Review of Current Research for Disease Modifying Treatments for, and Symptom Management of Multiple Sclerosis

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1. Introduction

The purpose of this report is to review the current research supporting the use of disease modifying treatments (DMTs) in multiple sclerosis (MS), and to assess whether the access that is currently provided to New Zealanders with MS, is evidence based and in line with international standards. The report addresses the early treatment of MS with DMTs and the current stopping criteria for these treatments. The use of cannabis based medicine for people with MS is also examined along with the current requirement for the patient to self-fund this therapeutic option.
2. Disease modifying treatments

2.1 Early Intervention

It is well established that the Costs of Illness (COI) for MS increase as the level of disability increases. While the costs at the earlier stages of MS are largely represented by medications, they are far outweighed by indirect costs in later stages, mainly due to relapses and productivity losses. Considering these increased costs that are associated with relapse and increasing disability, pharmaceutical interventions which delay the progression of disease are essential not only to improve the quality of life for the person with MS, but also to reduce the economic burden. Between 50% and 80% of patients are unemployed 10 years after the onset of the disease, with only 15% due to physical restrictions. Not only is it important to invest in DMTs for people with MS very early in the disease progression, but it has also been reported that the higher the cost of the DMT used in the initial stages of the disease, the slower the disease progression.

The current entry criteria for all disease modifying treatments (DMTs) funded by Pharmac require that a patient must have Clinically Definite Relapsing Remitting MS, at least 1 significant relapse in 12 months (or 2 in 24 months) and evidence of new inflammatory activity on MRI scan within the past 24 months. This is not only in conflict with international best practice, the 2010 McDonald criteria and the current published evidence, but also the needs of the patient. The widely accepted 2010 McDonald criteria for MS diagnosis allow a diagnosis to be made on some patients with only a single MRI and require that a relapse or attack last only at least 24 hours. Patients diagnosed with MS by this criteria are not able to be treated in New Zealand, despite having active, ongoing disease, due to the Pharmac entry criteria. Regardless of the criteria used, once Multiple Sclerosis or Clinically Isolated Syndrome (CIS) is diagnosed, damage has already occurred in the central nervous system. Although not always clinically evident, neurodegeneration has been proven to occur early in the disease course, even before the subtlest of symptoms are present. This damage is not only within active focal lesions, but also in chronic silent plaques, axons and normal-appearing white and grey matter. All of these processes occur early, silently, and to contribute to ongoing disease. In order to reduce the impact of this damage, early treatment of MS is crucial.

2.2 Clinically Isolated Syndrome and Early Multiple Sclerosis

Clinically Isolated Syndrome can be considered as the earliest manifestation of MS. It is defined as an acute or subacute first demyelinating event caused by either inflammation or demyelination in the central nervous system (CNS). The acute attack, which lasts for at least 24 hours, is classified according to its localisation in the CNS as cerebral, optic nerve, brainstem or spinal cord. CIS does not always develop into MS, and therefore does not always warrant treatment. However, when it occurs in conjunction with lesions on an initial MRI, this combination is highly predictive of developing further inflammation and future definite MS within ten years. A 10 year follow-up study published in 1998 showed that 83% of patients with CIS and an initial abnormal MRI, progressed to develop clinically definite MS.

There have been several large, noteworthy, clinical trials done as early as 2000, which looked at early treatment of CIS patients with abnormal MRI scans. These have all showed that the earlier the treatment of CIS, the more effective it is in delaying conversion to clinically definite MS. The earliest study was the CHAMPS study, which took place 17 years ago. Patients diagnosed with CIS were randomly allocated to receive either Interferon-β or placebo, to test whether early treatment could
prevent those with CIS developing clinically definite MS. The results were so overwhelmingly in favour of treatment, that the study was stopped early as it became unethical to deprive the patients on placebo of meaningful treatment. Other studies have replicated and built upon these results including the TOPIC study. This was a randomised, double-blind, placebo-controlled, phase 3 trial where CIS patients were given either once-daily oral teriflunomide 14 mg, teriflunomide 7 mg, or placebo, for up to 108 weeks. Compared to placebo, teriflunomide at both doses significantly reduced the risk of conversion to clinically definite MS (hazard ratio of 0·574 at the 14 mg dose and 0·628 at the 7mg dose) and reduced the number of new MRI lesions (hazard ratio of 0·651 at 14mg and 0·686 at 7mg)\(^{17}\). More recently, Tintore et al \(^{18}\) showed that the three most predictive factors of further relapses in CIS are; the baseline lesion load, development of new lesions during the first year, and not starting a DMT before a second attack. Based on the evidence from the aforementioned studies, the US FDA has currently approved Avonex, Betaseron, Extavia, and Copaxone for use in CIS.

The current entry criteria for DMTs for treatment of MS in New Zealand need to be revised immediately to allow those with CIS or early signs of MS to be treated early. Patients with clearly active disease are currently disallowed beneficial disease modifying treatments. Treating patients with early MS would minimise further inflammation and axonal damage in their nervous system, thereby reducing the number of patients who develop Clinically Definite MS and in those who do, slowing the disease progression and delaying disability, fatigue and cognitive issues. Clearly, not all patients with CIS need treatment. However, the decision to treat CIS or early MS should be at the discretion of the treating neurologist, on a case by case basis, taking into account the clinical presentation, MRI data and circumstances of the patient.

2.3 Stopping Criteria

2.3.1 Disease progression

The current stopping criteria for Dimethyl Fumerate, Fingolimod, Natalizumab, Teriflunomide, the Interferons and Glatiramer Acetate all allow for a small amount of progression on the expanded disability status scale (EDSS) – but only at the lower end of the scale. Once a patient reaches EDSS 3.5 only a one point EDSS change, (or a 0.5 change at 4.0) will result in the patient reaching the stopping criteria for all DMTs. And no DMTs are funded beyond EDSS 4.5. An Australian paper was published last year by Lizak et al., using patient data from the global MSBase registry cohort \(^{19}\). This examined patients with a baseline EDSS of 3, 4 or 6 on highly effective immunomodulatory therapies (defined as; natalizumab and fingolimod, alemtuzumab, dimethyl fumarate, cladribine, rituximab and mitoxantrone) versus lower efficacy therapies (interferon β preparations, glatiramer acetate, and teriflunomide) and compared the time taken to reach either an outcome EDSS of 4, 6, or 6.5, respectively. Data from a total of 4,295 patients were included in the analysis. It was found that lower relapse rates and greater time on higher efficacy immunomodulatory therapy after reaching EDSS steps 3, 4 and 6 are associated with a decreased risk of accumulating further disability. Highly effective immunomodulatory therapy ameliorates accumulation of disability in moderately advanced and advanced relapse-onset MS. Additionally, Lizak et al. found that disability trajectory in moderately advanced and advanced disease is independent of earlier disease characteristics. This means that regardless of the person with MS’s previous disability trajectory, relapse activity or previous exposure to DMTs, once they reach a...
moderately advanced stage (defined as EDSS 3.0 by Lizak et al.), treatment with one of the
aforementioned highly effective DMTs reduces the risk of accumulating further disability.

Unfortunately, a person with MS living in New Zealand is severely disadvantaged compared to their
Australian, European and American counterparts. Not only are they unable to access this complete
range of treatments, but if persons from these same EDSS epochs progress along the EDSS scale (3.0 –
4.5, 4.0 – 4.5) they will no longer have access to drugs which have been proven to prevent them from
accumulating further disability.

The MS Society of NZ has obtained data from Roche Pharmaceuticals from their OPERA/RMS
randomised controlled trials, which breaks down the results data for ocrelizumab in MS patients by
EDSS. In this study patients are randomised to receive blinded treatment with either ocrelizumab or
interferon beta-1a as an active comparator. Patients in the trial had an EDSS of 0 to 5.5 at screening
and this new data analyses the reported outcomes by EDSS in two groups: <4 or ≥4. The outcomes
examined were time to confirmed disability progression (CDP) for greater than 12 weeks and greater
than 24 weeks, annual relapse rate and number of MRI lesions. For all of these outcomes, there is no
difference in the results between the two groups. Patients on ocrelizumab with an EDSS of <4 at
screening and those with an EDSS ≥4, all showed lower rates of disease activity and progression than
interferon beta-1a over a period of 96 weeks. These results add to the ever-growing evidence that
DMTs are not only effective in the early stages of MS, but also in moderate to severe RRMS.

The stopping criteria for the DMTs available in New Zealand need to change to reflect this new
knowledge about the benefits of such treatments beyond the scope for which they are prescribed in
New Zealand. It is now clear that all people with RRMS should be able to access highly effective DMTs at
least up to EDSS 6.5, and to require discontinuation prior to EDSS 6.5 is detrimental to the patient and
contradictory to current published evidence.

### 2.3.2 Fatigue and cognition in MS

All types of MS can greatly affect cognitive function, mood, fatigue and quality of life. Many studies
have illustrated that cognitive dysfunction contributes significantly to disability status and fatigue is
known to affect 80% of people with MS. Fatigue is a multifactorial symptom with both physiological
and psychological causes. Because it is more difficult to quantify than relapse rate or number of MRI
visible lesions, it is often ignored in the outcome measures of clinical trials sponsored by pharmaceutical
companies. However, subsequent studies have shown that DMTs are effective at treating fatigue in MS.

Treatment with Interferon β in the BENEFIT clinical trial had a beneficial effect on cognition. This
improvement became more pronounced over time, as shown in the five year data from the study. In
the TYNERGY clinical trial, natalizumab was proven to have improved fatigue after 12 months of
treatment. Both the physical and the mental component of fatigue improved and the average
improvement corresponded to a reduction from severe to moderate fatigue (measurements based on
the Fatigue Scale for Motor and Cognitive functions (FSMC)).

Under the current guidelines, patients experiencing benefits of a DMT on their non-physical symptoms,
such as fatigue and cognition, can be a position where the DMT is no longer funded for them as they
have progressed to the physical-based EDSS stopping criteria. A change to the stopping criteria is
necessary to best serve the needs of the person with MS.
Eventually, the development of biomarker testing to ascertain therapeutic response may supersede the use of disability scales\textsuperscript{28}, in the meantime, there are other disability scales available which may be more suitable than the EDSS.

2.3.3 Expanded Disability Status Scale

Currently a disability score on the Expanded Disability Status Scale\textsuperscript{29} is used as part of the entry and stopping criteria for prescribing DMTs to people with MS. Although it is widely used to measure status and progression, mostly in clinical research, it has well documented limitations which make it unsuitable as a criterion for which to prescribe medications. Firstly, it has high inter- and intra-rater variability\textsuperscript{30–33} which can result in up to a 1-step change (on a 10-step scale) depending on the rater. This can be attributed to the subjective nature of a neurological examination, but also to the complex and ambiguous rules for scoring the functional systems (FS).

Although it may appear so, the scale is not linear in practice. The rate of disease progression and disability status is dependent on the baseline score. Amongst a cross-sectional population of MS patients, the middle scores are moved through more quickly than the initial or final, and the majority of patients fall within two areas of the scale; 1.0-3.0 and 6.0-7.0\textsuperscript{34}. This means that stopping criteria for DMTs may be reached sooner if the person with MS has a higher baseline score, or than if an alternative disability scale was used. Clinical phenotypes are also unevenly distributed along the scale. This is largely due to the different types of MS exhibiting different predominant symptoms. For example, the symptoms of primary and secondary progressive (PPMS and SPMS) MS largely cause ambulatory dysfunction, and therefore PPMS and SPMS patients rate higher on the scale. Whereas with relapsing remitting MS (RRMS), the predominant symptoms are fatigue, numbness, spasticity and disturbed vision and so despite significant disability, may rate lower on the scale. However, none of these areas are directly assessed by the EDSS. This results in a skewed perspective of the level of disability of the patient, the progression of the MS and, importantly, the medication needs of the patient. Cognitive Function is one of the most significant disabilities restricting people to both work and social engagement and is poorly represented when measuring with EDSS.

2.3.4 Multiple Sclerosis Functional Composite (MSFC)

The MSFC was created by a Task Force appointed by the American National Multiple Sclerosis Society's Advisory Committee on Clinical Trials of New Agents in Multiple Sclerosis\textsuperscript{35}. The aim was to develop a scale which would improve on the disability scoring of the EDSS and aid in clinical assessment of MS\textsuperscript{36}. The MSFC consists of three objective quantitative tests of neurological function including cognitive (Paced Auditory Serial Addition Test), dexterity (Nine-Hole Peg Test), ambulatory (Timed 25-Foot Walk Test) and visual function. It has been shown to have excellent test-retest reliability and to be more sensitive to change than the EDSS\textsuperscript{37,38}. Although the MSFC improves on the EDSS with the addition of a test of cognition, it still lacks a measure of fatigue. MSFC is also time consuming, requires training and patients often dislike this measurement scale meaning it is rarely used outside of clinical trials.

2.3.5 Multiple Sclerosis Impact Profile

The MSIP is a self-report scale which assesses physical and psychological symptoms and covers a broad range of clinically relevant aspects of MS in 11 domains\textsuperscript{39}. It is a reliable and valid outcome measure based on the International Classification of Functioning, Disability and Health (ICF). This measure has been extensively tested, including in a five year longitudinal study where it was used to examine the course of a broad spectrum of MS-related disabilities and quality of life in relation to disease severity\textsuperscript{40}. 

June 2017 – Report compiled for Multiple Sclerosis New Zealand by Kerry Walker
2.3.6 Performance Scales
The Performance Scales was created in 1993 to assess multi-dimensional disability and to consider more relevant domains of MS-specific disability. It is a self-report measure specifically for MS-associated disability, assessing mobility, bowel/bladder, fatigue, sensory, vision, cognition, spasticity and hand function. The measure was validated in a multi-site, cross sectional study involving 274 MS patients and 296 healthy controls in 13 MS centres in the United States and Canada. And a recent review showed that 82 studies have used the tool in empirical research so far and that it is a highly sensitive tool with high test-retest reliability. If the Performance Scales were substituted for the EDSS in the stopping criteria for DMTs, this would allow patients who are experiencing real benefits from the medication, but still progressing physically to remain on treatment.

2.3.7 Removing the disability measure from the stopping criteria
The argument can be made that including any disability measure as a stopping criteria for DMTs for MS does a disservice to the patient. As MS symptoms and disease progression varies greatly from person to person, the decision about stopping medication should also be on a case-by-case basis. Like the decision of what DMT is best suited, or when to switch to a different DMT, the decision to stop treatment should be made between the person with MS and their neurologist. Best practice guidelines created by Association of British Neurologists advise the recognition of “the central importance of patient choice”. These guidelines also emphasise that it is not feasible to have a mandatory stopping criteria that apply in all cases.

2.0 Cannabinoids and Multiple Sclerosis
2.1 Cost to access
The cannabis based medicine, Nabiximols (trade name Sativex) has been approved for treatment of spasticity in MS by MedSafe New Zealand. However, because it is not funded by Pharmac, a prescription through a District Health Board can cost the user $1200 per month, or $1500 per month if ordered through a chemist. Information obtained under the Official Information Act by MS NZ from Pharmac shows that although five people with MS have applied for funded access to Sativex, no one has to date been approved to receive this. Although these applications were not declined, the applications were classified as “not progressed” and the same result is achieved. In the year ended June 2015, Statistics NZ reported that the average household income was $93,880 and average weekly housing costs were $295.40. A prescription medication that costs more than a person’s rent or mortgage is simply not an option. Particularly for a person with severe spasticity, who is also therefore unlikely to be able to work full time. This would be somewhat detrimental to the person with MS if Sativex was simply one of the treatment options for spasticity, however, this is not the case. Sativex is approved for use in persons with severe spasticity who have not responded to any other anti-spasticity medication and who show a clinically significant improvement in spasticity-related symptoms during an initial trial of therapy. That is, people for whom there is no choice in treatment and who are experiencing real symptom relief from Sativex. Speaking to the media in November 2016, Pharmac stated Sativex was not yet funded as “the Pharmacology and Therapeutics Advisory Committee had advised there was not yet sufficient evidence that Sativex was effective.”
2.2 Therapeutic benefits

2.2.1 Spasticity

The therapeutic benefits of cannabinoids have been intensively investigated in MS. Of the symptoms most studied, spasticity is at the forefront. As early as 2007 it was becoming evident that Sativex was an effective treatment for spasticity. A randomised controlled trial which allocated patients to either a cannabis based medicine containing delta-9 tetrahydrocannabinol (THC) and cannabidiol (CBD) or placebo, showed that those on the active medication showed a $\geq 30\%$ reduction in spasticity over the study, a statistically significant result compared to the placebo group ($p=0.014$). In 2010, a meta-analysis was completed of all the then-published randomised control trial evidence of the efficacy and safety of Sativex, on spasticity in people with MS. In this review, patients from all studies achieving a greater than or equal to 30% improvement from their baseline spasticity score, were defined as 'responders'. The results from 666 MS patients with spasticity were included and a statistically significant greater proportion of treated patients were responders. In the years since this 2010 review, there have been long term extension studies that have shown Sativex is effective in the treatment of spasticity in the long term, a phase IV safety extension study that showed that long-term treatment with Sativex was not associated with cognitive decline or significant changes in mood, and clinical use data which showed that Sativex can be a useful and safe option for those with moderate to severe spasticity resistant to common antispasmodic drugs. Following the approval of Sativex for management of MS symptoms, a postmarketing safety registry was set up to follow patients on the treatment, in Germany, the UK and Switzerland. This registry contains data from 941 patients with 2,213.98 patient-years of exposure, and results from this were published last year. Within this cohort, 60% continued treatment and 83% were reported as benefiting from the treatment. There were no new identified safety issues with the treatment, and based on this data, the risk/benefit profile remains positive.

In 2011, a study was done that closer resembles the real life clinical situation of prescribing Sativex. Subjects were treated with Sativex, as add-on therapy, in a single-blind manner for 4 weeks. After this period, those who showed an improvement in spasticity of $\geq 20\%$ were then enrolled in a 12-week randomised, placebo-controlled phase. 572 subjects were enrolled in the initial 4-week stage, and of those 272 showed a $\geq 20\%$ improvement after 4 weeks and 241 of those were randomised for the final 12-week stage. Those who received Sativex in the second phase showed a highly significant ($P = 0.0002$) difference in mean spasticity Numeric Rating Scale from the placebo group. This study is important not only because it demonstrates unequivocally that Sativex is an effective drug for the treatment of MS spasticity, but also because it recreated the real-life conditions in which Sativex would be used in the clinic. Namely, the drug was used as an add-on to current medications, and an initial trial was done to assess efficacy in the individual patient prior to committing to treatment.

There are also individual case studies of exactly how effective Sativex can be in MS patients, such as the example of a 54-year-old SPMS patient published in 2013. The patient had advancing disability due to MS spasticity despite having an implantation of an electronically adjustable intrathecal baclofen pump, being administered intermittent botulinum toxin, and both intravenous and intrathecal steroids. The treatment regime was failing and the side effects prevented continuation. Administration of Sativex restored some functioning, reduced disability, pain and paroxysmal cramps. The patient’s EDSS score stabilised and the patient was able to self-care again. This case study is representative, rather than exceptional. Similar case studies have also described the benefits of Sativex in MS.
2.2.2 Pain

Unfortunately, pain is a frequent and debilitating component of MS. It is particularly prevalent in the harder to treat, progressive forms of MS. Pharmacological treatment of pain in MS is challenging due to the many underlying pathophysiological mechanisms and as with other forms of neuropathic pain, the pain of MS is often refractory to treatment. Although in New Zealand Sativex is not strictly approved for the treatment of pain in MS, it is certainly one of its well proven therapeutic benefits.

In the 1980’s, the cannabinoid receptor type 1 and 2 (CB1 and CB2) were discovered, opening doors for research into cannabinoid receptor agonist and antagonist ligands. In 1989, lab experiments using cannabinoid receptor agonists to relieve the effects of experimental allergic encephalomyelitis (an animal model of MS) showed promising results and lead the way for randomised controlled trials in human subjects. The Rog et al. study in 2005 was the first to examine oromucosal delivery of a cannabis based medicine (CBM) with a 1:1 ratio of THC:CBD, the same formula as Sativex. The results from this early study showed a significant reduction in multiple sclerosis-related central neuropathic pain, and that the treatment was mostly well tolerated. In this initial trial, the CBM was added to the pain medication that the patients were already taking. In a 2-year follow-up of this trial, patients were able to adjust their other pain medications as required and continued on the initial dose of CBM. After two years of treatment, the patients continued to experience a reduction in pain and although a large proportion of participants experienced adverse events, these were largely reported to be mild or moderate.

In the SAFEX study, a Phase III, double-blind randomised controlled trial extension in 160 subjects with various symptoms of MS, 137 patients elected to continue on Sativex after the conclusion of the initial study. Rapid declines were noted in the first twelve weeks in pain as recorded on a Visual Analogue Scale (VAS) (N = 47) with slower sustained improvements for more than one year. During that time, there was no escalation of dose indicating an absence of tolerance to the preparation. Similarly, no withdrawal effects were noted in a subset of patients who voluntarily stopped the medicine abruptly. Upon resumption, benefits resumed at the prior established dosages.

In a more recent randomised controlled trial, MS patients who had failed to achieve adequate pain relief from existing medication were treated with either Sativex or placebo as an add-on treatment. This was done in a double-blind manner, to investigate the efficacy of the medication in MS-induced neuropathic pain. Despite the results being equivocal at the 14-week point, an analysis at the 10-week point, showed a statistically significant difference between the number of patients on Sativex reporting a 30% or greater improvement in pain, versus those in the placebo arm. After 14 weeks of treatment, participants in both arms were followed for a further 4-week withdrawal period to investigate the time to treatment failure. In this second part of the study, 57% of patients receiving placebo did not achieve a 30% or greater improvement in pain versus 24% of patients from the Sativex group.

2.2.3 Progression of disease

There is growing evidence that cannabis-based medicine could influence the progression of disease in MS. This started in the animal model where endocannabinoid augmentation has been shown to attenuate inflammatory events at a cellular level which resulted in slowed progression of this experimental version of the disease. Cannabinoids acting via the CB2 receptor control the inflammatory cascade which causes neuronal damage in MS. They may also act via the CB1 receptor to help limit excitotoxic damage to neurons by suppressing neuronal release of glutamate and the neuronal...
depolarisation response to glutamate. Cannabinoids may also be able to stimulate myelination, a nerve cell property lost in MS.

These effects have been seen in human subjects in the CAMS study. The 12-month follow-up data showed that participants had a continued improvement in disability scores over time, suggesting that there was a longer-term treatment effect of cannabinoids.

2.2.5 The argument against cannabis-based medicines

An examination of the arguments against cannabis-based medicines reveals that there is little logic or fact to support them.

Insufficient evidence

It has been previously stated that the reason for not funding Sativex in New Zealand is that there is not enough evidence to show efficacy. This is clearly not the case. As described here, there are many randomised controlled trials resulting in peer-reviewed papers published in reputable scientific journals which prove the efficacy of Sativex on MS symptoms. Sativex is now approved for use in many countries throughout the world including the UK, the USA, Spain, the Czech Republic, Germany, Denmark, Sweden, Switzerland, Italy, Austria, Canada, Poland and France. Therefore, there are many thousands of patient hours of experience of clinical use, and many different regulatory guidelines created and available for review. This allows New Zealand to benefit from the experience of other countries in the use of Sativex.

Potential for abuse

There is the suggestion that perhaps allowing use of cannabis-based medicine opens the door for abuse. Like all countries, we allow the use of opioid analgesics in New Zealand, which have the potential for abuse, misuse and overdose. Yet we allow their use because they are extremely useful for the treatment of pain and we, as a society, consider that the benefits of these medications outweigh the potential for harm. Much like with opioids, there must be tight controls for the use of Sativex which help to prevent potential abuse. However, it has been shown that Sativex can even be safely used even in recreational cannabis users. A randomised controlled trial of Sativex use in subjects with a history of recreational cannabis use was done in order to assess the subjective abuse potential and cognitive effects. It was found that even in these participants who recreationally use cannabis, Sativex can be used safely at low doses. At higher doses, Sativex showed the same potential for abuse as Dronabinol, a synthetic THC product.

Psychiatric disorders

There is a well-known link between cannabis and psychosis or psychotic disorders, including schizophrenia (reviewed by D’Souza et al., 2009). An open-label trial of Sativex examined participants for adverse events after long-term use, including psychiatric events. A total of 146 patients entered this open-label follow-up safety trial and the mean treatment exposure was 334 days. Of those patients, one reported two psychiatric events and no psychoses, psychiatric adverse event (AE) trends, or withdrawal symptoms occurred following abrupt cessation of treatment. The study concluded that there no new safety concerns were identified, that serious AEs were uncommon and that there was no evidence of tolerance developing. As an added protection, the NZ MedSafe Data Sheet states that Sativex is contraindicated in patients “with any known or suspected history or family history of
schizophrenia, or other psychotic illness; history of severe personality disorder or other significant psychiatric disorder other than depression associated with their underlying condition.”

Why not just smoke?
Although it is illegal to sell, possess or use cannabis in New Zealand, it remains widely available. According to the Ministry of Health’s 2012/2013 Cannabis Use Survey, 11% of adults aged 15 years and over reported using cannabis in the last 12 months, and 42% of those cannabis users reported using the drug for medicinal purposes. Clearly some patients are taking matters into their own hands. However, as described by Russo, 2016, this presents a myriad of problems. Firstly, there is a large biochemical variability between the available products. Users have no way of ascertaining the amount of THC or CBD they are receiving from a smoked product. Secondly, as the growth of cannabis is unregulated, products may contain pesticide residues, mould, bacteria or heavy metals. And thirdly, smoking cannabis poses risks such as chronic cough, bronchitis, and cannabis smoke is known to contain carcinogens. Smoking cannabis is not a viable option for people with MS looking for symptom relief. As MS patient and cannabis-based medicine advocate, Dr Huhana Hickey, put it “I really don't want to go to jail. They can't look after me in jail, they have no prison hospitals, they have no real proper treatment and care.”

New Zealand MS patients have a right to access this safe, effective and evidence-based pharmaceutical product and to expect that physicians can prescribe it without financial considerations. Associate Health Minister Peter Dunne urged physicians to “consider the prescribing of cannabis-based products with an open mind” in his February 2017 press release. However narrow-minded physicians are not the problem. Physicians in New Zealand strive to practise evidence-based medicine and they must not be prevented from doing so because a clearly beneficial medicine is prohibitively expensive for patients.


