AN EXAMINATION OF MEDICAL AND LEGAL ISSUES IN THE STRUGGLE OF NEW ZEALAND SAWMILL WORKERS AND ACC COVER FOR PCP POISONING

BY BERNHARD KREBER*

I INTRODUCTION

Since the 1940s chlorinated phenols, specifically pentachlorophenol (PCP), have found wide application in many industry sectors throughout the world including their use as pesticides to protect wood from fungal degradation.[1] Chlorophenol-based treatments were also widely used by the New Zealand timber industry for wood protection from the early 1950s. Furthermore, a host of products containing chlorinated phenols were available from retailers for domestic purposes, for example, to control moss and algae.[2] Exterior and interior wood stains containing PCP were also used widely for residential homes.[3] While large quantities of PCP were sold worldwide an estimated 5000 tonnes was used by the timber industry over a 40 year period in New Zealand.[4] Because of widespread, historical use of PCP products for industrial and domestic purposes PCP contamination has become ubiquitous in the environment, namely in soil, drinking and surface water, vegetable, fruits and livestock.[5]

Concern about the potential health risk of PCP exposure to European sawmill workers arose in the 1970s through findings that commercial PCP formulations contained a variety of contaminants, for example, polychlorinated dibenzo-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). PCDDs and PCDFs represent two groups of compounds with over 200 isomers, with some known for their high acute toxicity.[6] For example, the most toxic and biologically active of the 75 PCDD isomers is 2, 3, 7, 8–tetrachlorodibenzo–p–dioxin (2,3,7,8–TCDD).[7] The most toxic of the 135 PCDF isomers is 2, 3, 4, 7, 8 pentachlorodibenzofuran (4–PeCDF) which is half as

* PhD, MSc. LLB, Enrolled Barrister & Solicitor of the High Court of New Zealand

* The author acknowledges Mr Doug Tennent, Lecturer, School of Law, University of Waikato who offered invaluable guidance and knowledge to this research project.

1 D Graham, 'History of wood preservatives' in D Nicholas (ed) *Wood deterioration and its prevention by preservative treatments* (1973) 1, 17.

2 P Stevenson, 'The scandal of the PCP dumps.' *Terra Nova*, August 1992, 10–15, 11.

3 C Krause, M Chutsch, N Englert, 'Pentachlorophenol exposure through indoor use of wood preservatives in the Federal Republic of Germany' (1989) 15 *Environment International* 445–447.

4 K Dew, 'National identity and controversy: New Zealand's clean green image and pentachlorophenol' (March 1999) Vol 5 No 1 *Health and Place* 45–57, 48.

5 R Chhabra, R Maronpot, J Bucher, et al., **'**Toxicology and carcinogenesis studies of pentachlorophenol in rats**'** (1999) Vol 48 *Toxicological Sciences*, 14–20**.**

6 U C Ahlborg, 'Health and environmental aspects of the use of chlorophenols with particular references to chlorophenol and dioxin toxicity' in *Chlorophenol wood treatments – safety and the environment* (1978) 11.

7 Ibid.

toxic as 3, 4, 7, 8–TCDD.[8] Acute toxicity varies greatly between closely related PCDD and PCDF isomers. Because of potential health risks to sawmill workers Sweden became the first country to effectively de register all PCP based pesticides in 1978.[9] Many European countries followed in the 1980s imposing bans on PCP based wood treatments. Other countries, for example Canada and the United States, have continued using PCP based products for pressure treatment of wood but have put in place strict management procedures that are thought to greatly reduce the risk of adverse effects to human health and the environment.[10]

In New Zealand, PCP pollution became the focus of attention when alarmingly high concentrations were detected in the sediment from Manukau harbour in 1988. The fear of widespread PCP pollution stipulated the set up of a National Task Group (NTG) in 1990 to determine the extent of PCP contaminated sites in the timber industry. Following growing local and international concern over PCP contamination New Zealand also banned the sale of PCP in 1991.[11] Subsequently, regulatory bodies attempted to develop guidelines to clean up contamination of the land in the 1990s, arguably in an attempt to maintain New Zealand's 'clean green' image.[12] In contrast, workers who claimed that occupational PCP exposure resulted in debilitating health effects receded into the background in such environmentally focused debate. Consequently, little support has been offered by governmental bodies to identify and compensate workers for illness suffered from working with PCP. In essence, sawmill workers claiming compensation from the Accident Compensation Corporation (ACC), the state run no fault insurance scheme covering industrial accidents, must demonstrate that the health effect suffered is both real and linked to PCP exposure during the course of employment, and is not substantially caused by other agents, or life style choices. Undoubtedly, that is a very heavy burden of proof and it is not surprising that workers have largely failed in doing this.

The object of the present research is to examine medical and legal issues that sawmill workers encounter in their struggle for compensation from ACC for ill health effects caused by PCP poisoning.

II. Background on PCP use in the timber industry

PCP, one of the most important biocides used in the timber industry in the last century, is a crystalline phenolic compound. It has five substituted chlorine atoms at its phenolic ring, is largely water insoluble, has a vapour pressure of 10^{-4} mm Hg at 20 degrees Celsius, and exhibits a strong phenolic smell.[13] PCP is soluble in organic solvents, for example, petroleum oil distillate such as white spirit or odourless kerosene. Wood treatments using PCP were typically performed in a treatment vessel using different pressure treatment processes. In New Zealand, a 5 per cent solu-

8 National Task Group on Site Contamination from the Use of Timber Treatment Chemicals, Study Team Report, NTG (1992), 'NTG Pentachlorophenol Risk Assessment Pilot Study' Camp Scott Furphy Pty. Ltd. 6–3.

9 Swedish Product Control Board, 'Press Release' (Stockholm), 27 May 1977

10 J Wilkinson, 'Pentachlorophenol. The US and Canadian experience' International Wood Preservation Symposium. The Challenge – Safety and Environment, Cannes-Mandelieu (France), 6–7 Feb 1995, 357.

11 'Still selling PCP's', (June 1992) *Terra Nova* 11.

12 Dew, above n 4, 45.

13 S Wood, W Rom, G White, et al., 'Pentachlorophenol poisoning' (July 1983) 25 *Journal of Occupational Medicine* 527.

tion of PCP in fuel oil replaced creosote in the 1950s to pressure treat poles.[14] From the early 1960s PCP/oil treatments were performed using the Rueping process.[15] At the Waipa sawmill in Rotorua, the Rueping process involved placing air dried wood into the treatment vessel, applying an initial pressure, flooding the vessel with 4 per cent weight/weight (%w/w) of PCP in oil solution, heating the vessel to 90 degrees Celsius, and then increasing the pressure to 950 to 1050 kilonewton per square meter (kN/m^2). After about eight hours, at which time up to 150 litres per square meter (L/m^2) was absorbed by the timber, the pressure was released causing the air trapped inside the wood to force excess solution from the timber. The treatment solution was withdrawn from the vessel and a final vacuum was then drawn to reduce bleeding of the PCP/oil solution from the timber. This process effected deeper chemical penetration of refractory wood species and also produced cleaner poles which reduced PCP bleeding in service.[16] However, bleeding of excess PCP from treated wood still occurred during subsequent timber storage and from wood in service. Undoubtedly, PCP bleeding contributed to environmental pollution at treatment sites, and likely posed a health risk to workers handling treated wood.

PCP/oil treatments were used to provide permanent protection to wood from decay fungi in situations of moderate and high decay hazard, for example, railway sleepers, pilings, transmission poles, cross arms and fencing posts. However, PCP/oil was also used for exterior and occasionally interior wood stains to prevent growth of mould and sapstain fungi on decorative timber in service; it was usually applied by brush.

Undoubtedly, PCP is very toxic to a wide range of fungi and insects making it a highly effective wood preservative. Other advantageous properties of PCP are that it has a low vapour pressure, is stable and resistant to high treating temperatures, non corrosive, retains the natural colour of wood, can be over painted, and importantly, was very cost effective.[17]

Because of consumers' demand for clean wood showing no fungal discolouration antisapstain chemicals have been widely used since the early 20th century.

Sodium salt of PCP, commonly known as sodium pentachlorophenate (NaPCP), is water soluble. NaPCP was widely applied as a prophylactic, short term wood treatment to control development of a plethora of mould and sapstain fungi that colonise sapwood of freshly felled logs and unseasoned sawn lumber. Mould and sapstain fungi can utilize readily accessible wood compounds as food source, for example, simple wood sugars. As they penetrate fresh sapwood they form pigmented hyphae and spores; this causes aesthetic damage, commonly referred to as sapstain or bluestain, which does not affect the structural integrity of wood.

NaPCP is a highly effective wood surface treatment and was used at concentrations of up to 2.5 per cent w/w. At the Waipa sawmill, a mixture of 0.5 %w/w NaPCP and 1.5 per cent w/w pentahydrated borax was used as antisapstain treatment.[18] NaPCP was commonly applied by immersion, by passing processed timber through large dip tanks, and occasionally by spray application. Following antisapstain treatment, wood is stacked and ideally allowed drying of excess solution under cover for 24 hours.

---

14 M Hedley and P Mills, Forest Research Institute New Zealand Forest Service (New Zealand) Technical Paper 64'Service tests of softwood transmission poles in New Zealand' (1977) 5.

15 National Task Group, above n 8, 2–4.

16 Hedley and Mills, above n 14, 4.

17 A Bravery, 'Present uses of chlorophenols in wood treatments' in (1978) 3.

18 National Task Group, above n 8, 2–5.

In New Zealand, the boron diffusion process used for treating construction timber also contained NaPCP, for example, 0.2 %w/w was used at Waipa.[19] Timber was dipped into a hot borate solution, and then block stacked under cover for six to eights weeks to allow diffusion of borates throughout the timber. NaPCP was added to prevent fungal infections occurring during the diffusion process when wood moisture content was high enough to encourage fungal growth.

III. Brief description of workers' PCP exposure and reported symptoms

Significant amounts of PCP contamination including some dioxins were found at the Waipa sawmills.[20] This likely reflected the situation at other New Zealand sawmills that used PCP based wood treatment. As outlined above, sawmill workers came into contact with PCP when performing various jobs at sawmills with PCP exposure occurring through direct contact (PCP solution), dust, vapour and mist. Men working at the green chain, a site where freshly sawn timber was passed through a dip tank containing NaPCP solution and then stacked according to timber grade and size, had direct exposure which inevitably caused skin soakage by the PCP solution. Workers reported that after a day of work on the green chain they went home absolutely saturated from the sap running off the freshly treated timber, mainly in the areas of their thighs and feet.[21] Although workers were given some protective equipment, for example, PVC gloves and aprons, the measures were generally inadequate. Workers reported that gloves tore open after a while and aprons funnelled chemicals into their boots, further promoting skin soakage.[22]

Aprons were also said to impair freedom of movement and were thus discarded by some workers. Workers at the green chain wore shorts in the mild season of the year, and on hot days took their shirts off. This illustrates the general lax health and safety attitude workers had at that time. A Labour Department officer visiting the Waipa sawmill in 1990 when PCP use had discontinued, also observed very poor practices of workers and management dealing with wood treatment chemicals and treated wood, and commented that 'it was exactly the same when PCP was used.'[23]

Another high (PCP) risk task also performed by graders was to manually mix the NaPCP solution.[24] Mixing of NaPCP solution was required daily and could be required three to four times a day depending on what timber size was in demand.[25] Workers also had to clean the dipping tank daily by scrubbing it out by hand to remove sludge accumulating at the bottom of the tank. The sludge was then stored onsite around the green chain area and periodically dumped somewhere offsite, for example, in a farmer's paddock.[26] In this context, farmland containing toxic waste (dumped sludge) from a Whakatane sawmill has been implicated in serious heath problems of occupants.[27] Interestingly, a 1992 information sheet by the Occupational Safety and Health Service (OSH) issued a warning to sawmill workers of the health risks associated with PCP sludge, and advised on precautionary measures when handling and disposing of PCP sludge and PCP contam-

19 Ibid.

20 Ibid, ii.

21 'The poisoning of Papatuanuku', (March 1996) *Pu Kaea* 20, 22.

22 A Spence, 'The chain gang: nightmare at the mill' (March 2001) *North & South* 62, 66.

23 P Stevenson, 'PCPs: Crunch time for the timber industry' (August 1992) *Terra Nova* 20.

24 Spence, above n 22, 65.

25 'The poisoning of Papatuanuku' above n 21, 22.

26 Spence, above n 22, 65.

27 'When dreams turn poisonous' (8 June 2003) *Sunday Star Times* 5.

inated soil.[28] It is clear from workers' reports that none of those precautions, for example, chemically resistant impermeable overalls and respirators, recommended in the 1992 OSH information were used by workers when removing sludge contaminated with PCP from dip tanks. In the view of occupational physicians the nature of PCP exposure of green chain workers performing the different tasks mentioned above produced a constant high level of exposure.[29] Other tasks, for example, filleting of NaPCP treated timber, were thought to produce an intermittent low degree of exposure.[30]

While NaPCP was used in nearly all sawmills leading to widespread exposure of workers, oil based PCP wood treatments were far less common in New Zealand. There were only five sawmill sites in New Zealand pressure treating wood with PCP/oil. However, reports from operators of pressure treatment plants and also responsible for preparing PCP/oil solution and removal of PCP sludge from the treatment cylinder, paint a grim picture of the working conditions. In essence, operators were constantly exposed to PCP dust and hot and smelly fumes.[31] It is likely that oil fumes posed an additional burden on the health of operators. Protective equipment for these workers was usually inadequate, for example, safety goggles fogged up which caused workers to remove them to see what they were doing.[32] Also, operators dealt with large quantities of PCP, reportedly handling up to 1,000 kilograms during a normal eight hour shift.[33] PCP spills which inevitably occurred, PCP bleeding of treated wood, and onsite PCP sludge disposal resulted in significant soil contamination, for example, adjacent to pressure treatment facilities and in sawmill well water that workers used for drinking.[34] Unsurprisingly, the nature of PCP exposure of treatment operators was considered constantly high.[35] It is also worthwhile mentioning that treatment plant operators worked with a range of different wood preservatives including chrome copper arsenate (CCA) and creosote.[36] Recently, the former wood preservative received considerable media attention as the public expressed concerns relating to potential health risks associated with use of CCA treated wood.[37] For sawmill workers however, the prolonged exposure to different wood preservatives likely caused an increased body burden. From an occupational health assessment view, workers with a history of exposure to *multiple* wood preservatives may face even greater difficulties establishing a causal relationship between PCP exposure and health problems. Exposure to other wood preservatives then becomes a confounding factor in workers' occupational history; ACC legislation however, requires workers to prove on a balance of probability that occupational exposure to a physical agent, such as PCP, caused alleged health problems. The issue of confounding factors will be discussed later.

---

28 Occupational Safety and Health Service [OSH], *Handling and Disposal of PCP Sludges at Timber Treatment Plants* (September 1992) available at <http://www.osh.govt.nz/order/catalogue/pdf/pcp-i.pdf>

29 Occupational Safety and Health Services [OSH], *An investigation into the health effects of previous occupational pentachlorophenol exposure on timber sawmill employees* (1996) 9.

30 Ibid.

31 P Stevenson, *No risk to Employees' Health* available at <http://www.stevenson.net.nz/reup.html>.

32 Ibid.

33 Ibid.

34 D Williams, 'On the PCP trail' (June 1002) *Terra Nova* 13.

35 OSH, above n 29, 9.

36 'The poisoning of Papatuanuka', above n 21, 22.

37 E Kwon, H Zhang, Z Wang, et al., **'**Arsenic on the Hands of Children after Playing in Playgrounds**'** (2004 Oct) 112(14) *Environmental Health Perspectives* 1375–80.

There is little doubt that work related exposure to PCP has affected the quality of life of sawmill workers and their families. However, at the time of actually working with PCP workers largely had little or no awareness of the potential health consequences of PCP exposure. Concerns raised by workers, for example with their general practitioners, were often brushed aside.[38] Many sawmill workers reported various degrees of skin irritation, burning eye sensations, eye watering, and dizziness working around PCP.[39] Furthermore, many workers experienced on going problems with skin rashes and eye burning for many years following exposure to PCP. Interestingly, some family members experienced similar skin rashes and eye watering.[40] Other common symptoms included severe headaches, constant sinus problems and fever,[41] extreme tiredness,[42] reeking night sweets,[43] and severe weight loss[44]. Some workers also reported coughing up blood and passing it in their faeces.[45] The workers also believe that PCP caused other diseases, for example, asthma, diabetes, heart problems, kidney and liver ailments and cancer.[46] As will be discussed below, medical evidence has not been able to support the workers' notion that PCP triggered these diseases.

IV. Selected medical studies on PCP poisoning

A relatively large body of medical publication has focused on establishing the effect of occupational PCP exposure on human health. For a layperson, medical research is confusing and often difficult to understand because of unfamiliar terminology. However, one gets a sense of the complexity and difficulty of medical science establishing a clear causal relationship between occupational PCP exposure and persistent health problems claimed by workers.

Medical research draws a distinction between acute and chronic consequences of PCP exposure. The Concise Medical Dictionary offers two meanings for acute, namely, 'disease of rapid onset, severe symptoms and brief duration' [and] 'any intense symptom, such as severe pain'.[47] Acute PCP consequences can occur though a single dose of exposure of sufficient severity. For example, skin irritation is well recognized as an acute symptom of PCP exposure. Severe instances of acute PCP poisoning have resulted in death of workers involved in preparing wood preservatives.[48] In contrast chronic is defined as 'disease of long duration involving very slow changes [and] often of gradual onset'.[49] Also, the term chronic makes no reference to the severity of the disease, but simply refers to a persistent health problem.[50] For chronic symptoms in workers, the frequency of PCP exposure and PCP concentrations used are likely important contributing factors.

The distinction between acute and chronic consequences of PCP exposure is not clear cut because some chronic effects are the persistence of acute health effects. While the medical com-

38 Stevenson, 'PCP's: crunch time for the industry', above n 23, 19.

39 Stevenson, above n 31.

40 Spence, above n 22, 69.

41 Spence, above n 22, 65.

42 'The poisoning of Papatuanuka', above n 21, 21.

43 H Murdoch, 'Ex-timber worker battles on with PCP poisoning' (15 May 2001) *The Nelson Mail* 6.

44 'The poisoning of Papatuanuka', above n 21, 22.

45 Stevenson, above n 31.

46 Spence, above n 22, 69.

47 E Martin, (ed) *Concise Medical Dictionary* (2002) 8.

48 Wood et al, above n 13, 528.

49 Martin, above n 47, 133.

50 Ibid.

munity has never questioned that PCP exposure can trigger a range of acute symptoms in the human body, chronic health effects have been a lot more problematic.[51] I provide in Table 1 a list of some acute and chronic effects reported in humans following PCP exposure. Those effects have been observed following domestic and occupational PCP exposure. Not all effects listed in Table 1 were associated to PCP exposure of sawmill workers. Then, I give a snapshot of selected international and domestic medical studies to illustrate aspects of current medical knowledge on PCP poisoning in people. I further indicate why those medical findings have somewhat impeded the cause of New Zealand sawmill workers to get public acknowledgement that PCP poisoning severely impacted on their quality of life.

Table 1: Acute and alleged chronic health consequences in humans exposed to PCP.52

Health Effect	Acute	Chronic
Skin	Irritation or burning of the skin after a single exposure to strong PCP solutions or prolonged and repeated exposure to lower PCP solutions	Chloracne, low grade skin inflammation and infection
Eyes	Eye irritation and itching	Conjunctivitis and/or eye discomfort
Respiratory tract	Irritation of nasal airways and upper respiratory tract	Sinusitis and irritation of the upper respiratory tract; bronchitis
Endocrine and metabolic systems	Fever, sweating, weakness, tachycardia, dyspnoea, hyperthermia, anorexia, diaphoresis, nausea, vomiting	Hyperpyrexia, diabetes, disturbances of lipid metabolism
Nervous system	Headaches, mental fatigue, dizziness, balance loss, ataxia	Dizziness, headache, personality and mood changes, peripheral neuropathy
Cardiovascular system	Increased heart rate, cardiac arrhythmia or arrest at acute PCP poisoning	
Kidneys	PCP accumulates in the kidneys	Reduced glomerular filtration rate and tubular reabsorption.
Haemopoietic system		Haemolysis, thrombocytopenia, aplastic anaemia
Reproductive systems		Increased risk to father off spring with congenital anomalies, including dislocation of hip, cleft lip, eye, genital organs
Immune system		Activated T–cell and B–cell dysfunction, Decrease in Ig G and Ig A immunoglobins
Liver	Increase in AST & ALT levels, hepatomegaly	
Cancer		Kidney, gastric, duodenal ulcer, soft tissue sarcoma

51 V Edwards, 'The danger of PCP exposure' (27 November 1996) *GP Weekly* 12.

52 Table 1 is a summary of some acute and chronic health effects reported in the 1996 OSH report.

A. Cancer

Several epidemiological studies have been undertaken to determine the relationship between chlorinated phenols including PCP and cancer. In the 1980s health care professionals and epidemiologists at the University of British Columbia in Canada (UBC) undertook the largest cohort study (more than 20,000 workers) to date to determine whether workers exposed to PCP and ,3,4,6–tetrachlorophenol (TCP) where at an increased risk of cancer. PCP and TCP were widely used in British Columbia and by 1987 the province had consumed 1,100 tonnes annually to which over 100,000 workers were exposed.[53] A significant trend of increased risk of non-Hodgkin's lymphoma associated with increased exposure and small excesses in overall cancer incidences and lung cancer was observed, but none of the cancers of interest caused elevated mortality.[54] The UBC study could not establish a risk between childhood cancer and parental PCP exposure.[55] Finnish research observed an excess of skin cancer and leukaemia in sawmill workers.[56] Wolf and colleagues investigating malignant nasal tumours in the German wood working industry reported that PCP is genotoxic in nasal cells of human beings.[57] Except for the UBC study, a general limitation of epidemiologic studies is that the majority are based on relatively small sample sizes lacking statistical power to detect excessive cancer risk. Furthermore, most studies lack specific information on the types of chlorinated phenols workers were exposed to and cannot exclude confounding by other occupational carcinogenic agents. Clearly, exposure misclassification leading to underestimation of cancer risk of workers is an important consideration when interpreting findings.[58] Based on these limitations the International Agency for Research on Cancer considers there is sufficient scientific evidence from animal studies for carcinogenicity of PCP but has classified the evidence regarding human carcinogenicity as limited.[59] Animal carcinogenesis studies are the prime indicators of potential carcinogenicity risk to humans. Furthermore, a perfect correlation has been found for all human carcinogens that have been tested in animals.[60] However, animal bioassays often centre on individual agents. In reality however, human cancer is probably caused by multiple factors including individual genetic susceptibility and lifestyles.

B. Reproductive effects.

Offspring of male sawmill workers in British Columbia were at an increased risk of developing congenital anomalies, for example, congenital cataracts, but no association was found with low

53 C Hertzman, K Teschke, A Ostry, et al., 'Mortality and cancer incidence among sawmill workers exposed to chlorophenate wood preservatives' (1997) 87(1) *American Journal of Public Health,* 71–9.

54 Ibid.

55 H Heacock, C Hertzman, P Demers, et al., 'Childhood cancer in the offspring of male sawmill workers occupationally exposed to chlorophenate fungicides' (2000 Jun) 108(6) *Environmental Health Perspectives* 499–503.

56 P Jappinen, et al., Cancer Incidence of Workers in Finnish Sawmill (1989) 15 *Scand. J. Work Environ. Health* 18–23.

57 J Wolf, P Schmelzer, D Fengel, et al., 'The Role of Combination Effects on the Etiology of Malignant Nasal Tumours in the Wood-Working Industry: Most Recent Findings and Analysis of 147 Indemnified Cases of Adenocarcinomas' (30 July 1998) Volume 118, Supplement 535 *Acta Oto-Laryngologica* 3–16, 15.

58 M Kogevinas, H Becher, T Benn, et al., 'Cancer mortality in workers exposed to phenoxy herbicides, chlorophenols, and dioxins: an expanded and updated international cohort study' (1997) 145 *American Journal of Epidemiology* 1061–75, 1073.

59 J Huff, 'Sawmill Chemicals and Carcinogenesis' (2001) 109 *Environmental Health Perspectives* 209–212, 211.

60 Huff, above n 59, 209.

birth weight, still birth or prematurity.[61] Significantly reduced birth weight and length were found in offspring of female day care workers exposed to wood preservatives including PCP; reduced birth weight was suggested to be a childhood risk factor for some adverse health effects.[62] PCP has been detected in semen of exposed sawmill workers.[63] In this context, New Zealand sawmill workers believe that some health problems suffered by their children, for example, persistent irritations of the skin, are related to PCP poisoning. New Zealand sawmill workers took their work clothes home where they were often washed together with other family clothes; thus indirect PCP exposure of children is theoretically plausible.

C. Neurological problems

Peper and colleagues suggested that long term domestic exposure (inhalation) to wood preservatives including PCP has adverse effects on neurobehavioral performance, for example, working memory, and is further related to frequent subjective complaints including increased fatigue, distractibility and mood swings in women.[64] However, considerable heterogeneity of exposure conditions between and within exposed subjects and confounding factors such as solvents, pigments, and other contaminants found in wood preservatives was noted. New Zealand studies have also noted neurological dysfunctions in exposed sawmill workers as discussed below.

D. New Zealand research on PCP effects

Overseas research, starting in the 1970s, indicated serious health and environmental problems associated with occupational PCP use. Research in New Zealand, despite the widespread use of PCP in the timber industry and for domestic purposes, did not commence until the late 1980s when findings of high PCP sediment levels in the Manukau harbour caused the government to set up a National Task Group (NTG) to examine environmental issues. Specifically, NTG's mandate was to assess the extent of PCP contaminated sites and advise the government and industry on policies concerning liability and clean up.[65] In contrast, the impact of widespread PCP use on workers' health has received much less attention, and did not gain momentum until 1995 when several timber companies commissioned medical experts to undertake a literature review on the health effects of PCP.[66] The review confirmed that exposure to PCP in the timber industry causes a range of acute health effects (Table 1), but the literature review did not provide conclusive evidence of long term health effects. An immediate action arising out of the literature review was the set up of an OSH initiated, questionnaire based study by medical experts, involving current and ex workers

61 H Dimich-Ward, C Hertzman, K Teschke, et al., 'Reproductive effects of paternal exposure to chlorophenate wood preservatives in the sawmill industry' (1996) vol 22 no 4 *Scandinavian journal of work, environment and health* 267–273.

62 W Karmaus and N Wolf, 'Reduced birthweight and length in the offspring of females exposed to PCDFs, PCP, and lindane' (1995 December) 103(12) *Environmental Health Perspectives* 1120–1125.

63 Dimich-Ward, et al., above n 61, 271.

64 M Peper, M Ertl and I Gerhard 'Long-term exposure to wood-preserving chemicals containing pentachlorophenol and lindane is related to neurobehavioral performance in women' (1999) Vol 35 Iss 6 *American Journal of Industrial Medicine* 632–641.

65 National Task Group, above n 8, 1–2.

66 OSH, above n 29, 18.

who felt their symptoms of ill health was attributed to PCP use.[67] The study investigating disease and symptom prevalence in a non random sample of sawmill workers found a strong association between PCP exposure estimates of individuals and the frequency of acute symptoms. The study further showed an apparent close relationship of some acute effects of PCP, specifically, persistent fever (sweating), weight loss, fatigue, nausea as well as a screening measure for neuropsychological dysfunctions. In essence, the OSH study provided strong evidence of long term effects of PCP and also mirrored the clinical experience of two authors.[68] Limitations of the OSH study noted were the small sample size of 127 workers, the self selective nature of the population investigated and the lack of controls.[69] In 1997, the New Zealand Engineers Printing and Manufactures Union (EPMU) undertook a survey of members primarily involved in mill maintenance, for example, welding tanks containing PCP residue, and suggested a range of persistent health effects, for example headaches, fatigue, breathing difficulties and mood swings, which were attributed to past PCP exposure.[70] Subsequently, EPMU with the input of the Wood Industries Union, set up a register to identify sawmill workers who alleged ill health due to PCP exposure and assist with ACC claims. A 1999 telephone questionnaire study conducted by 5th year medical students found that the majority of workers suffered from a high per centage of symptoms at relatively low exposure levels and problems of neurological origin; headaches, mood changes and depression were the leading complaints.[71] An assessment of 62 PCP exposed workers undertaken to determine clinical syndromes that could be related to PCP exposure identified three groups of syndromes as follows:

i) acute symptoms of fever, headaches, upper and lower respiratory tract and eye irritation, skin disease and foul smelling and discoloured sweat. These symptoms, with the exception of sweating and skin disease, often resolved after workers left the timber industry;

ii) a chronic fatigue syndrome in workers starting during PCP exposure and often persisting following their PCP exposure;

iii) a delayed encephalopathy (various diseases affecting the function of the brain) developing well after workers had left the timber industry including anxiety, depression, behavioural, cognitive and personality problems, and confusion.[72]

This latter syndrome complex was found in more than a third of the cohorts studied. The authors suggested thought that none of the syndromes were characteristic of PCP poisoning because many confounders identified questioned the specificity of symptoms.[73] A study commissioned by Sawmill Workers Against Poison (SWAP) showed a large number of sawmill workers had symptoms

67 C Walls, W Glass, N Pearce, 'Health effects of occupational pentachlorophenol exposure in timber sawmill employees: a preliminary study' (1998) 111 *NZ Medical Journal* 362–4.

68 Ibid, 364.

69 D Gorman, J Monigatti, B Glass, B., et al 'Assessment of pentachlorophenol-exposed timber workers using a test-of-poisoning model' (2001) 7 *Int. J. Occup. Environ. Health* 189–194.

70 N Bandaranayake, B Caldwell, F Connell, et al., *PCP in the timber industry: A follow-up of exposed workers* (1999) Appendix 3.

71 Ibid, 25.

72 Gorman, above n 69, 189

73 Gorman, above n 69, 193.

attributed to PCP exposure including high blood pressure, depression, mood swings, blood disorders and cancer.[74]

E. Presence of PCP in human urine and blood

Indoor use of wood preservatives containing PCP resulted in occupants showing three times the levels of PCP in their urine than the controls. But with the exception of reddening of tonsils in men, depression in women and slightly elevated basophil counts, no pathological symptoms or alterations suggesting an association to PCP exposure were detected in persons with high PCP exposure despite credible complaints of severe health problems.[75] Increased blood levels of PCP found in patients with long term, low dose PCP exposure were associated with cellular and humoral immunodeficiency leading the researcher to suggest a causal relationship between immune dysfunction and clinical symptoms, for example, recurrent respiratory infections (colds) and chronic fatigue.[76] Triebig, in a response to the former study, argued that many confounding factors, for example, age, gender, medication, viral infection, stress, smoking and alcohol consumption can influence immune dysfunction, thus high PCP levels in blood does not prove causation.[77] New Zealand sawmill workers who had constant high PCP exposure, for example, at the green chain, showed high PCP levels in urine specimens prompting mill management to shift workers to areas with less PCP exposure. However due to insufficient medical data, occupational physicians, are unclear how to interpret PCP levels found in humans, specifically at what levels a person can expect health problems.[78] This is in contrast to other well known occupational diseases, for example, lead poisoning, where biological measurements can be related to workers' health outcomes.

The research summarized above shows that PCP exposure can be associated by the worker with 'non specific, difficult to measure symptoms of ill health or unusual disease entities'[79]. Medical experts in New Zealand maintain that long term health effects of occupational PCP exposure remain uncertain and they are supported in their view by the general lack of published, scientific information linking cause and effect(s).[80] Calls from the medical science community for further studies have now been answered in New Zealand, and a study is underway at Massey University to investigate health outcomes of former timber workers exposed to PCP.[81] As PCP use in the timber industry stopped in 1988 many of the worst affected workers have reportedly already died. In my view, it is questionable whether this study can establish a clear link between PCP poisoning and ill health effects considering the subjectivity of some of the symptoms which make it very difficult to establish scientific certainty for PCP poisoning. Scientific certainty may not be possible in the face of the inherent difficulty associated with correct determination of the amount and type of exposure, symptoms not uniquely associated with exposure, latency between exposure and ef-

74 Daily Post Rotorua 'Survey of sawmillers and families face "health risk"'(26 June 2002) *The New Zealand Herald* available at <http://www.nzherald.co.nz/section/1/story.cfm?c_id=1&objectid=2048748>

75 Krause, above n 3, 446.

76 V Daniel, W Huber, K Bauer, et al., 'Association of Elevated Blood Levels of Pentachlorophenol (PCP) with Cellular and Humoral Immunodeficiencies' (2001) 1 *Archives of Environmental Health* 77.

77 G Triebig, 'Letter to the Editor' (1997) 2 *Archives of Environmental Health* 148.

78 Edwards, above n 51, 12.

79 Walls et al, above n 67, 364.

80 Ibid.

81 Massey University, 'Health outcomes of former New Zealand timber workers exposed to pentachlorophenol (PCP)', available at <http://publichealth.massey.ac.nz/research/CPHRBased/Completed/resoccpcp.htm>

fect, and conflicting scientific evidence on the effects of exposure.[82] Arguably, there can be no certainty in science. Science however, can offer a story that explains best a particular phenomenon at a certain point in time.

Former timber workers argue there is no need for further research to tell them what health problems they suffer and they believe sufficient evidence is already available that PCP poisoning has harmed them.[83] In the following I will examine the legal barriers that sawmill workers face to get cover under the New Zealand accident compensation scheme.

V. New Zealand accident compensation scheme(s)

A. The Beginning

Prior to ACC the common law provided one avenue to claim compensation for personal injury where the personal injury could be attributed to negligence.[84] The common law remedy however, was considered flawed; for example, it was unable to compensate large numbers of victims and guarantee damages thus impeding rehabilitation of injured people, and took a long time to deliver benefits to those who did secure them.[85] Statutory compensation systems supplemented the common law, for example, the Workers' Compensation Act 1908 further amended in 1922 and 1956, which was backed by compulsory but privately administered insurance; it offered compensation for workers if injured at, but not out of, work.[86] It is interesting to note that the 1908 Act already included occupational diseases such as anthrax and lead, mercury and arsenic poisoning, and lump sum payment for loss of function.[87] In addition to Workers' Compensation Act, the Social Security Act 1964 offered some assistance with pressing needs, if the means test was met.[88] However, the common law and workers' compensation were regarded as highly inefficient, fragmented and capricious. Perhaps the most important criticism of the pre ACC system was that large amounts of money were absorbed by legal and administrative costs.[89] The Woodhouse Commission (Woodhouse) bluntly documented the general inadequacy associated with remedies available to workers. Specifically, Woodhouse recognized that injuries result in costs; for example, lost income, loss of work and production capacity, and medical costs, and 'the community as a whole has a responsi-

82 K Dew, 'Accident insurance, sickness and science: New Zealand's no-fault system' (2002) 32(1) *International Journal of Health Services* 163–178, 173.

83 N Boyes, 'Former timber workers say there's no need for new dioxin study', (27 September 2004) *The New Zealand Herald* available at <http://www.nzherald.co.nz/section/1/story.cfm?c_id=1&objectid=3595005>.

84 P McKenzie, 'The compensation scheme no one asked for: The origins of ACC in New Zealand' (2003) *Victoria University of Wellington Law Report* 193, 195 available at http://www.austlii.edu.au/nz/journals/VUWLRev/2003/10.html.

85 G Palmer, 'The Nineteen-Seventies: Summary for presentation to the Accident Compensation Symposium' (2003) *Victoria University of Wellington Law Report* 239, 241 available at http://www.austlii.edu.au/nz/journals/VUWLRev/2003/13.html.

86 McKenzie, above n 84, 195.

87 I Campbell, 'Compensation for personal injury in New Zealand: its rise and fall' (1996) 17.

88 McKenzie, above n 84, 195.

89 B Easton, 'The historical context of the Woodhouse commission' (2003) *Victoria University of Wellington Law Report* 207, 211 available at http://www.austlii.edu.au/nz/journals/VUWLRev/2003/11.html.

bility to distribute those costs according to the principles of social equity'.[90] The new system was designed to remove 'once and for all the perceived delays, waste and unfairness of disparate systems'.[91] Woodhouse recommended a set of five coherent and acceptable principles as the foundation of a new social insurance regime to alleviate mounting social problems arising from personal injury.[92] Proposing a new standard of public entitlement Woodhouse advocated real compensation similar to common law damages to all accident victims to compensate for economic and physical loss regardless of whether the injury was caused by fault.[93] Removing the element of fault which determines common law damages, Woodhouse suggested that compensation for personal injury was a matter of public welfare, thus the responsibility of the community. After long debate, policy makers enacted the first accident compensation legislation in 1973 which was to be administered by a government department.[94] It has been suggested that with this enactment New Zealand became the first country in the world to set up a coordinated public response to victims suffering personal injuries.[95]

A unique feature of ACC is a comprehensive, non fault compensation system providing 24 hour coverage in respect of all personal injuries regardless of cause.[96] ACC's focus was on the victim and not 'the culpability or fault of whoever has caused the events giving rise to cover'.[97] That 24 hour coverage on a no fault basis has largely remained the grundnorm throughout the scheme's history.[98] In return for ACC entitlements the Accident Compensation Act 1972 prohibited the right to sue in a New Zealand Court to recover damages for personal injury suffered in New Zealand to those persons covered by ACC.[99] That prohibition, which has been largely carried forward in subsequent accident compensation legislation, is frequently referred to as a social contract.

ACC was celebrated as a revolutionary measure providing certainty and encouraging early rehabilitation of victims.[100] In the first 18 years, ACC was administered generously providing real compensation (lump sum, pain and suffering) generally leaving recipients of ACC benefits reasonably content.[101]

B. ACC after 1992

In the late 1970s and 1980s many countries recognised the growing power of market systems and concepts such as free market and free choice increasingly dominated public debate. Neo-liberal

90 R Gaskins, 'Rebuilding ACC: An International View' in *Rebuilding ACC beyond 2000 : papers from a conference on reformulating the Woodhouse principles for a modern social insurance scheme in the new century, July 1999, Michael Fowler Centre, Wellington* (1999) 13, 18.

91 O Woodhouse, 'ACC: Integration or demarcation' in *Rebuilding ACC* above n 90, 6.

92 McKenzie, above n 84, 203.

93 J Ferguson, 'Medical Misadventure under Accident Compensation: Diagnosis and Treatment of a Problem' [2003] *NZLR* 485, 490.

94 Palmer, above n 85, 241.

95 Gaskin, above n 90, 15.

96 A Clayton, 'Some reflections on the Woodhouse and ACC legacy' (2003) *Victoria University of Wellington Law Report* 450, 456 available at http://www.austlii.edu.au/nz/journals/VUWLRev/2003/28.html.

97 Ferguson, above n 93, 491.

98 Clayton, above n 96, 456.

99 D Rennie, 'Comprehensive entitlement' in *Rebuilding ACC* above n 90, 27.

100 A Duffy, 'The common-law response to the accident compensation scheme' (2003) *Victoria University of Wellington Law Report* 367 available at http://www.austlii.edu.au/nz/journals/VUWLRev/2003/21.html.

101 Ferguson, above n 93, 493.

market concepts also gained control in New Zealand in the late 1970s and 1980s causing major ideological shifts in public policy 'away from state building policies and towards market systems as the new guardians of public welfare'.[102]

As public policies are ultimately driven by political priorities of the party in government, liberal market ideologies did not seriously impact on ACC until the national government enacted the 1992 Accident Rehabilitation and Compensation Insurance Act (1992 Act).[103] The long title of that Act claimed that persons suffering personal injury will be 'compensate[d] in an equitable and financially affordable manner'.[104] Arguably, the words 'financially affordable manner' implied National's view that that the two previous accident compensation legislation had granted ACC too much discretion resulting in an escalation in cost. Thus, National restricted ACC's discretion by enacting tighter definitions, for example, of accident, personal injury and medical misadventure, and the removal of two lump sum entitlements; one for permanent disability and the other for loss of amenity and pain suffering. The 1992 Act reflected the general trend in government's policies of the 1990s; user pays, cut backs in the level of benefits and the delivery of services under contractual arrangements to government funding organizations.

As ACC's focus has changed from a needs based to a cost savings approach it has greatly impacted on the generosity of ACC. This has caused widespread public discontent and a call for justice from some victims claiming the nature of the social contract has been damaged. Sawmill workers alleging ill health due to PCP poisoning arising out of their employment represent one such group of victims facing great injustice. Sawmill workers firmly believe they have suffered a personal injury due to occupational PCP exposure and should be granted full ACC entitlements. ACC's hard rules however have continued to disregard the needs of affected workers. In the following discussion I will look at the specific provisions relating to diseases arising out of employment.

VI. Personal injury caused by work related gradual process, disease or infection

Personal injury caused exclusively or substantially by gradual process, disease or infection has never been part of New Zealand's accident compensation schemes unless the disease was personal injury by accident; in which case personal injury included the physical manifestation of the accident, namely the disease.[105] This separation between sickness and accident suggests an inconsistency in social policy considering that disease, like personal injury, is a mishap to the person, and not a choice one makes in life. There is little doubt that a disease can have serious consequences for the individual and their family, and also the wider community. Fortunately, the ambit of Accident Compensation Act 1972, and the 1973 Amendment to that Act and the Accident Compensation Act 1982 (1982 Act) gave wide discretion to ACC to deal with unusual circumstances and reasonable doubt in favour of the applicant; this is clearly exemplified in *ACC v E*.[106]

---

102 R Gaskins, 'Recalling the future of ACC' (2000) *Victoria University of Wellington Law Report* 215, 222 available at http://www.austlii.edu.au/nz/journals/VUWLRev/2000/17.html.

103 Accident Rehabilitation and Compensation Insurance Act 1992.

104 Campbell, above n 87, 92.

105 *Stok v Accident Compensation Corporation* [1995] NZAR 396.

106 [1992] NZLR 426.

While personal injury caused by disease exclusively does not qualify for ACC entitlement ACC has long acknowledged, for example, in s28 of the 1982 Act, that workers are eligible for compensation for diseases arising out of employment. The 1992 Act however, introduced new legislation through s7 to qualify personal injury that is the consequence of occupational or work related disease. The essential focus of s7 of the 1992 Act is upon causation. That focus has been carried forward in s30(2) of the Injury Prevention, Rehabilitation and Compensation Act (IPRCA) 2001. It is undisputed that the 1992 Act has severely constrained the scope of coverage for work related disease or infection and has added further complexity.[107]

Determination of compensation for work related process, disease or infection is often very difficult as it involves consideration of complex, multiple factors. Following the 1992 Act however, workers claiming a particular type of personal injury, namely that caused by gradual process, disease or infection in the course of employment, had to meet strict qualifying conditions that assess the employment risk as outlined in s7(1) of the 1992 Act and s30(2) of the present IPRCA. In essence these sections prescribe when particular types of personal injury due to gradual process, disease or infection contracted in a work place are established.

Before I discuss the specific qualifying conditions it is important to note that all claims for personal injury caused by gradual process, disease or infection must satisfy the qualifications defined in the relevant ACC legislation unless the personal injury is derived from exposure in employment to dangerous substances described in the Schedule(s) of the Act. For example, mesothelioma caused by exposure to asbestos is an acknowledged personal injury listed in Schedule 2 IPRCA. Therefore, if mesothelioma is contracted in employment a claimant suffering such personal injury is entitled to compensation providing the disease results in the person's incapacity. Importantly, a person suffering a Schedule 2 injury is not required to undertake an assessment of causation pursuant to s30(4).[108] The implications from that are that once a claimant has established a Schedule 2 personal injury then the onus is on ACC to prove that the person's personal injury has a cause other than employment or falls outside a Schedule 2 personal injury as stated in s60 IPRCA.

The situation however, is very different for timber workers experiencing a wide range of symptoms due to prolonged, occupational PCP exposure because the list of dangerous substances and occupational diseases in Schedule 2 makes no reference to any diseases relating to PCP exposure. This means timber workers are not only required to establish that the personal injury or alleged disease is a consequence of PCP exposure in the course of employment but must also meet strict qualifying conditions before ACC grants cover.[109] Panckhurst J summarized the three qualifying, cumulative pre conditions set out in s7(1)(a),(b) and (c) of the 1992 Act as follows:

First, the employment task had a particular causative property or characteristic. Next that such property or characteristic is not materially found in the person's non employment activities. Third that persons performing the particular employment task are known to be at significantly greater risk of suffering the injury in question.[110]

The onus on the claimant to satisfy the three cumulative pre conditions heavy suggesting that parliament intended to compensate for personal injury said to be caused by work related gradual

107 Campbell, above n 87, 109.

108 *ACC v Estate of Lehmann* [2004] District Court, Wellington 225/04 (Unreported, Ongley DCJ, 11 August 2004).

109 Ibid.

110 *JBDB v ARCIC* [2000] NZAR 385.

process, disease or infection, only in clear cases.[111] It is logical that to apply the tests in s7(1) it is necessary to define the injury.

With regard to the 7(1)(a) inquiry, Ongley J stated in *Mallia v ARCIC,* (a case on sick building syndrome, an umbrella term for patients with a variety of symptoms, controversial in nature and cause but volatile organic compounds, for example, formaldehyde in the building environment which has been implicated in the literature) that the inquiry considers whether there is sufficient evidence to demonstrate that the environment in which the claimant performed the employment task had the property or characteristic of exceeding the formaldehyde levels of 0.1pm, a guideline comfort limit, more or less continuously.[112] Because formaldehyde levels determined were consistently above the upper limit of 0.1pm, Ongley J decided that on the balance of probabilities the levels where sufficiently high to cause the appellant's discomfort.[113] Applying *Mallia*, sawmill workers must provide sufficient evidence that on the balance of probability occupational PCP exposure consistently exceeded acceptable PCP levels, and caused adverse human health effects. Threshold levels below which no adverse effects will be experienced have also been estimated for PCP and PCDDs/PCDFs.[114] For example, the NTG for the Waipa study adopted an Acceptable Daily Intake (ADI) of 0.03 mg PCP per kilogram of bodyweight per day as appropriate for non carcinogenic human health effects.[115] An estimated minimal oral lethal dose of about 30mg/kg in humans has been reported for PCP.[116] Similar dose levels administered through inhalation and skin contact, the chief routes of exposure to PCP in an industrial setting, are thought to have a similar degree of toxicity.[117]

It seems that not only scientific information but also considerable value judgment is required to determine whether PCP levels in sawmills exceeded threshold limits below which no observable health effects may be expected. In my view there is circumstantial evidence that PCP levels were sufficiently high to cause worker's discomfort, at least. I support that proposition using published data as follows:

1. The NTG determined significant concentrations of PCP in the soil at Waipa ranging from 0.35–3600 mg/kg in the vicinity of the green chain and 50–1250 mg/kg in the vicinity of the Rueping plant.[118] Highest concentrations were associated with soil surfaces (0.5 cm). Furthermore, marked PCP levels were confined to Waipa sawmill and an adjacent stream.[119]

2. In the environment, PCP rapidly degrades by exposure to the sun but low oxygen levels in soil cause PCP to persist. For example, PCP has a half life of 10–70 days in flooded soil.[120] However, sawmill soil is not water logged for extended periods of the year. Therefore PCP degradation would occur at a faster rate for most times of the year.

---

111 Ibid.

112 *Mallia v ARCIC* [1996] 1 BACR 386.

113 Ibid.

114 National Task Group, above n 8, 6–19.

115 National Task Group, above n 8, 6–3.

116 P Jorens and P Schepens, 'Human pentachlorophenol poisoning' (1993 Nov) 12(6) *Human & Experimental Toxicology* 479–95.

117 OSH above n 29, 25.

118 National Task Group, above n 8, ii.

119 S Anderson, S Buckland, J Gifford, et al., 'Pentachlorophenol (PCP), PCDD, PCDF and pesticide concentrations in a fresh-water lake catchment' (1996) 32(11) *Chemosphere* 02097–02113, 2111.

120 National Task Group, above n 8, 6–2.

3. Considering the reported half life of PCP in soil and the fact that the PCP measurements at Waipa were undertaken three years after PCP was used, it is reasonable to conclude that those PCP levels found are unlikely to have been less when workers were using PCP for timber treatments.

4. The PCP levels determined at Waipa likely reflect the situation at other sawmills with high PCP use, for example, in Whakatane.

5. Theoretically, the lethal oral dose for a worker weighing 100kg would be at least 3g of PCP. Using the Waipa data one kilogram of soil could contain up to 3.6g of PCP. It is plausible that at the upper PCP levels found in Waipa soil, workers were placed at a significantly high risk. For example, inhalation of PCP tainted soil particles (dust) is a known route of human exposure in industrial settings.[121] NTG also documented significant concentrations of PCDD/PCDF in soil and suggested that Waipa workers in the green chain area exceeded the ADI for PCDD/PCDF through inhalation and ingestion of contaminated dust.[122]

6. Tests of individual timber workers confirmed PCP in their urine.[123] PCP in the human body can trigger a range of acute health effects which indicates it is acting as a human toxicant. It is accepted that medical experts have insufficient understanding of what these levels mean in terms of advising a patient whether they will get a disease in the long term.[124]

7. Test on three sawmill workers performed seven years after the Whakatane mill closure showed dioxin blood levels that were four to five times above levels the World Health Organisation regards as safe.[125]

From the above it seems reasonable to propose that the property or characteristic in sawmills causing or significantly contributing to worker's ill health was constant exposure to high levels of PCP. This resulted in cumulative body burden through inhalation and skin absorption, and ultimately in personal injury.

The proposition just described is not sufficient to satisfy the qualifying condition under 7(1)(a) unless a claimant can document an occupational exposure history revealing long and intense exposure to PCP.[126] Such exposure history takes into account months of exposure, task(s) undertaken, what PCP process was used (water based or oil based), adequacy of personal protection and severity of exposure.[127] Furthermore, the appellant must show relevant symptoms in keeping with associations documented for PCP poisoning in medical literature.[128] For example, persistent fatigue has been associated with sawmill workers who showed a high PCP exposure history.[129]

Importantly, appellants must show that there is a physical injury resulting in a range of symptoms, and that but for the original physical injury, the symptoms or illness from which they suffer would not have occurred.[130] Ongley J stated in *ACC v Smith*, a case on occupational chemical poisoning, that the existence of physical injury being irritation (red mucosae), is not to be decided from physical consequences that may only be symptoms and not caused by any physical

121 Wood et al, above n 13, 527.

122 National Task Group, above n 8, Appendix H at 24.

123 Spence, above n 22, 65.

124 Edwards, above n 51, 12.

125 V Edwards, 'Frustrated workers' (15 May 2002) *New Zealand GP* 20.

126 OSH, above n 29, 40.

127 Ibid, 9.

128 Ibid, 40.

129 Walls et al, above n 67, 362.

130 *ACC v Smith* [2002] District Court, Palmerston North 76/02 (Unreported, Ongley J, 7 March 2002).

effect.[131] For example, some people will faint at the sight of blood without any physical effect but the symptom itself. Following *ACC v Smith* a noxious element, for example, PCP, must cause or significantly contribute to physical damage. In my opinion, sawmill workers could argue that irritations of skin, eyes and respiratory tract which are well documented, were immediate physical manifestations of injuries caused by PCP exposure. I further suggest that those physical injuries suffered by sawmill workers are personal injuries that fall within the definition of s7 of the 1992 Act.[132] In *Flay v ARCIC*, another case on chemical poisoning, Ongley J decided that the appellant's long term consequences which did not fit into any know pattern of illness, would unlikely have occurred but for the physical effect experienced in the first place.[133] Thus, Ongley J, while accepting the medical contention that physical consequences of exposure should have abated in a short time and the remaining illness could have stemmed from other causes that were of psychogenic origins, held that those other causes could not be separated from the physical consequences of the appellant's exposure.[134] In essence, Ongley J held that the defendant's whole health problems stemmed from exposure to a noxious element and rejected the possibility that, if the exposure had not caused physical distress, her serious illness would have occurred in any case. Sawmill workers also suffer from a range of long term consequences that do not fit into any characteristic pattern of disease. The question is whether those persistent health problems stem from a separate cause that would have occurred in any event. In my view, it is highly improbable that the general debilitating conditions sawmill workers suffer would have occurred in any event if PCP had not caused physical distress; this distress then set off a chain of persistent symptoms resulting in the debilitating health of workers.

The second qualifying pre condition set out in s7(1)(b) of the 1992 Act requires an appellant to demonstrate that the property or characteristic, namely high PCP levels, were not found to any material extent in their non work environment. While PCP has become ubiquitous in the environment including the food chain, those levels are very much lower than found at sawmill sites. According to Ongley J a claimant is entitled to cover if 'a contributory cause from non employment activities with marginal effect is not material'.[135] In my opinion, there is insufficient evidence to indicate that non work related PCP exposure would have substantially caused those syndromes in sawmill workers.

Under the s7(1)(b) inquiry however, any other factor which could equally cause the symptoms claimants display must be considered and discounted as being material. Beattie J held in *Thomas v ARCIC and Carter Holt Harvey Ltd,* a case where the appellant suffered from solvent neurotoxicity from PCP exposure, that drug and alcohol abuse, which medical evidence suggested could also cause the appellant's symptoms, are two such factors that fall under the s7(1)(b) inquiry.[136] Additional factors that must be discounted as being material to the symptoms claimed could include exposure to other wood preservatives and concurrent diseases, for example, diabetes, medication, head injuries or depression. For example, in *Thomas v ARCIC and Carter Holt Harvey Ltd* a

131 Ibid.

132 *Flay v ARCIC* [1996] District Court, Whangarei 144/96 (Unreported, Middleton J, 29 November 1996).

133 *ACC v Doyle* [2002] District Court, Wellington 270/2004 (Unreported, Ongley J, 7 March 2002).

134 Ibid.

135 *Mallia a*bove n 112, 398.

136 *Thomas v ARCIC and Carter Holt Harvey Limited* [1999] Christchurch District Court 373/99 (Unreported, Beattie J, 25 November 1999).

claim that PCP exposure at work was causative of asthma was not accepted because the appellant showed a predisposition to this malady since early childhood.[137]

The final statutory precondition that must be satisfied concerns a risk assessment in general terms. It compares the risk of persons, but not the claimant, to contract the condition(s) when performing the specific type of work with that property or characteristic and the general population. Young J in *Knox v ARCIC* decided that a medical expert has to make three assessments:

i) The risk to a person carrying out the relevant task in the relevant work environment of developing the injury concerned (classified as 'X');

ii) The risk to persons not performing that task in the environment of suffering from that personal injury (classified as 'Y');

iii) If 'X' were determined to be significantly greater than 'Y' section 7(1)(c) was satisfied.[138]

Medical experts accept that any sawmill worker falling into the high category of PCP exposure would have a good case for a connection between PCP exposure and current medical symptoms.[139] For example, workers in the high category of PCP exposure showed a strong association with persistent fever (sweating).[140] An assessment of the risk of the general population to contract a 'PCP related condition' such as persistent sweating is complicated by the fact that we are generally dealing with non specific, difficult to measure signs of ill health. Medical experts must also provide an opinion determining the risk of the general public (Y) who did not use PCP for the intensity and duration identified for sawmill workers of acquiring, for example, persistent sweating,[141] It is my opinion for the general public working in occupations that do not involve on going high PCP exposure, for example, constant handling of PCP treated timber or daily mixing of PCP treatment solutions, the risk of suffering from that personal injury, for example, persistent fewer, is much lower. If the risk of persons exposed to constant high PCP is much greater than for the general public having no occupational PCP exposure then s7(1)(c) is satisfied.

From the above discussion it is evident that the issue of causation is established through consideration of s7(1)(a) and (b) while s7(c) is a risk assessment in more general terms. A claimant in a work related disease case such as PCP poisoning must satisfy the three pre condition in s7(1) before ACC entitlements can be regarded as reasonably unambiguous. Because of the uncertain nature of PCP poisoning, specialists and in particularly medical experts, play a key role in assisting ACC in the decision making process. The role of specialist will be considered in the following discussion.

VII. Role of Experts in the Legal Decision Making Process

The 1992 Act introduced new legislation for personal injury caused by work related gradual process, disease or infection requiring claimants to establish causation, on the balance of probabilities, that a work related task caused personal injury to justify ACC entitlements. Thus, the 1992 Act has introduced an adversarial system into a no fault accident compensation system where a claimant is required to make a proposition why ACC cover should be granted. In complicated cases,

137 Ibid.

138 *Knox v ARCIC* [2000] NZAR 609.

139 OSH, above n 29, 40.

140 Walls et al, above n 67, 364.

141 *Knox v ARCIC*, above n 138, 620.

claimants depend heavily on the views of specialists from different disciplines, in particularly medical experts, to support their theory of causation.[142]

Studies in New Zealand have documented associations between various syndromes of disease and PCP exposure. Medical experts however, argue that determination of ill health effects arising from PCP exposure involves a diagnosis of exclusion based on detailed occupational and medical investigations to exclude other causes.[143] For example, experts must determine whether any confounding factor contributed to workers' illness and whether that factor was substantial in the causation of the alleged symptoms. The argument against ACC cover generally is not that PCP is entirely excluded as a cause of a claimant's illness but that there are more likely causes for the condition. It is undeniable that medical diagnosis and etiology have become critical factors in determining eligibility for ACC cover in this particular field.[144] A diagnosis of PCP poisoning cannot be made with certainty because symptoms, for example, persistent fatigue, is non specific, present in the general public, and there is no specific diagnostic test available. Consequently, considerable disagreement can exist among medical experts regarding causation of symptoms which may result in delays in the decision process due to litigations. This puts further financial and emotional distress onto the claimant, in addition to the health problems suffered.

VIII. Test of poisoning

As discussed above medical research has not shown a causal link between PCP exposure and chronic ill health symptoms of timber workers. In other words, scientific validation has failed to conclusively demonstrate causation of long term effects of PCP poisoning of timber workers, and thus is of little aid to workers claiming compensation. Because it is not proven from a medical science point of view that PCP exposure causes long term health effects ACC has used an assessment procedure, called a test of poisoning (TOP).[145] For ACC compensation any worker alleging a causal relationship between PCP exposure and chronic illness must take the TOP.

TOP is a tool used by the PCP medical expert panel to decide whether recommendation can be made to ACC to grant compensation to workers claiming personal injury due to PCP poisoning. The TOP has been criticised by some occupational physicians because it has not been validated.[146] Other critics of the TOP suggested that for some occupational disorders, medical experts have to rely on probability 'based on symptoms and clinical findings, where the results obtained at functional assessment are not positive'.[147] Gorman and colleagues however, argued that the TOP estimates the likelihood that a person's syndromes being due to chemical poisoning; thus it is a particularly useful tool in conditions of uncertainty. In other words, the TOP does not require proving the cause of an illness but what is required is to accept or reject the hypothesis that a chemical agent has caused the illness.[148]

142 *Primary Producers Co-Operative Society Ltd v ARCIC* [1999] District Court, Christchurch (Unreported, Beattie J, 17 August 1999).

143 OSH, above n 29, 40.

144 G Duncan, 'Moral hazard and medical assessment' (2003) *Victoria University of Wellington Law Report* 433, 435 available at http://www.austlii.edu.au/nz/journals/VUWLRev/2003/26.html.

145 V Edwards, 'PCP poisoning: the uncertainty lingers', (15 May 2002) *New Zealand GP* 20.

146 C Walls, 'Diagnosis of chemical poisoning' (1998 Jul 10) 111(1069) *New Zealand Medical Journal* 258–9.

147 T O'Donnell, 'Chemical poisoning and occupational asthma: Diagnosis and/or acceptance for current compensation. (1998) 111(1074) *New Zealand Medical Journal* 372–3.

148 Dew, above n 82 at 175.

The TOP is divided into three parts classifying a patient's symptoms into major, intermittent and minor criteria categories.[149] It awards points scored for each criterion of the test which are summed up to determine the likelihood that health problems experienced are the consequence of chemical poisoning. If a patient scores at least nine points the expert panel accept the likelihood of chemical poisoning and will make a recommendation to ACC to grant cover for personal injury.[150] The TOP is a very strict, and in my opinion unfair, assessment tool because the standard of proof is very high to establish that PCP poisoning is causative of workers' illness. In the following paragraphs I will discuss selected criteria of the TOP to illustrate that it disadvantages timber workers seeking ACC cover for PCP poisoning.

The TOP may award points if workers suffered symptoms, for example, excessive sweating, on the day of the ACC assessment. ACC's assessments for PCP poisoning however, did not start until about 1998; this is ten years after timber workers used PCP for various wood treatments. The medical community accepts that PCP exposure can cause acute excessive sweating which constitutes one of a host of measurable reactions of the human body dealing with acute PCP stress.[151] Clearly, former timber workers have reported on going problems with sweating.[152] While ACC may accept excessive sweating as an indication of PCP poisoning its absence at the day of assessment does not disprove that the health problems of timber workers are not the consequence of PCP exposure. Sweating may be intermittent but still chronic in some workers. Since the TOP awards only points for symptoms measurable on the assessment day it ignores workers' evidence of severe sweating in the past.

Furthermore, points are not awarded if symptoms failed to meet ACC's specification ascribed to PCP poisoning. For example, excessive sweating, which ACC may accept as a specific symptom of PCP poisoning, must have a particular foul smell and rot clothes.[153] This means a worker having excessive sweating on the assessment day but with sweat lacking a particular smell would not qualify for any points. ACC believes that foul smelling and discoloured sweat only have a clear temporal relationship to PCP exposure which will abate after PCP exposure ceases. Again, the example above illustrates the rigor of the ACC's test of poisoning.

Also, the TOP further grants points to workers that have had an appropriate exposure to the chemical at which levels chronic effects of PCP poisoning could be possible.[154] To determine exposure a formula was developed that takes into account the job task, length of time at that task and the type of PCP formulation (oil vs. water based) to estimate the level of PCP exposure in workers, also called exposure index.[155] However, the toxic potency of technical grade PCP used at sawmills was not included in calculating the exposure index because it is unknown. It is well known that PCP based wood treatments contained dioxin contaminants of variable nature and quantities. It is likely that contaminants found in PCP based products which were purchased from

149 D Gorman and E Dryson, 'Diagnosis of chemical poisoning: report of a working party established by the Australasian Faculty of Occupational Medicine, Royal Australasian College of Physicians, Auckland, 9 May 1997' (1998 Feb 13) 111 (1059) *New Zealand Medical Journal* 34–7.

150 Ibid.

151 Wood et al, above n 13, 528.

152 Gorman, above n 149, 35.

153 Dew, above n 82 at 171.

154 Gorman, above n 69, 191.

155 Gorman, above n 69, 190.

different overseas suppliers varied depending on the source of supply.[156] Also, workers' recall bias may result in miscalculating PCP exposure levels. It seems reasonable to suggest that the calculated exposure index may grossly underestimate the 'real' risk timber workers encountered when working with PCP based wood treatments.

Furthermore, it is likely that individual workers have different thresholds or tolerance levels to PCP. The consequence of variable PCP tolerance is that individual workers may still experience persistent health problems at relatively low PCP exposure levels. According to the TOP however, chronic affects are not plausible at those lower PCP levels. Exposure level is significantly correlated to increased mood changes, rhinorrhoea and breathing difficulties but not to other symptoms timber workers experience.[157] Also, workers with relative low PCP exposure reportedly suffer from a large number of symptoms.[158] While there is great uncertainty at what level of exposure PCP is harmful to humans the TOP defines a somewhat arbitrary exposure index above which ACC accepts that chronic effects would be plausible.[159]

TOP further awards ten points if a patient has levels of chemicals in their body in excess of what is considered to be toxic and objective biological markers of the poisoning effect.[160] An example is lead poisoning where body levels of lead and haemoglobin precursors can be concurrently detected.[161] ACC however argues that there is no objective biological effect marker for PCP poisoning and bodily PCP levels in workers are not available due to the relative short half life of PCP.[162] PCP had a urinary half life of 33 hours for a human volunteer.[163] This means timber workers alleging PCP poisoning are unable to get any points based on this criterion. They are clearly disadvantaged because medical science has not yet discovered evidence based measures of toxicity, namely biological effect markers of PCP poisoning. On the other hand dioxin contaminants of PCP based wood treatments vary in nature and extent and have an extensive half life in humans, reportedly between 3 and 20 years.[164] Thus, dioxin can be determined in human blood and fat many years after PCP exposure. However, interpretation of dioxin data with regard to long term human diseases is difficult because it is not clear at what levels human health is at risk.[165] ACC has not determined dioxin levels in serum of timber workers. It is interesting to note that a poor correlation was observed between dioxin levels determined in four sawmill workers and the ACC exposure index, casting serious doubt that the latter measure can estimate the amount of dioxin absorbed in the body.[166]

---

156 National Task Group, above n 8, 2–6.

157 Bandaranayake, above n 70, 21.

158 Ibid.

159 Gorman, above n 69, 191.

160 Gorman, above n 149, 35.

161 Ibid.

162 Edwards, above n 145, 20.

163 G Kleinman, S Horstman, D Kalman, et al., 'Industrial Hygiene, Chemical and Biological Assessments of Exposures to a Chlorinated Phenolic Sapstain Control Agent' (December 1986) Volume 47, Issue 12 *American Industrial Hygiene Association Journal* 731–741, 732.

164 H Kontsas, C Rosenberg, J Tornaeus, et al., 'Exposure of workers to 2,3,7,8-substituted polychlorinated dibenzo-p-dioxin (PCDD) and dibenzofuran (PCDF) compounds in sawmills previously using chlorophenol-containing antistain agents' (1998) *Archives of Environmental Health* 99, 100.

165 Gorman, above n 69, 192.

166 Bandaranayake, above n 70, 34.

The TOP further awards points for symptoms that are characteristic of the chemical agent. Chloracne is the only symptom ACC has accepted as characteristic or proven of chronic PCP poisoning in humans.[167] Timber workers however, cannot provide objective data linking PCP exposure to other, alleged chronic diseases, thus points will not be granted. However, two points will be awarded if any symptoms alleged are biological possible effects of PCP poisoning. For example, the medical expert panel has accepted PCP as a plausible cause of brain injury.[168] This plausible cause is accepted because PCP uncouples oxidative phosphorylation producing cellular disturbance of energy production and utilization.[169]

In summary, timber workers alleging chronic effects due to PCP poisoning, unless they suffer from chronic chloracne, are not able to score any points for four out of eight categories of the TOP. This is because medical research has failed to provide conclusive scientific evidence for PCP poisoning. The remaining four categories grant five and two points respectively, yielding a maximum score of 11. As mentioned above timber workers require at least nine points before the medical expert panel can recommend ACC to accept cover for personal injury. By 2002 the medical expert panel recommended to ACC that 20–25 per cent of all claims be accepted.[170] This further indicates that the TOP poses a huge hurdle to timber workers in their battle to get compensation for chronic health problems. However, even if the medical expert panel accepts the hypothesis that PCP poisoning caused a worker's health problem this does not guarantee that ACC will grant compensation. As discussed above other criteria outlined in the ACC legislation must be met before cover is granted for any occupational diseases.

IX. Standard of Proof

As discussed above the 1992 ACC Act introduced an adversarial system for personal injuries caused by work related gradual process, disease and infection. The onus falls on sawmill workers seeking ACC compensation to establish a nexus between occupational PCP exposure and illness. Essentially, in order to succeed sawmill workers are required to make propositions which provide sufficient, persuasive evidence for this nexus to satisfy ACC on the balance of probability.

Before I elaborate further on standard of proof a clear distinction must be drawn between scientific and legal standard of proof. In science, including medical science, proof showing a causal effect is determined in terms of certainties which basically means beyond reasonable doubt. Researchers establish scientific certainty using various statistical tests to measure significance of a particular effect observed. In an instance where an effect is significant causation is said to be proven at a certain level of probability, for example, 99 per cent. While medical science in New Zealand has shown high levels of probability relating certain symptoms to PCP exposure, calls were made again and again for further studies.

One argument is that better experimental design could provide greater statistical certainty as to the possibility of cause and effect. Public funding has now been granted to investigate health outcomes of former timber workers exposed to PCP.

The legal (civil) standard of proof differs from that applied in medical science. That civil standard is on balance of probability which essential means greater than 50 per cent. There is no

167 Edwards, above n 145, 20.

168 Ibid.

169 Wood et al, above n 13, 529.

170 Edwards, above n 145, 20.

doubt that the Courts, considering the evidence submitted by medical experts and laypersons, apply the appropriate legal test to reach a decision.[171] Likewise, ACC decision makers will evaluate the propositions (evidence) made by the parties and then decide the validity of a claim applying the civil standard of proof.[172] Sawmill workers are not required to prove their claim with absolute certainty because it clearly is impossible to ascertain that PCP poisoning caused workers' illnesses. On the other hand ACC will not accept any claim based on mere speculation. Sawmill workers must provide direct evidence or offer other objective proven facts (circumstantial evidence) that enables the decision maker to draw a reasonable deduction from the evidence.[173] Sawmill workers will fail to discharge the burden of proof unless they create a state of belief in the mind of the ACC decision maker that allows the decision maker to accept that the worker's evidence as submitted is more probable than evidence presented by the opposing party. Within the context of evidence the TOP provides one piece of evidence that may support or reject workers' claim. It must be kept in mind however, that the TOP is a likelihood model for use in situations of uncertainty relating to cause and effect.[174] In other words, the TOP can only indicate whether it is probable that a claimant suffers the alleged personal injury due to PCP poisoning. Thus the TOP represents one view of medical evidence which ACC has to consider within the whole context (evidence) of the appellant's claim. Because, in my view, the TOP is a very strict assessment tool it is highly recommended that sawmill workers seek additional, independent medical evidence to support their claim.

Clearly, the role of ACC decision makers and also judges (in case of litigations) is to critically examine the evidence, including medical, presented by the parties. For example, they will examine the clarity of expression, impartiality, and supporting scientific evidence. Responsible expert opinion cannot be rejected unless there is some clear indication that it is based on mistake or oversight.[175] In weighing up the different medical opinions, a judge will specifically consider whether a medical expert comments on the reasoning or conclusions reached by other specialists.[176] In *Knox v ARCIC* Young J rejected medical evidence by Dr Monigatti because it was 'too conclusive in that it did not lay out the steps in his reasoning'.[177] Ongley J rejected the contention of Dr Monigatti who argued a psychogenic diagnosis following negative TOP results of the appellant, because there was no medical evidence supporting her predisposition to a psychoneurotic illness.[178] Likewise, in *Hawkins* Gorman's strong conviction that the appellant had a somatoform disorder, meaning a physical complaint with no physical basis, was rejected due to 'an absence of clear and unequivocal evidence to that effect from a physiological or psychiatric source.'[179]

Within the PCP debate medical experts like Gorman have continued to question the specificity of acute and persistent symptoms that have been documented in the population of former timber

171 *Davidson v ACC* [1995] NZAR 299.

172 *Wilde v ACC* [2001] Accident Compensation Appeal Authority, Auckland 10/02 (Unreported, Cartwright J, 8 August 2001).

173 *Hawkins ET UX v Dominion Breweries, Ltd*. [1948] NZLR 15.

174 Gorman, above n 69, 190.

175 *Doyle a*bove n 133.

176 *Marshall v ACC* [2004] District Court, Wellington 169/2004 (Unreported, Ongley J, 8 June 2004).

177 *Knox v ARCIC*, above n 138, 619.

178 *Doyle* above n 133.

179 *Hawkins* above n 173, 29.

workers.[180] For example, pre morbid education level, alcohol use, head injuries, depression and senility were thought to confound the identification of neuropsychiatric disorders of PCP in 21 of 48 workers showing significant psychometric abnormalities.[181] While Gorman's contention is theoretically plausible there must be sufficient evidence for it. For example, did workers consume alcohol at levels above which the medical community accepts could cause psychometric abnormalities? Although it is difficult to generalize, it is my view that, on balance of probability, deterioration of workers' health, most of whom were physically very fit due to the strenuous nature of the work, was due to exposure to PCP.

X. Conclusion

Sawmill workers have been largely battling unsuccessfully to get full ACC entitlement for personal injuries caused by PCP poisoning occurring during the course of employment. To gain ACC cover sawmill workers must prove a nexus of causation between occupational PCP exposure involving inhalation, skin absorption and oral ingestion and the subsequent personal injury affecting their health. ACC relies heavily on medical evidence to decide whether such nexus of causation exists. Because of the complex nature of PCP poisoning decision makers will use considerable value judgments and not just the rational application of scientific knowledge. Any ACC decision however, must be founded on sufficient evidence, not mere speculation, to discharge the legal burden of proof on the balance of probability.

Medical and legal requirements have undoubtedly put many obstacles onto sawmill workers in their attempt to get ACC cover. Ideally, a policy change in ACC compensation would be required to address these grave injustices of the no fault ACC system. For example, the onus could be on ACC to show that occupational PCP poisoning is not causative of personal injury. In my view, sawmill workers suffering debilitating health from PCP poisoning should also receive public acknowledgement to assist in the healing process of past injustices.

180 Gorman, above n 69, 192.

181 Gorman, above n 69, 193.