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East Midlands RSN NEWS

from the East Midlands Research Support Network

*** February 2014 / No 13 ***

Welcome to this edition of RSN News.

This sees the launch of the new feature, "Has this been researched?", with an article by Lionel on Depression. Accompanying this is a contribution from Bob Raeburn on his personal experiences - our thanks to him for his frank revelations. Bob has also sent other contributions, one on the Ronnie Gardner Rhythm Method (RGRM) which has been used in treating stroke victims. This appears to be a variation on "Walking with Music", which Fiona Lindop spoke about at our Research Forum in Derby, but could also be used for treating Parkinson's – to view this, go to http://rgrminternational.com/node/16

("Musical Instrument Group" by nuttakit: freedigitalphotos.net)

We are very pleased to have received a number of other contributions in response to our request for input from you, our readers. One, an article from Harry Pearman, appears on page 5 (my apologies, Harry, for the need to edit this). Others will be answered in subsequent editions. Thank you for your responses and please keep them coming.

For those who wish to obtain more information than we can provide in our EMRSN News, it is worth re-

Inside this issue:

… and much more!

(News word on mobile screen, by Stuart Miles: freedigitalphotos.net)

iterating that the Parkinson's UK website is a major source of news about research into Parkinson's. Going to http://www.parkinsons.org.uk, clicking on 'Research' on the menu along the top and then using the drop-down menu on the left hand side of the page gives access to a mine of information.

Another valuable source of information is the European Parkinson's Disease Association (EPDA). This can be accessed at http://www.epda.eu.com/en/ and again using the menu across the top of the page.

On a general note, not related to research, but of great interest to movie fans, PwP can obtain a free ticket for someone accompanying them to the cinema by getting a Cinema Exhibitors' Association Card. For full details and application form go to http://www.ceacard.co.uk/.

Many thanks to all our contributors to this edition.

Ian Billcliff, Editor

News & Events

BREAKING NEWS 1

Simple way to make stem cells in half an hour!!!

This was a startling headline in The Guardian on 29 January.

("Red And White Blood Cells" by Victor Habbick: freedigitalphotos.net)

Haruko Obokata and colleagues in Japan have discovered that somatic cells (any biological cell forming the body of an organism) from mice can be converted into pluripotent stem cells just by exposing them to a slightly acidic solution for 30 minutes.

Her work has been reported in two papers in Nature (references below), in which she describes the phenomenon, called stimulus-triggered acquisition of pluripotency (STAP).

Dr Obokata started work on this project 5 years ago after noticing that cells which had been squeezed through a thin tube shrank to the size of stem cells. In subsequent research she discovered that she could convert white blood cells from newborn mice into cells that behaved very like stem cells and she then continued to do the same with cells from other organs in the body.

The team went on to create mice which have tissues grown from these STAP cells. After one to two years these mice appear to be healthy, normal and fertile.

Stem cell researchers in the UK consider this to be a major breakthrough, heralding “a new era in stem-cell biology”. If it could be repeated in human tissue it could lead to a simple and cheap procedure to produce patient-matched stem cells that could repair damaged or diseased organs.

References:

http://www.theguardian.com/science/2014/jan/29/make-stem-cells-major-discovery-acid-technique
http://www.nature.com/nature/journal/v505/n7485/full/nature12968.html
http://www.nature.com/nature/journal/v505/n7485/full/nature12969.html

BREAKING NEWS 2

In experiments on fruit flies with a genetic mutation linked to a rare form of Parkinson’s, in which problems with the mitochondria lead to death of the dopamine producing nerve cells, Dr. Miguel Martins and his team at Leicester University have discovered that feeding folic acid to the fruit flies helped their mitochondria to function better, prevented nerve cell death and completely restored their mobility.

This work has been reported in ‘Nature’ and the team has already started exploring the effect of Folic acid on human cells. However, a lot more work is needed to discover whether taking folic acid has any benefits for people living with Parkinson’s

Folic acid is one of the B vitamins and is found in foods such as broccoli, sprouts and liver. Adults need 0.2 mg of folic acid per day, which most people should get from a normal balanced diet. It must be remembered **that taking too much can be harmful** and **current guidelines recommend that people should not take more than 1mg of folic acid per day.**

("Broccoli " by zirconicusso: freedigitalphotos.net)

See http://www.parkinsons.org.uk/researchnews and also the article on ‘East Midlands research’ by John Telford on page 4.

Other News

The Winter 2014 edition of ‘Progress’ has just been published, with some excellent articles, particularly the one on Clinical Trials, the main feature of this edition. For anyone who does not receive this publication, it can be viewed on the Parkinson’s UK website at http://www.parkinsons.org.uk/content/progress-magazine

The AllTrials Campaign

Following the controversy surrounding information supplied by pharmaceutical companies about clinical trials they have conducted, with allegations that information on negative data has been suppressed, it is good to learn that Johnson & Johnson have followed the lead of GlaxoSmithKline in announcing that they will give all the pharmaceutical clinical trial data they hold to researchers.

The Campaign is gaining momentum worldwide and new clinical trials regulations have been approved by the Public Health Committee of MEPs in Brussels.

Caroline Maxwell wrote about this in EMRSN News in March 2013 in an article called ‘Good Pharma’ at: http://www.parkinsons.org.uk/sites/default/files/emrsn_newslettermarch2013.pdf - We will report further on this in the next edition.

The Research Support Network Strategy

A meeting of the RSN Development Team was held on 3 February with the purpose of developing a strategy for the period 2015 -19. We were represented at this meeting by our Chairman, John Telford, who reports that it was a good meeting, showing a determination that members should play a bigger role in development of the strategy.

This was the first in a series of meetings planned throughout this year and it will be the end of the year before the strategy is finally revealed. We will report further as things progress

Parkinson's research in the East Midlands

We are very privileged to have a lot of good Parkinson's-related research going on in the East Midlands.

Much of it is at the 'heavy' end – cell biology involving a lot of complex biology and genetics which is not easily understandable by the non-specialist.

But some of it is much more accessible, such as research into speech therapy, the provision of dedicated Parkinson's hospital wards, music and mobility and changes in blood flow through the brain. Adam Brown in Leicester, Rob Skelly and Fiona Lindop in Derby and Martha Hanby, also in Leicester, are running these projects in which anyone, either with and without Parkinson's, can be involved. It is easy to imagine how the findings in this sort of area could have immediate implications on the quality of everyday life for people living with Parkinson's.

Yet this could also be the case for some of the lab-based studies. A good case in point is the project of Miguel Martin's group at Leicester University. While it is about the health of mitochondria and biochemical pathways that signal that sick ones need rescuing, the significant discovery is that, in laboratory conditions, the well-known and safe vitamin folic acid can do just that: save mitochondria and stop neurons dying. The next stage, after a bit more preliminary work, is to see whether the same would be true in patients with Parkinson's. There are a couple of other research projects at Leicester University which are also discovering substances that are neuroprotective in other ways; there's real hope that one that works well as a therapy in human patients will eventually be found.

There has also been work in Nottingham that has thrown much light on what goes wrong in cells when one has Parkinson's; to over-simplify it, it is a problem with garbage disposal! Misfunctioning mitochondria also feature in the discoveries of Lynn Bedford who has been doing this research, so a more complete pattern than ever before is emerging regarding the basis of the disease.

Nottingham is also the place where Dorothee Auer, Nin Bajaj and Stephan Schwarz are involved in imaging research, using MRI scanning. This work is enabling a much more detailed picture of parts of the Parkinsonian brain to be seen and should enable the

progress of the condition to be monitored more reliably, one benefit of this being to know whether a prospective treatment is actually working.

So the East Midlands is making its contribution and holding its own in comparison with the big centres like Oxford, Cambridge, London and Sheffield! But, of course, it is not a competition.

The projects mentioned above will be discussed by their researchers at the EM RSN Researchers Meeting on 22nd March at Leicester University. Some places at this meeting will be reserved for research supporters. If you are interested in attending and can handle the science, please email me at jntelford@ntlworld.com.

John Telford

Coming Up …

22 March 2014
10.30 am to 1.00 pm

East Midlands Researchers Meeting at Leicester University

The main purpose of this is for researchers to exchange information on their work on Parkinson's amongst themselves and to discuss areas of mutual interest.
There will be a small audience of lay people, who will be present mainly to 'listen in' on the discussions. Because of time constraints, a basic understanding of Parkinson's biology by attendees will be assumed and some of the projects described will require some background knowledge to understand them fully. Time will be reserved for input from RSN members present. If you wish to take up this open invitation to attend, please e-mail John Telford on jntelford@ntlworld.com or Ian Billcliff on imb248@outlook.com

September / October 2014:

East Midlands RSN 3rd Annual Conference

2 November 2014

3rd National Research Supporters Day

Royal York Hotel, Station Parade, York
More details later in the year, but you can register your interest by e-mailing researchevents@parkinsons.org.uk or phoning 0207 963 9356

You might like to know

Parkinson's disease and melatonin deficiency – a hypothesis

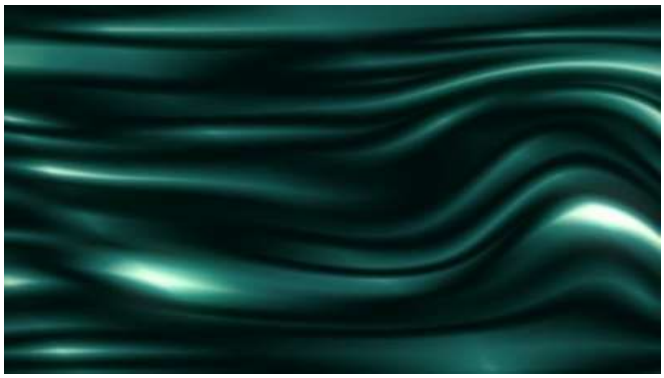
In humans one of the main functions of the pineal gland is the production of the hormone melatonin. Over the years, much research has been carried out on melatonin and its potential use in the treatment of Parkinson's, on occasion being compared against other treatments. Quite a lot of this research, but not all, has resulted in positive conclusions. It

was discovered as a powerful antioxidant in 1993 and since then its ability to protect all cells and organs from oxidative damage has been confirmed in over 1,000 publications.

The pineal gland is about the size of a grain of rice, reddish-grey in colour, shaped like a pine cone, and tucked away in the centre of the brain at the top of the spinal column between the two hemispheres. It reaches its maximum size at the age of 2 and is the only part of the brain not to have bipolar symmetry. Also, unlike the rest of the brain, the pineal gland is not insulated from the rest of the body by the blood/brain barrier. It is rich in trace elements (zinc, iron, manganese, magnesium, strontium and copper) and its function could be affected if any of these prove to be deficient. It also accounts for a vigorous blood flow.

Melatonin has been shown to have anti-ageing properties. Its use as a supplement has been shown to increase the levels of gene expression in older people. However, the pineal gland has been shown to be susceptible to calcification and the degree of calcification increases steadily with age, which leads to a melatonin deficiency.

The production of melatonin is geared to light reception in the retinas such that it follows a 24 hour cycle, known as the Circadian Rhythm. The pineal gland is active primarily during the hours of darkness, secretion of melatonin, as well as its level in the blood, peaking in the middle of the night and gradually falling during the second half of the night. 5mg of melatonin taken half an hour before retiring is sometimes prescribed as a treatment for sleep disorders. Its use at other times of the day is not recommended because of its potential narcoleptic effects.



("Lighting background" by phanlop88: freedigitalphotos.net)

Melatonin was isolated and named by Aaron B. Lerner and colleagues at Yale University in 1958. It performs a number of functions around the body, not all of which are fully understood. It has been shown that melatonin may play a significant role in modulating the effects of drugs of abuse such as cocaine. It also determines the daily sleep pattern. Some supplemental melatonin users report an increase in vivid dreaming. Extremely high doses of melatonin (50 mg) dramatically increased REM sleep time and many psychoactive drugs, such as cannabis and LSD, increase melatonin synthesis.

Incomplete clinical trials have shown that melatonin may interact with the immune system and induce the production of cytokines, a broad category of small proteins that are released by cells and affect the behaviour of other cells; they are especially important in the immune system. Some studies also suggest that melatonin might be useful in fighting infections, including viral diseases, such as HIV, and bacterial infections, and potentially in the treatment of cancer.

Research was carried out in 1999, in which rats were given either an implant of slow release melatonin, removal of the pineal gland, or exposure to constant light and then injected with either of two neurotoxins. In this research it was found that the melatonin implant actually exacerbated the resulting Parkinsonian symptoms, whereas the pinealectomy and exposure to constant light significantly reduced the severity of the symptoms.

In 2002, a review of evidence for the neuroprotective qualities of melatonin concluded that it showed important antioxidant properties and exerted important anti-inflammatory actions. Further research in 2003 concluded that the experimental data obtained suggested that the

use of melatonin in PwP would reduce the burden of the condition and proposed that epidemiological studies of individuals who use it on a regular daily basis should be carried out. Because of its low toxicity and cost it should be tested against the development or progression of Parkinson's.

Some 90% of PwP report sleep disorders among their prominent non-motor symptoms. Research was conducted in 2005, in which 40 subjects were treated with doses of melatonin between 5 and 50 mg, over a 10 week period, while continuing to take their Parkinson's medication. A significant improvement in night-time sleep was seen on a 50mg dose and significant improvement in subjective sleep disturbance, sleep quantity and daytime sleepiness was seen on a 5mg dose.

In an experiment in 2007, involving the exposure of 12 PwP to white fluorescent light for 1 to 1.5 hours shortly before bedtime, it was found that within 2 weeks of commencing the treatment there was a marked improvement in bradykinesia and rigidity in most patients. Tremor was not affected, but agitation, dyskinesia and psychiatric side effects were reduced. Other improvements included elevated mood, improved sleep and increased appetite and some patients were able to reduce their dosage of L-dopa and other medication by up to 50%.

At the same time a series of pieces of research were carried out in which neurons were exposed to toxins, such as marine acids, metals and cyanide and then treated with melatonin. The research concluded that, although there were exceptions, melatonin demonstrated its efficacy in safeguarding neurons and glia (which provide support and protection for neurons in the brain) from the persistent molecular damage that would otherwise have occurred. Neuron precursors in the brain in those select areas where cellular proliferation occurs were also stimulated by melatonin.

Research in 2010 described the toxic residue left after the processing of oxygen by cells. It stated that, although oxygen is important for the survival of neurons and glia, it also indirectly contributes to their destruction and death over time. The reason for this is that a small percentage (an estimated 1-4%) of the oxygen that enters cells is metabolised to free radicals or other oxidative compounds (known as ROS) that gradually erode and destroy essential molecules. The antioxidant properties of melatonin come in useful here.

("Neuron" by renjith krishnan: freedigitalphotos.net)

In 2011 a good description of the physiology of melatonin was published. Although there appears to be no consensus at present on levels of melatonin to be found in PwP compared to controls, it was concluded that melatonin and melatonin agonists can be useful tools in treating sleep and associated disorders in Parkinson's. Available evidence supports the inference that melatonin activity in Parkinson's can be substantial and that clinical investigations into the nature of this activity are warranted.

Summary

From the results of all these studies, the hypothesis that age-related calcification of the pineal gland leading to melatonin deficiency is a primary cause of Parkinson's may be summed up thus:-

Oxygen, the principal fuel that drives animal cells is a toxin. However evolution has produced a system whereby individual cells metabolise oxygen into Adenosine

TriPhosphate (ATP), which is better tolerated. There is, however, a toxic residue in the form of Reactive Oxygen Species (ROS), which have the capability of inducing cell death. Melatonin plays a vital neuroprotective role in rendering ROS harmless.

The substantia nigra is a major consumer of melatonin. The pineal gland, which is the prime source of melatonin and is not protected by the blood/brain barrier, calcifies through ageing. This can reduce its production of melatonin to the point where neuroprotection of the substantia nigra breaks down and all of the other Parkinson's causative factors (toxins, infection, inflammation, genetic anomalies etc), which are waiting in the wings, are given free rein to attack the dopaminergic neurons in a variety of ways.

A Holy Grail of Parkinson's research has been the search for an environmental toxin which initiates sporadic forms of the diseases. This hypothesis offers a candidate for such a toxin - it is air!

("Digital Human Cell" by rajcreationzs: freedigitalphotos.net)

The hypothesis offers scope for further research in the pathological examination of the pineal glands of Parkinson's patients, the use of light therapy, and the clinical trials of melatonin-based drugs.

Two references to scientific papers are included for those who wish to read further on this topic. There is also a trial currently being run by Dr Jalesh Panicker at University College, London, on the use of melatonin to treat nocturia, the need to get up more than once overnight to pass urine – see http://www.parkinsons.org.uk/content/latest-research-projects

Harry Pearman

References

Reiter, Russel J.; Machester, Lucien C. and Dun-Xian, Tan (2010) Curr. Neuropharmacol 8 (3) 194-210 *Neurotoxins: Free Radical Mechanisms and Melatonin Protection* (see: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3001213/)

Srinivasan, Venkatramanujam; Cardinali,Daniel P.; Uddanapalli S.; Kaur, Charanjit; Brown, Gregory M.; Spence, D.Warren; Hardeland, Rudiger and Pandi-Perumal, Seithikurippu R. (2011) Ther. Adv. Neurol. Disord 4 (5) 297 – 317. *Therapeutic potential of melatonin and its analogs in Parkinson's disease: focus on sleep and neuroprotection* (go to: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3187674/)

"Has this been researched?"

A question and answer forum on Parkinson's research

Just to remind everyone, this new feature opens up the newsletter to readers' input. You are invited to ask questions on any aspect of research or tell us about your personal experiences with, or concerns about, anything to do with research. This can be done anonymously if you wish. We will endeavour to find out answers about the topics raised, to be printed in future editions. Readers' responses and further comments are also invited for inclusion in future editions. Alternatively, we would welcome your own articles on topics that you have looked into yourself.

Questions or articles can be sent by e-mail to Ian Billcliff at imb248@outlook.com

As a starting point we are covering 'Depression'. This problem that many Parkinson's people live with has been chosen as the first subject to discuss.

Lionel's lessons

A series of plain English explanations of aspects of Parkinson's research

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Lesson 4 - Depression: A major non-motor problem for people with Parkinson's

Non-motor symptoms, such as insomnia, depression and loss of sense of smell or taste, can negatively impact on the lives of people with Parkinson's (PwP) just as badly as struggling to move properly. Yet, traditionally, researchers have tended to focus their attention on movement and tremor difficulties. To redress the balance a little, and following reports in Newsletter 12 of other non-motor symptom studies this article is a layman's guide to one of Parkinson's most common non-motor symptoms, depression.

Clinical depression is not a character flaw or weakness and it is a world away from simply saying you feel depressed because you're having a bad day. It is an illness and is a challenge to cope with, no matter how tough you are. (Winston Churchill was known for his never-give-up attitude; even so he struggled to control depression which he called his 'black dog').

Symptoms of depression

You will probably have experienced some of the symptoms below during bad times in your life, but short term distress is not depression. However, if symptoms persist for weeks, months or even years then depression is, indeed, affecting your life.

('Stressed figure with question mark' by Master isolated images; freedigitalphotos.net)

- Feelings of sadness, chronic anxiety, or hopelessness, as though life is empty and without purpose.
- Feeling guilty even though there is nothing to feel guilty about.
- An absence of self-esteem, feeling that one is worthless or helpless at coping with anything in life.
- Irritable moods or a restlessness that won't let you sit down and relax.
- Loss of interest and, in extreme cases, total apathy towards activities or hobbies that used to be very enjoyable.
- Feeling tired all the time yet experiencing difficulty with falling asleep or staying asleep – or sleeping all the time and struggling to stay awake.
- Lacking concentration, poor short term memory and struggling to make decisions.
- Overeating or loss of appetite: some people are 'comfort eaters,' others feel 'sick with worry' and cannot eat.
- Loss of libido.
- Morbid thoughts of death and suicide or suicide attempts.
- Unexplained aches and pains such as headaches, cramps or digestive problems that do not improve with treatment. (See reference 2)

Depression and Parkinson's: possible causes

Parkinson's alters the proportions of neurotransmitters (brain chemicals) inside the brain, some of which have a considerable influence on how we feel. Most of us know that dopamine and the receptors that enable it to work are reduced in the PD brain and this affects movement. But another chemical, serotonin, is also very important for regulating (good) mood. PD affects the area that produces serotonin, and studies show that in PwP the levels of 5-hydroxyindoleacetic acid (5-HIAA) in the cerebrospinal fluid that surrounds the brain and spinal cord are lower than normal. This tongue-twisting acid is a waste product of serotonin activity, and lower levels indicate less serotonin in the brain. Brain imaging studies have revealed that PwP may have an unusually high number of serotonin re-uptake 'pumps' in their brains, which are literally pumping their serotonin away. (See references 2 & 3) Another very important part of the brain for mood regulation is the frontal lobe, also known to be under-active in PwP.

The possibility that PwP experience depression in response to the chronic problems that come with Parkinson's, was investigated as long ago as 1989. Depressive symptoms were compared in 45 PwP and 24 disabled controls who had the same level of disability but did not have Parkinson's. PwP achieved significantly higher scores than the control group on the Beck Depression Inventory (BDI), especially on BDI items grouped to reflect cognitive and depressive symptoms. Regardless of age, sex, length of time with Parkinson's or **clinical ratings** of disability, the strongest predictors of depression for PwP were **self-rated** disability (i.e. individuals were affected by their own perception of their disability) and the number of recent medical problems being endured. The study concluded that depression in PD is probably not a reaction solely to disability.

Depression: Risk factors

Not everyone with Parkinson's suffers from depression. Scientific studies into how many PwP are affected have produced figures varying from 15% to 60%. Approximately half of these numbers meet the criteria for severe episodes of depression, the other half experiencing a slightly less severe form known as dysthymia.

Lower levels of 5-HIAA, a past history of depression and greater functional disability are all associated with a higher risk of depression in PwP. Other possible risk factors include early-onset Parkinson's and being female (see reference 3).

Effects and treatments

People with both depression and Parkinson's tend to experience greater levels of anxiety and difficulties with concentration, but lower levels of sadness and guilt than those with depression alone. PwP with depression also experience more difficulty with movement and daily activities, making them more likely to begin medication for motor symptoms sooner than those without depressive symptoms. Most importantly depression decreased their quality of life and made motor symptoms worse - **but treating the depression rather than the movement problems gave the best results, improving both quality of life and movement abilities (see reference 1).**

("Think Positive" by jesadaphorn: freedigitalphotos.net)

This article cannot suggest courses of treatment, only your specialist nurse or consultant can do that, but I will mention a couple of remedies that can be prescribed these days: jargon refers to them as CBT and SSRIs.

Cognitive behavioural therapy (CBT) is a type of psychotherapy that helps people to change negative thinking patterns and behaviours which may be contributing to their depression. Please be aware this only works if you seriously apply yourself! It takes determination and persistence, but if you work hard at it, it can be very effective.

Selective Serotonin Re-uptake Inhibitors (SSRIs) and Serotonin and Norepinephrine Re-uptake Inhibitors (SNRIs), are antidepressants designed to moderate the activity of the 'pumps' which are removing too much serotonin from the brain. These medications are not an instant treatment; they can take weeks to begin making an appreciable difference and then have to be maintained, often for long periods, until gradually reduced under medical supervision. But they too can be very helpful. (See reference 2)

DIY treatments

Much has been said about exercise (any kind of exercise). Many people find it helps improve their mood (I am one of them), but there is surprisingly little hard evidence to show whether it has an actual physical influence on the areas of the brain that deteriorate. In the same way that stroke victims brains 're-route' transmission in the brain to unaffected areas, some believe that exercise does the same, by creating and using new pathways as the old ones fail. This is a huge subject and I invite your comments and experiences with exercise.

And finally, unscientific as this may sound, the old saying 'it is better to give than receive' is very true! Help somebody else out and realise you are not hopeless and useless. Between you and me, that's why I became a volunteer for the Steering Group.

Reference 1: BMJ 2000; 320 doi: http://dx.doi.org/10.1136/bmj.320.7245.1287
Reference 2: http://www.nimh.nih.gov/health/publications/depression-and-parkinsons-disease/index.shtml
Reference 3: http://psycnet.apa.org/psycinfo/1992-28033-001

A personal perspective

I was diagnosed with Parkinson's in 2003, but in the 1990's I had two severe bouts of Depression the second of which had me off work for 6 months and I had a (self referred) week in a Psychiatric Hospital during that time - I have to say that experience was horrible and I just got myself together and talked my way out.

I did not know anything about Parkinson's then (I was under severe pressure at work and had some personal issues at the time of the second bout - not sure that was true of the first bout), but there is much current evidence to suggest that these bouts were the start of my Parkinson's.

("Train Tunnel" by Sura Nualpradid: freedigitalphotos.net)

During this second bout, I was prescribed Seroxat (paroxetine) anti depressant tablets - I was told by my GP that I had a shortage of serotonin in my brain and would probably have to take these for life and I take them to this day. I was also prescribed Zimovane (zopiclone) sleeping tablets, which I also take to this day. I had weekly counselling sessions with my GP who was marvellous. I found no benefit from psychiatrists.

I have to say that my depression is very much a thing of the past now by continuing to take the tablets. Even the diagnosis of Parkinson's did not cause me to get depressed (there was the usual period of denial of course for about 18 months to two years). The only symptom similar today is that when I go "Off" I sometimes feel very nervous and anxious but that passes as soon as I medicate.

When I was lucky enough to meet Roger Barker of Cambridge six years ago, he recommended Azilect (rasagaline) to compliment my drugs cocktail. He did warn there might be contra indications with my anti depressant medication and indeed there was, but I contacted as many neurologists that I could (it is amazing how many are very happy to respond to Email, phone calls) and was told the risks (of Serotonin Syndrome) were very small. I took the Azilect and it changed my life - it made everything work!

I now lead a very full and active life in Derbyshire and do boxing training twice a week, circuit training once a week, yoga once a week, weight training twice a week and still use my chainsaw to lop off branches so that I can chop up logs to fuel the fire.

Speech, Walking and Balance are my major symptoms of "The Beast" but with a positive attitude - so very important for PwP - everything is possible.

Bob Raeburn

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EMRSN News is published monthly by the Steering Group of the East Midlands Research Support Network (RSN). The RSN brings together people driven to help find a cure and better treatments for Parkinson's. Through our network, anyone can get involved in research and raise funds and awareness for Parkinson's research.

The views expressed EMRSN News are not necessarily those of the Editor, the Editorial Group, the EMRSN Steering Group or Parkinson's UK.

The next deadline for the March edition is **Monday 3 March**. Please send us a copy of your newsletter and event notices.

Editor: Ian Billcliff (imb248@outlook.com)

Editorial Group: Lionel Paulo, John Telford, Chris Johnson

We look forward to hearing from you!

Our page on the Parkinson's UK web site:
http://www.parkinsons.org.uk/content/east-midlands-research-support-network