Parkinson's Disease (PD) or Parkinsonism, has been reported all over the world in manganese (Mn) mine workers, and in some engineering welders. Waikato farmer clients have suffered it because of Mn in their farm drinking water. Thousands of Waikato cows have suffered stress from drinking it, some more so because they were fed American designed minerals containing Mn where it is low. Parts of USA are very low in Mn with only 15 ppm in pasture leaves which should contain about 40 ppm. I have never seen a low Mn level in New Zealand, but some people who are American influenced, and lack New Zealand practical knowledge and experience, add it to animal mineral mixes and fertiliser mixes with drastic results of animal stress. This is followed by stress in those milking the stressed cows which kick, pass manure and shuffle more, possibly causing the workers to then abuse the cows, more so if they are also subjected to excess Mn in their own water and/or food. PD worsens over time and has no cure, but has preventions.

Zinc deficiency is associated with Parkinson's disease.

Parkinson's disease is an age-related disorder characterised by movement disorders such as stiffness of the body, slowing of movement, and trembling of limbs when they are not in use. In advanced stages it progresses to dementia and eventually death. The main symptoms are caused by the loss of dopamine-secreting cells in the substantiar niagara. These dopamine cells are especially vulnerable to damage, and a variety of insults, including encephalitis (as depicted in the book and movie "Awakenings"), and repeated sports-related concussions. Some forms of chemical poisoning such as MPTP, can lead to substantial cell loss, producing a parkinsonian syndrome that is similar in its main features to Parkinson's disease. Most cases of Parkinson's disease, however, are idiopathic, meaning that the cause of cell death cannot be identified.

Numerous studies have shown that PD is worse in rural areas because of Mn in well water and herbicide/pesticide exposure. Nearly a dozen commonly used pesticides have been linked to Parkinson's, for example, avoid Mn and consume clean organic foods from well limed soils as much as possible. Manganese and other bad heavy metals, do better in wet acid soils, while magnesium and other neutral minerals enjoy soils that have been correctly limed and drained.

Prolonged exposure to lead can double the likelihood of developing Parkinson's Disease.

A study found that high levels of manganese and iron could make people twice as likely as those with low levels of heavy metals to suffer Parkinsonism.

Iron is abundant in red meat, some fish and poultry.

In New Zealand never apply Mn as a fertiliser, and avoid fertilisers that make soils acid such as superphosphate and urea because acid soils release Mn and other bad heavy metals. Read the two chapters on Mn and Lime (Calcium) and on fertilisers.

High lead and copper increases the likelihood of Parkinson's disease as do over work and insufficient sleep.

Parkinsonism to Parkinson's disease is still classified as idiopathic, meaning it has no identifiable cause. But one reason it is likely on the rise is due to many environmental toxins that now bombard your body on a daily basis, with pesticide exposure becoming an undeniable risk factor.

Avoiding pesticide exposure, around your home, in your community, and in the food you eat, is clearly important for reducing your Parkinson's risk, as is reducing your exposure to environmental toxins of all kinds. Another important and often overlooked environmental risk factor is amalgam dental fillings, 50 percent of which are mercury - a known neurotoxin.

Mercury becomes a biochemical train wreck in your body, causing your cell membranes to leak, and inhibits key enzymes your body needs for energy production and removal of toxins. Mercury toxicity can lead to major inflammation and chronic illnesses such as PD.

Nearly a dozen commonly used pesticides have been linked to PD, suggesting your best bet is to stick to an organic diet as much as possible.

If you take supplemental vitamin D, make sure you're getting enough vitamin K2 and magnesium as well.

Get plenty of animal-based Omega-3 fats, such as that found in krill oil, which serve an important role in protecting your brain cells.

Read more - http://www.dailymail.co.uk/health/article-184184/Excess-iron-raises-risk-Parkinsons.html#ixzz3ASQA7rYH

Read more - http://www.dailymail.co.uk/health/article-184184/Excess-iron-raises-risk-Parkinsons.html#ixzz3ASLv404P

According to the USA National Safety Council, inhalation or ingestion of manganese dust or fumes can cause symptoms similar to those of Parkinson's disease including tremors, rigidity, slowed movement, loss of muscular control and impaired mental function. It also can cause coughing, flu-like fever, vomiting and fatigue.

The Greatest Medical Blunder, by Autoimmune Specialist Noel Batten.

From http://www.parkinsonsdiseasecure.com/go/index.html

“The Greatest Medical Blunder is a book that reveals all the information your doctor and neurologist should be telling you about Parkinson's, but aren't, simply because they are trained by pharmaceutical drug company lecturers at medical schools, to focus on the sales of pharmaceutical drugs.

“While contemplating a medical career, I spent considerable time researching the medical approach to health and attending workshops at Mount Olivet Hospital.

“During this time I soon realised, any medical research that explains how to overcome disorders like Parkinson's disease without the use of pharmaceutical drugs is totally ignored and the medical research available is overwhelming to say the least.

“The medical evidence proves without any doubt, this is more than just an innocent mistake, it is a blunder of major proportion that is steering patients in the wrong direction and robbing them of their potential to recover.

“It is my opinion, that neurologists are misleading patients with false information.

“There is so much medically documented evidence that proves Parkinson's disease is just a normal response to emotional stress that can be overcome through personal effort, the treatment most neurologists apply is, in my opinion, nothing short of criminal.”

Preventions

Avoid manganese of more than 2 to 3 ppm in human foods, water, and in supplements, some of which are high, when there should be none.

Green tea twice a day is reported to reduce the chances of getting Parkinson's disease.

Medical Reports that prove, neurologists are giving false information

This first medical journal report explains that lack of blood flow in the prefrontal cortex causes Parkinson's disease, not dying neurons of the substantia nigra.

This medical journal gives details on how the prefrontal cortex is the area of the brain that actually causes Parkinson's disease and the prefrontal cortex is the area which transfers emotional stress and mental overactivity to the stomach, neck muscles and lungs, in the form of tension, which also interferes with the ability to achieve the deep stage of Rapid Eye Movement (R.E.M.) healing sleep.

Journal Of The Royal Society Of Medicine, June 1991, volume 84, pages 349-356

“It is my opinion, this second medical journal report reveals, the drug companies are aware that dying neurons in the substantia nigra are not the cause of Parkinson's disease, because the medication they supply for Parkinson's disease is not designed to treat the substantia nigra, it is designed to treat the prefrontal cortex. This fuels my opinion there is a purposeful conspiracy by the drug companies to

ensure Parkinson's patients don't overcome their disease and are thus deceived into purchasing a lifetime supply of drugs that never actually solve the problem.

“This medical journal confirms, the pharmaceutical drug used to treat Parkinson's disease is designed to improve blood flow in the prefrontal cortex, not the substantia nigra, so why do neurologists tell patients, dying neurons in the substantia nigra is the cause?

“The Brain Medical Journal, March 2002, volume 125, book 3, pages 584-594

“Research by Dr Ronald Mandel indicates, the substantia nigra does not cause Parkinson's

“Dr Ronald J. Mandel was successful in regenerating the cells of the substantia nigra and noticed after doing so, it made no difference to the presence of Parkinson's disease: QUOTE: "But when we were able to do so, (save the cells of the substantia nigra) we still saw all the signs of the disease". Ronald J. Mandel is an associate professor in the College of Medicine's neuroscience department at a University in America. Even though Dr Mandell may not agree with my application of his findings, I merely intend to report the facts so that Parkinson patients can consider all possibilities in their efforts of recovery.

“The following medical journal explains, an artificially induced lesion of the substantia nigra created purposely to diminish dopamine neuron activity, did not produce the motor symptoms of Parkinson's disease. This experiment also revealed, dysfunction of the substantia nigra does not cause Parkinson's disease.

“The Journal of Brain Research, May 2002, volume 58, book 1, pages 41-47

“This following medical report explains, when autopsies were carried out on four people who passed away with Parkinson's disease, the substantia nigra was found to be relatively free of cell damage, however severe cell damage was found in a part of the brain called the locus ceruleus. This part of the brain is affected by emotional stress and mental overactivity which takes place in the prefrontal cortex during sleep.

“The Journal of Neuropathology, June 2002, volume 22, book 2, pages 77-84

“The medical journals I refer to in this article and in my book, only scratch the surface of the pallet loads of medical reports available world wide that expose this blunder of medical inefficiency.

“With persistence, you "CAN" overcome Parkinson's disease

“Overcoming Parkinson's disease begins with improving your R.E.M. sleep

“This medical journal documents that the tremors in Parkinson's disease almost always disappear after good sleep. Good sleep can only be achieved if the mind is calm and the body is high in oxygen, as peace of mind and oxygen, regulate our ability to relax and achieve R.E.M. sleep.

“Journal of Homeostasis in Health and Disease, April 1999, vol 39, pages 107-111

“The results of this medical report should be displayed on every neurologist's waiting room wall to steer Parkinson's patients in the right direction and allow them to realise, through personal effort, Parkinson's disease can be overcome. Instead, neurologists are supplying misleading information that Parkinson's disease is incurable, it is caused by substantia nigra neurons dying and taking a lifetime of pharmaceutical drugs is the only option.

“Medical researchers world wide have documented indisputable evidence that mental overactivity in the prefrontal cortex causes tension in the neck, lungs and stomach, which leads to the development of Parkinson's disease.

“By using my stress management techniques to overcome an overactive mind and improve R.E.M. sleep, relax the neck and stomach and improve lung efficiency, it is possible to overcome Parkinson's disease with complete and lasting success. You "CAN" overcome Parkinson's disease providing you are willing to follow the techniques I outline in my book "Parkinson's Disease, The Greatest Medical Blunder".

“The real problem is that doctors and neurologists are so busy keeping up with the ever-increasing drugs and are so deeply influenced by drug company representatives, they never have time to review the research that proves emotional stress such as grief, grudge, depression and fear are the cause of Parkinson's and that overcoming these emotions is the only way to achieve remission.

The arm tremors experienced by Parkinson's patients are simply a stress reaction to lack of R.E.M. sleep caused by suppressed grief etc which creates excessive tension in the muscles of the neck that

strangle nerves which operate the arms. Once the emotional grief etc is overcome, and R.E.M. sleep is improved and the tension eliminated, the tremors disappear completely.

This journal reports, the symptoms of Parkinson's, Multiple sclerosis and Alzheimer's can all be caused by vertebral misalignment, muscular swelling, or degeneration at the C6 or C7 vertebra in the neck, which reduces blood and oxygen to the face and brain.

“Panminerva Medical Journal, March 2002, vol 44, book 1, pages 47-59.

“The fact that neurologists refer patients to brain surgeons to have a brain operation that permanently damages a section of the thalamus to stop excessive arm shaking, when all the patient needs to do is improve R.E.M. sleep, bring the neck back to a relaxed state and improve digestion, is nothing short of criminal.

“If you have Parkinson's disease and your doctor and your neurologist are telling you Parkinson's disease is caused by dying neurons in the substantia nigra, and your only option is a lifetime supply of pharmaceutical drugs, don't listen!

“When I had epilepsy at age thirteen, my neurologist misinformed me and started me on a lifetime supply of pharmaceutical drugs that did nothing but turn me into a zombie. After overcoming my condition completely through natural means, and studying autoimmune disorders, I found medical journals that proved this neurologist's ignorance was cheating me out of my life.

The vast majority of studies have been able to link Parkinson's only to broad classes of pesticides. However, investigations of some individual Parkinson's cases have shown links to specific pesticides. These include paraquat, maneb, diquat and dieldrin (Le Couteur et al. 1999) - not forgetting the New Zealand case linking Parkinson's to 2,4-D reported in Soil & Health last year (Watts 1999). Other pesticides have been linked with acute but reversible parkinsonism (parkinsonism is a disorder with symptoms like Parkinson's disease, but which may be reversible). These include the organophosphates malathion, dimethoate and chlorpyrifos, and the carbamate propoxur (Bhatt et al. 1999). One interesting case of reversible parkinsonism supports the study by Nelson on household insecticide use. It involved a 64-year-old woman who used an aerosol organophosphate insecticide twice daily in her kitchen and bedroom over a period of ten years. She developed progressive symptoms of parkinsonism, but recovered after a period in hospital. On returning home she became ill again. The pattern recurred every time she went home. She eventually moved to a new home, then after two years without symptoms she wore clothing that had been stored in her previous home. She again developed parkinsonism, which subsided when she removed the clothing. Her sister and daughter who came to care for her were also affected, but her husband, son, daughter-in-law and grandchildren were unaffected (Bhatt et al. 1999). This episode supports the notion that there may be familial susceptibility to Parkinson's disease as a result of genetic variation in the enzymes involved in detoxifying pesticides (Le Couteur et al. 1999). It also provides a sharp warning for those people who think flysprays are harmless.

A patient with Parkinson's disease steadily improved when treated daily with 4,000 IU of vitamin D. At the same time, two new meta-analysis studies (which combine data from multiple reports) have found even more evidence that vitamin D is an important cancer-fighting tool. The first study examined nearly 1,800 records and found that women with the highest blood levels of vitamin D had the lowest risk of breast cancer, and those with the lowest vitamin D levels had the highest rates of breast cancer.

A study which looked at nearly 1,500 people, showed similar results. Considerable evidence that vitamin D **deficiency** is a cause, and possibly the major cause, of Parkinson's disease.

The researchers reviewed a 1997 case report in which a patient with Parkinson's disease steadily improved when treated daily with 4,000 IU of vitamin D.

People with Parkinson's disease often experience trembling, muscle rigidity, difficulty walking, problems with balance and slowed movements. These symptoms usually develop after age 60, although some people affected by Parkinson's disease are younger than age 50.

James Parkinson (1755-1824), renowned for first describing the shaking palsy in 1817. His

activities led to his being hauled before the Privy Council to give evidence about a plot to assassinate King George III - but he sensibly refused to testify until he was certain he would not be made to incriminate himself. Parkinson also contributed extensively to medical journals and wrote several books on medical subjects and palaeontology. However, his fame sprang from his major opus, "An Essay on the Shaking Palsy", in which he vividly described the disease and its different stages and gave it the Latin name of "paralysis agitans".

Not Inherited?

The disease is not normally inherited. The rate of dizygous and monozygous twins is identical. Although familial PD occurs, the disease in such families is severe and of early onset. No biological marker for the condition has been found, although a 1997 report suggests that an abnormality on chromosome 4 is linked to one cluster of familial PD.

MPTP: One intriguing chemical cause of parkinsonism has been discovered: 1-methyl-4-phenyl-1,2,3,6-tetrahydropyidine (M.P.T.P). This occasional contaminant of certain designer drugs can produce irreversible full blown Parkinsonism within a few days. The drug is toxic to the substantia nigra, does not produce Lewy Bodies and has similarities to several pesticides in common use. Selegiline, a drug which has been studied for its role in slowing the progression of PD, seems to protect against the toxic affects of MPTP.

Numerous case control studies have indicated that PD is associated with rural residence, farming, well water drinking and herbicide/pesticide exposure. Furthermore, families have been identified in which age of onset in parent and child correspond to a past common environmental trigger. The similarity of numerous pesticides with MPTP is intriguing.

Insufficient neuro protection?

There has been speculation that anti-oxidant vitamins could protect against PD. Two case control studies suggested that tocopherol may decrease the risk of developing PD.

Risk Factors for Parkinson's Disease

Increasing age, rural residence and exposure to manganese and pesticides. A very good friend who owned an orchard and admitted to spraying a lot with pesticides and fungicides, got and died of Parkinson’s disease. He acknowledged about using powerful machine spraying equipment and not using protective clothing.

Non specific early features

Patients complain of tiredness, lethargy, mild depression or restlessness. Many will have non specific limb pain, aching muscles and vague paeasthesias.

Resting tremor

Found in about 75% of patients at presentation, tremor is the commonest early symptom. In early stages it rarely interferes with movements. To the patient it feels "like a motor running all the time". It is most frequently confused with essential tremor.

Early features of Parkinson's Disease

- Tiredness
- Lethargy
- Mild depression
- Restlessness
- Tremor at rest
- Non specific limb pain

Bradykinesia

Is slowness in initiating movement. This includes -

- Large voluntary movements (standing up or hurrying)
- Fine movements (difficulty fastening buttons or cutting food; writing may become smaller and more cramped)
- Involuntary movements (slow blinking, reduced arm swinging and loss of facial expression)
- Automatic movements (constipation, unstable bladder, dysphagia)

Rigidity

Rigidity produces the perplexing muscle pains described by many sufferers. Muscle tone increases, producing cramps, stiffness and aches in arm or leg muscles.

Postural instability

The gait and balance is affected. Patients may feel unsteady, possibly limp, and may begin to stoop. Falls are so frequent in the elderly PD patient that anyone with a fractured neck or femur should be examined for PD.

The four major manifestations of PD

- Tremor
- Bradykinesia
- Rigidity
- Postural instability

Secondary clinical features

Postural hypotension

Postural hypotension in PD is thought to result mainly from automatic dysfunction. It is potentiated by most anti parkinsonian drugs.

Restless legs

This primary sleep disorder is so common in PD patients that it should always raise suspicion of the condition. It consists of uncomfortable sensations in the legs and an overwhelming desire to stretch or walk.

Speech problems

Speech difficulty may be an early problem. Bradykinesia of the speech mechanism makes the voice quiet, monotonous and often slow despite patients feeling that they are speaking normally. They find it hard to shout and have difficulty using the phone. Paradoxically some patients may have rapid (festinating) speech. Stuttering is also common.

End

A 63 year old university professor with a history of previous anxiety became rapidly disabled by inability to raise his voice and express himself when lecturing. His anxiety over the symptom made it worse. He responded very well to intermittent doses of Madopar Q,, and was able to return to work with his confidence restored.

Other "soft" symptoms

Problems with body functions are frequent. Anosmia (loss of sense of smell) is remarkably common. Sufferers sometimes have dysphagia and take a long time to eat their meals. Impotence is not unusual.

“I wasn't able to cope with even things like getting family meals without great difficulty - I was just flopping about the place. The other things that I thought were relatively minor things, and related to the menopause, were incontinence, bowel problems, constipation and bleeding. It was when my

left leg started to behave strangely that I was diagnosed" (Sarah, age 46)

Produced for: Parkinson's Association of WA Inc, 320 Rokeby Road, SUBIACO WA 6008

Further Patient Information Sheets & Updates at:- http://www.nevdgp.org.au/tbbase.htm

Some of this comes from the Parkinson's Association of WA Inc, 320 Rokeby Road, SUBIACO Western Australia 6008. More Information & Updates are at one of the best sites -

http://www.nevdgp.org.au/tbbase.htm

The Parkinson's Association of New Zealand wrote that no one knew the cause.

Causes

PD is more of an environmental problem. It is not inherited.

Proven causes of Parkinson's disease (a brain fault) and Alzheimers is excess manganese. See both Manganese chapters.

Medical research scientists have discovered a correlation between pesticide poisoning and Parkinson's Disease (BBC news, Monday 6 November 2000).

Zinc deficiency and excess manganese (Mn) are associated with PD. Levels of zinc in victims are substantially lower than those without Parkinson's.

Parkinsonism in manganese exposed workers has been reported all over the world.

Numerous control studies have indicated that PD is associated with rural residence, farming, well water drinking and herbicide/pesticide exposure.

Vegetables grown in wet, low pH, acid soils low in calcium (lime) are usually very high in Mn. The ryegrass leaves should not be above 40 ppm.

Medications known to make tremors worse should be eliminated or minimised when possible. These include lithium, several antipsychotics, valproic acid, corticosteroids, some anti-depressants and a class of drugs called adrenergic agonists. People with tremor also may benefit from avoiding dietary stimulants, such as caffeine. They should also be evaluated for hyperthyroidism, which can produce tremors that mimic ET.

Symptoms

The earliest symptoms are tiredness, lethargy, mild depression or restlessness. Many will have non-specific limb pain and aching muscles. Tremors are a common early symptom. A 30 year-old man's little finger shook for years before being diagnosed. Problems with body functions are frequent. Anosmia (loss of sense of smell) is common with zinc deficiency. Sufferers sometimes have dysphagia (difficulty swallowing) and take a long time to eat their meals. Impotence is not unusual.

Restless legs is a primary sleep disorder common in PD patients. It consists of uncomfortable sensations in the legs and an overwhelming desire to stretch or walk.

It was when my left leg started to behave strangely that I was diagnosed as low in zinc.

Speech difficulty may be an early problem. Bradykinesia of the speech mechanism makes the voice quiet, monotonous and often slow despite patients feeling that they are speaking normally. They find it hard to shout and have difficulty using the phone. Paradoxically some patients may have rapid (festinating) speech. Stuttering is also common.

Rigidity produces the perplexing muscle pains described by many sufferers. Muscle tone increases producing cramps, stiffness and aches in arm or leg muscles.

The gait and balance is affected. Patients may feel unsteady, possibly limp, and may begin to stoop. Falls are frequent in the elderly.

A 63 year old university professor with a history of previous anxiety became rapidly disabled by inability to raise his voice and express himself when lecturing. His anxiety over the symptom made it worse. He responded very well to intermittent doses of Madopar Q and was able to return to work with his confidence restored.

http://www.memorylossonline.com/winter2010/mind_parkinsons.html

USA, world famous actor/comedian Robin Williams, took his life in 2014 after suffering Parkinson's

disease.

"It's not just a movement disorder. Besides causing tremors and other motion-related symptoms, Parkinson's disease affects memory, learning, and behaviour."

by Daniel Pendick

Parkinson's disease is notorious for so-called motor symptoms like muscle rigidity, tremor, slowed movement, and unsteady posture and gait. Less well known, even to some doctors who treat the disease, are the effects of Parkinson's on thinking. These "cognitive" signs include a general slowness of thought, "tip of the tongue" forgetfulness of words, and difficulty juggling multiple mental tasks.

Parkinson's disease and the medications used to treat it may also affect how the brain learns. And even stranger, certain Parkinson's drugs can trigger compulsive behaviours such as pathological gambling or uncontrolled shopping. Understanding these and other aspects of how Parkinson's disease affects the mind offers hope of a better life for people with a disease estimated to affect more than a half million Americans.

Parkinson's is caused by the death of brain cells that produce dopamine, one of the chemicals that carry messages between neurons. Low dopamine impairs the basal ganglia, which are brain regions that control movement and coordination.

Drug treatments try to shore up dopamine levels. For example, medications containing the chemical L-dopa provide extra raw materials to produce natural dopamine. Another, newer class of drugs, dopamine "agonists," mimic the action of natural dopamine on motor-control brain cells.

Dopamine-boosting drugs address motor symptoms, and this allows people to function better. But realisation is growing that some patients need help with non-motor symptoms. These include depression, anxiety, daytime sleepiness, insomnia, lightheadedness, urinary incontinence, nerve pain and loss of smell. Some patients develop memory loss and dementia, generally late in the disease's progression.

Research priorities

In 2001 and again in 2006, the National Institute of Neurological Disorders and Stroke (NINDS) held meetings at which scientists, doctors, and patients discussed priorities in Parkinson's disease research. Non-motor symptoms emerged as a major concern.

"In both summits, patient advocates and the clinical community identified it as one of the most important under-addressed areas for patients with Parkinson's disease," notes neurologist Debra Babcock, MD. She heads the NINDS program on Cognitive Neuroscience that funds research on non-motor Parkinson's symptoms. "What's worse is that some non-motor symptoms are actually aggravated by the treatments used for the motor symptoms."

What is needed, Babcock says, is more research. "It's understudied," she says. "Less than 5 percent of our Parkinson's disease grants are looking at cognitive dysfunction. This is an improvement over prior years though, and we continue to actively encourage the research community to focus on this issue."

Impulse-control disorders

The latest non-motor symptoms to come to light are impulse-control disorders. These occur in at least 14 to 17 percent of people who take dopamine agonists, says neurologist Melissa J. Nirenberg, MD, of Weill Cornell Medical College in New York. The most common are compulsions for gambling, sex, shopping, food, eating, or even hobbies. Some people exhibit "punding," or repetitive, purposeless

behaviours such as sorting objects. Frequently the compulsion involves a behaviour the person "previously enjoyed in moderation," Nirenberg notes.

Nirenberg is an expert in impulse-control disorders associated with Parkinson's medications. One factor that still obscures this problem even from experienced neurologists is the sensitive nature of the behaviours.

Some patients might be willing to bring up the fact that they have been eating uncontrollably. But it's harder to uncover repeated and financially disastrous trips to the casino, or all-night internet pornography-viewing sessions and visits to prostitutes.

A frank discussion with a spouse or partner can help. Then the medications can be changed to reduce or eliminate the problem.

Changes in learning

In recent years, researchers have uncovered another odd and unexpected effect of Parkinson's disease on the mind. Depending on whether people are taking dopamine-boosting medications or not, their mode of learning changes.

Normally we learn from both "rewards" and "punishment." In reward learning, we receive positive feedback (a reward) for doing the right thing. In punishment learning, we receive negative feedback for doing the wrong thing. For example, studying for an exam brings the reward of a high grade, but not studying brings a failing mark.

Remarkably, people with PD who are **not** taking dopamine-boosting medication learn better when they are punished for making the wrong choices, rather than being rewarded for making the right choices. However, on dopamine medication, the scales tip completely the opposite way, and rewards had a higher effect.

In a study published online in May 2009, in the journal 'Brain', researchers led by Hungarian psychiatrist Szabolcs Kéri and U.S. neuroscientist Mark Gluck probed this reversible learning bias in a special group of test subjects: relatively young patients (in their 40s) recently diagnosed with Parkinson's, but who were not yet on medication. This unique group of patients allowed the scientists to explore the effects of dopamine medication on positive learning and improve on previous research in several ways.

In previous studies, patients were commonly older and farther along in the disease. They were more likely to suffer from memory problems and mood disorders, such as depression, that could affect the study outcomes. In contrast, the younger and healthier Hungarian group provided a way to study the effects of Parkinson's on learning with fewer potentially confounding factors — both before and after the patients started on medications.

Gluck, professor of neuroscience at the Center for Molecular and Behavioral Neuroscience at Rutgers University, Newark, worked with researchers at Rutgers and New York University to develop a new testing technique for the study. Compared to previous methods, the new test more directly assessed the degree to which the patients learned from punishment or reward. It takes just 20 minutes to complete and runs on any laptop computer.

Nikoletta Bódi, a graduate student in Kéri's lab, performed most of the testing on the Hungarian patients for the study. The results were consistent with previous research. "We demonstrated that these newly diagnosed patients have a very specific deficit in learning from reward but are normal at learning from punishment," Gluck explains. "In contrast, when they are placed on medication this learning sensitivity reverses: They become impaired at learning from punishment, but are normal in their ability to learn from reward."

The results confirmed the reversible learning bias on and off medication. The study also extended previous research by examining the effect of medication on certain personality traits associated with Parkinson's disease.

One trait is "novelty seeking," or a preference for things that are new and different, rather than comfort with the familiar. In the study, never-medicated patients with Parkinson's disease show much less novelty seeking behaviour. But once they started taking the dopamine-boosting drugs, novelty seeking increased.

This and other research on the interactions of Parkinson's, dopamine medication, and learning could help have some practical benefits for patients. "The research should motivate neurologists to keep an eye on these cognitive effects and impulse control disorders that until recently were largely ignored because the doctors were trying to treat the motor dysfunction," says Michael Frank, a cognitive neuroscientist at Brown University and one of the first to document the affect of dopamine medications on learning in people with Parkinson's. "But it's becoming more clear that these cognitive effects significantly impact the quality of life."

Effects on behaviour and thinking are just "another factor they could use to determine which patient should be on which drug and at what dose," Frank says. Genes may someday come into play, too. Frank and his colleagues have studied gene variations associated with the sites on neurons that interact with dopamine. Certain dopamine gene variants are associated with whether a person learns better from positive or negative experience. In the coming age of "genomic medicine," genetic testing could identify people at risk of impulsive behaviour when taking a given medication.

So could tests for changes in personality and learning, Gluck adds. "It is generally believed that at the time of initial diagnosis, most Parkinson's patients have already lost up to 70 percent of their dopamine cells. Thus, there is surely a long period in which the loss of dopamine cells may cause cognitive and personality changes before the motor symptoms are apparent."

In September 2015 we reported on the medical finding of Parkinson's disease as a result of exposure to 2,4-D in Northland. In November, the mainstream media published the results of a laboratory study linking the disease to rotenone, more commonly known as Derris Dust. There is a gathering body of evidence and worldwide concern that exposure to pesticides might be an important triggering factor in the development of this devastating and irreversible condition of the nervous system. We take a closer look at the problem

For much of the last 20 years, concerns about pesticides have been focussed on cancer, the "dread disease". However, attention has recently begun to switch to the complicated effects of pesticides on the nervous system, in particular the chronic neurological problems, of which Parkinson's disease is just one. It has been projected that neurodegenerative diseases will overtake cancer as the second most common cause of death among the elderly by the year 2040 (Lang & Lozano 1998). What is Parkinson's disease? Parkinson's disease is the most common degenerative disease of the nervous system, currently affecting about one per cent of the population. It is usually a late onset disease characterised by progressive and irreversible symptoms of muscular rigidity, shaking hands, facial tics, and tremors. Muhammad Ali, Michael J Fox, and athlete John Walker exhibit these characteristic symptoms. As the disease progresses, speaking becomes difficult, a shuffling gait develops, and there may be symptoms of senility and severe depression: the suffering can go on for many years. It also shortens life span. The symptoms result from the death of neurons in the brain, particularly dopaminergic neurons - that part of the brain involved in producing dopamine, a chemical messenger that helps control muscle activity. The cause of this death of neurons is still largely unknown, even though the disease was first described in 1817 by James Parkinson. Early studies concentrated on finding a possible genetic link to the disease, but a massive study of nearly 20,000 identical twins has virtually ruled out the genetic causation theory, with fewer than 10 per cent of cases thought to have a genetic component (Tanner et al. 1999). Environmental causes have long been suspected and attention has increasingly turned towards pesticides (Giasson & Lee 2000). In fact, one of James

Parkinson's original patients was a gardener thought to have been exposed to arsenic and nicotine pesticides (Le Couteur et al. 1999). One of the classic symptoms of Parkinson's, the muscle tremor, is also a classic symptom of exposure to organophosphate insecticides. The current hypothesis, backed by strong evidence from scientific studies, is that damage to mitochondria is the cause of Parkinson's disease. Mitochondria are tiny organelles that exist in all cells and are involved in metabolism and the provision of energy for cells to carry out their functions. There is substantial evidence that a number of plant extracts, drugs, pesticides and other chemicals damage mitochondria, and in particularly interfere with its enzyme complex, known as "complex I", and hence can cause Parkinson's disease (Schapira 1998; Le Couteur et al. 1999; Betarbet et al. 2000). Suspect pesticides "A steady stream of studies from around the world have shown again and again that a common thread among victims of Parkinson's is a history of exposure to insecticides and herbicides" (Montague 1999). Fungicides do not appear to be quite so heavily implicated, although there is some evidence of the involvement of dithiocarbamate fungicides like maneb (Ritz & Yu 2000). Industrial solvents are also implicated, and many pesticide formulations contain solvents. Studies on the link between Parkinson's and pesticides began in the early 1980s when it was discovered that users of an opiate drug (MPTP), chemically similar to the herbicide paraquat, developed Parkinson's Disease (Pfohman 1992). Most of the evidence linking Parkinson's to pesticides comes from retrospective studies of populations with elevated levels of the disease. Because of the immense difficulties involved in establishing cause and effect, these studies have been able to establish links only to general classes of pesticides rather than to specific chemicals. The first reported association of this kind was in 1978 and involved a crop duster routinely exposed to organophosphate insecticides. Other studies have linked Parkinson's to carbamate insecticides, carbon disulphide-based fumigants, organochlorine insecticides, household fumigation, chlorphenoxy herbicides, or herbicides generally (Semchuk et al. 1993; Le Couteur et al. 1999; Ritz & Yu 2000). Many of these studies involved occupational exposure. However, in May 2000, neuroepidemiologist Dr Lorene Nelson presented to the American Academy of Neurology's annual meeting in San Diego the results of a study on the association between Parkinson's Disease and home use of pesticides. The study confirmed the occupational exposure findings, indicating that home users are just as susceptible to harm from pesticides as are farmers. The worse problem came with use of household insecticides, with a 70 per cent increase in risk of the disease, but exposure to garden insecticides and herbicides also significantly increased the risk. As mentioned already, There is also laboratory evidence that a number of pesticides are toxic to mitochondria or interfere with their function, and are thereby potentially able to cause Parkinson's disease. These include chlordane, cyanide, cyhalothrin, 2,4-D, DDT, dieldrin, dinoseb, endosulfan, ethaphos, glyphosate, heptachlor, paraquat, permethrin, rotenone and 1080 (Le Couteur et al. 1999). The new generation insecticides are worse New generation insecticides may be even more of a problem. Since scientists came to the conclusion that it is the target site insensitivity of the old style broad spectrum insecticides that was causing insect resistance, they have sought to develop insecticides that are much more specific in their biochemical activity. And one of the specific sites they have found to be effective in killing insects is that of the complex I enzyme system in mitochondria - the very one that appears to underlie Parkinson's disease (LŸmmen 1998). Such new generation insecticides include fenpyroximate, tebufenpyrad, fenzaquin, pyrimidifen, and pyridaben - all used for mite control and in some cases for other insects (LŸmmen 1998; Espositi 1998). Of these, only fenpyroximate and tebufenpyrad are currently registered in New Zealand. Do not expect that the new registration system soon to start up under ERMA will detect these problems: knowledge of the effects of pesticides on the complex I enzyme system of mitochondria is not required for registration of pesticides.

Derris Dust and Parkinson's

Despite the fact that there is so much evidence linking Parkinson's disease to a number of synthetic chemical pesticides, the subject only hit the news when a recent study linked Parkinson's to a pesticide that is permitted under organics standards: rotenone, more commonly known as Derris Dust, and used principally by home gardeners. Rotenone is extracted from plants belonging to the

legume family, mostly plants of the Derris and Lonchocarpus genera which come from Southeast Asia, Central and South America. Rotenone is found in at least 68 species of legume (Morris & Powell 2000). Apparently it has been known in scientific circles since 1961 that rotenone inhibits the complex I enzyme system of mitochondria (Esposti 1997), and since 1997 that it may be implicated in Parkinson's disease (Morris & Powell 2000). The latest study found that chronic intravenous exposure to rotenone induced the progressive degeneration of the dopaminergic neurons in the brains of rats, and the reduced movement, unsteady movement, hunched posture, rigidity and shaking paws characteristic of Parkinson's (Betardet et al. 2000). o NOTE: Do not take the list of pesticides provided here as any sort of definitive statement on those which cause Parkinson's disease. It is important to remember that there is an immense lack of scientific knowledge about the effects of pesticides on the nervous system and on the enyzme systems involved in metabolism, and that the work on linking specific pesticides to Parkinson's disease is in its infancy. Studies linking Parkinson's to pesticides have implicated most categories of insecticides and herbicides: organochlorines, organophosphates, carbamates, fumigants, synthetic pyrethroids, the new generation miticides, phenoxy herbicides, paraquat, glyphosate, and even natural plant extracts. Those who slavishly adhere to scientific proof before they stop using pesticides will argue there is no definitive proof. Those who do not want to run the risk of developing Parkinson's might take a different view. What to do instead of using Derris Dust As Derris Dust is a naturally occurring compound, it has long been allowed in organic growing systems. Under the current BIO-GRO standards, Derris Dust is a restricted material, meaning that it may be used "only in accordance with the principles specified in these Standards", with a reduction each year in the dependence on it, and only until more acceptable materials are available. It is also permitted under Demeter and Agriqual/Certenz standards. Its main use is in home gardening, particularly for controlling the caterpillar of white cabbage butterfly. In commercial horticulture it has largely been replaced by Bt preparations. Bt is the short name for Bacillus thuringiensis, the preparation that was used to eradicate the white-spotted tussock moth in Auckland several years ago. Soil & Health will be requesting that BIO-GRO, Demeter and Agriqual/ Certenz no longer allow Derris Dust in certified organic systems. We will report progress on this front as it occurs. The finding of such drastic health problems from exposure to an age old organic preparation should act as a wake-up call to organic growers. It is a reminder that many natural plant products can be toxic when they are extracted and concentrated. It is a reminder that the philosophy of organic growing is not that of replacing synthetic chemicals with natural chemicals. Rather it is to strive for balance in an agri-ecosystem, or home garden, in which the grower works with nature as much as possible rather than trying to control it. Pesticide preparations, even those of natural occurring substances, should be the last port of call not the first remedy. They may control the pest at that moment in time, but they do not encourage a balanced system that looks after itself. Derris Dust also kills valuable biological control insects like ladybirds. This is counterproductive. In our city garden, we have fed the birds to encourage their presence for many years. We have no problem with white cabbage butterfly, or any other insect pests. After dining on the birdseed and left over bread crusts, the birds make a bee-line to the garden and clean up any insects they can find. Mind you they are also partial to lettuce and silver beet - these we net. If your bird populations are not sufficient for natural control, try squashing the caterpillars with your fingers. It may be messy, but it is preferable to Parkinson's disease. Or use Bt. Finally, a little bit of good news for the coffee-drinkers out there: research indicates that coffee-drinkers are less likely to develop Parkinson's disease (Ross et al. 2000). It appears to be a direct effect of caffeine intake! On the other hand, high intake of total fats, saturated fats and cholesterol seems to increase the risk of Parkinson's (Johnson et al. 1999).

SUSPECT PESTICIDES 2,4-D, dieldrin, diquat, maneb, paraquat, rotenone.

Berkeley attorney Philip Hanley said his plaintiffs in the California cases are welders or pipe fitters who worked near welders for 20 to 40 years. He noted two major differences between California and the federal litigation. The state cases involved only ‘true Mn poisoning cases’ with symptoms similar to Parkinson's. Many of the federal cases alleged welding fumes caused actual

Parkinson's.