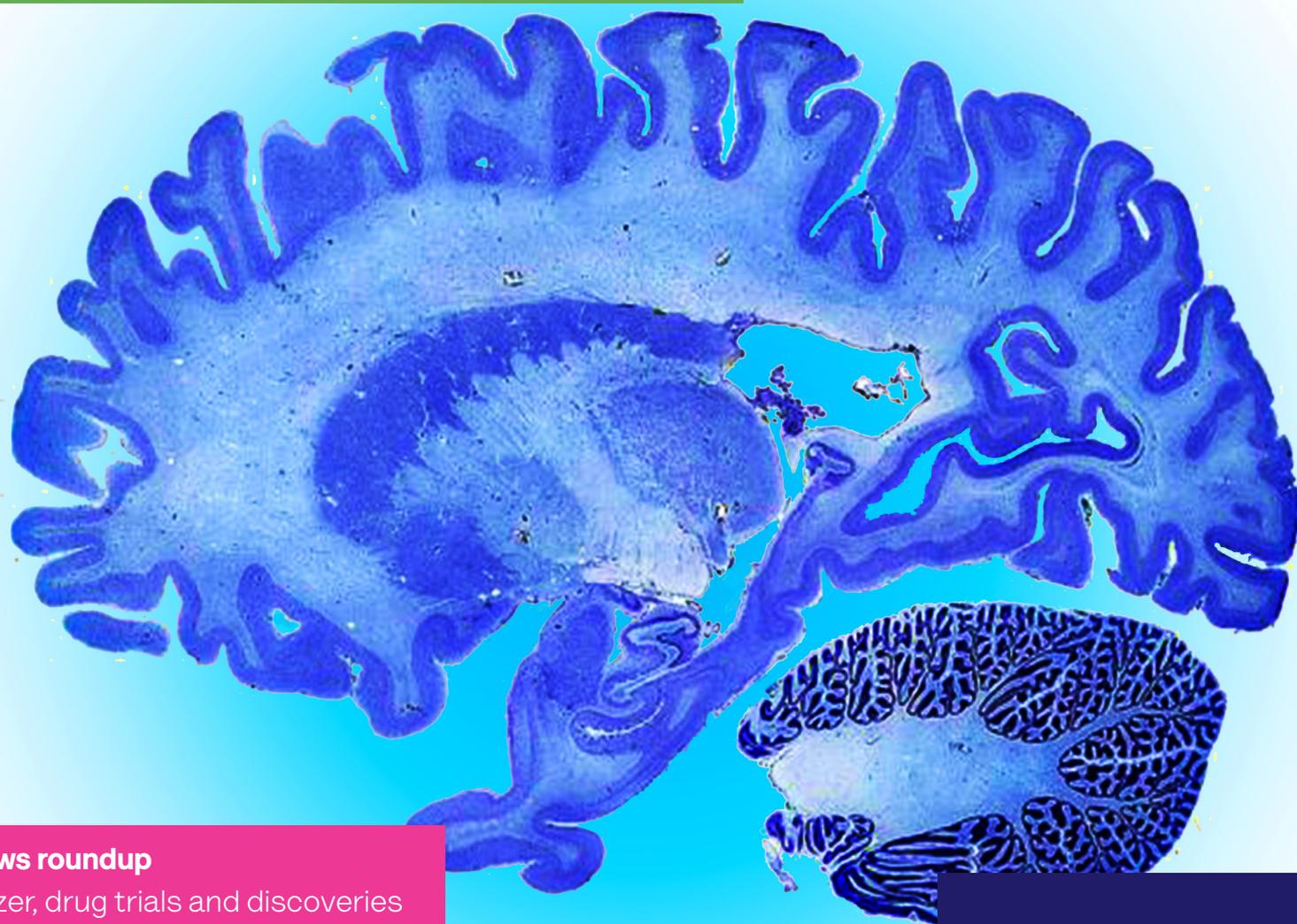


Care and cure

The Alzheimer's Society research magazine

Brains for Dementia Research

How brain donation helps research



News roundup

Pfizer, drug trials and discoveries behind the headlines

Diabetes drug for Alzheimer's

A new potential

Social withdrawal and Alzheimer's

Dr James Dachtler tells us about his research



Dr Aoife Kiely
Editor

Welcome to Care and cure magazine

This is my first issue as the new editor of Care and cure magazine. I recently joined Alzheimer's Society after years of working as a lab-based research scientist. I started my research career in my home country of Ireland, where I got my PhD in the biochemistry of Alzheimer's disease.

When I moved to the UK, I began working at a brain bank in London, helping to make sure researchers can make use of the brain tissues that people have chosen to donate after their death. This experience made me keen for my first issue to include an explanation of brain donation and brain banking. On page 6, you'll find our feature on the fascinating work of Brains for Dementia Research. I hope this will help to dispel some myths about this truly priceless gift to research.

We also cover the top dementia news stories and hear from Dr James Dachtler, who is investigating the link between social isolation and the risk of developing Alzheimer's disease (page 11). We learn more about tests to see whether diabetes drugs can treat Alzheimer's (page 8) and hear about the latest breakthrough in research into dementia with Lewy bodies (page 9).

Thank you for picking up this issue of our research magazine, I hope you enjoy it!

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About us

Since 1990, Alzheimer's Society has funded £50 million of cutting-edge dementia research. We aim to increase investment in our research programme to at least £150 million over the next decade. This money funds important research that helps to improve the quality of life of people with dementia by investigating prevention, improving practice in care and pursuing a cure.

[alzheimers.org.uk/research](https://www.alzheimers.org.uk/research)



Photo by Norbert Nagel, Mbrfelden-Waldorf, Germany

Pfizer drops out of dementia drug race

In January, news hit the headlines that pharmaceutical company Pfizer is pulling out of neuroscience research.

This means that it will no longer work on potential treatments for dementia including Alzheimer's and other conditions such as Parkinson's. Pfizer came to this decision after a number of setbacks in its research. This included the failure in 2012 of the drug bapineuzumab to help people in the mild to moderate stages of Alzheimer's. Dr James Pickett, Head of Research at Alzheimer's Society, described the move as, 'A heavy blow to the estimated 46.8 million people currently living with dementia across the globe.'

This is not a reason to lose hope. Financial giant KPMG thinks other companies are unlikely to give up, saying, 'It's too big a market to ignore. People are going to continue to go after it despite roadblocks.' Other pharmaceutical companies have since come forward to pledge their continued involvement in dementia research. For example, Lilly said that the last 30 years of hard work has taught it to better understand the condition and inspired new therapies to test. The US National Institutes of Health invested an estimated £2.9 billion into dementia research in 2017 alone, triple the amount it spent in 2013.

But it's not all about money and markets. Finding new ways to treat and cure the condition is an international effort, not only of pharmaceutical companies but of scientists working in labs in universities and hospitals.

For example, large co-operative research initiatives such as the EU-run AMYPAD, EPAD and MOPEAD are recruiting clinical study members from across Europe with the help of universities, hospitals and research institutes. These types of studies will help us learn more about why people develop dementia and why mild cognitive impairment becomes dementia in some people but not others.

'As we make progress in our understanding of the diseases that cause dementia, we hope pharma will unite with us to turn breakthroughs into treatments that could improve the lives of millions.'

At Alzheimer's Society, we continue to fund £10 million of biomedical and care-focused research each year. We have committed to raising £50 million to establish the UK Dementia Research Institute. We set this up together with Alzheimer's Research UK and the Medical Research Council to bring the brightest research minds together in excellent research facilities in the UK to tackle dementia.

'Every three seconds someone in the world develops dementia and, with this number set to rise, there has never been a more important time for such life-saving research,' said Dr Pickett. 'As we make progress in our understanding of the diseases that cause dementia, we hope pharma will unite with us to turn breakthroughs into treatments that could improve the lives of millions.'



The researchers found that, when they kept volunteers awake overnight, they had a third more amyloid-beta in their CSF than when they let them sleep. Amyloid-beta is a protein that is present in everyone's brains, but in Alzheimer's disease it builds up and forms dense clumps called 'plaques'.

This led to the question: Does a lack of sleep cause more amyloid-beta to be made, or does the process that would remove this protein need you to be asleep to function properly? To find out, the researchers 'tagged' the amyloid-beta in volunteers' brains with a fluorescent chemical so they could follow it from being made to being cleared away.

In general, they found that amyloid-beta levels were low in the morning after a good night's sleep and increased during the day up until bedtime. It was cleared out of the brain even if the volunteer wasn't allowed to sleep. This led the researchers to suggest that extra amyloid-beta must be being made in the brains of volunteers who were kept awake.

This study helps us to understand more about how amyloid-beta might be made and removed in the brains of people who don't have dementia. However, it was a very small study and isn't able to answer the question of whether a lack of sleep increases a person's risk of developing dementia. It will be interesting to follow this area of research to learn more about the mysterious function of sleep and its role in brain health.

Keep snoozing

Following on from our winter feature 'Can you sleep your way to a healthier brain?' more early stage research has come out that looks into any links between sleep and brain health.

Researchers at the Washington University School of Medicine in the US recruited a small group of eight volunteers to sleep for science. However, before the volunteers could enjoy a good night's rest they were kept awake for one whole night. Lead researchers Brendan Lucey and Randall Bateman sampled the cerebrospinal fluid (CSF) of each volunteer after one night awake and also after a restful night.

CSF is the liquid that surrounds the brain and spinal cord and that fills chambers within the brain. It can drain waste products from the brain, so seeing what is in the CSF can tell us how the brain is reacting to something.

Drug fails in Alzheimer's trial

In January we learned the disappointing news that the drug idalopirdine had failed to improve thinking ability or slow the progression of Alzheimer's in people with the condition who took part in a recent clinical trial.

In early trials, idalopirdine had looked promising. There were signs that it might improve cognition – or thinking ability – when taken alongside cholinesterase inhibitors, another type of drug commonly prescribed to treat the symptoms of dementia.

In the most recent trial, a team of international researchers tested the effects of idalopirdine in 2,525 people with mild to moderate Alzheimer's disease in

34 countries. For 24 weeks people received either the drug or a placebo, which has no effect as so is used to compare against any effect the drug treatment might have.

Unfortunately, the researchers found no improvement in cognition among people who received the drug compared to those who took the placebo. They also found no effect on the progression of people's Alzheimer's disease.

'These results indicate that this type of drug may not have the effects we were hoping for in people with Alzheimer's and we need to broaden our focus,' said Dr James Pickett, Head of Research at Alzheimer's Society.

Researchers learn from each research study and clinical trial. This means that even a negative result is something that researchers can use to help to focus on the next opportunity to understand, treat and cure dementia.



Amish fountain of youth?

The Berne Amish community is a secluded population living in north-east Indiana in the US. Isolated groups such as these are of great interest to geneticists. Their members tend to remain close-knit, rarely marrying outside the group. This means that unusual inherited features – which could make someone more or less likely to develop a disease – are more likely to be carried in the genes of both parents. These genetic features are then far more likely to be passed down to later generations than they would be otherwise.

Researchers from Chicago's Northwestern University tested 177 people from the Berne Amish community and found that some had inherited one mutated copy of a gene called SERPINE 1 along with one normal copy. People who had the mutated copy of this gene lived an average of 10 years longer than their neighbours who didn't have it. As well as living longer, the researchers noticed that these people were less likely to have type 2 diabetes or dementia compared to those without the mutated gene.

The body uses the SERPINE 1 gene to make a protein called PAI-1. During normal ageing, cells in the body receive signals from PAI-1 telling them to function very slowly. These slowed-down cells build up around the body and are part of the process that results in ageing. In people who have a mutated copy the SERPINE 1 gene, PAI-1 is turned off, so there is nothing telling their cells to slow down.

However, there is a downside. PAI-1 also works to stop the breakdown of blood clots. Most people with a mutated copy of SERPINE 1 also had a normal copy, meaning they still had half the usual amount of PAI-1 working in their bodies. For people who had two mutated copies of the gene – and no normal copy – there was no PAI-1 in their blood at all. This causes a bleeding disorder where their blood cannot clot properly when needed.

This tells us that the benefits of PAI-1 – reducing the effects of ageing and the risk of type 2 diabetes and dementia – must be weighed against the risk of causing a bleeding disorder. Trials are already underway in Japan, where researchers are using an existing diabetes drug called metformin that they hope can be used to gently 'turn down' the amount of PAI-1 in the blood.

News in brief

Ginkgo? No go

A study earlier this year showed that a combination of ginkgo biloba plant extract and aspirin helped thinking and memory problems after a stroke. Despite the researchers not testing this treatment in people with dementia, and the fact that the majority of previous research disagrees with them, the researchers guessed that this treatment could also be used to treat Alzheimer's. Our view remains that – unless there's better evidence of its effectiveness in future – this is one leaf you don't need in your salad.



A little help from your friends

Recent studies have reported that marriage could help to reduce the risk of dementia. However marriage itself may not be responsible. Instead, these studies are believed to be showing the benefits of daily social contact and meaningful friendship. Making friends and keeping a rich social life may help to reduce risk of developing dementia.

Dementia in past populations

We sometimes think of dementia as a condition of modern times. However, a fascinating study from researchers at Bournemouth and Sheffield universities suggests there may have been greater numbers of older people in the past than generally assumed and more people with dementia, going back to prehistoric times. They also explore how people with dementia may have been supported as societies changed.

Brains for Dementia Research

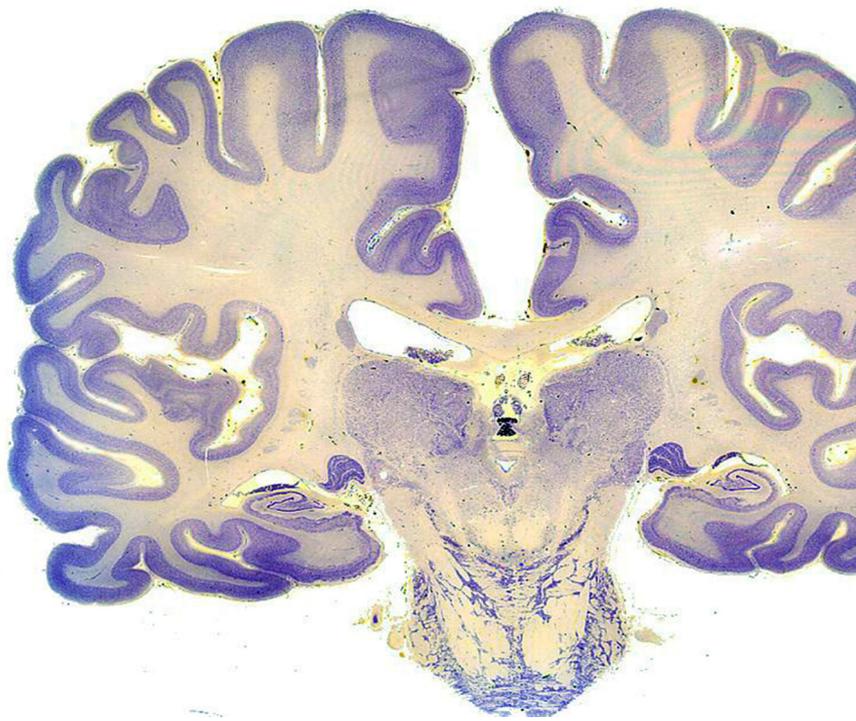
Brains for dementia research is a joint initiative of Alzheimer's Society and Alzheimer's Research UK. It receives brain donations and makes excellent quality brain tissue and medical information available to biomedical researchers.

A human brain contains billions of brain cells, connected through incredibly intricate networks. With every sensation, thought and emotion, these cells communicate with each other in many complex ways. Not surprisingly, the diseases that damage this elaborate system can also be complicated, and they are notoriously difficult to study.

The diseases that cause dementia are some of the most challenging for us to understand. They affect multiple regions of the brain and cause symptoms ranging from memory loss to personality changes and language difficulties. To learn more about these diseases, researchers can grow brain cells in a dish or use animals bred to have the symptoms of dementia. However, there are many things that we can only find out by studying the brains of people who actually lived with the condition.

Brain banks receive, examine and store brains from donors. They are an important resource for researchers to study the impact that dementia had on a person's brain while they were alive. For dementia researchers, the ideal brain bank would provide samples of brain tissue for them to study the many different diseases that cause the condition. However, this is impossible for a single brain bank to do.

Brains for Dementia Research was set up to provide researchers with the brain tissue they need by bringing together a network of excellent brain banks. A collaborative project between Alzheimer's Society and Alzheimer's Research UK, it consists of centres in Bristol, Cardiff, London, Manchester, Newcastle and Oxford. Each centre receives donations of brains from people who had dementia, but they also welcome brains donated by people who do not have dementia. Researchers use these to compare changes that have taken place in the brains of people who had dementia.



Since it was founded in 2007, the public response to Brains for Dementia Research has been very positive. Over 3,200 people have signed up to donate their brains to research, with over 700 brains received from people who have died. The value of each brain donated is immense – tissue from a single brain can be used for up to 200 research studies.

Why is this project special?

As well as providing brain tissue for research into a broader range of diseases, Brains for Dementia Research also makes sure the donated tissue is looked after well and that there is better information about each donation.

A big problem faced by other brain banks is that it is difficult to get detailed medical records and genetic information for each brain donated to them. This is frustrating for researchers, as it makes comparing different cases of dementia more difficult.

'Brains for Dementia Research is now the "gold standard" for brain tissue banking,' says Dr Helen Costello, the project's Co-ordinating Senior Manager. 'Monitoring each prospective donor throughout their later life provides researchers with extensive medical information, allowing them to see how brain changes match symptoms.'

After the brain donor has died, their brain is brought to a brain bank as quickly as possible. It is examined by a neuropathologist, a person who specialises in identifying disease and injury in the brain tissue of someone who has died. The neuropathologist examines the whole brain before

using a microscope to detect the tiny changes in the delicate architecture of the person's brain that have been caused by dementia. This is how the neuropathologist can confirm the diagnosis of the type of dementia that the person had – examining the brain after death is still the only way to do this conclusively. As well as being vital information for researchers, an accurate diagnosis can be important to family members and friends, who may be able to better understand the condition that affected the person they lost.

How does brain donation help?

Since Brains for Dementia Research was established, it has supplied researchers with over 80,000 samples of brain tissue. Each sample is typically very small, weighing no more than a postage stamp. The majority of tissue has been used in research based within the UK. Around 20% of samples have been sent to research groups across the globe.

The extensive list of breakthroughs made using tissue from this project is impressive. They include advances to improve the diagnosis of vascular dementia and the identification of new risk factors for dementia. Researchers discovered a chemical interaction that is unique to Alzheimer's, and are developing therapies to target this to prevent the disease from progressing. In a large-scale study, researchers uncovered the unique genetic profile of dementia with Lewy bodies that will inform years of future research (see page 9).

How do researchers apply for tissue?

Brains for Dementia Research makes sure that the donated tissue in its care is used ethically in good quality research. Researchers submit a detailed application that is reviewed by an ethics committee so that the brain tissue, information about the donor and the wishes of their family are all respected.

Although this review process is thorough and done with great care, it is also efficient. While other brain banks can take weeks or even months to review applications, the Brains for Dementia Research committee reviews them within one week.

All going well, staff at the project co-ordinate how the researcher receives the brain tissue they need for their work. To study some rarer types of dementia, tissue may come from several different brain banks in the network so that the researchers have enough samples.

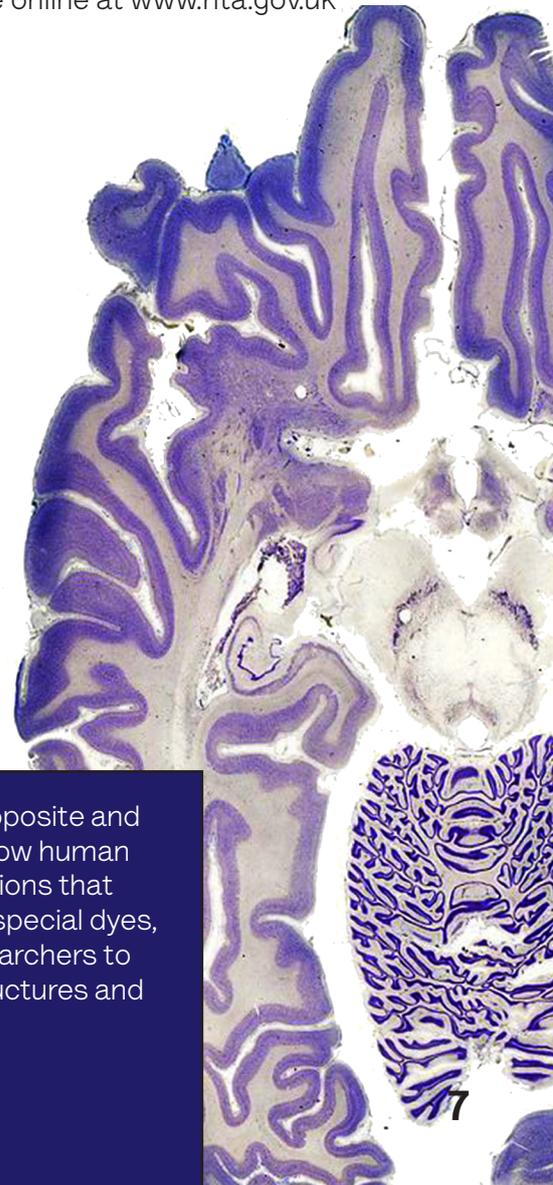
What does 2018 hold?

The focus of Brains for Dementia Research has now switched from recruiting brain donors towards promoting the quality and availability of tissue for researchers.

'In 2018 we move to our next exciting stage, during which further important information on participants will be collected, including more detailed measures of frailty and vascular disease,' says Professor Alan Thomas, Brains for Dementia Research Director. 'This will enhance the usefulness of the brain tissue to researchers, as well as providing important clues about the relationship of such factors to ageing and dementia.'

Running a network of brain banking centres is a worthwhile but very expensive task. We would not be able to run Brains for Dementia Research if it wasn't for the generosity of our supporters. If you would like to give a gift today to support this essential resource, you can donate online at alzheimers.org.uk/brainbanks or call 0330 333 0804.

Although Brains for Dementia Research is no longer seeking donors other brain banks are still are, find out more online at www.hta.gov.uk



Images (right, opposite and on the cover) show human brain cross-sections that are coloured by special dyes, which allow researchers to look at brain structures and cells.



Professor Christian Hölscher.

Diabetes drug for Alzheimer's

A drug normally prescribed for people with diabetes has been shown to reverse memory loss in mice that were bred to develop the symptoms of Alzheimer's disease.

Researchers have known for some time that having type 2 diabetes increases your risk of developing Alzheimer's disease. It can also make Alzheimer's progress more quickly. Type 2 diabetes is caused by someone either not having enough insulin or the cells of the brain no longer responding to insulin. Insulin is important, as it controls the amount of sugar in the blood and has been shown to protect brain cells. Researchers had noticed that, in the brains of people with Alzheimer's, some of their brain cells were not responding to insulin. This led them to become interested in whether Alzheimer's disease might be a sort of 'diabetes of the brain', that might be possible to treat it with modified diabetes drugs.

In the recent study, Professor Christian Hölscher's laboratory treated the 'mouse model' of Alzheimer's disease with a drug that activates three chemical chains of events affect the body in a similar way to insulin. When these are switched on, they trigger processes that help to protect brain cells.

The mice from this 'mouse model' have the same clumping of toxic amyloid protein in the brain that people with Alzheimer's do, along with similar changes in how their cells make energy and communicate with one another. They treated the mice with either a placebo that would produce no effect or the triple-action drug.

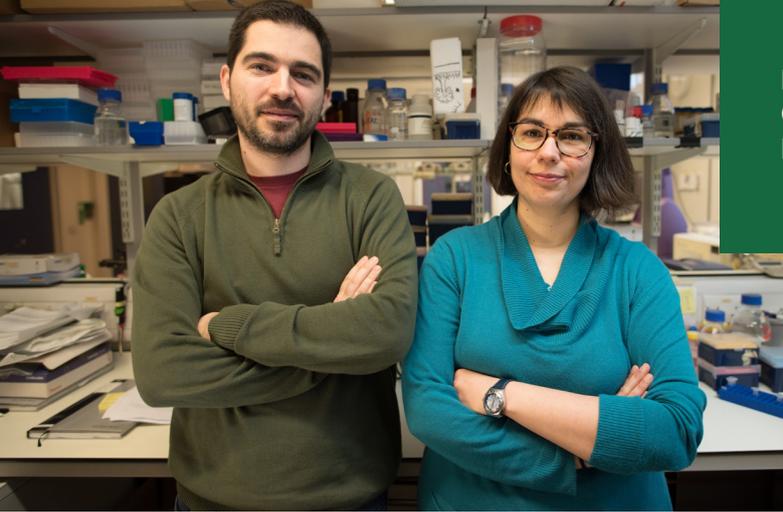
After two months of daily treatment with the triple-action drug, mice performed better on memory tests than untreated mice. The scientists also found that the brains of treated animals showed higher levels of a chemical that works to keep brain cells functioning. In the treated mice, they saw less clumping of amyloid protein, fewer signs of stress and more surviving brains cells than in mice that were given the placebo.

'This new approach shows great promise. Initial pilot studies in people with Alzheimer's or Parkinson's disease testing earlier versions of this drug type have already shown promising results,' said Professor Hölscher. 'Hopefully we will be able to find a treatment that can stop this dreadful disease.'

We must remember that this is only the early stages of testing and the drug has only been shown to work in mice so far. Humans are far more complex than mice, so we will need to wait a few more years for the results of clinical trials to tell us if the same positive effects are found in people. All going well, this is a finding could revolutionise the search for a cure for Alzheimer's. Up until now, the treatments undergoing clinical trials have focused on targeting toxic amyloid directly.

'This new approach shows great promise. Initial pilot studies in people with Alzheimer's or Parkinson's disease testing earlier versions of this drug type have already shown promising results.'

This research has opened up a whole new area of potential new treatments for Alzheimer's disease. The next steps is for the triple-action drug to be tested in healthy humans. This will confirm whether it is safe for people to use. A trial currently testing a similar drug in people with Alzheimer's disease, is partially funded by Alzheimer's Society. In a few years a new, more advanced drug may be ready to be tested in people who have dementia. We look forward to hearing about the future of this exciting field of research.



Dr Jose Bras and Dr Rita Guerreiro in their UCL lab in London.

Breakthrough in dementia with Lewy bodies

Researchers in London make a landmark genetic discovery about dementia with Lewy bodies.

Dementia with Lewy bodies (DLB) is a type of dementia that shares symptoms of Alzheimer's disease and Parkinson's disease. A person with DLB might have symptoms including dementia, visual hallucinations, slow movement and stiff limbs. Like those who have Parkinson's disease people with dementia with Lewy bodies have dense round spots and wispy threads of alpha-synuclein protein in their brains. People with DLB can also show the signs of Alzheimer's disease in their brains such as twisted tangles of tau protein and in some cases clumps of sticky amyloid-beta protein.

Until now, it has been unclear whether your genes might play a role in how likely you are to develop DLB. In a study that is the first of its kind, Alzheimer's Society-supported researchers Dr Rita Guerreiro and Dr Jose Bras have discovered a group of genetic factors that increase the risk of having DLB. They propose that as many as 36% of cases in the sample studied might have been influenced by these and other, still to discovered, genetic risk factors.

The aim of this study was to discover any potential genetic factors that could change a person's risk of developing DLB. Geneticists tackle this sort of question by looking at the genes of as large a group of people living with the disease as they can. The

researchers took on the huge task of testing 8.3 million genetic factors in 5,778 people (1,324 people with DLB and 4,454 without) from 10 countries.

The results were organised into what is called a 'Manhattan plot', a type of graph that resembles the densely packed New York skyline. Each 'skyscraper' in this graph represents a genetic marker that the researchers tested. The tallest represent the factors that are most strongly linked to DLB.

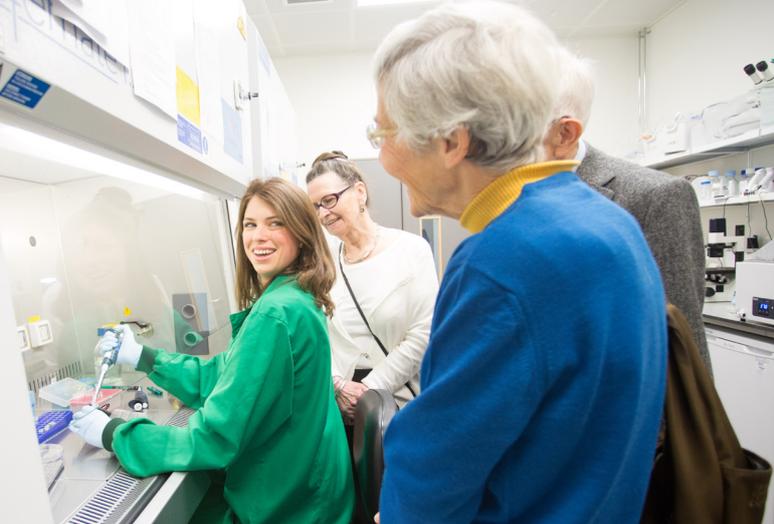
'These results offer us, for the first time, a way to start to differentiate DLB from Alzheimer's and Parkinson's from the most relevant perspective – their molecular basis,' said Dr Guerreiro.

Drs Guerreiro and Bras discovered four genetic factors that are linked to people developing DLB. This finding has been welcomed as a breakthrough, since it shows that DLB is a genetically unique disease. DLB will no longer be assumed to be caused by a mixture of the genetic risk factors for Alzheimer's and Parkinson's.

DLB has not been researched as much as Alzheimer's disease and this study is a big step forward. It highlights our commitment to funding research to understand and treat all forms of dementia.

Genetic factors like these can increase risk of developing DLB, but you can reduce the risk of developing dementia by making lifestyle changes such as staying active, keeping up social interactions and not smoking.





Network volunteers said that learning more about dementia made them feel better able to cope with their own experiences of the condition, and optimistic that research will help others in the future.

‘Just seeing the development of the research from the beginning, and how encouraging that it was actually going to fulfill its potential,’ said Elizabeth.

Researchers also told us about the positive impact that the monitors have on their work. They described how this kind of contact with volunteers had changed their views on how best to conduct research and involve people affected by the condition.

‘Just seeing the development of the research from the beginning, and how encouraging that it was actually going to fulfill its potential,’

Mario a biomedical researcher said, ‘In a single interaction the wealth of knowledge, information and feedback that you can gather is incredible as compared to other contexts’.

We encourage researchers to speak to the network at every stage of their project’s design, and to involve volunteers as collaborators and equal partners.

Members of the network felt that the range of their backgrounds strengthened their contribution. Whether former school teachers, occupational therapists, engineers or home makers, the volunteers all have skills and experience to contribute. By sharing their different perspectives, they are able to shape the research landscape to meet the needs of everyone affected by dementia.

We hope to grow the Research Network and to encourage more people with dementia to volunteer by making sure that the roles of members are accessible.

If you are interested in becoming a member of the network, visit alzheimers.org.uk/researchnetwork or call 020 7423 3603.

Nothing about us without us

The phrase ‘nothing about us without us’ sums up a lot about the spirit behind our Research Network.

Since it was established in 1999, the network has put people affected by dementia at the centre of everything we do in our research programme. The Research Network currently consists of 250 volunteers who actively contribute to improving Alzheimer’s Society-funded research. They include people with dementia, their carers and former carers.

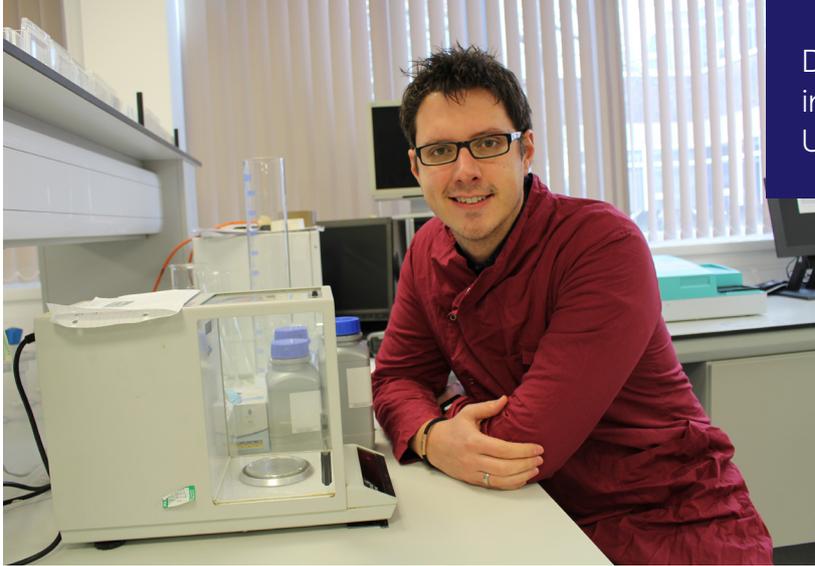
Network volunteers read short summaries of research grant applications submitted to us and give their opinion on whether they think we should fund them, providing over 5,300 reviews in the past year. They also work with researchers, with 137 volunteers acting as ‘monitors’ – visiting the researchers they have been paired with and providing input into their research.

Anna Grinbergs-Saull, our Research Engagement Officer, interviewed members of the network to learn more about their motivations for taking part.

Keith said, ‘I didn’t want to be a participant in a drug trial – I didn’t feel entirely comfortable with that – but I did want to do something linked to this, so the network was an ideal way of doing it. I wanted to broaden the network to include people with dementia like myself.’

‘If research doesn’t have any practical implications, then it is research for research’s sake,’ said Kieran, another volunteer. ‘Is it going to help or is it going to make a difference?’

Dr James Dachtler
in his lab in Durham
University.



called electrophysiology to see how well cells in the amygdala communicate with each other. Then we will use a brain scan technique called diffusion tensor imaging to see how well the mouse's amygdala connects to other brain regions (see the image below). If the cells aren't communicating clearly with each other inside the amygdala, and if the connections that link the amygdala to other brain areas are weak, then the instructions that help the mouse know how to behave and react to others aren't been sent.

Social withdrawal and Alzheimer's

Who are you?

My name is Dr James Dachtler. I am a researcher who is interested in understanding the role of social withdrawal in Alzheimer's disease. I am in my second year of a three-year research fellowship at Durham University.

What are you doing?

We tend to think of Alzheimer's disease as mainly affecting memory. While this is true, we know that changes in behaviour happen too. In some people, social withdrawal – avoiding friends, family and activities that they liked before – occurs years before Alzheimer's is diagnosed. Other researchers have shown that, while living with Alzheimer's disease, the more people you meaningfully interact with, the slower your onset of memory problems. An area of the brain called the amygdala is affected in the earliest stages of Alzheimer's. This has been called the 'social area' of the brain because it helps us to engage in interactions with other people.

So far, researchers have not been able to explain why social withdrawal happens in Alzheimer's. We want to see if social withdrawal relates to changes taking place inside the amygdala. Using a mouse model of Alzheimer's – mice that have been bred to have the symptoms of the disease – we will watch how they interact with each other and how well they remember each other.

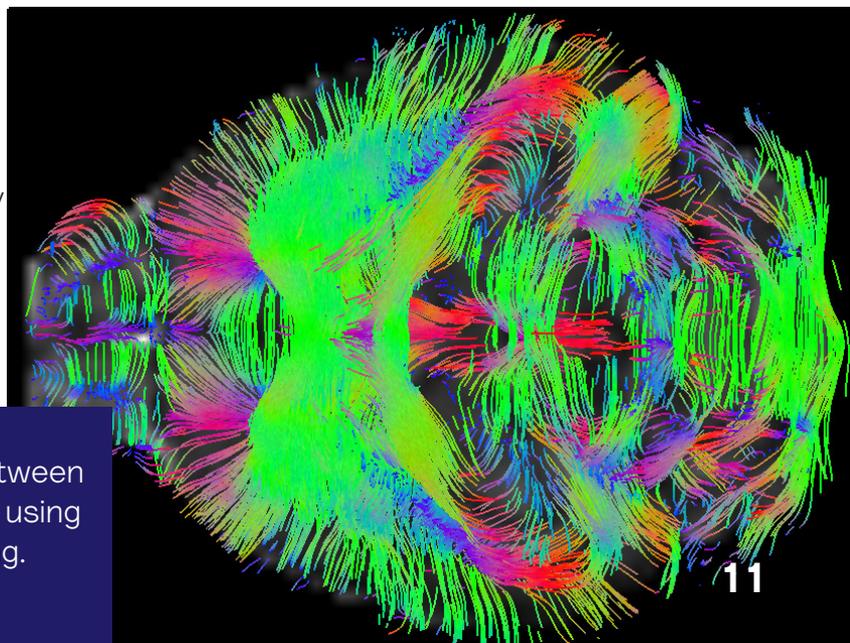
Then, to see if social withdrawal in these mice is caused by brain changes, we will use a method

Why do this research?

Last year, a study showed that being married lowered your risk of developing dementia. Alzheimer's Society activities such as Singing for the Brain are also beneficial for the symptoms of dementia. Why? We believe that this is because of meaningful social engagement. This means it is important to know whether social withdrawal is a symptom of Alzheimer's disease. By investigating the effects of dementia on the amygdala, we will be able to find out how to support people living with the condition more effectively.

What's next?

Since starting this study we are now running a clinical trial. This trial looks at whether changes in social behaviour show that mild memory issues will progress to Alzheimer's disease. If this trial goes well, we will apply for funding for larger clinical trials with the NHS for drug therapies that improve social motivation and so may slow the progression of the disease.



This image shows the connections between different areas of a mouse brain, seen using diffusion tensor imaging brain scanning.



**Research
is our
only hope
to beat
dementia**

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Call 0300 222 1122 and ask about Join Dementia Research.

Join our Research Network

Involving people with dementia, carers and former carers to influence our research.
alzheimers.org.uk/researchnetwork

General enquiries

enquiries@alzheimers.org.uk

Aoife Kiely

Research Communications Officer
aoife.kiely@alzheimers.org.uk
020 7423 3522

