exposure to trichloroethane has caused or contributed to the cause of his Parkinson's Disease?"

[95] In this case, the evidence is that both his parents suffered from Parkinson's Disease, with his mother being diagnosed with it in her mid-50's.

[96] While the research papers show an increased risk of developing Parkinson's Disease at odds of 1.63, it necessarily follows from that statistic that many people who have been exposed to trichloroethane will not develop Parkinson's Disease. It is for that sensible reason that Schedule 2 effectively has two categories when it comes to

qualification for cover. The issue of cover is straightforward for those conditions which are “generally accepted by the medical profession as caused by the particular substances, however, for other diseases, including ours, in order to qualify they must be diagnosed as caused by particular exposures. That then requires proof on the balance of probabilities of such causation.

[97] Given the strong familial history of Parkinson’s Disease in the appellant’s case, I am unable to find that on the balance of probabilities that his Parkinson’s Disease has been diagnosed as caused by trichloroethane exposure.

[98] In concluding thus, I am mindful of what *Ambros*⁴ has to say regarding causation: at paragraph 70, where it said:

... The generous and unniggardly approach referred to in *Harrild* may, however, support the drawing of “robust” inferences in individual cases. It must, however, always be borne in mind that there must be sufficient material pointing to proof of causation on the balance of probabilities for a court to draw even a robust inference on causation. Risk of causation does not suffice.

[99] Accordingly, therefore, I conclude that ACC’s decision of 6 December 2021 declining cover for a work related gradual process injury was correct. I must therefore dismiss the appeal.

[100] Costs are reserved.



CJ McGuire
District Court Judge

Solicitors: Schmidt & Peart Law, Ellerslie
Medico Law Limited, Grey Lynn

⁴ *Accident Compensation Corporation v Ambros* [2007] NZCA 304.