Ian Freckelton*

Huntington's disease (HD) is a relentlessly progressive and fatal neurological condition that is inherited. It has serious and disabling physical and mental components. As such, it impacts upon those who have HD, those with the potential to inherit it, and those who care for those with HD in a wide variety of ways. These can have many legal ramifications including in relation to evolving impairments of capacity which can have an outcome in terms of involuntary status as mental health patients, testamentary capacity and the need for guardianship and administration. It can have effects upon fitness for parenting, obligations for spousal maintenance, and the quantum of compensation from a tortious incident to which a person is entitled. It has repercussions for criminal liability and culpability. This article reviews case law from a number of countries in relation to such matters, noting the broader radiation to others of the effects of HD, and reflecting on the need for legal and medical professionals to be aware of the legal consequences of HD for them to be able to discharge their responsibilities holistically, sensitively and informedly.

HUNTINGTON'S DISEASE AND THE LAW

The opinion expressed by the expert witnesses in the trial was that Huntington's disease is perhaps the most unpleasant disease of all.¹

[Huntington's] is a real terrorist specializing in the slow poisoning, the time bomb ticking away that will inevitably explode. You're born with the fuse, and you live with it for years, without knowing that it's slowly burning down.²

Our fate is not in our genes, irrevocable and inexorable as the illness may be. Whatever the size of our Huntington's disease gene – since we all have this gene – we are not defined by it. We and our genes are not identical.³

INTRODUCTION

There are many different lenses through which deteriorating capacity to make decisions about one's life and through which worsening conditions of physical health can be viewed. All of them have in common sadness and suffering, albeit with the potential for residual opportunities for the exercise of autonomy and for dignity and personal growth in the form of coming to terms with life, how it is lived and how it concludes. Huntington's disease (HD) is particularly poignant and confronting in this regard, both personally and, in some instances, legally.

HD is an inherited, degenerative neurological condition that affects a significant percentage of the population: most obviously those who contract it, but also those at risk of inheriting it, as well as those who live with and care about such persons. As yet, HD has no cure and medications are unable to halt

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¹ Kakoschke v Draper [2006] QSC 386 at [30] (Skoien AJ).

² Barema J, The Test: Living in the Shadow of Huntington's Disease (Franklin Square Press, New York, 2005) p 14.

³ Wexler N, "Foreword" in Wexler A, *The Woman Who Walked into the Sea: Huntington's and the Making of a Genetic Disease* (Yale University Press, New Haven, 2008).

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or slow its progression – they simply attempt to manage its symptoms. However, since the gene responsible for HD was discovered in 1993, concerted research efforts have attempted (as yet with only modest success) to identify ways to address its onset and its effects.⁴

HD particularly came to public attention with the death of folk singer Woodie Guthrie⁵ who died of the condition in 1967. His widow Marjorie Guthrie founded the Committee to Combat Huntington's Disease in the same year. It later evolved into the Huntington's Disease Society of America.⁶ Between 1976 and 1978 the Commission for the Control of Huntington's Disease and its Consequences,⁷ established by the United States Congress, undertook inquiries into the measures needed to address the disease more effectively.

Alice Wexler⁸ has also written and spoken extensively about a wide range of issues about HD that have affected her family, among other things chronicling her mother's diagnosis with the disease in 1968 and her father's organisation of the Hereditary Disease Foundation.⁹ A number of books have been published too, giving voice to those with HD and providing support, guidance and a focus for reflection to persons with HD and to family members and friends of those with HD.¹⁰ A secondary benefit of such publications is their educative value for the medical and legal communities, and the wider community generally, about the experience of those with and affected by HD.

Aside from its physical symptomatology which, among other things, is characterised by distinctive choreic (writhing) movements,¹¹ adversely affected gait and a reduced capacity to swallow, HD produces impaired executive functioning and a host of affective symptoms which can mimic psychotic and dementing conditions. A consequence is that HD has the potential to generate multiple difficulties with legal ramifications for those diagnosed with it or at risk of contracting it. It was identified as a focus of United States eugenics research and debate in the 1920s and 1930s¹² and then singled out for eugenic extermination by the Nazis along with other inherited and congenital conditions that were seen to weaken the gene pool.¹³ A major issue in relation to HD continues to be

⁴ See eg Huntington's Disease Society of America, *Research*, <u>http://www.hdsa.org/research.html</u> viewed 5 July 2010.

⁵ See Woody Guthrie (2010), <u>http://www.woodyguthrie.org</u> viewed 6 July 2010.

⁶ See Huntington's Disease Society of America, <u>http://www.hdsa.org</u> viewed 31 July 2010.

⁷ Commission for the Control of Huntington's Disease and its Consequences, *Report* (Dept of Health, Education, and Welfare, Public Health Service, National Institutes of Health, 1978).

⁸ See Wexler A, *Mapping Fate: A Memoir of Family, Risk and Genetic Research* (University of California Press, Berkeley, 1995); Wexler, n 3.

⁹ See Hereditary Disease Foundation, About HDF, <u>http://www.hdfoundation.org/aboutus.php</u> viewed 5 July 2010.

¹⁰ See eg Sulaiman S, *Learning to Live with Huntington's Disease: One Family's Story* (Jessica Kingsley Publishers, London, 2007); Barema, n 2. See also Grey A, *Huntington's and Me: A Guide for Young People* (Huntington's Disease Associations of New Zealand, Wellington, 2000); Ferguson F, *Ocean of Dreams* (Zeus Publishing, Melbourne, 2010). See also the newsletters of the Huntington's Disease Associations of New Zealand, <u>http://www.huntingtons.org.nz/news_pdf.php</u> viewed 6 July 2010 and of Huntington's Victoria, <u>http://www.ahda.com.au/images/stories/HV%20Contact%20June%202010.pdf</u> viewed 10 August 2010.

¹¹ Compare Sydenham's chorea, also known as Chorea minor or St Vitus' dance: see Waller J, *A Time to Dance, A Time to Die* (Icon Books, Cambridge, 2008); Hannah P, *Huntington's Chorea or Sydenham's Chorea: Two Major Forms of Chorea* (4 November 2009), <u>http://www.associatedcontent.com/article/2354268/huntingtons_chorea_and_sydenhams_chorea.html</u> viewed 6 July 2010.

¹² Wexler, n 3, p 174ff; see also Davenport CB, "Huntington's Chorea in Relation to Heredity and Eugenics" (1910) 1(5) Proc Natl Acad Sci USA 283. See too in relation to the sterilisation of persons with, among other things, HD, pursuant to the *Sexual Sterilization Act 1928* (Alta); *Muir v Alberta* (1996) 132 DLR (4th) 695.

¹³ See discussion in *Soulitopoulos v La Trobe University Liberal Club* (2002) 120 FCR 584 at [35]-[38]; Wexler, n 3, p 175. Article I of the *Law for the Prevention of Genetically Diseased Offspring 1933* (Ger) (<u>http://frank.mtsu.edu/~baustin/</u><u>nurmlaw1.html</u> viewed 5 July 2010) defined who was to be examined and then sterilised: "(1) Anyone who suffers from an inheritable disease may be surgically sterilized if, in the judgment of medical science, it could be expected that his descendants will suffer from serious inherited mental or physical defects. (2) Anyone who suffers from one of the following is to be regarded as inheritably diseased within the meaning of this law: congenital feeble-mindedness; schizophrenia; manic-depression; congenital epilepsy; inheritable St Vitus' dance (Huntington's Chorea); hereditary blindness; hereditary deafness; serious inheritable malformations." See further Lifton RJ, *The Nazi Doctors: Medical Killing and the Psychology of Genocide* (Basic



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discrimination and genetic stigmatisation in the workplace, including access to insurance for those with HD, within social relationships and during a range of ordinary encounters.¹⁴ However, this aspect of HD will not be the focus of this editorial.

Instead, this editorial reviews a range of areas that legal decision-making in a number of countries has identified as having a relevance for HD in terms of administrative, criminal, civil and family law litigation. It does not purport to be an exhaustive treatment of HD in courts and tribunals but it has selected cases to illustrate a cross-section of relevant legal issues for persons with HD or at risk of developing it. The editorial argues that greater awareness of the disease is needed within the legal system so as to better inform court and tribunal processes, as well as the protections that need to be accorded under the law to this vulnerable category of persons in the community. It also argues that cognisance of the legal repercussions of the disease is important for health practitioners working with HD and those affected by it.

CLINICAL ISSUES

Huntington's disease (HD), also known as Huntington's Chorea (HC),¹⁵ chorea major,¹⁶ or inherited St Vitus' Dance,¹⁷ because of its dance-like symptomatology, was identified in 1872 by United States physician, Dr George Huntington.¹⁸ However, it had long been recognised by physicians,¹⁹ the German physician Thilenius²⁰ having written about it in some detail in 1816, and it having been described for some time as "magrums" or "megrims", a name given to it by early Dutch settlers in North America.²¹ One of the theories about the behaviour of the witches in the Salem area was that at least some of them may have had HD, resulting in involuntary movements which led to suspicions about their being demonically possessed.²²

HD, in fact, results from genetically programmed degeneration of neurons in certain areas of the brain. It is now understood that the disease is caused by a CAG (cytosine, adenine, guanine) trinucleotide expansion in the first exon of the HD gene.²³ The HD gene was mapped to chromosome 4 in 1983 and cloned in 1993; it codes for production of a protein called "huntingtin", whose function is still unknown. The altered huntingtin protein contains many more molecules of the amino acid glutamine than regular huntingtin. This is due to repetitive copies of the CAG codon in the Huntington gene: "The extended glutamine tracts of these proteins have affinity for one another, and tend to 'stick together', leading to the formation of 'clumps' or aggregations of the protein in the cell's nucleus.

Books, New York, 1986); Freckelton I, "Bioethics, Biopolitics and Medical Regulation: Learning from the Nazi Doctor Experience" (2009) 16 JLM 555 (Editorial).

¹⁴ See Australian Law Reform Commission, *Essentially Yours: The Protection of Human Genetic Information* (ALRC 96, AGPS, Canberra, 2003), <u>http://www.austlii.edu.au/au/other/alrc/publications/reports/96</u> viewed 30 June 2010; Williams JK et al, "In Their Own Words: Reports of Stigma and Genetic Discrimination by People at Risk for Huntington Disease in the International RESPOND-HD Study" (2010) Am J Med Genet B Neuropsychiatr Genet (in press); Kirby M, "Human Genome Project: Legal Issues", paper to Australian Lawyers' Conference, Hanoi, Vietnam, 10 July 1999, <u>http://www.hcourt.gov.au/speeches/kirbyj/kirbyj_humang.htm</u> viewed 5 July 2010; Jensen P, "Genetic Privacy: The Potential for Genetic Discrimination in Insurance" (1999) 33 VUWLR 379.

¹⁵ From the Greek word, choreia, dance.

¹⁶ See Hannah, n 11.

¹⁷ See Tabrizi SJ, "The Differential Diagnosis of Chorea" (2007) 7 Pract Neurol 360.

¹⁸ Huntington G, "On Chorea" (1872) 26 Med Surg Report 320. See too Wexler, n 3.

¹⁹ For a helpful history see Hayden MR, "Reflections on the History of Huntington's Chorea" (1983) 6(1) Trends in Neurosciences 122.

²⁰ See Stone TT and Falstein EI, "Genealogical Studies in Huntington's Chorea" (1935) 89(7) Journal of Nervous and Mental Disease 795.

²¹ See Rosenbaum R, "Psychosis with Huntington's Chorea" (1941) 15(1) Psychiatric Quarterly 93.

²² See eg Bellows A, *Bad Rye and the Salem Witches* (9 March 2007), <u>http://www.damninteresting.com/bad-rye-and-the-salem-witches</u> viewed 10 August 2010; Tytler C, *The History of Huntington's Disease* (6 March 2009), <u>http://</u>www.associatedcontent.com/article/1540511/the_history_of_huntingtons_disease.html viewed 10 August 2010.

²³ See Knowles J, *Huntington's Disease* (Rosen Publishing, New York, 2006).

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These protein aggregations are often referred to as neuronal inclusions (NI)."²⁴ It remains to be determined whether NIs are a cause²⁵ or a result of HD, or even a defence mechanism against it.

The CAG repeat is highly polymorphic and varies from 6 to 37 repeats on chromosomes of unaffected individuals and from more than 30 to 180 repeats on chromosomes of those with HD.²⁶ Persons with 28 or fewer repeats will not develop HD; nor will their children. Those with more than 40 generally will develop HD and their children have a 50% chance of doing so.

It appears that people who have 29-35 repeats will not develop HD themselves, but a small percentage of their children may. People with repeats in the range of 36 to 39 are considered to be in a "grey zone"; they may or may not develop HD and, likewise, their children may or may not be affected by the mutant gene.²⁷

This latter situation frequently engenders particular distress arising from its uncertainty and the consequent decision as to whether to submit to testing.

Characteristic neuropathology in HD occurs within the neostriatum or striate nucleus (a subcortical part of the forebrain), in which gross atrophy of the caudate nucleus and putamen²⁸ is accompanied by selective neuronal loss and astrogliosis, an abnormal increase in the number of astrocytes due to the destruction of nearby neurons. Marked neuronal loss is also identifiable in deep layers of the cerebral cortex. Other regions, including the globus pallidus, thalamus, subthalamic nucleus, substantia nigra, and cerebellum, show varying degrees of atrophy depending on the stage of a patient's development of HD.²⁹

HD has many health consequences. Among other things, it causes uncontrolled movements, deterioration of cognitive function, and emotional disturbance. This can affect coordination, thought, perception and memory. Many with HD experience involuntary movements of the arms, legs, body, and face (chorea). Often these symptoms are accompanied by mood swings, depression, irritability, slurred speech and clumsiness.³⁰ Wild and Tabrizi³¹ have summarised the physical features of HD as follows:

Minor motor abnormalities seen early in the disease are general restlessness, hyperreflexia, and fidgety movements of the fingers, hands and toes during stress or when walking. Oculomotor abnormalities are a cardinal feature of the disease and often the earliest motor sign; delayed initiation and slowing of saccades, and inability to suppress glances at novel stimuli. As the disease progresses, blinking and head thrusting are seen during saccade initiation. Later, impaired pursuit and gaze impersistence are seen. Patients then develop more obvious extrapyramidal signs – chorea is seen in 90% of adult-onset patients with varying degrees of dystonia, parkinsonism and bradykinesia. A key motor abnormality is impairment of voluntary motor function with clumsiness, motor impersistence (inability to sustain motor actions or gestures, such as eye closure or tongue protrusion), disturbances in fine motor control and motor speed. Gait disturbance is common with impairment of postural reflexes making patients prone to falling. Dysarthria and dysphagia are common.

²⁴ Huntington's Outreach Project for Education at Stanford, *Protein Aggregation* (2009), <u>http://hopes.stanford.edu/treatmts/</u> pbuildup/h0.html viewed 5 July 2010.

²⁵ Gharibzadeh et al have argued that glutamate toxicity is the cause of HD: see Gharibzadeh S, Sarbaz Y, Banaie M and Towhidshah F, "Two Novel Comments on the Treatment of Huntington's Disease" (2009) 21 *Journal of Clinical Neuropsychiatry and Clinical Neurosciences* 98.

²⁶ See Monche-Buchner E et al, "Counting CAG Repeats in the Huntington's Disease Gene by Restriction Endonuclease *Eco*P151 Cleavage" (2002) 30(16) *Nucleic Acids Research* e83. See too National Human Genome Research Institute, *Learning about Huntington's Disease*, <u>http://www.genome.gov/10001215</u> viewed 28 June 2010.

²⁷ Better Health, *Huntington's Disease – Genetics*, <u>http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Huntingtons_disease_genetics?open_viewed 5_July 2010.</u>

 28 A structure in the forebrain whose main function is to regulate movement and influence various types of learning, utilising dopamine.

²⁹ Revilla FJ and Grutzendler J, *Huntington Disease* (4 September 2008), <u>http://emedicine.medscape.com/article/1150165-overview</u> viewed 27 June 2010.

³⁰ See Folstein SE, Huntington's Disease: A Disorder of Families (Johns Hopkins University Press, Boston, 1989).

³¹ Wild EJ and Tabrizi SJ, "Differential Diagnosis of Chorea" (2007) 7 Pract Neurol 360 at 362.



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HD manifests in its early phase in lability of mood, depression, irritability, and difficulties in learning new information, remembering a fact, or making a decision. Quarrell³² stresses this aspect of the disease, observing that "many families find it more difficult to cope with the wide range of behavioural and emotional problems than with the physical aspects of the condition". As the disease progresses, concentration on intellectual tasks becomes increasingly difficult and the patient may have difficulty feeding himself or herself and in swallowing. Many display considerable apathy. The rate of disease progression and the age of onset vary from person to person. When the disease progresses, a variety of symptoms emerge, including increasing problems in swallowing, loss of balance (ataxia), impaired reasoning, and memory problems. HD is a progressive condition that affects people differently, often quite slowly, leading to a significant incidence of misdiagnosis in its early phases. This can be problematic in the forensic context. A person with HD may live for 15 to 25 years after developing the first symptoms.³³ Death is commonly caused by a complication of the disease such as choking, or an injury related to a fall.³⁴

Each child of a parent with HD symptoms has a 50-50 chance of inheriting the HD gene.³⁵ Those with the HD gene experience cell damage and destruction in the brain's basal ganglia and cortex.

When a child does not inherit the HD gene, he or she will not develop the disease and cannot pass it to subsequent generations but for those who inherit the HD gene it is a matter of time until they develop the disease. Whether one child within a family inherits the gene has no bearing on whether others will or will not inherit the gene. In between 1% and 3% of those ultimately diagnosed with HD, no family history of HD is found. The symptoms of the juvenile form of HD, or "Westphal variant", are somewhat different from adult-onset HD:

Initial symptoms usually involve slow, stiff and awkward walking and talking, choking, clumsiness and falling. Later, the child may become slow to respond and performance at school may become erratic. The course of the juvenile form is generally more rapid than the adult-onset HD.³⁶

Testing prior to the development of symptoms is available for those who are at risk for carrying the HD gene.³⁷ There are three categories of testing:

- presymptomatic for those at risk but not showing symptoms;
- confirmatory for those exhibiting possible symptoms; and
- prenatal testing to determine whether a fetus is at risk for HD.

The testing has a very high rate of accuracy.

Generally HD has an adult onset with its mean age of onset being 40 years but, as noted above, in 5 to 10% of cases its symptoms commence in persons under 20 years of age.³⁸ In the United States about 30,000 people have HD, while some 200,000 are at risk of contracting it.³⁹ In addition, 35,000 people exhibit some symptoms and 75,000 people carry the abnormal gene that will cause them to develop the disease.⁴⁰ In the United Kingdom there are between 4,200 and 6,000 patients with HD at

³⁶ International Huntington Association, Juvenile Huntington's Disease, <u>http://www.huntington-assoc.com</u> viewed 6 July 2010.

³⁷ See Huntington's Disease Society of America, *Genetic Testing for Huntington's Disease*, <u>http://www.lkwdpl.org/hdsa/</u> <u>hdtest.htm</u> viewed 5 July 2010.

³⁸ See Quarrell OWJ, Juvenile Huntington's Disease and Other Trinucleotide Repeat Disorders (Oxford University Press, Melbourne, 2009).

³⁹ Family Caregiver Alliance, *Huntington's Disease*, <u>http://www.caregiver.org/caregiver/jsp/content_node.jsp?nodeid=574</u> viewed 5 July 2010.

⁴⁰ National Human Genome Research Institute, n 26.

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³² Quarrell O, *Huntington's Disease* (2nd ed, Oxford University Press, Oxford, 2008) p 28.

³³ Quarrell, n 32, p 7; Gerics J, *Huntington's Disease Basics* (25 August 2007), <u>http://neurologicalillness.suite101.com/</u> <u>article.cfm/huntingtons_disease_basics</u> viewed 30 June 2010.

³⁴ See Wexler, n 8, p 152.

³⁵ National Institute of Neurological Disorders and Stroke, *Huntington's Disease Information Page*, <u>http://www.ninds.nih.gov/</u> <u>disorders/huntington.htm</u> viewed 12 June 2010.

any time.⁴¹ The incidence of HD is somewhat variable across different populations, with the Lake Maracaibo region in Venezuela being 700 per 100,000 people, the island of Mauritius 46 per 100,000 people, and Tasmania 17.4 per 100,000 people. The prevalence in most European countries ranges from 1.63 to 9.95 per 100,000 people but the prevalence of HD in Finland and Japan is less than 1 per 100,000 people.⁴² In Australia HD is said by Huntingtons Australia⁴³ and the Howard Florey Institute⁴⁴ to affect six to seven people in every 100,000.

Treatment of HD (there being no cure as yet) targets different aspects of its symptomatology, including involuntary movements, psychosis, depression, anxiety and swallowing difficulties. Research is being conducted with a view to reducing the amount of NIs in the cell;⁴⁵ this includes both medications and stem cell research. A number of drugs are also prescribed to address emotional and movement problems associated with HD. In August 2008 the United States Food and Drug Administration approved tetrabenazine to treat Huntington's chorea (the involuntary writhing movements), making it the first drug approved for use in the United States specifically to treat the disease. Most drugs used to treat the symptoms of HD have side-effects such as fatigue, restlessness, or hyperexcitability. Recent research suggests that the defective protein produced by the HD gene interferes with additional types of gene activity that normally help cells survive, among other functions. HDAC inhibitors appear to counteract these gene activity problems and have potential for aiding HD. This is leading to optimism in relation to the identification of a cure or at least for useful treatments for HD.⁴⁶ However, for the present it is only symptoms that are treated.

A number of such symptoms are psychiatric, with depression and anxiety being common, as well as (less often) irritability and aggression.⁴⁷ Obsessions are encountered and suicide is the cause of death for those with HD in about 5% of cases – it is about four times more common in HD patients than in the general population.⁴⁸

Treatments for HD without any evidence base have been promoted by the unscrupulous, resulting, for instance, in *Commissioner for Fair Trading, Department of Commerce v Perrett* [2007] NSWSC 1130, in determinations that spurious representations about substances distributed to address the symptoms of HD were misleading and deceptive.

IMPOSITION OF INVOLUNTARY STATUS

HD is not itself a mental illness but, as noted above, it gives rise first to impaired executive functions and then to dementia, the symptoms of which constitute significant disturbances to thought,⁴⁹ capacity for rational thought⁵⁰ or thought form⁵¹ or stream of thought⁵² and memory,⁵³ often involving limited

⁴⁶ Society for Neuroscience, *Huntington's Disease*, <u>http://www.sfn.org/index.cfm?pagename=brainBriefings_huntingtons_</u> <u>disease</u> viewed 12 June 2010.

⁴⁷ Wild and Tabrizi, n 31 at 362-363.

⁴⁸ Wild and Tabrizi, n 31 at 363.

⁴⁹ Mental Health and Related Services Act (NT), s 6(1); Mental Health Act 2000 (Qld), s 12(1); Mental Health Act 1986 (Vic), s 8(1); Mental Health Act 1996 (WA), s 6(1).

⁵⁰ Mental Health Act 1996 (Tas), s 4.

⁵¹ See Mental Health (Treatment and Care) Act 1994 (ACT), Dictionary; Mental Health Act 2007 (NSW), s 4.

⁵² See Mental Health (Treatment and Care) Act 1994 (ACT), Dictionary; Mental Health Act 2007 (NSW), s 4.

53 See Mental Health Act 1986 (Vic), s 8(1); Mental Health Act 2000 (Qld), s 12; Mental Health Act 1996 (WA), s 4.



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⁴¹ Quarrell, n 32, p 5.

⁴² Revilla and Grutzendler, n 29.

⁴³ Huntingtons Australia, <u>http://www.huntingtonsaustralia.asn.au</u> viewed 17 July 2010.

⁴⁴ Howard Florey Institute, *Huntington's Disease*, <u>http://www.florey.edu.au/the-brain/brain-disorders/huntingtons-disease</u> viewed 5 July 2010.

⁴⁵ Huntington's Outreach Project for Education at Stanford, *Treatment vs Cure*, <u>http://www.hopes.stanford.edu/treatmts/</u> <u>findcure/g0.html</u> viewed 5 July 2010.

capacity to consent to needed treatment, and posing a risk to the health and safety of the person and, sometimes, to others. This means that the criteria for involuntariness either as an inpatient or in the community may be satisfied.

For clinicians and mental health review tribunals determining whether a person should be made involuntary, HD gives rise to a number of challenges. These range from clear identification of the disorder to overlap in its symptomatology with other conditions.

In *Re B* 2009 Can LII 47221 (ONCCB), for instance, B was a 52-year-old woman who had appeared before the Ontario Consent and Capacity Board on six occasions since 2008. Her HD was "declining" and she had lost her capacity to care for herself and had been found incapable to consent to long-term care. A neurologist stated in a report:

Disturbances of cognition are characterized by memory loss, anosognosia (lack of self awareness), loss of cognitive speed and poor judgment. Difficulties are apparent in the person's ability to plan, organize, prioritize and initiate. Because these executive functions are no longer intact, the person with HD loses the ability to problem-solve and to judge situations appropriately. Normal day-to-day activity also becomes difficult because, quite quickly after the onset of symptoms, the ability to think sequentially becomes impaired.

However, the board found there to be "no objective or even substantive evidence of cognitive decline, or in fact mental disorder" in B. As the onus was not satisfied by the attending physician to persuade the board of the conditions for involuntary status, the board declined to make such an order.

By contrast, in *Re LM* 2007 CanLII 20015 (ONCCB) LM, a 56-year-old homeless male, had been diagnosed with HD 10 years previously. A psychiatrist gave evidence that LM suffered a disorder secondary to HD or possibly dementia secondary to HD. He was irritable, aggressive, paranoid, on occasions threatening, and displaying cognitive deficits. LM was described as having no insight into his illness or its impact upon him and had repeatedly stated that he was not prepared to take medications prescribed for his mental state. Psychiatric evidence before the board was that LM lacked the ability to process information relevant to his making decisions about his condition and was unable to appreciate the reasonably foreseeable consequences of his decision-making. The board concluded that he lacked the capacity to consent to anti-psychotic medication and to benzodiazepines that he clinically required.

An issue that arises in a number of different contexts in relation to HD is the toll taken by the uncertainty for persons as to whether they will inherit the disease. Deciding whether to have the genetic test to determine whether HD has been inherited can be extremely emotionally fraught. In *Re S* 2005 CanLII 56704 (ONCCB), S was a 23-year-old woman who had been sexually abused by her father and whose mother was murdered by another psychiatric patient when she was 12 years old. She was pregnant and had significant alcohol dependency problems. Her mother and her mother's twin sister had HD but S had declined to ascertain whether she had inherited the condition. She was diagnosed as having paranoid schizophrenia characterised by a range of delusions. The interplay between her self-destructive lifestyle and her anxieties relating to inheriting HD was unclear. Ultimately, the board confirmed her involuntary status.

GUARDIANSHIP HEARINGS

The most common scenario in which those with HD come into contact with the legal system is when an application is made for a guardian and/or an administrator to be appointed for them. In *Re FH* [2005] QGAAT 42, for instance, an application was made by a case manager to the Queensland Guardianship and Administration Tribunal for appointment of the Public Trustee of Queensland as administrator over the financial affairs of a 65-year-old man, FH, who had HD.⁵⁴ He had been admitted shortly previously to hospital after being found paranoid, living in premises that were in disarray and unhygienic – they had also been barricaded by him against entry by others. FH had a history of alcohol dependence and of setting fires while intoxicated. He had declined home help

⁵⁴ See too *Ps and Ss* [2005] WASAT 231; *Re WSF* [2006] QGAAT 79; *Re HVM* [2006] QGAAT 37; *Re WAB* [2007] QGAAT 12; *Re PAC* [2007] QGAAT 54; *Re WSF* [2010] QCAT 12.

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services. On admission to hospital, FH was noted to have cognitive deficits, particularly with regard to orientation and short-term memory; the deficits were consistent with a mild to moderate dementia. He was found to have HD.

FH was described by clinical staff as insightless into his illness and guarded, suspicious and distrustful. Occupational therapy, psychology and social work assessments all recommended supported accommodation for FH. A speech therapy assessment noted early disturbance of his swallow, a risk factor for his developing aspiration pneumonia. A psychiatrist expressed the view that FH lacked the capacity to make informed decisions regarding personal, health and financial matters. FH was adamant he did not have HD. A neurocognitive assessment determined that FH demonstrated significant deficits in visuospatial abilities and that he had widespread deficits including frontal network functions such as planning, problem-solving, generation of response alternatives, and the ability to evaluate and modify behaviours: "his adaptive type behaviours were assessed as being so poor that he will experience difficulty in day to day functioning including well-learned tasks" (at [21]). Already he had involuntary movements, restlessness, gait disturbance, poor coordination and balance problems.

The tribunal found that FH was not able to make complex financial decisions and could not understand the nature and effects of decisions about his financial matters; as well as that, he had impaired concentration. This meant that in relevant respects he lacked capacity to make financial decisions,⁵⁵ so it appointed the Public Trustee of Queensland to make financial decisions for FH.

Similarly, in *Re AB* [2008] WASAT 25 the Western Australian State Administrative Tribunal (WASAT) was asked by a social worker from an adult mental health centre to appoint the Public Advocate as limited guardian for a 64-year-old woman with HD who was resident in a psychiatric hostel and in a relationship with another hostel resident who informed the WASAT that he and AB intended to marry. She and her partner proposed to leave the hostel and live in a flat which the partner owned. They proposed to receive assistance from a weekly cleaner and said that they would receive family support. They also expressed the wish to travel to visit the family of the partner in the Eastern states of Australia and possibly to settle there if the partner obtained employment.

However, the expert evidence was that AB was experiencing significant and increasing difficulties in personal care due to HD and lacked insight into her escalating needs. An Aged Care Assessment Team report noted that AB was displaying short-term memory problems, occasional "at risk" and aggressive behaviours, disturbed sleep, confusion and disorientation. He had been noticed choking on food and required speech therapy assessment. The WASAT concluded that the lack of insight on the part of AB into the effects of her illness potentially placed her at risk "because she requires care and supervision of her medication, her personal hygiene and of her eating because of the risk of choking but does not understand this to be the case" (at [32]). It concluded that AB was unable to make a judgment about her person and because of her disability was in need of oversight, care and control in relation to her health and safety (at [36]). It found that, in spite of AB's expressed wishes and those of her partner, it was in AB's best interests that a guardian be appointed to make decisions in relation to her accommodation, care needs and her travel plans. It appointed the Public Advocate for this function for five years.

FAMILY LAW

In a variety of circumstances HD can impact upon fitness to parent issues⁵⁶ and upon spousal maintenance and division of asset issues. In S(ER) v S(HC) 1998 CanLII 4619 (BCSC) an application was brought by a husband for division of family assets, spousal maintenance and custody.⁵⁷ The wife had HD. Evidence was placed before Levine J from documentation produced by the Huntington Society of Canada, among other things stating (at [8]):

Adult-onset HD can be roughly divided into three stages. Early in the disease, manifestations include subtle changes in coordination, perhaps some minor involuntary movements, difficulty thinking through

⁵⁵ Under Sch 4 of the Guardianship and Administration Act 2000 (Qld), he lacked capacity.

⁵⁷ See too Jenkins v Repatriation Commission (1999) 56 ALD 379; [1999] FCA 269.



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 $^{^{56}}$ See eg the arguments raised in KM v GEM 2005 ABQB 651.

problems, and, often, a depressed or irritable mood. In the middle stage, chorea may become prominent, and difficulty with voluntary motor activities will be more evident with worsening dysarthria and dysphagia. As cognitive deficits increase, the patient will be unable to hold a job or carry out most household responsibilities. Patients with late-stage disease may have severe chorea, but are more often rigid and bradykinetic. They are largely nonverbal and bedridden, with a more global dementia, although retaining a significant degree of comprehension. Psychiatric disorders may appear at any time during the course of the disease, even years before motor symptoms develop.

Evidence before the Supreme Court of British Columbia was to the effect that the wife was in the middle stages of HD. The husband had custody of their daughter. The division of the spousal assets took account of the fact that he was caring for their child and that the wife required spousal support to pay for her care and living expenses. However, Levine J noted (at [25]): "The difficulty is to anticipate her expenses, as they will depend on the progress of her illness." The evidence before the court (at [26]) was that her accommodation was too small, that she needed more care and that she required a companion "to take her on outings, but also for homemaking and personal care. Ultimately, she will require care in a long-term care facility. She is on a waiting list for a government-subsidized room in such a facility." Taking such factors into account, Levine J ordered a division of assets and spousal maintenance and ordered that the husband's obligation to pay spousal support to his wife, should he die before her, would become the obligation of his estate until she passed away.

CRIMINAL LAW

HD has the potential to affect both criminal responsibility and criminal culpability in significant ways. However, expert evidence that clearly addresses the relevance of the disorder to either criminal responsibility or criminal culpability is necessary.⁵⁸

In R v Norman [2009] EWCA Crim 1810; [2009] Crim LR 346 the appellant, a 50-year-old man, was arrested and charged with child abduction for detaining a child, CM, to keep him out of the lawful control of the child's mother. Norman had been diagnosed in 2003 as suffering from HD. The disease had had a progressive impact upon him. He was examined by three psychiatrists and a clinical neuro-psychologist, all of whom concluded that he was not fit to plead and that there was no prospect that he could become so in light of his illness. Evidence before the trial judge established that Norman had been subject to "unpleasant treatment" by his fellow prisoners while on remand. In due course he was removed from prison and made the subject of a hospital order after having been found to have committed the offences with which he was charged. However, a crucial aspect to this finding related to the interpretation of a conversation with the child's mother. The Court of Appeal concluded that the jury should have been permitted to hear evidence about the effects of HD on the intentions of the appellant and what he meant in the course of that conversation. This led to the finding of guilt against him being quashed.

In R v Baird [2002] EWCA 737 the appellant pleaded guilty to causing death by dangerous driving. He contended that at the time of the offence he was suffering depression as a result of his father having been diagnosed with HD and his concern that he too may be affected by the disease. A psychiatric report suggested that his depression led to a lack of concentration in an otherwise meticulous person and possibly a state of dissociation. A pre-sentence report recommended that sentencing be deferred pending his being tested for HD. However, the sentencing proceeded and he received a sentence of two years' detention in a young offender institution. The Court of Appeal substituted a term of 18 months' detention.

In *R v Larmour* [2004] NICC 4 the effects of HD were considered in a sentencing context.⁵⁹ At first instance Larmour was sentenced to two terms of life imprisonment for murders he committed in 1987. In 2004 Kerr LCJ and Nicholson LJ heard submissions on the tariff to be set under Art 11 of the *Life Sentences (NI) Order 2001* to represent the appropriate sentence for retribution and deterrence before the Life Sentence Review Commissioners could assess his suitability for release on the basis of risk. The killings that he committed were in the company of a loyalist gang associated with the Ulster

⁵⁹ Compare R v Abbotts [1997] EWCA Crim 959.

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⁵⁸ Compare *R v Belcourt* 2000 BCAA 441 (CanLII).

Freedom Fighters and were premeditated and brutal. However, by 2001 he had been diagnosed as suffering from HD. He was already in need of full-time care "and will become increasingly disabled" (at [12]). The Crown accepted that his HD could be taken into account in fixing the tariff in his case. It, along with his youth when he committed the offences, was factored into the decision, resulting in a 15-year tariff being set.

In R v Jans [2000] NSWSC 525 the prisoner pleaded guilty to the manslaughter of his wife whom he stabbed to death with a knife. The evidence was that the prisoner and his wife enjoyed a satisfactory married life of apparently mutual devotion until she commenced to suffer HD. A significant symptom for her was paranoia, characterised by her making multiple accusations against him of infidelity, bisexuality, sexual abuse, trauma and manipulation and by her engaging in multiple acts of physical violence against him. She announced that she intended to seek a divorce from him. Unfortunately, for a considerable period of time she was not diagnosed with HD, in part because of her symptoms of breast cancer which resulted in a mastectomy. Grove J accepted that the wife's mental state was "a contributing factor in the development of depression in the prisoner. At the time of the killing he was undergoing a major depressive episode" (at [13]). He concluded that the abnormality of mind that the prisoner suffered was of such a scale and extent as to markedly reduce his level of culpability, resulting in Grove J taking the unusual step of sentencing him only to a four-year good behaviour bond for the killing of his wife.

PROBATE

The capacity of a person with HD to make a will – to have testamentary capacity – can be complex.⁶⁰ In addition, there can be difficult issues in relation to the needs of a person with HD after the death of a spouse or partner. In *Gully v Dix* [2004] 1 WLR 1399 Mrs Gully had left her husband to live with the deceased in 1974 but they never married although they lived in a bona fide marriage-like relationship. However, the deceased became addicted to alcohol which led to multiple separations between the parties and an attempt on his part to commit suicide, after which Mrs Gully looked after him in conditions that were increasingly squalid. Her difficulties were compounded by her being diagnosed in 1991 with HD. In 2001 he separated from Mrs Gully after he attacked her in the night with a knife. The separation was short-lived as he was found dead in his garden some three months later.

This led to an inquiry into whether, at the time of his death, the deceased was living in his household with Mrs Gully for the purposes on the *Inheritance (Provision for Family and Dependents)* Act 1975 (UK). The Court of Appeal found (at [24]) that "the steadfastness of a commitment to live together may wax and wane, but so long as it is not extinguished, it survives". This permitted the claim by Mrs Gully. What was not canvassed was the extent of Mrs Gully's needs in light of her having becoming symptomatic with HD.

COMPENSATION

Whether a person previous to a tortious event had or later developed HD^{61} can impact upon damages awarded, given that while the tortfeasor takes their victim as they find them, they are also generally only responsible for the harm that they cause.

In *Kakoschke v Draper* [2006] QSC 386 the plaintiff, a woman of 43, suffered injuries in a motor vehicle collision. The physical sequelae from the collision were minor and the basis for the plaintiff's economic loss claim was a claim that she suffered from a psychiatric illness brought on by the collision. A complicating factor was that she had been sexually abused as a child by her grandfather. She also carried the gene for HD and was either symptomatic or in due course would become so. Psychiatric opinions about her condition varied. One expert expressed the view that the plaintiff had

⁶⁰ See In Estate of Stefan Miegoch No 119295/93 Wills – Solicitors [1996] NSWSC 68.

 61 See eg *Hefferland v Fink* 1995 CanLII 2047 (BC SC) where the plaintiff in an action for damages arising from a motor car accident had HD and the court found (at [30]) that "The accident has compounded and aggravated the problems he had with Huntington's Disease. His walking is now extremely slow and awkward. Now his speech is such that it is difficult to comprehend him. He is now unable to swim. He now has no sexual relations and doesn't expect he will in the future."



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PTSD. Another said she did not. One psychiatrist opined that her knowledge that she was carrying the HD gene was causing depression, including at the time of the accident. A neurologist expressed the view that she was showing mild choreiform movements and that "her recent development of mood changes, depression and anxiety, probably represents the early stages of Huntington's disease" (at [36]). Skoien AJ did not accept this latter view on the basis of there not being sufficient evidence to support it.

The plaintiff was assessed by Queensland Health as a person able to cope with learning that she was a carrier of the gene after her family learned that her father had HD. In 2003 she learned that she too carried the gene and had a "repeat count" of 43, much the same as the count of 42 of her father with diagnosed HD. Skoien AJ commented (at [32]): "The 'repeat count' indicates the abnormality which, if it exceeds 37 means that the carrier will contract the disease. Her increased repeat count is minor, considering it may be measured in the 80's or higher."

Ultimately Skoien AJ accepted that the only cause of her serious psychiatric disability was the motor vehicle accident.⁶² However, Skoien AJ also needed to consider its relevance in terms of being a supervening factor in her ability to return to work. He noted (at [30]) that the early manifestations of HD "may include difficulty in co-ordination, involuntary movements, difficulty in planning and often a depressed or irritable mood". He noted that the plaintiff's father suffered the onset of HD to a noticeable degree probably when he was in his mid-60s. He concluded that it was likely that the plaintiff was likely to contract HD at a similar stage to that of her father or perhaps a little earlier, given that she already had a slightly increased repeat count.

CONCLUSIONS

Huntington's disease is a profoundly confronting condition. It is conventionally said that, once its symptoms manifest, it is relentlessly progressive. It generally leads to death in under two decades, after considerable suffering. It affects both the mind and the body in inexorable and devastating ways. Awareness of the potential to have inherited it, and/or passed it on to one's offspring, can be extraordinarily distressing. As the International Huntington Association puts it, "HD is a family disease because of its impact on every family member".⁶³ Finding perspective in relation to HD is difficult; not everyone reaches the point of Barema: "It all depends on your angle, your take on life. Because eventually, we are all going to come apart. We'll all go through it. Yes, we've all got some Huntington's in us."⁶⁴

In the legal context, HD has multiple repercussions of which it is constructive for those with the condition, those at risk of the condition, and those caring for those with the condition, to be conscious. It can result in unfitness to give evidence,⁶⁵ unfitness to make decisions as a litigant and unfitness to stand trial. A consequence of HD symptomatology at some point is increasingly impaired capacity in multiple other areas as well, including capacity to make decisions in the context of involuntary inpatient or outpatient status, the need for a guardian or administrator, and capacity to exercise testamentary capacity. Consciousness of this emphasises the need for encouragement by medical and legal personnel toward forward planning, while it is still possible, for those with HD so that as much autonomy as possible can be exercised by advance directives, powers of attorney and other means of communication on the part of the person with the disease. In this way, they can take as much control for as long as possible in relation to their lives and the management of their condition.

HD can eliminate criminal responsibility and reduce criminal culpability, but this depends on the extent of symptomatology at the time of commission of the offence and the time of sentencing. Courts are dependent in this regard upon well-focused and argued expert reports (and oral testimony) from health practitioners, often those who are treating the accused person.

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⁶² See the similar issue which arose in a different compensation context in Hales v Repatriation Commission [2003] AATA 530.

⁶³ International Huntington Association, <u>http://www.huntington-assoc.com</u> viewed 6 July 2010.

⁶⁴ Barema, n 2, p 160.

⁶⁵ Caddy v Mcinnes (1995) 58 FCR 570; see too Langridge v Nifort 1991 CanLII 4477 (NSSC).

The special needs of a person with HD can impact upon their entitlement to damages arising out of a tortious incident and can increase their need for special provision under spousal maintenance obligations in family law, and their own ability to pay child support or spousal maintenance. They can also be relevant in the context of a testator's family maintenance claims.

The decided cases reveal the difficulty of identifying with clarity the status of a person's HD symptomatology at any given time, especially when alternative aetiologies exist for symptoms experienced by a patient, including when the symptoms are principally affective.

An issue which has arisen in the course of litigation has been the impact of a person experiencing stress in relation to HD – whether it is as to whether they have HD or whether they may have transmitted it to a child or grandchild. Many persons do not wish definitive diagnosis but ongoing potential to be HD-positive can wreak its own damage and can be very difficult to live with. This can result in lifestyle decisions that are reckless and risky⁶⁶ or that are the product of denial. In addition, living with a person with HD can be exceptionally problematic because of the risk of paranoid, violent and disordered ideation at some stage on the part of the person with HD. This in itself can be psychogenic and corrosive on the wellbeing of loved ones; the disorder has secondary victims too. Even survivor guilt for those who do not inherit HD can have its own difficulties.⁶⁷ However, there are elements to the HD experience that have little to do with the law but from which lawyers and clinicians alike can learn. After all, a person is not their disease; we are more than our genes. There are perspectives other than the wholly negative in relation to HD. As a wife of a middle-aged man with HD⁶⁸ has said, after emphasising the grim realities of HD:

Being able to live in the moment is Huntington's greatest gift. ... After you've finished grieving for your lost future (which can take years), you realize that the imagined future you are grieving for was never real anyway. Huntington's shocks you into an appreciation that all we have, really, is "now". Your plans, even your subconscious ones, aren't your life. Moments are your life.

Ian Freckelton SC

66 See eg Re S 2005 CanLII 56704 (ON CCB).

⁶⁷ See Stauffer J, "Dealing with Survivor Guilt" (2010) 109 *Huntington's News* 3, <u>http://www.huntingtons.org.nz/news_109.pdf</u> viewed 20 July 2010.

68 See Sulaiman, n 10, pp 132-133.



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