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If you’ve been following our progress (please forgive the pun), you’ll no doubt have noticed that we’re back with a new look and feel. This is because the charity is moving forward with a fresh, new identity that includes changing our name to Parkinson’s UK. I’d like to welcome you to this issue with some thoughts on what this means for Parkinson’s research.

We haven’t just changed our name, we’ve changed our whole approach. Parkinson’s is a very complex condition that has a significant effect on the lives of hundreds of thousands of people in the UK. But everyone’s different, so in the last couple of years we’ve spent a lot of time listening to people and finding out what they think are the most important things we can do for everyone affected by Parkinson’s.

Top of the list is to find a cure, so I’m delighted to tell you that we’ve made this even more central to our research priorities. We have a new five-year plan for research to take us through to 2014 – you can read more about it on page 8 – and we’ll be investing heavily in the search for a cure for Parkinson’s.

We also need to continue our effort to improve lives here and now, so we’ll make sure we keep funding research that leads to practical help in the areas that matter most.

Inside this and future issues of Progress, you’ll find information about the grants we’ve awarded to Parkinson’s researchers in the UK. As before, this will include details on new research grants and projects that are under way, or have recently finished. We’ll also keep bringing you news about the research that looks most promising, such as our feature on gene therapy on page 4.

If you have Parkinson’s, or you are a carer, partner, family member or friend of someone with the condition, I hope you’ll find some interesting information in the new issue of Progress. Do let me know what you think. We’ve got a new, easier way for you to give us feedback (details are on the inside back cover) so we can continue to improve.

All the very best,

Dr Kieran Breen
Director of Research and Development
The science of gene therapy for neurological diseases has come a long way in a short time, particularly so for Parkinson’s. We’ll take you through some of the most exciting developments, and explain how the latest human clinical trials are using ‘reprogrammed viruses’ to potentially beat the condition.

Why use gene therapy?
Levodopa is currently the standard drug treatment for people with Parkinson’s. People with the condition don’t have enough of a chemical called dopamine, because some of the nerve cells in their brain have died. When someone with Parkinson’s takes levodopa, the remaining nerve cells turn the drug into dopamine, helping to replace what has been lost through nerve cell death. In the early stages of Parkinson’s, levodopa does well at treating the common symptoms of tremor, stiffness and slowed movement. But as the condition progresses, the drug becomes less effective. Long-term use of levodopa can also lead to uncontrollable movements (known as dyskinesia). Levodopa isn’t
a cure for Parkinson’s – it can only be used to ease some of the symptoms. We don’t yet have any effective ways to treat Parkinson’s that will stop the underlying condition from getting worse. This is where gene therapy comes in and offers a real hope for the future.

So how does it work?
Genes are strings of DNA found inside all the cells in the body. They are the instruction manuals that tell different cells around the body what to do to keep us alive and well. Humans have around 20,000 of them. If a gene becomes faulty – for example through ageing, exposure to toxic substances or from an inherited condition – cells can stop working normally and may even die. This may happen in some cases of Parkinson’s.

The idea of gene therapy is to insert DNA for specific genes into the cells, in order to treat them or perhaps prevent them from dying, as happens in Parkinson’s. If it works, it could be a long-term solution for some people with Parkinson’s. However, getting the lab-made DNA inside nerve cells is tricky.

Right now, the most effective way of putting DNA into a cell is to ‘hijack’ a virus. Some viruses are very good at infecting a cell by getting inside and inserting their own DNA into the DNA already in the cell. This can cause the cells to stop working or even die.

Scientists have found a way to re-programme viruses by removing the part of the DNA that allows them to reproduce and cause disease. The trick is to let them keep the ability to infect a cell. They can then be used as a carrier to take the replacement gene into the cell, where it’s needed (Figure 1).

After many lab-based studies, gene therapy research has now entered a really exciting stage. Several clinical trials using viruses to piggy-back the genes into the nerve cells of people with Parkinson’s have been approved in the last five years. Three different approaches to the therapy have been tested, and the results are now becoming available.

1. Re-starting dopamine production
Dopamine is an important chemical that’s used all over the brain as well as in the areas that control movement. To make dopamine, the brain needs three specific genes (called TH, AADC and GCH1) to be in good working order. One approach to gene therapy has been to transport these genes into nerve cells. Having these genes inside the cell improves its ability to make dopamine. The result should be that dopamine is brought back up to typical levels within the part of the brain that’s been treated (Figure 2).
So far, one very early stage clinical trial (known as a phase 1 trial) has been done, with the main aim of making sure the treatment is safe. Five people with Parkinson’s were treated with just one of the genes – AADC. Results showed the treatment was safe and that the participants had a slight improvement in their symptoms. A second trial has been started using a larger group of people to find out more about safety and the right dose. This larger trial is the first one in which all three genes have been given together. Hopes are quite high, as previous studies that have given all three genes to animals with symptoms similar to Parkinson’s have worked well.

2. Preventing nerve cells from dying

A second approach to gene therapy is to target chemicals called growth factors that protect nerve cells, such as Dr Maeve Caldwell’s study on page 15 of this issue. Growth factors could be used to slow down the progression of Parkinson’s by preventing more dopamine-producing nerve cells from dying after the treatment has begun. The good news here is that a phase 1 trial that used genes to produce a growth factor called nuerturin to treat people with advanced Parkinson’s has shown some positive effects. People in this trial had significant benefits from the treatment without

**Figure 2:** The three main approaches to gene therapy for Parkinson’s are: 1) Re-starting dopamine production, 2) Preventing nerve cells from dying and 3) Calming overactivity in the brain.
any major side effects. However, phase 1 trials are always small and only 12 people took part. So, we don’t know yet how well it will work for a larger group or people at different stages of Parkinson’s. A phase 2 study of 60 people with Parkinson’s started recently in the US to find out more about how well nuerturin gene therapy works.

3. Calming an overactive brain

The third approach that has reached clinical trials in people aims to restore the activity of nerve cells back to normal within a brain region called the basal ganglia. This region helps to control movement, and some of the nerve cells here become overactive in people with Parkinson’s. It’s also the region that contains the group of dopamine-producing nerve cells that die.

Nerve cell activity can be turned down by a chemical called GABA that’s produced naturally in the brain. This approach to gene therapy transfers a gene needed to make GABA into nerve cells in the basal ganglia so the brain can make more of it.

The very first gene therapy clinical trial for Parkinson’s, which took place in New York, used this approach. All 12 participants did well, with significant improvements in their symptoms and no negative side effects. Their progress was followed up regularly – some people for more than three years – and they stayed free of ill effects. Four participants showed 40% improvement in their movement when they were tested in the clinic.

Later this year we should get the results of a larger phase 2 study with 44 participants that was based on the success of this first GABA trial.

Hope for the future

So at the moment it looks promising. Gene therapy for Parkinson’s has come through some small clinical trials, improving symptoms without causing dangerous side effects. But it’s still early days and research can take a long time. Clinical trials come in three phases before a treatment can be approved for routine use. Phase 3 trials need to take place in several locations, testing large groups of people over months or years to see how the new therapy compares to existing ones. For now, we can say that gene therapy has real potential to make a big impact on treatment for people with Parkinson’s.

Although none of the current gene therapy trials is taking place in the UK, you can visit parkinsons.org.uk/takepartinresearch to find out more about the different ways to get involved with research. And we’ll keep you updated on gene therapy both on the website and in future issues of Progress.

Further reading articles freely available online:


Our plans for research
2010–2014

By the end of 2009, Parkinson’s UK had spent over £45million on research. However, it is vital that we increase our investment in research to bring us closer to a cure.

Over the past couple of years, we’ve been developing a new five-year plan for research to drive our work forward through to 2014. We spoke to Parkinson’s researchers both in the UK and abroad. We also consulted with other research charities such as the Michael J. Fox Foundation in the US. Our plans set out what we need to do to make sure we’re funding the kind of groundbreaking research that will improve
treatments and find a cure. Dr Kieran Breen, our Director of Research and Development, spent the winter of 2008/09 consulting scientists and clinical practitioners with expert knowledge in many different areas of Parkinson’s and Parkinson’s research.

As a result, we’ve developed the new five-year plan for research that will benefit everyone affected by Parkinson’s. We need to be very focused so we can turn the vision of a cure into a reality.

The plan takes several important factors into account. These include the call for a cure from people affected by Parkinson’s, the expertise of Parkinson’s researchers in the UK, the research gaps that need filling, and the economic climate. All these factors influence how we can support research of the highest quality, which will benefit people with Parkinson’s in both the short and long term.

We believe we can strike an effective balance between working towards discovering a cure, and also finding better treatments to improve lives here and now.

**Our key priorities in the next five years**

- Understanding why nerve cells die in Parkinson’s
- Developing better animal models of Parkinson’s (where an animal is used for research about Parkinson’s)
- Rapid testing of new potential drug treatments
- Diagnosing Parkinson’s as early as possible

**By 2015:**

- we’ll have invested more than £70million in innovative, high-quality research, and so will substantially increase the money spent on Parkinson’s research within the UK
- we’ll drive forward the UK Parkinson’s research agenda, making sure it delivers maximum impact and investment in treating and curing Parkinson’s
- we’ll increase the proportion of money we spend on cure-related research
- we’ll effectively communicate the outcomes and benefits of our research to raise our research profile, build momentum and inspire support

**We will:**

- work with other funding bodies to make sure Parkinson’s research gets more funding
- monitor Parkinson’s research taking place around the world in order to identify pivotal developments
- communicate our research priorities to researchers
- promote innovation grants to accelerate the progress of research
- publish annual reports on the charity’s research grants to monitor the quality of the research
- communicate the impact our research has on the lives of people with Parkinson’s

Our research strategy pinpoints four major challenges in Parkinson’s research. By tackling these four priorities together over the next five years, we will move closer to a cure. However, while we are focusing on these four priority areas, it’s important to recognise that we cannot predict where the next research breakthrough will occur. We will therefore maintain a flexible and dynamic approach to make sure we stay at the cutting edge of Parkinson’s research.
The Monument Discovery Award

In the last issue we were delighted to announce The Monument Discovery Award – our biggest-ever research grant. We’re giving £5 million over the next five years to a world-class team of 12 researchers at the University of Oxford in our drive to find a cure for Parkinson’s. The award is thanks to a generous donation from The Monument Trust – one of the Sainsbury Family Trusts – and is the largest donation we’ve ever received. The research team launched the Oxford Parkinson’s Disease Centre in October 2009. This means that we can kick off our new research strategy for 2010–2014 with a truly groundbreaking contribution to Parkinson’s research. Dr Kevin Talbot is a consultant neurologist at the Oxford Radcliffe Hospitals Trust and is the deputy leader of the Discovery Award team. He told us more about why their research is vital to finding a cure for Parkinson’s.

“Only about 5% of people with Parkinson’s have genes that directly lead them to inherit the condition,” said Dr Talbot. “But most people have what’s known as ‘idiopathic’ Parkinson’s, which means that we don’t know exactly why their condition developed.”
“But it’s possible that people who develop Parkinson’s have one or more genes that make them more at risk than others. We can say this because some people get idiopathic Parkinson’s, but others don’t – even if they’ve been exposed to similar environments all their lives.”

**Three linked research themes will form the core of the research at the Centre:**

**Theme 1 – Understanding what happens inside the nerve cells that die in Parkinson’s**

The research team will be looking for rare genetic changes that may increase a person’s risk of developing Parkinson’s. Then they’ll investigate what any genes they find inside human nerve cells are doing. This will help them to work out why certain nerve cells are lost in Parkinson’s. This will complement other studies by Professor John Hardy in London which have also been funded by Parkinson’s UK.

Finding out exactly what goes wrong inside the nerve cells will give us a good chance of developing new treatments. We want to make sure the remaining nerve cells survive and work for longer inside the brain.

**Theme 2 – Developing new and better animal models of Parkinson’s**

Dr Talbot said: “Parkinson’s is a progressive brain disorder. This means that over time, the damage to cells that begins in one area of the brain will slowly become more severe. It will also spread to other regions of the brain, although this takes varying amounts of time for different people.

“Most of the current animal models of Parkinson’s are created by damaging the same type of dopamine-producing nerve cells that die in Parkinson’s. But this doesn’t really match the way the condition develops in people.”

So this strand of research will follow on from theme 1, using any likely genes they find to improve models of Parkinson’s in the mouse and rat.

“Studying the animals will give us vital information about how Parkinson’s develops in the ageing brain. The models will also make a good testing ground for new drug treatments before they reach clinical trials in people.”

**Theme 3 – Diagnosing Parkinson’s earlier – before symptoms develop**

Dr Talbot’s approach to theme 3 is based on his work in the clinic.

“At the moment there is no definitive test for Parkinson’s, so people are diagnosed based on their symptoms. And everyone with Parkinson’s is different, so while one person may have the ‘typical’ symptoms of slowed movement, stiffness or a tremor, others may not.

“This means the condition can sometimes be difficult to identify, and diagnosis may be a long time coming. People with young onset Parkinson’s can have the extra problem that it’s often just not considered as a possibility – they’re ‘too young’!”

In the third strand of research, 1,700 people with Parkinson’s, 300 people without Parkinson’s, and another 300 people ‘at risk’ of developing the condition will be recruited. The team will search for any subtle difference between the groups. DNA from skin cells, samples of blood and cerebrospinal fluid, and brain imaging will help the team find new ‘biomarkers’ – ways to spot Parkinson’s earlier.

If we can detect Parkinson’s early on, we’ll have the best chance of protecting the nerve cells that are still healthy. We may be able to slow its progress or even stop Parkinson’s altogether. So Parkinson’s UK and The Monument Trust are wishing the Oxford Parkinson’s Disease Centre every success!
Putting a stop to dyskinesia

At the University of Cardiff, Professor Riccardo Brambilla’s research group is starting work to develop a treatment for involuntary movements (dyskinesia). This is a very common problem for people with Parkinson’s. The problem isn’t caused by the condition itself; it is a side effect of some of the drugs used to control the symptoms. It is particularly a problem for people taking levodopa. We’ve given the research group a three-year grant of £251,496 to look into new ways to stop this problem without affecting the benefits of the drugs.

Levodopa has been used since the 1960s and is the main drug treatment for Parkinson’s. There is less dopamine in the brains of people with Parkinson’s, because dopamine-producing nerve cells have died. Levodopa stimulates the brain to make more of the chemical dopamine. When people with Parkinson’s start taking levodopa, it is very good at controlling their motor symptoms. But over time, most people who take it will experience the side effect of uncontrollable movements (dyskinesia). Some people can find it just as distressing as their Parkinson’s symptoms. So it’s high on the list of areas we need to understand better to improve the lives of people with Parkinson’s.

Professor Brambilla’s team has been working on some recent discoveries about how levodopa causes dyskinesia. He tells Progress:

“We’ve known for a while that long-term use of levodopa excites nerve cells in some parts of the brain. Parkinson’s happens because of the death of dopamine-producing nerve cells in a small area in the middle of the brain called the substantia nigra. This loss of cells directly affects what’s known as ‘the striatum’, which is a part of a larger section of the brain called the basal ganglia. The basal ganglia controls voluntary movement. When nerve cells in these areas are overactive, they send the wrong signals to the muscles, causing dyskinesia. As someone’s Parkinson’s progresses and more of
their dopamine-producing nerve cells die, they may have to take more levodopa. This will make the dyskinesia worse.

“New research has begun to tell us exactly which proteins and genes are involved in increasing the activity of particular nerve cells.

“We’ve got some great first results showing how some nerve cell activity (a chain of events called the Ras-ERK cellular ‘pathway’) in the striatum part of the brain might be blocked to reduce the overactivity of the nerve cells and reduce or stop unwanted movement. A cellular pathway is a sequence of proteins and molecules that can change how cells behave.

“But there is a catch. The Ras-ERK pathway is also involved in cell survival and cancer. Most of the parts of this sequence are found all over the body. So it’s very important that any treatment for Parkinson’s that affects this sequence only works on the right nerve cells in the brain. If molecules elsewhere in the body are affected, it could have a serious effect.

“Very luckily for us, there are a few of these molecules, like one called Ras-GRF1, that are found only in nerve cells in the brain. And Ras-GRF1 isn’t involved in cell survival at all, so it’s an ideal molecule to start looking at. Its normal job is to keep dopamine and glutamate levels under control. Glutamate is one of the other chemicals affected by Parkinson’s. Now, with the help of Parkinson’s UK, we are going to try to find out how well Ras-GRF1 can reduce overactive chemical signals in a mouse with Parkinson’s symptoms.

“I know that putting a stop to uncontrollable movement would have a big impact on the people with Parkinson’s who experience it. So it is a great honour and privilege to be able to work on this exciting project.”

**Targeting proteins for a new drug treatment for Parkinson’s**

We don’t fully know how and why dopamine-producing nerve cells die in Parkinson’s. But one suggestion is that something goes wrong inside the cells, which leads to a harmful build-up of unwanted protein that eventually leads to the death of the cells. We’ve given £120,894 to a two-year project led by Chris Moody, the Sir Jesse Boot Professor at the University of Nottingham. His research team will try to design and make new molecules that might stop protein build-up. This could ultimately save dopamine cells.

*Continued on next page.*
“Proteins are complex substances that take part in almost everything that happens inside the body. They’re involved in everything from making our muscles work to controlling how nerve cells work together,” says Professor Moody.

“To stay healthy, our bodies need to keep the right balance between about 25,000 different proteins. It’s a very complicated system. For example, proteins have to be folded into the right shape to work properly. This is controlled by other proteins known as chaperones. Meanwhile, unwanted proteins, such as any that are folded the wrong way, have to be broken down and removed. When this happens, things can go wrong.”

In this project, Professor Moody’s team will concentrate on chaperones that are involved in shaping, breaking down and removing proteins.

The best-known chaperone is called heat shock protein 90, or Hsp90 for short. More Hsp90 than normal has been found in the brains of people with Parkinson’s, as well as in an animal model (where an animal is used for research into Parkinson’s). One animal model has shown that stopping Hsp90 from working when there’s too much of it around can protect dopamine-producing nerve cells from dying.

“Before the dopamine-producing nerve cells die, clumps of protein called Lewy bodies start to form inside the cells,” continues Professor Moody. “We’re going to try to stop the protein from forming Lewy bodies by blocking Hsp90 with new molecules.” You can read more about Lewy bodies in the Autumn 2009 edition of Progress.

“We want to know more about how blocking Hsp90 stops dopamine-producing nerve cells from dying. We’ll use different ways to make new molecules using a natural substance that can stop Hsp90 from working. Once we have designed and made a few molecules, we’ll compare them to see which work best.

“Ultimately, our aim is to make new drug treatments that can then be tested and used in the clinic. If we can stop the cells from dying, it could become possible to stop the progress of Parkinson’s and let people live free of symptoms.”

How does Parkinson’s spread through the brain?

Parkinson’s is a progressive condition. This means that symptoms are likely to get worse over time. The movement symptoms that people with Parkinson’s experience are caused by the death of dopamine-producing nerve cells. Current treatments help to control symptoms, but there aren’t any that can slow or stop the nerve cells from dying. As time goes on, more of the brain is affected and new symptoms can appear. In a three-year project at University College London, Professor Tamas Revesz will use our award of £319,334 to map the spread of Parkinson’s through the brain.
As Parkinson’s progresses, it spreads further than the areas of the brain that control voluntary movement. As it affects other parts of the brain, problems such as memory loss, depression and trouble sleeping can add to the symptoms that each person with Parkinson’s has.

Instead of looking at how dopamine-producing nerve cells die, Professor Revesz and the UCL team will look at the different paths that Parkinson’s takes through the brain as the condition progresses. They hope to discover a treatment that can stop it from spreading.

We already know that Parkinson’s spreads slowly from one part of the brain to the next in a similar pattern in everyone with the condition. But we don’t know what leads to this spread, because the research is usually done once the condition has already damaged many cells. By this time it’s too late to find out what could predict how the condition spreads. And the condition doesn’t spread in the same way in the brains of animals as it does in humans, so there isn’t an animal model (where an animal is used for research about Parkinson’s) that shows this.

We also know that some people who have had tissue transplanted into their brains to replace the lost dopamine-producing nerve cells have later shown signs of Parkinson’s in the transplanted tissue. So we need to know how each part of the brain affects the areas nearby, to find out why this happens.

In this project, the team will study human brain tissue from people who had Parkinson’s. They’ll focus on 11 key areas known to be affected by the condition, but that were not yet showing any symptoms. By comparing the proteins at work in the tissue that is and isn’t yet affected by Parkinson’s, the team hope to discover any differences that could explain how the condition spreads. This would pave the way for a treatment.

**Can CDNF stop dopamine nerve cell death?**

We’ve awarded a three-year grant of £216,686 to Dr Maeve Caldwell (in collaboration with Professor James Uney and Dr Liang Fong-Wong) at the University of Bristol. They aim to find out if gene therapy with a protein called ‘conserved dopamine neurotrophic factor’ (CDNF) can stop dopamine-producing nerve cells from dying.

At the moment there are no treatments that can stop or reverse Parkinson’s. Even though the drugs used, including levodopa, can ease some of the symptoms, they don’t stop nerve cells from dying.

We’re very excited about this project because the development of novel treatments such as gene therapy gives real hope for a future cure. You can read more about this in our feature on page 4. Dr Caldwell tells Progress what she and her research team plan to do:

“CDNF is a neurotrophic factor. That means that it helps to maintain the conditions that let nerve cells stay alive in a place where cells are dying,” she explains. “We know about a couple of neurotrophic factors that do this, but CDNF seems the most promising for treating Parkinson’s. In an animal study it has already shown it can protect dopamine-producing nerve cells in the part of the brain affected by Parkinson’s. It may also have fewer side effects than other similar proteins. But we really need to know more about it before it can be used to help people with Parkinson’s.

“We’re going to do two sets of experiments. First, we want to find out if CDNF can protect dopamine-producing nerve cells just after they’ve been exposed to a toxic chemical that produces some of the effects of Parkinson’s in animals. Then we’ll look at what CDNF can do after longer-term exposure to the toxin, when the nerve cells have already started dying.”

By the end of the project, the team aim to have developed a way to protect the dopamine cells and to stop them from dying. They also want to have a plan for making damaged brain cells better. We’ll let you know how they get on!
Risk factors for Parkinson’s

Which comes first – Parkinson’s or dry eye?

One of the non-motor symptoms of Parkinson’s you don’t hear much about is the problem of dry, inflamed and itchy eyes. But people with Parkinson’s tell us that this has a big effect on their lives. Dr Tara Moore at the University of Ulster and her colleagues at the Mater and Royal Victoria Hospitals in Belfast have been given £32,962 to look into eye trouble in Parkinson’s.

Having sore, inflamed eyelids (known as blepharitis) and getting ‘dry eye’ (when the tear ducts are drier than usual) are both problems that are often seen in people with Parkinson’s. It could be that Parkinson’s is the cause. Perhaps it’s because people with Parkinson’s blink less often. Or, it might be that some people who have a history of this kind of eye trouble go on to develop Parkinson’s. At the moment we don’t know, and this will be the first study that aims to find out.

Five hundred people with Parkinson’s will be compared to people without the condition to find out just how often blepharitis and dry eye occur. The team will also find out when the problems start (before or after Parkinson’s is diagnosed), and monitor how both the Parkinson’s and the eye problems develop. Blood samples from participants will let the team look for any genetic differences that make these eye problems more or less likely.

Connecting the eyes and the brain

“Our previous work, which we did with other research groups, has shown that eye infections can have a big effect on people’s health,” says Dr Moore. “And what we think is that if you’re prone to blepharitis and dry eye, this is linked to immune system problems. The eye is connected to the brain by nerve cells, so it may even be that some eye infections play a part in triggering Parkinson’s via a chain of events inside the cells.

“We’re going to use the innovation grant to test this new idea, so of course the results could go either way. But we can at least find out more about how these eye conditions affect people with Parkinson’s, which will help to ensure that treatment gets the right priority. And if it turns out that what happens in the eye comes before Parkinson’s, we’ll have data from this study to pave the way for some in-depth genetic research. It could open up new areas for both finding and preventing Parkinson’s early on.”

If you have any questions about the study you can email Dr Moore on t.moore@ulster.ac.uk or text/phone her on 07793 226 873.
Do other Lewy body conditions share genes that can predict Parkinson’s?

Lewy bodies are clumps of protein found inside the dopamine-producing nerve cells of people with Parkinson’s. They are also found in the brains of people with some other conditions. If we can find out which genetic changes are the same in some of the Lewy body conditions, we might be able to predict the risk of developing Parkinson’s. Professor John Hardy at the Institute of Neurology and his colleagues have been awarded £34,845 to investigate.

Goucher disease is a condition caused by a genetic fault inherited from both parents. The fault stops unwanted proteins from being removed from inside someone’s nerve cells. As a result, Lewy bodies start to form. We know that several genes are involved in cleaning up nerve cells. There are also other conditions that make Lewy bodies when two copies of a particular gene are inherited. Professor Hardy’s team will try to find out whether inheriting just one copy of a gene involved in other Lewy body conditions increases the risk of Parkinson’s. Their results could mean we have new genes to target for treatment. It could also help to shed light on the risk of Lewy body dementia, a condition where someone has the symptoms of dementia first, before developing Parkinson’s-like symptoms.

How important are the support cells in the brain in Parkinson’s?

Our brains are packed full of nerve cells, including the dopamine-producing cells that die in people with Parkinson’s. Among these are cells called astrocytes, which create the right environment to keep nerve cells alive and working properly. Dr Anita Hall and her colleagues at Imperial College London have been looking at the part astrocytes play in keeping dopamine-producing cells alive. We’ve given them a grant of £19,720 over 12 months to find out more about any differences between how astrocytes work in brains with and without Parkinson’s.

Most research into Parkinson’s has focused on nerve cells. This means we don’t yet know much about how other cells such as astrocytes might be involved in the condition. We do know that astrocytes regularly communicate with dopamine-producing nerve cells. What we need to find out next is exactly what the astrocytes do for dopamine-producing cells, and the effect that Parkinson’s has on this.

The team will study astrocytes and dopamine cells which are grown together within a dish, using new techniques that make it easier to specifically monitor the astrocytes. They’ll try to find out if there are any new targets for drug treatment, and if Parkinson’s drugs such as levodopa help or stop astrocytes from supporting dopamine cells. They will also ask whether changes to astrocytes could be used to help identify and diagnose Parkinson’s earlier.
Towards a cure

New ways to make dopamine-producing cells for studying Parkinson’s

If we’re going to find a cure, one of the most important things we need to know is how and why dopamine-producing cells die in the brains of people with Parkinson’s. We’ve given Professor Matthew Wood at the University of Oxford a 12-month grant of £35,000 to develop a ready supply of the nerve cells researchers need to find the answer.

Making dopamine-producing nerve cells in the lab is a complex process and at the moment the success rate is quite low. Professor Wood and his team will try using molecules called microRNAs to help turn stem cells into dopamine cells. MicroRNAs can control the way cells develop and one particular type, called miR-124a, specifically helps to stimulate the development of dopamine cells. People with Parkinson’s typically have less of it than other people do, and this may also help us to understand why the cells die.

The research team will try combining miR-124a with another type of microRNA, called miR-133b, which generally helps nerve cells develop. They want to know whether using the two together can up the success rate for producing dopamine nerve cells in the lab. They’ll also try to find out whether any other types of microRNA can help push human stem cells into becoming working dopamine nerve cells.

Which genes are involved in non-inherited Parkinson’s?

We’ve helped to discover some of the genes involved in inherited Parkinson’s. But much less is known about which genes might increase the risk of getting the non-inherited form of Parkinson’s that affects the majority of people with the condition. A team at Cardiff University and University Hospital of Wales, led by Dr Nigel Williams, will spend a year studying the links between specific genes that could be involved. The project will cost £35,000.

The Wellcome Trust is currently funding a study comparing the genetic code of more than 2,000 people with Parkinson’s with the codes of people without the condition. The research is designed to find key genes that differ between the groups. This could mean a big step forward in understanding Parkinson’s. But it’s likely that in addition to individual genes there will also be combinations of genes that interact with each other to increase the risk of Parkinson’s. The study might not pick up these combinations, which is where Dr Williams’ team comes in!

The research team will use the genetic data to study the way these genes work together, which might be less easy to notice than those in inherited Parkinson’s. It’s a great chance to increase knowledge of Parkinson’s genetics even further and to find new paths for future research and improved treatments.
How do dopamine nerve cells made in the brain compare with ones created from stem cells?

Human stem cells have often been in the news as a possible future treatment or cure for Parkinson’s. When you put stem cells into a lab dish with the right mix of chemicals, they can grow into nerve cells that seem very like the ones that die in Parkinson’s. But how well do lab-grown cells match those found naturally in the human brain? We’ve given Dr Patrick Lewis at the Institute of Neurology £30,593 to find out.

In a six-month project, the London-based research team will work with colleagues at the University of Edinburgh to study the two types of nerve cell. They can do this by comparing which genes are at work inside the cells.

Continued on next page.
Improving lives

Can non-invasive brain stimulation help walking and turning in people with Parkinson’s?

Most people with Parkinson’s have some trouble with getting around in everyday situations. Moving, turning and walking usually become more difficult as time goes on. So we’ve given £33,608 to Dr Geert Verheyden at the University of Southampton for a nine-month project to find out whether a new type of brain stimulation can help people with Parkinson’s to move more easily.

The drug levodopa tends to help make movement easier at first, but after a while the benefits seem to wear off and moving becomes harder again. Increasing the dose isn’t always a good option because the drug can have unwanted side effects, such as uncontrollable movement, or dyskinesia. So Dr Verheyden’s team are trying a treatment to help the symptoms of Parkinson’s that doesn’t use drugs or surgery.

Brain cells use electrical signals to communicate with each other. This means it’s possible to change how they work by passing a small electric current through them. The research team will use a technique called ‘transcranial direct current stimulation’, or tDCS, to stimulate the nerve cells in the brain.

Below: LRRK2, one of the proteins mutated in inherited forms of Parkinson’s

Continued from previous page.

Genes control which proteins each cell makes, and different types of cell do their various jobs (like making dopamine or other chemicals) because of the specific proteins they contain. Using new technology that counts the different types of protein being made by the nerve cells, the team will find out which genes are active. If it turns out that both the lab-based nerve cells and those that are in the brain make the same mix of proteins in similar amounts, then they will probably be a good match and it is likely that they will work in the same way.

Having the right sort of cell will help us to understand why the cells die in Parkinson’s, and will also let us screen for new drugs. This would also bring treatment to replace the lost dopamine nerve cells one step closer.

Breaking news: Based on some initial results gained from this project, Dr Lewis has been awarded a Parkinson’s UK Career Development Award. You can read more about it in the next issue of Progress.
Why do people with Parkinson’s have trouble swallowing?

Swallowing is a vital bodily function that usually happens about a thousand times every day. Almost everyone with Parkinson’s will have trouble swallowing food and drink as time goes on, but at the moment we don’t know enough about why. Dr Shaheen Hamdy at the University of Manchester has received £15,576 from us for a 12-month investigation into how and why swallowing difficulties develop in Parkinson’s.

Being unable to swallow can lead to serious complications, from not getting enough nutrition to life-threatening pneumonia resulting from food or drink going down the wrong way. One mystery that hasn’t been solved is why the drugs used to treat other symptoms of Parkinson’s don’t seem to improve problems with swallowing.

Dr Shaheen Hamdy

The Manchester team will use a non-surgical technique called ‘transcranial magnetic stimulation’, or TMS, to find out how the brain controls swallowing. By turning on a TMS magnetic field above a particular part of the brain that helps control movement, it is possible to make specific muscles respond. Participants will have their muscle activity and breathing monitored during stimulation, when they are either on or off levodopa medication.

At the end of the study we should find out what effect levodopa has on the amount of activity in the areas of the brain that will be stimulated with TMS. The team should also be able to see what effect the drug has on the muscles that are used for swallowing.

Abdominal massage to ease constipation in people with Parkinson’s

Constipation is one of the most common problems for people with Parkinson’s, affecting an estimated 30–80% of those with the condition. Dr Doreen McClurg at Glasgow Caledonian University has been provided with a 12-month Parkinson’s UK grant of £33,576 to test how well abdominal massage works to ease the symptoms.

Constipation may not be life-threatening but it can make people feel sick or bloated, get cramps or feel unwell. It can put pressure on the bladder, leading to embarrassing accidents, with some people then drinking less liquid, which makes the constipation even worse. It may also affect the way the drugs used to treat Parkinson’s are absorbed by the body, with the result that they don’t work as well.

Eating fibre can make bowel movements easier. But some people with Parkinson’s have trouble with chewing and swallowing, so they may not eat enough fibre. Even when people with Parkinson’s do eat ‘enough’ fibre, it may only lead to feeling bloated without helping their constipation.

Dr McClurg aims to find out if it’s practical to teach either people with Parkinson’s or their carers to treat constipation with abdominal massage. Her team will also build up evidence on the effect constipation has on people’s quality of life, and what people with Parkinson’s think of abdominal massage as a treatment.

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At the end of the study we should find out what effect levodopa has on the amount of activity in the areas of the brain that will be stimulated with TMS. The team should also be able to see what effect the drug has on the muscles that are used for swallowing.
Ongoing and completed research

An update on PROMS–PD

PROMS–PD is a major five-year study of depression and mood in people with Parkinson’s. We awarded £972,430 to a team spread across research centres around the UK for the largest single study of its kind ever to take place. Professor Richard Brown of the Institute of Psychiatry in London is leading the project, which is now in its fourth year. He spoke to Progress to let us know how it’s all going.

When someone has a long-term condition such as Parkinson’s, it’s not surprising that they’ll have worries or low mood from time to time. What did you aim to find out?

“For some people, these problems can be severe and last for long periods of time. If this happens, mood and anxiety can start to affect daily life as much or more than Parkinson’s itself. Although we now recognise the importance of depression and anxiety, we’re only just starting to understand what causes them and how they should best be treated. We’re particularly unsure whether depression or anxiety are the same in everyone, or whether they affect people in individual ways that need different approaches to treatment.”
What does PROMS stand for?
“Prospective Study of Mood States in Parkinson’s – in other words, we’re finding people and following them over the years to see who becomes depressed or anxious and who stays well. By looking at the many ways that depression and anxiety can combine at different ages or stages of Parkinson’s, we can also try to identify any groups of people that share the same pattern of problems with mood.”

So does everyone with Parkinson’s become depressed?
“We recruited over 500 people of all ages and stages of Parkinson’s. As part of the assessment, they were interviewed to find out about any problems they might have had related to depression or anxiety. The good news was that 60% of participants did not report any major problems. They tended to be people who’d developed Parkinson’s later in life and who still had relatively mild symptoms. However, almost 20% of people mentioned problems related to depression.”

What about anxiety?
“Anxiety was more common than depression. About one in three people reported severe worry, tension and anxiety, sometimes in combination with depression. Most of this group had been anxious for over a year. The anxious participants tended to be younger than the group with no problems or those with depression alone, and to have had Parkinson’s for longer with more severe symptoms. However, they tended to have fewer problems with memory and attention than the participants with depression but without anxiety.”

What do your results show so far and where does that leave us?
“These results tell us a number of important things. Firstly, it’s possible to have Parkinson’s without also being depressed or anxious. Secondly, long-term anxiety is common, and may be more of a problem for people who’ve had Parkinson’s from a younger age. Finally (for now), depression in Parkinson’s can either be part of a wider problem including anxiety, or can be part of a decline in memory and attention. We may find there’s no one-size-fits-all treatment. Instead, each group of people may need its own tailored approach. By following these people over time and seeing what happens to their problems, we’ll be in a good position to find more effective ways to manage these important non-motor aspects of Parkinson’s.”

How do you think the study will benefit people with Parkinson’s?
“Probably the most immediate benefit is increased awareness of the importance of ‘everyday’ problems such as worry. We all worry, and many people have plenty to worry about. However, when severe, as in many people that we have studied, it can be a considerable source of distress and we think it may increase the risk of developing more severe problems. Fortunately there are effective ways to manage worry, and we are starting to look into whether these prove useful in Parkinson’s. If so, we hope they can be made available to help people manage their own anxiety.

“More widely, our findings suggest that it may be misleading to lump together all people with Parkinson’s who are ‘depressed’. We suspect that those who are anxious and depressed probably need different treatments to those who are ‘only’ depressed. We hope that our results will encourage researchers to think more carefully in planning clinical trials to provide more useful evidence.”

Could ‘heat shock proteins’ be a new target for Parkinson’s treatment?
Back in summer 2007, we gave Professor Jacqueline de Belleroche and Dr David Dexter at Imperial College London £124,633 to find out whether a protein that’s one of the brain’s natural defences could be used to halt the progress of Parkinson’s. First results from the study show that nerve cell death in the most affected brain region can be significantly reduced by treatment with the protein. This opens the door for a new type of therapy.

Continued on next page.
No one knows exactly why nerve cells in a part of the brain called the substantia nigra begin to die. These cells contain the chemical dopamine, which is vital for controlling voluntary movement. Without them, people develop the motor symptoms of Parkinson’s.

We do know that a protein called alpha-synuclein forms clumps known as Lewy bodies inside the dopamine nerve cells before they die. This could well be a sign that something has gone wrong inside the cell. (You can read more about Lewy bodies in our feature in the Autumn 2009 issue of Progress.)

Two other proteins, known as ‘heat shock proteins’, Hsp70 and Hsp27, are also found inside the dopamine nerve cells that contain Lewy bodies in the brains of people with Parkinson’s. Hsp70 prevents alpha-synuclein from building up in the nerve cells of animals that have symptoms similar to Parkinson’s. And we know that Hsp27 reduces nerve cell death both ‘in vitro’ (in a lab dish) and in animal studies of other conditions such as epilepsy.

Identifying dopamine-producing cells

Being able to make the right kind of dopamine-producing nerve cell is critical for Parkinson’s research. It can help scientists develop new Parkinson’s treatments, such as cell replacement therapy, discover new drugs and find out what goes on inside the dopamine-producing cells that die in Parkinson’s.

Three years ago we awarded a SPRING (Special Parkinson’s Research Interest Group) PhD studentship grant of £53,415 to Dr Meng Li. Her team were planning to find out more about what makes up a dopamine-producing nerve cell.

A gene called Pitx3 is part of a family of genes that is essential for brain development. Pitx3 is interesting because it’s only found in dopamine-producing nerve cells. Most genes of its kind aren’t nearly as picky. This may mean that Pitx3 does a specific job within dopamine-producing cells. And if so, we need to know what that job is, and which other genes or proteins it affects. So the main aim of the project was to identify genes found inside the dopamine-producing nerve cells that are affected in Parkinson’s which could perhaps be controlled by Pitx3. Dr Li explained how they went about doing the research.

Hundreds of genes

“We worked with stem cells and mice that had been genetically engineered to produce green fluorescent protein only in nerve cells that contain Pitx3. This meant we could see and separate out the dopamine cells in the same brain area, in order to compare the two types of cell,” said Dr Li. “We were looking to find out whether there were differences between the genes each type of cell contained. Any such differences could mean that Pitx3 was involved in controlling whether some genes were active or not.”
and stroke. But until this project, Hsp27 hadn’t been studied in an animal model of Parkinson’s. Professor de Belleroche tested its potential for protecting dopamine cells. She told us:

“The in vitro work with Hsp27 led to our hypothesis that increasing amounts of the protein would prevent the dopamine nerve cells from dying and this was tested in our animal study. We thought this could be true whether the cells were under threat from the build-up of alpha-synuclein or from what’s known as ‘oxidative stress’. That’s essentially when toxic by-products, generated when cells produce energy, are not removed as efficiently as would occur typically in the brain.

“What we found was that some of the nerve cells still died when treated with Hsp27 after being exposed to a chemical that leads to the production of Lewy bodies. But what’s really encouraging is that cell death was significantly reduced by the Hsp27 treatment. This could have major implications in the development of new treatments for Parkinson’s. Our results strongly suggest that we should consider developing drugs that increase the amount of Hsp27 in the brain.”

Professor de Belleroche and the team are still analysing the results on oxidative stress. We’ll update you with any interesting news in a future issue of Progress. Meanwhile, they’ve published one report on their work in the scientific journal Trends in Molecular Medicine (on pages 1–10 of issue 584) and they have another report in preparation. The team have also presented their work at a number of scientific meetings. This means that the worldwide Parkinson’s research community can make use of the research findings from this Parkinson’s UK-funded study. We’re also funding Professor Chris Moody at the University of Nottingham to study a heat shock protein called Hsp90. You can read about it on page 14 of this issue.

Preventing the dopamine cells from dying could one day slow or halt Parkinson’s, something that is not possible with current treatments.

Current work in the lab is focusing on the new genes mda1 and mda5 and is a continuation of the Parkinson’s UK-funded project. In fact, based on the work done in this project, Dr Li has been awarded grants for further research from both the Medical Research Council here in the UK, and the European Commission.

**Publications resulting from the project**


More effective treatment

Several research studies have shown that in order to repair the loss of dopamine-producing cells in the substantia nigra (the brain area in which dopamine cells die in Parkinson’s), you need to use the type of dopamine-producing cells that are specifically found in this region. Dopamine nerve cells from other parts of the brain just don’t work as well. So finding new ways to be sure that the right kind of cell is produced means better research now, and potentially more effective treatment in future.
In late March, a group of our Research Network members and staff visited Professor Ann Ashburn and her team at the University of Southampton to see how their Parkinson’s UK-funded research was going. We awarded £75,861 for an 18-month project, now coming to an end, to learn more about how people with Parkinson’s move and turn.

People with Parkinson’s are more likely to freeze or fall when they are turning around, but we don’t know why this happens. Professor Ashburn talked to us about studying the problems people with Parkinson’s have with balance:

“We are particularly interested in the sequence of movements people use when they turn. Studies in younger people without Parkinson’s show that they start turning by moving their eyes first, followed by their head, shoulders, trunk and finally their legs. This ‘eyes-first’ sequence may be important for maintaining our balance as we turn.

“We are comparing people with and without Parkinson’s to see if there are any differences in their sequence of motions. Understanding where turning goes wrong will help us develop techniques and therapies to help people to turn more safely.”

Tracking body movements with sensors
The research team invited people from local Parkinson’s UK groups to take part in their study. Thirty-one people with Parkinson’s and 15 people without Parkinson’s volunteered. Each person completed questionnaires about falling and mobility and participated in tests of their posture and turning.

To monitor how the body moves as people turn, everyone wore a special helmet with a camera attached to it to track their eye movements. They also had movement sensors attached to their shoes, safety belt, shoulders and the helmet. Then each person was asked to complete a series of different turns, both to the left and right, while standing on a ‘pressure plate’. All the information from the camera, movement sensors and pressure plate was then fed into a computer to be analysed.

Which way to turn?
Some interesting findings have already emerged from the first stages of analysing the data:

- People with Parkinson’s in the study reacted more slowly to being asked to turn, and they took longer to complete turns.
- People with Parkinson’s seem to prefer turning in one direction rather than the other. When asked to turn in the other direction it took
people longer and they had much more difficulty keeping their balance.

- Measuring posture showed that people with Parkinson’s are bent forward at an angle that may make balancing harder.

Communicating the results
Pam Evans and Janice Russell-Taylor from the Research Network found the day interesting and were pleased with the progress the research team are making.

“This study has thrown up some very intriguing results that we’re very keen to explore further,” said Professor Ashburn. “I was struck by the preference people with Parkinson’s have to turn in one direction over the other. I think that understanding this better could help us develop strategies to help people who have difficulties with turning.

Putting a spring in your step
“I was also inspired when I attended an exercise conference hosted last year by the Parkinson’s UK group SPRING (Special Parkinson’s Research Interest Group) to study the effects that certain types of activity may have on people’s mobility. I’m currently planning a study to see if different types of dance can help people with Parkinson’s to turn.”

You can read more about dance and Parkinson’s in the Spring 2010 issue of The Parkinson.
Our supporters are the heart of Parkinson’s UK. As you may know, the work we do is entirely funded by voluntary donations. So without our supporters we would not be able to continue to fund research projects across the UK in the search for better treatments and a cure. And Parkinson’s Awareness Week is a key time when people go the extra mile to raise funds. Taking place in April, Parkinson’s Awareness Week 2010 was packed with a varied mix of fundraising activities – all helping to make sure our work can progress. Here are just a few things people got up to.

Teeing off in style
A keen golfer, Richard Lawrance chose to get involved by taking on his very own ‘Golfathon’ challenge. Richard has Parkinson’s, making this no easy task. Starting at his local golf club in Edenbridge at 7.30am, he played one hole at 18 different courses throughout the day, finally returning to play the 18th at his local club. He has already raised over £5,000 in sponsorship, with further funds expected to follow.

Raising the roof
The Wessex Male Choir – the leading choir at the charity’s annual carol concert – were welcomed by over 330 guests at a choral concert in Plymouth at the start of Parkinson’s Awareness Week. They were also joined by the Montpelier Primary School Choir.

The members of the Wessex Male Choir are committed supporters of the charity and have raised over £40,000 and counting.
Keep on running
The Virgin London Marathon falls on the last day of Parkinson’s Awareness Week and we were delighted to have our largest-ever team with 212 runners. One of our runners, Sarah Stanhope, completed the run in just over four hours. Her mother and grandmother both have Parkinson’s and she raised more than £3,500 in sponsorship. She said: “I’m thrilled to have been a part of the Parkinson’s UK team. It was an amazing experience.” This year our London Marathon team are aiming to smash last year’s total of £260,000.

All change please!
For the first time this year, our local bucket shakers were out at 15 mainline railway stations across the UK during Parkinson’s Awareness Week, including London, Edinburgh and Newcastle. Volunteers spent all day encouraging commuters to part with some spare change and raised an incredible £14,000 for our work.

Thank you to everyone who has supported us.
Working together with:
The Freemasons’ Grand Charity

As the UK’s Parkinson’s support and research charity, we are leading the work to find a cure and we’re closer than ever. All of our research programmes are funded entirely through voluntary donations, whether it’s the combination of small, but together significant, donations by individuals or through larger contributions by community groups and organisations.

The Freemasons’ Grand Charity is one example of how organisations working together can make a difference to people with Parkinson’s. The Grand Charity was established in 1981 to continue a tradition of charitable support for people in need. Since then, it has given grants totalling over £80million, helping thousands of individuals and hundreds of charities. The Freemasons’ Grand Charity has just finished funding a three-year study into a gene involved in Parkinson’s called PINK1. Findings from the study could ultimately lead to better treatments for the condition.

Having supported our work for a number of years, this latest project funded by The Grand Charity was carried out by Professor Nick Wood at University College London. The research team aimed to find out more about how mutations in the PINK1 gene might be linked to the death of specific nerve cells within the brain. PINK1 is a gene that is involved in some cases of Parkinson’s. Understanding how the gene works, and how this can be changed in Parkinson’s, will help us to unravel further the puzzle of why nerve cells die.

Professor Wood and his group are now nearing the end of this three-year research study, and the team has made some major breakthroughs. The PINK1 gene gives rise to a protein that usually helps keep nerve cells healthy. But the protein needs to be changed from its original form before it can work. Through their research, Professor Wood’s group have discovered new factors that work with PINK1 to alter it in order to allow it to work correctly. Mutations in the PINK1 gene prevent that interaction, and this eventually leads to nerve cell death. But we still need to understand more about how this happens and how we can prevent the death of nerve cells from occurring.

The team’s discovery is a significant step towards understanding how the nerve cells die in Parkinson’s, and to finding out whether and how we can rescue the cells.

This research would not have been possible without funding from The Freemasons’ Grand Charity. Laura Chapman, their Chief Executive, said: “The Freemasons’ Grand Charity recognises the devastating effects of Parkinson’s and the need to find a cure, and we are delighted our grant of £177,056 is helping to do so.”

We rely heavily on the generosity of our supporters and charitable trusts and foundations such as The Freemasons’ Grand Charity. If you would like to know more about projects funded by Parkinson’s UK or how you can help fund groundbreaking research like this then please contact us on 020 7963 9313.
Brain Donor Appeal update

In spring last year, we launched our nationwide Brain Donor Appeal. Our aim was to get 1,000 more people with or without Parkinson’s to pledge to donate their brains for Parkinson’s research – and it worked. We’re delighted to say that the appeal was a huge success and we more than doubled the target, with over 2,300 people signing up.

People from all over the UK joined celebrities Jane Asher, Jeremy Paxman and John Stapleton in pledging to donate their brain to the Parkinson’s Brain Bank. The bank collects brain tissue which can then be provided to researchers around the world.

By studying the brain tissue together with the medical and lifestyle histories of the donors, scientists can learn more about how the condition develops at different stages. This could help diagnose and treat the symptoms earlier and ultimately help find a cure.

Research using donated brain tissue has already led to major breakthroughs in the treatment and understanding of Parkinson’s. This includes the development of Parkinson’s drugs which have revolutionised the way symptoms of the condition are controlled.

We could not have done this without the generous people who have signed up. But we still need more donors – especially black and minority ethnic people and younger people. Carol Freestone, 54, lives in the Cotswolds and was only diagnosed in 2009. It took about three years of experiencing her initial symptoms before a neurologist finally confirmed that Carol has young onset Parkinson’s.

After hearing about the Brain Donor Appeal on Jeremy Vine’s Radio 2 show, Carol quickly signed up to the register. She said:

“I want to do as much as I can for others in the same position as me. If by donating my brain I can help researchers learn more about Parkinson’s, it will give me hope for the future.

“My husband David, who doesn’t have Parkinson’s, has also signed up.”

So a big thank you to Carol, David and the thousands of others who have joined us to help find a cure.
The World Parkinson Congress 2010

The World Parkinson Congress is taking place in Glasgow from 28 September to 1 October 2010. Here, our Trustee Liz Wolstenholme, CBE, looks forward to four days of all things Parkinson’s, research and beyond.

“When Steve Ford, our Chief Executive, asked me if I would like to play a part in helping to shape the World Parkinson Congress, I was pleased to accept. I’ve had Parkinson’s since 1994, and been a trustee of Parkinson’s UK for five years.

“Over the last year I have been involved with planning groups consisting of people from all across the world. We ‘meet’ by telephone. This brings some interesting challenges – a meeting called for a civilised start in Washington can be 6 am in California and even earlier in Australia! Luckily for me it has been a civilised mid-afternoon.

“What’s different about the Congress from other Parkinson’s conferences? Well, it’s an opportunity for everybody from the Parkinson’s global community to get together, to ensure that the voices of people with Parkinson’s are heard, and our interests addressed.

“This year is only the second time that the Congress has gathered, and people with Parkinson’s, carers, health professionals and researchers will meet to share the latest in cutting – edge research, and all aspect of living with Parkinson’s. For this reason, many of the sessions will be aimed at people with Parkinson’s and carers, using everyday language.

“I will be chairing a session on day two, ‘Taking charge of your Parkinson’s and making a difference in your community’, with speakers from the US, Ireland and Spain.

“I’m looking forward to meeting many Parkinson’s UK members at the Congress, and to four days of stimulating and lively sessions and debates.”

The full programme and details of how to book your place can be found online at parkinsons.org.uk/wpc. And you can watch Steve and Liz’s short film about the Congress at www.worldpdcongress.org
Take part in Parkinson’s research!

Caring for a person with Parkinson’s can be a considerable cause of stress for some people. Over time, this stress can start to affect the carer’s own health and wellbeing and even interfere with their ability to look after their partner or family member with Parkinson’s.

Although there is often little that the carer can do to change the symptoms of the Parkinson’s, there are ways to help reduce the amount of stress that they cause. Caring for Carers groups offer carers the opportunity to join together with a local Parkinson’s nurse to learn new ways to cope with the challenges of living with and looking after a person with Parkinson’s. If you live in South London or East Kent, you may be able to join one of the groups that will be running over the coming months. If you would like to find out more, speak to your local Parkinson’s nurse to see if they are involved, or call Rachael Lee on 020 7848 0749 or email her on Rachael.lee@kcl.ac.uk

Caring for Carers groups are being run as part of a research project at King’s College London, supported by Parkinson’s UK and the Edmund J Safra Foundation.

In the next issue of Progress

• Dr Iracema Leroi of the University of Manchester gives us the latest on compulsive behaviours

• We’ll report back from the 2nd Parkinson’s UK Research Conference which takes place in York on 1–2 November. And during the conference you can follow us on twitter – www.twitter.com/parkinsonsuk or read our conference blog at http://talkparkinsons.blogspot.com
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<td>Which genes are involved in non-inherited Parkinson's?</td>
<td>Dr Nigel Williams</td>
<td>Cardiff University</td>
<td>Innovation</td>
<td>£35,000</td>
<td>Dec 09</td>
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<td>K-0912</td>
<td>New ways to make dopamine-producing cells for studying Parkinson's</td>
<td>Prof. Matthew Wood</td>
<td>University of Oxford</td>
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<td>Feb 10</td>
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<td>K-0911</td>
<td>How do dopamine nerve cells made in the brain compare with ones created from stem cells?</td>
<td>Dr Patrick Lewis</td>
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<td>Innovation</td>
<td>£30,593</td>
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<td>K-0909</td>
<td>Can non-invasive brain stimulation help walking and turning in people with Parkinson's?</td>
<td>Dr Geert Verheyden</td>
<td>University of Southampton</td>
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<td>Abdominal massage to ease constipation in people with Parkinson's</td>
<td>Dr Doreen McClurg</td>
<td>Glasgow Caledonian University</td>
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<td>K-0910</td>
<td>Why do people with Parkinson's have trouble swallowing?</td>
<td>Dr Shaheen Hamdy</td>
<td>University of Manchester</td>
<td>Innovation</td>
<td>£15,576</td>
<td>May 10</td>
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<td>J-0601</td>
<td>An update on PROMS-PD</td>
<td>Prof. Richard Brown</td>
<td>Institute of Psychiatry</td>
<td>Programme</td>
<td>£972,430</td>
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<td>G-0609</td>
<td>Could 'heat shock proteins' be a new target for Parkinson's treatment?</td>
<td>Prof. Jacqueline de Belleruche</td>
<td>Imperial College London</td>
<td>Project</td>
<td>£124,633</td>
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<td>H-5001</td>
<td>Identifying dopamine-producing cells</td>
<td>Dr Meng Li</td>
<td>Imperial College London</td>
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<td>G-0802</td>
<td>Turning heads in Southampton</td>
<td>Prof. Ann Ashburn</td>
<td>University of Southampton</td>
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<td>G-0612</td>
<td>Working together with: The Freemasons' Grand Charity</td>
<td>Prof. Nick Wood</td>
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<td>£177,056</td>
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<td>G-0805</td>
<td>Caring for Carers</td>
<td>Prof. Richard Brown</td>
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### Recently started

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<th>Parkinson’s UK ref</th>
<th>Project title</th>
<th>Lead researcher</th>
<th>Institution</th>
<th>Award type</th>
<th>£ awarded</th>
<th>Start date</th>
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<td>G-0902</td>
<td>What role does the protein DJ-1 play in Parkinson’s?</td>
<td>Dr Flaviano Giorgini</td>
<td>University of Leicester</td>
<td>Project</td>
<td>£242,759</td>
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<td>H-0901</td>
<td>Finding new drug targets controlled by PINK1</td>
<td>Prof. Dario Alessi</td>
<td>University of Dundee</td>
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<td>H-0902</td>
<td>Stopping nerve cell overactivity: a new drug target for Parkinson’s?</td>
<td>Dr Susan Jones</td>
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<td>K-0903</td>
<td>Generating dopamine nerve cells from hairy skin stem cells</td>
<td>Prof. Maya Sieber–Blum</td>
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<td>G-0905</td>
<td>How does the protein DJ-1 protect nerve cells from damage?</td>
<td>Dr Gyorgy Szabadkai</td>
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<td>G-0911</td>
<td>Which proteins help dopamine-producing nerve cells develop from stem cells?</td>
<td>Dr Rosemary Fricker–Gates</td>
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<td>G-0904</td>
<td>What role does inflammation play in Parkinson’s?</td>
<td>Dr Matthew Wood</td>
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<td>F-0902</td>
<td>Producing adult stem cells from people with Parkinson’s</td>
<td>Dr Tilo Kunath</td>
<td>University of Edinburgh</td>
<td>Senior Research Fellowship</td>
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<td>G-0912</td>
<td>Using nematode worms to develop a cure for Parkinson’s</td>
<td>Dr Anton Gartner</td>
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<td>G-0914</td>
<td>What changes happen over time for people with Parkinson’s and their carers?</td>
<td>Dr Carl Counsell</td>
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<td>F-0901</td>
<td>Preventing activity-related falls in Parkinson’s</td>
<td>Dr Emma Stack</td>
<td>University of Southampton</td>
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<td>G-0913</td>
<td>A review of assistive technology for people with Parkinson’s</td>
<td>Prof. Sheila Kitchen</td>
<td>King’s College London</td>
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<td>£163,223</td>
<td>May 10</td>
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</tbody>
</table>

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*Progress* is produced by the Parkinson’s UK Research and Development team in collaboration with the Information Resources and Communications teams

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