IN THE DISTRICT COURT
AT WELLINGTON

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

[2023] NZACC 59 **ACR 304/21**

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

Hearing: 29 March 2023
Held at: Wellington/Te Whanganui-a-Tara by AVL

Appearances: The Appellant is self-represented
L Hansen for the respondent

Judgment: 6 April 2023

RESERVED JUDGMENT OF JUDGE P R SPILLER
[Claim for treatment injury - s 32,
Accident Compensation Act 2001 ("the Act")]

Introduction

[1] This is an appeal from the decision of a Reviewer dated 22 December 2021. The Reviewer dismissed an application for review of the Corporation's decision dated 4 April 2016 accepting cover for a treatment injury.

Background

[2] Mr Austin was born in 1952. In 1982, he was prescribed Roaccutane (the brand name for the drug isotretinoin in tablet form) to treat his acne condition. From 1991 to 1996, and again in 2005, Mr Austin was prescribed Roaccutane by Dr David

Downey, Dermatologist. Mr Austin also suffered spinal pain and stiffness for which he sought chiropractic and osteopathic treatment.

[3] On 29 March 2015, Dr Lucy-May Holtzhausen, Musculoskeletal Medicine Specialist, lodged a treatment injury claim with the Corporation. This claim sought cover for diffuse idiopathic skeletal hyperostosis (DISH)[1] as well as a C6/7disc protrusion as personal injuries, resulting from Roaccutane treatment. The Corporation sought comment on Mr Austin's claim from Dr Downey.

[4] On 28 April 2015, Professor Carl Burgess, Clinical Pharmacologist, commented on Mr Austin's claim. Professor Burgess said that Mr Austin was treated with isotretinoin in the 1980s and 1990s. His initial dosing was not known but the dosing in the 1990s was 20 mg/day. Professor Burgess advised that the first report of hyperostosis with the use of isotretinoin was by Pittsley and Yoder in 1983. He described the findings of that study, as well as other studies. Professor Burgess noted that DISH is a relatively common condition, occurring in approximately 19% of men over age 50 years. He said that it was important to keep this in mind when considering the role of retinoids.

[5] As to whether the extent to which the risk of hyperostosis as a side effect of treatment would have reasonably been expected to be known in the 1980s and 1990s by prescribers, Professor Burgess advised:

> In regard to knowing about hyperostosis following treatment for acne, I would have thought that the earliest this would have been considered would have been in the 1990s, but a recent publication in 2014 (Graf and Whittle) points out that it is often a diagnosis that is overlooked. As the hyperostosis usually does not cause any symptoms there has not been a call to routinely x-ray patients.

[6] Professor Burgess also discussed how this medication came to be used to treat cystic acne. He said that Roche abandoned the drug for use in skin cancer treatment when it was shown to cause major teratogenic effects, but two dermatologists (Pittsley and Yoder) discovered that it was highly effective for treatment of cystic acne. Professor Burgess listed known side effects as ch-y eyes, liver abnormalities,

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1 A form of arthritis that involves the tendons and ligaments around the spine.

and elevation in blood lipids, in addition to the teratogenic effects. For these reasons, Professor Burgess advised:

> … all registration bodies have definite rules on how this drug should be used, namely in short courses with great care to prevent reproduction as the risk of teratogenicity is very high. The risk of bone changes has stressed that the use in children is associated with closure of the epiphyses. All these effects are effects that one might expect with excessive vitamin A.

[7] On 6 May 2015, Dr Downey reported, setting out the doses he had prescribed to Mr Austen during 1991 to 1996 and in 2005. Dr Downey advised that Mr Austen had been prescribed isotretinoin earlier from Dr Gray in Takapuna, but Dr Downey did not have information about that dosage. Dr Downey stated:

> His approximate cumulative dose, based on 80kgs body weight, is somewhere between 160 to 180mg per kg. This is just outside the internationally recognised cumulative dose of 120mg to 140mg per kg.

[8] Dr Downey advised that there was no recommended length of time an individual should remain on isotretinoin, but nine to 12 months is a standard regime. Severe persistent cases are not often controlled on a low dose of Isotretinoin, such as 10-20 mg per kg. Dr Downey noted that Mr Austin was on this medication on an intermittent basis from 1991 to 1996. Dr Downey stated that he had comprehensive knowledge of the side effects of isotretinoin at the time he prescribed it:

> My clinical knowledge of the side effects of this drug at the time of prescribing was comprehensive and included discussion of birth defects, mucocutaneous side effects, ophthalmologic, neuromuscular and gastrointestinal side effects. Side effects relating to dry skin on mucous membranes as well as elevated serum lipids and hepatotoxicity were also known. Bone abnormalities were documented with a high dose of Isotretinoin in paediatric population at doses exceeding 2-4 mg per kg on a daily dose. In the paediatric population prolonged high dose daily use (2 mg per kg per day) for prolonged periods over two years had been possibly thought to be related to premature closing of the epiphyses and DISH. Cases report for DISH and Isotretinoin were mainly for longer term use of high dose Isotretinoin and in diseases other than acne.
>
> Little is known about the pathogenesis of DISH, which can also occur in the absence of Isotretinoin treatment and can be furtl1er confounded by conditions such as ankylosing spondylitis. As the patient is at an age where other factors could be appearing association to Isotretinoin is more difficult.

[9] Dr Downey advised that he informed Mr Austin of the known side effects at the time as they related to the adult population. Dr Downey did not consider that the

causal relationship between Mr Austin's use of this medication and his development of DISH could be firmly established.

[10] On 7 August 2015, Dr Chris Maughan, GP, Corporation Medical Advisor, reviewed Mr Austin's claim and reported to the Corporation. He considered Mr Austin's treatment records from Dr Downey, Dr Holtzhausen, and Mr Ferguson (Spinal Surgeon), as well as an MRI scan. Dr Maughan questioned whether Mr Austin had any significant hyperostosis at all:

> There is no note of hyperostosis in report of spinal surgeon or in the MRI scan. This begs the question of whether there is objective evidence of significant hyperostosis? And if significant hyperostosis is confirmed:
>
> DISH by definition is idiopathic i.e. of unknown cause.
>
> However, Roaccutane is a retinoid drug associated with hyperostosis. But Professor Burgess notes the spontaneous occurrence of DISH as relatively common - estimated as occurring in 19% of men over the age of 50 years and that it is important to keep this in mind when considering any role of retinoids. Hence if significant hyperostosis is confirmed:
>
> > The injury may represent only underlying DISH hyperostosis.
> >
> > Or the injury may be retinoid induced diffuse skeletal hyperostosis (versus idiopathic DISH).
> >
> > Or could the injury constitute a defined progression of underlying DISH hyperostosis?
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> Dr Burgess was not asked to address the injury in relation to hyperostosis, and he was not asked whether or not treatment with the retinoid Roaccutane likely caused its development in this case.

[11] Dr Maughan considered the available evidence about the dose of Roaccutane taken by Mr Austin, noting Dr Downey's advice that this was just outside the internationally recognised cumulative dose of 120 to 140 mg per kg. Dr Maughan referred to information from Medscape describing DISH, noting that it appeared to be a phenomenon rather than a disease. But, Dr Maughan said, if significant hyperostosis were confirmed, it would likely constitute physical change/bodily damage and therefore meet the Act's definition of personal injury. Dr Maughan considered Professor Burgess' advice about the causal link between isotretinoin and hyperostosis. Dr Maughan concluded:

> The figures would not appear to support prescription in this case at the low cumulative dose noted between 160 to 180mg per kg just outside the internationally recognised cumulative dose of 120 to 140mg per kg) as a probable cause of hyperostosis taking also into account that hyperostosis may occur in nearly 20% of men over the age of 50.

[12] Dr Maughan suggested that the Corporation first needed to confirm that Mr Austin had significant hyperostosis (that is, a personal injury), and then ascertain the timing of its onset in relation to his treatment with isotretinoin. Dr Maughan said that the Corporation should ask Dr Holtzhausen whether there were specific findings for DISH, outside the range of ossification of ligaments and osteophyte development that might ordinarily occur in a man of Mr Austin's age. If so, the question would then be when these specific signs first developed. If significant hyperostosis could be confirmed, Dr Moughan advised:

> It will be necessary to ask Professor Burgess as expert for ACC to address the hyperostosis - whether he is able to say that treatment with the retinoid Roaccutane is likely to have caused development or defined progression of hyperostosis in this case - versus underlying "idiopathic" development?

[13] On 22 August 2015, Dr Holtzhausen responded to the Corporation's questions. She addressed the nature of DISH and its prevalence and explained the diagnostic criteria. Concerning Mr Austin's diagnosis, she said that, after delving back into his past radiological reports, she found a chest x-ray taken in November 2013 which had reported moderate flowing ossification of the anterior longitudinal ligament suggestive of DISH. Dr Holtzhausen said that she had reviewed this imaging herself and that it supported a clear diagnosis of DISH. She said that more recent x-rays showed DISH also affecting Mr Austin's cervical spine. Dr Holtzhausen explained how DISH differed from spondylosis. She said that Mr Austin was aware of only one other set of spine x-rays taken and this was by his chiropractor, Mr Alley, in 2011. Dr Holtzhausen said that Mr Alley apparently undertook a full chiropractic spine x-ray series in November 2011, which demonstrated flowing osteophytosis of DISH-like nature in his cervical and thoracic spine segments. Dr Holtzhausen said that x-rays of Mr Austin's left hand, taken in 2012 and 2013, provided some evidence for peripheral enthesopathy involvement. In short, Dr Holtzhausen advised that Mr Austin had DISH. She was unable to say, however, when these changes first occurred because there were no x-rays taken before 2011.

[14] Dr Holtzhausen then went on to discuss the pathogenesis and aetiology of DISH. She described it as a bone-forming disease, and explained that vitamin A and its derivatives have the ability to promote new bone formation. She said:

> The main skeletal abnormality described in synthetic retinoid treated patients is identical to DISH. Yoder was the first to draw attention to this problem in the early 1980s when he reported symptoms and radiological evidence of DISH in 5 out of 7 patients treated with a high dose of isotretinoin (Roaccutane-UK, Accutane-US: 3 mg/kg/ day) for 3 years [Yoder FW. J. Am. Med. Ass. 249: 350-351 (1983)]. This initial report was followed up by his classic paper with Pittsley, which reported the development of DISH-like changes in 4 patients receiving high dose long term isotretinoin [citation omitted]. McGuire and colleagues confirmed these findings describing flowing ossification of the anterior longitudinal ligament in patents receiving hi dose synthetic retinoids. …
>
> Since Mr Austin weighed 75kg in the 1990s when he was on a dose of between 30mg and 60mg/ day he would have been receiving 0.4mg/kg of Roaccutane per day for a 4 year period and then 0.8mg/ day for a further 4 years. So it is highly likely that Mr Austin's florid radiographic evidence of DISH-like spinal lesions could have been set in motion with these high doses of Roaccutane given the prolonged period he was on this medication. The recommended dose for short term isotretinoin use in acne in the 1990s following Yoder, Pittsley and McGuire's papers, was less than 0.5 mg/kg/ day to be given for no longer than a 6 month period to avoid developing DISH-like spinal lesions.

[15] Dr Holtzhausen advised that adults on isotretinoin have an increased tendency to develop hyperostosis and should be monitored by serial spinal x-rays. Dr Holtzhausen noted that Mr Austin was not warned of the risk of spinal bony side effects of his medication or given the option to have regular x-rays. Dr Holtzhausen also noted a study by Kilcoyne et al (1986) addressing the timing of the appearance of DISH-like changes after treatment with a synthetic retinoid. The study found 10 of 96 patients had DISH-like spinal lesions. In seven cases, the spurs were suspected at six months, but in all cases were visible 11-14 months after treatment started. Dr Holtzhausen described Mr Austin's history of spinal pain and chiropractic treatment from the late 1990s onwards. She said it was not inconceivable that Mr Austin's symptoms at that time were due to bouts of acute arthritis set in motion by his Roaccutane therapy.

[16] The Corporation referred Mr Austin’s claim back to Professor Burgess and asked him to consider two questions: whether Roaccutane had likely caused

Mr Austin's DISH, and, if so, whether this was an expected or ordinary outcome in this particular case.

[17] On 24 September 2015, Professor Burgess responded to the Corporation's questions. He agreed that there was radiological evidence of DISH, but said that it was difficult to quantitate the degree associated with age. Professor Burgess said that the first time Mr Austin complained of pain or discomfort was in 2013, when he was over age 60. Professor Burgess advised:

> As referred to in my first report the reports of this complication were in children and young adults given large doses of the retinoid. I think it played some role, but I don't think it is responsible for his lumbar problems as there is loss of disc height, a finding much more suggestive of spondylosis (osteoarthritis). Similarly the cord lesion in the cervical spine is related to herniation of the disc, not a finding with DISH. I note the MRI report of CS/ 6 also notes decrease in the invertebral disc. The spur indenting the larynx is undoubtedly due to DISH. So, it may be that age is the biggest determinant here. In a recent study (Yaniv et al 2014), where patients with DISH were followed between CT scans, just under a quarter of the patients did not show any increase in the degree of DISH, but these patients were not followed from the onset of DISH. My opinion is that isotretinoin (Roaccutane) may have played some role, but age is a significant factor here.

[18] Concerning whether hyperostosis was an ordinary consequence of the treatment, Professor Burgess said that, if isotretinoin were the major factor, this would not be an expected outcome, as this is a rare complication of the use of isotretinoin.

[19] On 20 October 2015, Mr William Taine, Orthopaedic Surgeon and the Corporation's Medical Advisor, reported that Mr Austin was currently 63 years old and began seeking treatment for spinal pain and stiffness in the early 2000s. Mr Taine reviewed the radiological findings, noting multilevel degenerative changes and a right C6/7 disc protrusion. Mr Taine noted the records relating to Mr Austin's use of Roaccutane in the 1990s and early 2000s, as well as mention of earlier use in the 1980s and as a youth.

[20] Mr Taine confirmed that Mr Austin had DISH. Mr Taine said that, as its name suggests, by the use of the term idiopathic, the cause of the condition is not known. He noted that it was not always symptomatic and was often an incidental finding. He explained that DISH is distinct from ossification of the posterior longitudinal

ligament and from spondylosis. Mr Taine said that the incidence of DISH among people in Mr Austin's demographic was between 18 to 30%. Mr Taine noted that Mr Austin's spinal symptoms experienced in his 50s may well have been due to the presence of spondylosis, rather than DISH. Mr Taine did not consider that there was a clearly established link between Roaccutane use and bone changes:

> The reports of hyperostosis in association with its use are not based on large numbers of patients, but appear often to be case reports or small groups, so incidence across all those treated with this medication cannot be established. Many of the reports concern younger adults where hyperostosis would be very unusual, so tl1at a causal link could be taken as probable. One report suggested that tl1ere was a high risk of progression of existing hyperostosis (40% compared to nearer 15% witl1 placebo): however this is progression of an existing process as opposed to causation....
>
> Without a figure providing the risk of occurrence de novo in a patient taking Roaccutane, and with a known incidence in this demographic, it cannot be said with any certainty that the development of DISH in this patient is due to the medication.

[21] Mr Taine said it was more likely in this case that the medication accelerated the progression of DISH:

> An alternative view is that the patient may have had or been going to develop the condition and that the medication accelerated its development. This is supported by the study showing a 40% progression in patients with existing hyperostosis. The reference is Tangrea et al (1992) Arch Dermatol. 128:921. Given this figure, which is higher than the base incidence of DISH in males in their 60s, this is I think the most appropriate conclusion: that the medication probably accelerated the progression of DISH in this particular patient.

[22] Mr Taine then considered whether this was an ordinary consequence of the treatment. He said that it was not possible to state definitively whether it was an ordinary consequence because the incidence of DISH caused by Roaccutane is not established. Still, he said, he tended toward the view that it was not an ordinary consequence:

> Given the absence of firm recommendations regarding screening in adult patients being treated with this medication, which would suggest a low incidence of symptomatic disease. This lack of recommendation may be due to that lack of symptoms but does suggest that it is not an expected clinical problem.

[23] Mr Taine emphasised that many of Mr Austin's symptoms could be attributed to the degenerative changes in his spine, rather than to DISH. But Mr Taine

confirmed the presence of DISH, noting that it was quite widespread. He said that it may have been caused by the medication, have been accelerated by the medication, or be entirely coincidental. Mr Taine concluded that the second option (acceleration) was the most probable scenario.

[24] On 1 December 2015, Dr Quentin Reeves, Diagnostic Radiologist, confirmed the presence of DISH. but said that it was impossible without sequential films to determine whether this is early or late onset. He added, however, that the appearances would not be atypical for this age group. Dr Reeves said that there were changes of DISH with superimposed degenerative changes of several of the facet joints. He said that the degenerative changes were minor. He was unable to comment on the cause of the disc protrusions at C5/6 and C6/7, but noted that they were very common in Mr Austin's age group. Dr Reeves said that DISH can typically cause variable degrees of stiffness in the spine.

[25] On 11 December 2015, the Corporation accepted cover for DISH, secondary to Roaccutane use in the 1980s and 1990s. The Corporation agreed that, on balance, the use of Roaccutane in the 1980s and 1990s had resulted in DISH and that this would not be an expected or ordinary outcome of treatment. In the same decision letter, the Corporation declined cover for spondylosis. The Corporation said that this was a degenerative condition and excluded from cover.

[26] Mr Austin presented further information in support of his claim that his spondylosis and cervical disc pathology were also linked to Roaccutane.

[27] On 4 April 2016, the Corporation issued a revised and amended decision on Mr Austin's claim to clarify the scope of his covered injury. The Corporation decided to accept cover for DISH manifested by osteophytes in the cervical spine and thoracic spine as a treatment injury resulting from Mr Austin's use of Roaccutane. The Corporation declined cover for spondylosis and for cervical disc protrusions at CS/ 6 and C6/7.

[28] On 5 April 2016, the Corporation advised that it owed Mr Austin backdated weekly compensation of $159,548.60. The Corporation paid Mr Austin ongoing

weekly compensation of $1,553.91, to cease when he reached superannuation age in 2017.

[29] On 25 August 2016, the Corporation received Mr Austin's application to review its decision declining cover for spondylosis and cervical disc protrusions.

[30] On 6 December 2016, at review, Mr Austin argued that his degenerative disc disease was the result of taking oral retinoids over a long period and should be covered as a treatment injury:

> First prescribed Roaccutane in mid-80's when I was 33 years old and living in Hamilton, for a greasy, infected skin. Course duration likely 6 months.
>
> In 1991 my GP Andrew Murley referred me to David Downey, a dermatologist practising on Auckland's North Shore.
>
> I went back on Roaccutane in 1991 and was taking the drug on and off through to 2005.
>
> At no time over this extended period did David Downey ever suggest I should have my spine x-rayed to see if any abnormalities were presenting.
>
> From the mid 90s's I started having neck and spinal issues, stiffness, pain, partially prolapsed discs and nerve compression etc.
>
> Bays Chiropractic x-rayed my spine in 2011 and I was told I had the neck of a 70 year old person.
>
> In 2014 I was finally forced to give up work, liquidate my golf importing business of 17 years and to try and find out what was going on with my health.
>
> Referred to Dr Lucy Holtzhausen in 2014 and had a MRI Scan of Lumbar Spine showing marked loss of disc height and dehydration of L4/ 5 with other levels reasonably well prese1-ved.
>
> In late 2014 I started having severe pain in the neck region and also swallowing difficulties.
>
> In February 2015 sent for a Cervical MRI which showed dramatic and profound abnormalities in the cervical spine.
>
> Advised by Dr Lucy Holtzhausen that this among of damage may have been cause by Roaccutane and she asked if I have ever been on Roaccutane medication. Dr Holzhausen advised that oral retinoids such as Roaccutane are highly toxic and can cause severe spinal abnormalities. This is especially evident with adult patients.

[31] On 21 December 2016, the Reviewer found that Mr Austin's spondylosis and cervical disc protrusions were not treatment injuries. The Reviewer said that the majority of the expert opinion agreed that causation had not been established.

[32] In 2016, Mr Austin initiated a claim in the High Court against Roche Products (New Zealand) Limited ("Roche"), the New Zealand distributor of Roaccutane. He sought compensatory and exemplary damages. Roche applied to strike out Mr Austin's claim for compensatory damages as barred by the Act. The Court of Appeal agreed and struck out the claim. The Supreme Court granted leave to appeal.

[33] On 21 March 2021, the Supreme Court issued its decision in the appeal.[2] The Court noted as follows:

> [20] Section 133(5) is … triggered by … the making of a claim for which there is a right of review or appeal. "Claim" is defined in s 6 as a claim—that is, an application—to ACC for coverage under s 48 of the Act. So a claim is an application for an entitlement under the Act. It is not actual entitlement. The effect of s 133(5) is therefore that once a person lodges a claim, they are locked into the Act's procedures. No court may "consider or grant remedies in relation to that matter if it is covered by [the] Act". …
>
> [35] Given the Act's comprehensive system for challenging coverage decisions, including a right of appeal on a point of law to the High Court, and in light of the terms of s 133(5), we conclude that this Court does not have jurisdiction to consider Mr Austin's appeal. Nor did the Courts below.

[34] On 19 April 2021, Mr Austin lodged an application to review the Corporation's decisions of 11 December 2015 and 4 April 2016. He asked for a finding that his injury was an ordinary consequence of the treatment he received, and therefore outside the scope of accident compensation.

[35] On 23 July 2021, the Corporation accepted that extenuating circumstances affected Mr Austin's ability to lodge his review in time.

[36] On 13 December 2021, review proceedings were held. The focus of the submissions presented on behalf of Mr Austin was that his injury should be excluded as an ordinary consequence of extended, high-dose treatment with Roaccutane. His advocate also noted in passing that some expert opinion did not find causation at all.

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2 *Austin v Roche Products (New Zealand) Limited* [2021] 1 NZLR 294.

[37] On 22 December 2021, the Reviewer dismissed the review on the basis that Mr Austin's skeletal hyperostosis is a personal injury caused by treatment. The Reviewer found that the evidence was sufficient to establish a causal link between Mr Austin's use of Roaccutane and his skeletal hyperostosis, and that it was not an ordinary consequence of the treatment.

[38] On 22 December 2021, a Notice of Appeal was lodged.

[39] On 11 August 2022, Dr Holtzhausen confirmed that it is highly probably that the action of Roaccutane therapy over a 20-year period has led to Mr Austin's disc degeneration. She noted that she initially considered Mr Austin's Roaccutane dosages to be high, "but on furthermore recent research it has shown that the dosages used were always quite low and it seems that the prolonged period of usage has been the determining factor with Mr Austin's outcomes". She added:

> I have been seeing Mr Austin since 2014, and have now concluded that all his spinal hyperostosis has been caused by prolonged use of retinoids such as isotretinoin (Roaccutane).
>
> His disc disease has been accelerated to a large degree by the calcified damage appearing all around the affected discs.
>
> It would appear that although that although Roaccutane was not a direct cause of Mr Austin's disc disease, the Roaccutane-caused calcified bony material, as reported in the spinal surgeon's operation note exhibit 0, has been the largest contributor to the deterioration in at least three disc levels of Mr Austin's spine.
>
> I also wish to confirm that all three spinal segments (cervical, thoracic, and lumbar) have suffered excessive bony lesion s from Roaccutane therapy and that in all three cases those early bony lesions ere an expected and ordinary consequence of the treatment and are, in no way accidental.
>
> It would have taken 10-15 years for those bony lesions to grow into large, clinically significant osteophytes, as finally diagnosed in 2015 by myself.

[40] On 10 November 2022, Dr Garry Brown, Medical Advisor, advised that the most appropriate description of physical injury in Mr Austin's treatment injury claim is skeletal hyperostosis. On the issue of whether the personal injury was an ordinary consequence of Mr Austin's retinoid therapy, Dr Brown said that it is not. He advised that the cumulative body of evidence indicated that skeletal hyperostosis is a rare adverse outcome. He stated:

> With respect to Dr Holtzhausen, whose expertise is in the diagnosis and treatment of musculoskeletal disorders, I note her conclusion that causally related to the use of Roaccutane/isotretinoin it is quite unusual in the wrist to see profuse hyperostosis.
>
> I do not agree with Dr Holtzhausen's conclusion that 'all three spinal segments (cervical, thoracic, and lumbar) have suffered excessive bony lesions from Roaccutane therapy and that in all three cases these early bony lesions were an expected and ordinary consequence of the treatment and are in no way accidental'.
>
> There is no expert evidence on file to indicate Mr Austin was considered more or less susceptible to skeletal hyperostosis following treatment with isotretinoin for acne.
>
> The available evidence indicates Mr Austin has been treated with an average daily dose less than 0.4 mg/kg. This sits within the range defined in a Cochrane Systematic Review as low dose treatment 0.25 0.40 mg/kg/day.
>
> There are infrequent reports of skeletal hyperostosis associated with the use of oral isotretinoin and related retinoids. These cases are consistently associated with considerably larger mean daily doses - in the order of between 3.8 and 10 times higher e.g.1.5 - 4.0 mg/kg/ day - tl1a11 that mean daily dose given to Mr Austin as treatment.
>
> Both the literature, and expert pharmacology opinion, support that the adverse outcome of skeletal hyperostosis is a rare event, which likely represents an individual reaction e.g. one that cannot be identified in advance.
>
> There are no factors to indicate tl1at Mr Austin was more or less susceptible to this adverse outcome at the time of his treatment. The injury outcome appears enduring - and of significant impact and severity. The medical evidence and the opinion of Dr Holtzhausen (excessive bony lesions... in all three spinal segments) supports that conclusion.
>
> Taking all of these circumstances into account, the adverse outcome in the case of Mr Austin is surprising, and in my opinion is not an ordinary outcome of treatment in his case.

[41] On 21 November 2022, Mr Austin filed evidence responding to Dr Brown's evidence. He said that the correct physical injury is retinoid hyperostosis. He said that Dr Downey's letter to the Corporation dated 6 May 2015 "answers the question of informed consent" and that he "does not wish to engage in pure speculation over informed consent or medical knowledge as it is felt that both issues are too arbitrary to be relied on". He took issue with Dr Brown's opinion that the diagnosis was not an ordinary consequence. Mr Austin disputed that Dr Brown considered the correct retinoid therapy applying to him. Mr Austin provided a table of his isotretinoin use which he said was use of fourteen months between 1982 and 2005. He said that he used it during 14 of those 23 years.

[42] Mr Austin emphasised Dr Hotzhausen's view that it was the prolonged nature of his retinoid therapy that has ultimately led to all his skeletal problems and that such outcomes are not rare for adults on prolonged retinoid therapy. He said that the evidence shows that adults on prolonged therapy almost without exception develop retinoid hyperostosis.

Relevant law

[43] Section 32 of the Accident Compensation Act 2001 ("the Act') provides:

> **32 Treatment injury**
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> (1) Treatment injury means personal injury that is—
>
> (a) suffered by a person—
>
> (i) seeking treatment from 1 or more registered health professionals; or
>
> (ii) receiving treatment from, or at the direction of, 1 or more registered health professionals; or
>
> (iii) referred to in subsection (7); and
>
> (b) caused by treatment; and
>
> (c) not a necessary part, or ordinary consequence, of the treatment, taking into account all the circumstances of treatment, including
>
> (i) the person's underlying health condition at the time of the treatment; and
>
> (ii) the clinical knowledge at the time of the treatment.
>
> (2) Treatment injury does not include the following kinds of personal injury:
>
> (a) personal injury that is wholly or substantially caused by a person's underlying health condition:
>
> (b) personal injury that is solely attributable to a resource allocation decision:
>
> (c) personal injury that is a result of a person unreasonably withholding or delaying their consent to undergo treatment.
>
> (3) The fact that treatment did not achieve a desired result does not, of itself, constitute a treatment injury.

[44] In the High Court judgment in *Adlam,* [3] Gendall J stated:

> [39] And, the ACC's interpretation here in my view is also consistent with these definitions and the context of the provision whereby s 32(1)(c) requires that treatment injury not be a necessary part or ordinary consequence of the

[3] *Accident Compensation Corporation v Adlam* [2016] NZHC 1487, [2016] 3 NZLR 497 at [39].

> treatment, taking into account the clinical knowledge at the time of the treatment. The Court of Appeal in *McEnteer v Accident Compensation Corporation* has held that s 32(1)(c) requires an analysis that is rooted in the facts of particular cases, requiring expert opinion reflecting what actually occurred.

[45] In the Court of Appeal judgment in *Adlam*,[4] Cooper J stated:

> [62] Taken as a whole the provisions indicate a legislative intent to limit cover for persons who suffer injury while undergoing treatment, rather than providing cover for all those who suffer. The injury said to be a treatment injury must be the consequence of a departure from appropriate treatment choices and treatment actions. The drafting could have simply provided for cover for all injury suffered while a person undergoes treatment. But that course was not taken. Rather, boundaries were set out that have the effect of limiting the availability of cover for injury during treatment. A failure in the sense of omitting to take a step required by an objective standard is necessary. …
>
> [65] As is always the case, it is necessary to focus on the words Parliament has actually used. It will be apparent from our reasoning that we have discerned a legislative policy that, while not requiring a finding of negligence, still operates on the basis that a treatment injury will only have occurred where there has been some departure from a standard and that departure has caused a personal injury.

[46] In *Ng*, [5] the Court of Appeal stated the following in relation to the phrase "not [an] ordinary consequence":

> [68] In our view, it should be interpreted as meaning an outcome that is outside of the normal range of outcomes, something out of the ordinary which occasions a measure of surprise. That is an interpretation that we consider, as did the Court in *Childs v Hillock*, best captures Parliament's intent in the context of a scheme which is underpinned by the concept of "personal injury by accident" and which does not provide universal compensation for sickness or ill-health. So, for example, side effects of chemotherapy of a nature and severity that are encountered reasonably often and occasion no surprise are ordinary consequences of that chemotherapy even if (as will often be the case) such side effects are not encountered in more than 50 per cent of cases.
>
> [69] Whether an adverse consequence is inside or outside the normal range of consequences of the medical treatment given to a particular claimant is ultimately a matter of judgment for the decisionmaker. It is to be exercised on a case specific basis taking into account all of the circumstances of the treatment and the particular claimant. Thus, relevant considerations will include not only the nature of the harm suffered but also its duration and severity as well as any other circumstances pertaining to the patient which may have rendered them more or less susceptible to the adverse consequence. The decision may be

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4 *Adlam v Accident Compensation Corporation* [2017] NZCA 457, [2018] 2 NZLR 102 at [62] and [65]; see also *McEnteer v Accident Compensation Corporation* [2010] NZCA 126, [2010] NZAR 301 at [20].

5 *Accident Compensation Corporation v Ng* [2020] NZCA 274, [2020] 2 NZLR 683.

> informed by medical studies including relevant statistical analysis … as well as the clinical experience of the treating physician(s) and other specialists.

[47] In *Ambros*,[6] the Court of Appeal stated the following in relation 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 …
>
> [70] … The generous and unniggardly approach referred to *Harrild* may, however, support the drawing of a robust inference in individual cases. It must, however, always been borne in mind that there must be sufficient evidence 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.

[48] In *Stewart*,[7] Barber DCJ stated:

> [33] The cases consistently highlight that the question of causation cannot be determined by a matter of supposition. There must be medical evidence to assist the respondent Corporation, and now the Court, to determine that question. A temporal connection, in itself, will be insufficient. There needs to be a medical explanation as to how the ongoing condition has been caused by the originally covered injury.

[49] In *Stanley*,[8] Justice Heath concluded there was sufficient evidence of a personal injury where the District Court had found that a failure to treat left the claimant with an exacerbated personal injury:

> [55] I have already found that there was sufficient evidence for Judge Joyce to conclude that the exacerbated personal injury occurred and prima facie, was caused by the treatment delay. Unless the exacerbated personal injury was

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6 *Accident Compensation Corporation v Ambros* [2007] NZCA 304, [2008] 1 NZLR 340.
7 *Stewart v Accident Compensation Corporation* [2003] NZACC 109.
8 *Accident Compensation Corporation v Stanley* [2013] NZHC 2765.

> wholly or substantially caused by Mr Stanley's underlying health condition, he is entitled to cover.

Discussion

[50] The issue on appeal is whether the Corporation's decision dated 4 April 2016 accepting cover for Mr Austin for a treatment injury was correct. The Corporation decided to accept cover for diffuse idiopathic skeletal hyperostosis (DISH) manifested by osteophytes in the cervical spine and thoracic spine as a treatment injury resulting from Mr Austin's use of Roaccutane (isotretinoin).

[51] A treatment injury is one which is caused when a person receives treatment from a registered health professional, and which was not a necessary part, or ordinary consequence, of the treatment.[9] The injury said to be a treatment injury must be the consequence of a departure from appropriate treatment choices and treatment actions, objectively assessed.[10] The test of "not an ordinary consequence" means an outcome that is outside of the normal range of outcomes, something out of the ordinary which occasions a measure of surprise.[11]

[52] Mr Austin submits as follows. His skeletal outcomes, spread over such a long period of time, cannot possibly be considered accidental. Retinoid hyperostosis is not accidental and it would be wrong for the Corporation to fund such a disease. Retinoid hyperostosis is a slow-developing bone forming disease which can take as long as 15 years to fully express itself. Bone forming diseases such as DISH and RH are usually benign and only become clinically significant if the osteophytes grow too large. Further, his main injuries have all eventuated from spondylosis, a degenerative disc disease for which the Corporation has declined cover, and not retinoid hyperostosis. Parliament could not have intended to cover his condition.

[53] Mr Austin further submits the Corporation did not consider the correct test for whether an outcome was an ordinary consequence of treatment, as recently set out by the Court of Appeal in *Ng*. The expert opinion does not establish treatment injury cover, and did not properly assess the evidence relating to the duration of treatment

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9 Section 32(1) of the Act and *Adlam*, see n 2 above.
10 See *Ng*, above n3.
11 ibid.

and the dosages he received, or consider the relevant literature of the era. His injury should be seen as an ordinary consequence of extended, high-dose treatment with Roaccutane, and so does not qualify as a treatment injury. None of the experts properly evaluated whether the injury was an ordinary consequence of the treatment, taking into account his particular circumstances and the clinical knowledge at the time.

[54] This Court acknowledges Mr Austin's submissions. However, the Court points to the following considerations.

[55] First, Mr Austin himself has previously maintained, over six years, that his skeletal hyperostosis qualified as a treatment injury caused by his Roaccutane use:

(a) In March 2015, Mr Austin claimed cover for skeletal hyperostosis for his health problems as a treatment injury caused by his Roaccutane use. In December 2015 (confirmed in April 2016), the Corporation accepted cover for a treatment injury, being satisfied that Mr Austin's use of Roaccutane had resulted in his skeletal hyperostosis and that this would not be an expected or ordinary outcome of treatment.

(b) Mr Austin accepted funding for his surgery, physiotherapy and consultation costs, backdated weekly compensation of $159,548.60, and weekly compensation of $1553.91 until he reached superannuation age in 2017.

(c) Mr Austin did not pursue any review options to challenge the Corporation's acceptance of his claim and its provision of benefits.

(d) In 2016, Mr Austin commenced proceedings against the New Zealand distributor of Roaccutane, claiming compensatory and exemplary damages. After extended legal proceedings, the Court of Appeal struck out Mr Austin's claim for compensatory damages as being covered by the Accident Compensation Act.[12] On 21 March 2021, the Supreme Court dismissed Mr Austin's appeal, noting that, once a person lodges an

[12] *Roche Products (New Zealand) Ltd v Austin* [2019] NZCA 660, at [50] and [54].

accident compensation claim, they are locked into the Act's procedures.[13]

(e) On 19 April 2021 (nineteen days after the Supreme Court decision), Mr Austin lodged an application to review the Corporation's decisions of December 2015 and April 2016, on the basis that his injury was an ordinary consequence of the treatment he received, and therefore outside the scope of accident compensation. The purpose of the present review and appeal proceedings is to allow Mr Austin to resume his civil proceedings for damages.

[56] Second, in relation whether Mr Austin's treatment caused his skeletal hyperostosis, this Court notes the following medical evidence:

(a) Dr Holtzhausen, Musculoskeletal Medicine Specialist and Mr Austin's treating physician, strongly supported a causal link between his Roaccutane use and his skeletal hyperostosis. Dr Holtzhausen advised that the evidence supported a finding that Mr Austin was prescribed significantly more Roaccutane than recommended for safe use. Dr Holtzhausen also advised that adults (such as Mr Austin who was in his 30s and 40s) taking this medication are at greater risk of developing hyperostosis than younger patients.

(b) Professor Burgess, Clinical Pharmacologist, agreed that Roaccutane use played some role in Mr Austin's hyperostosis.

(c) Mr Taine, Orthopaedic Surgeon, found it likely that Roaccutane had accelerated Mr Austin's hyperostosis.

(d) In light of this evidence, the Corporation found, on balance, that Mr Austin's use of Roaccutane had resulted in his skeletal hyperostosis.

[57] Third, in relation to whether Mr Austin's skeletal hyperostosis was not a necessary part, or ordinary consequence, of his treatment, this Court notes the following medical evidence:

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13 Above n 2, at [20].

(a) Professor Burgess, Clinical Pharmacologist, assessed that, if isotretinoin were the major factor, Mr Austin's skeletal hyperostosis would not be an expected outcome, as this is a rare complication of the use of isotretinoin.

(b) Mr Taine, Orthopaedic Surgeon, tended to the view that skeletal hyperostosis was not an ordinary consequence of Roaccutane. Mr Taine noted that the absence of firm recommendations, regarding screening in adult patients being treated with this medication, suggested a low incidence of symptomatic disease and that it is not an expected clinical problem.

(c) Dr Brown, Medical Advisor, advised that both the literature, and expert pharmacology opinion, support that the adverse outcome of skeletal hyperostosis following treatment with isotretinoin is a rare event, which likely represents an individual reaction (one that cannot be identified in advance). Dr Brown assessed that, taking all of the relevant circumstances into account, the adverse outcome in the case of Mr Austin was surprising, and not an ordinary outcome of treatment in his case.

Conclusion

[58] In light of the above evidence, this Court finds that Mr Austin's treatment injury was caused when he received treatment from a registered health professional, and which was not a necessary part, or ordinary consequence, of the treatment.

[59] As a result, the Court finds that the Corporation's decision dated 4 April 2016 accepting cover for Mr Austin for a treatment injury was correct. The decision of the Reviewer dated 22 December 2021 is therefore upheld. This appeal is dismissed.

[60] I make no order as to costs.

P R Spiller
District Court Judge