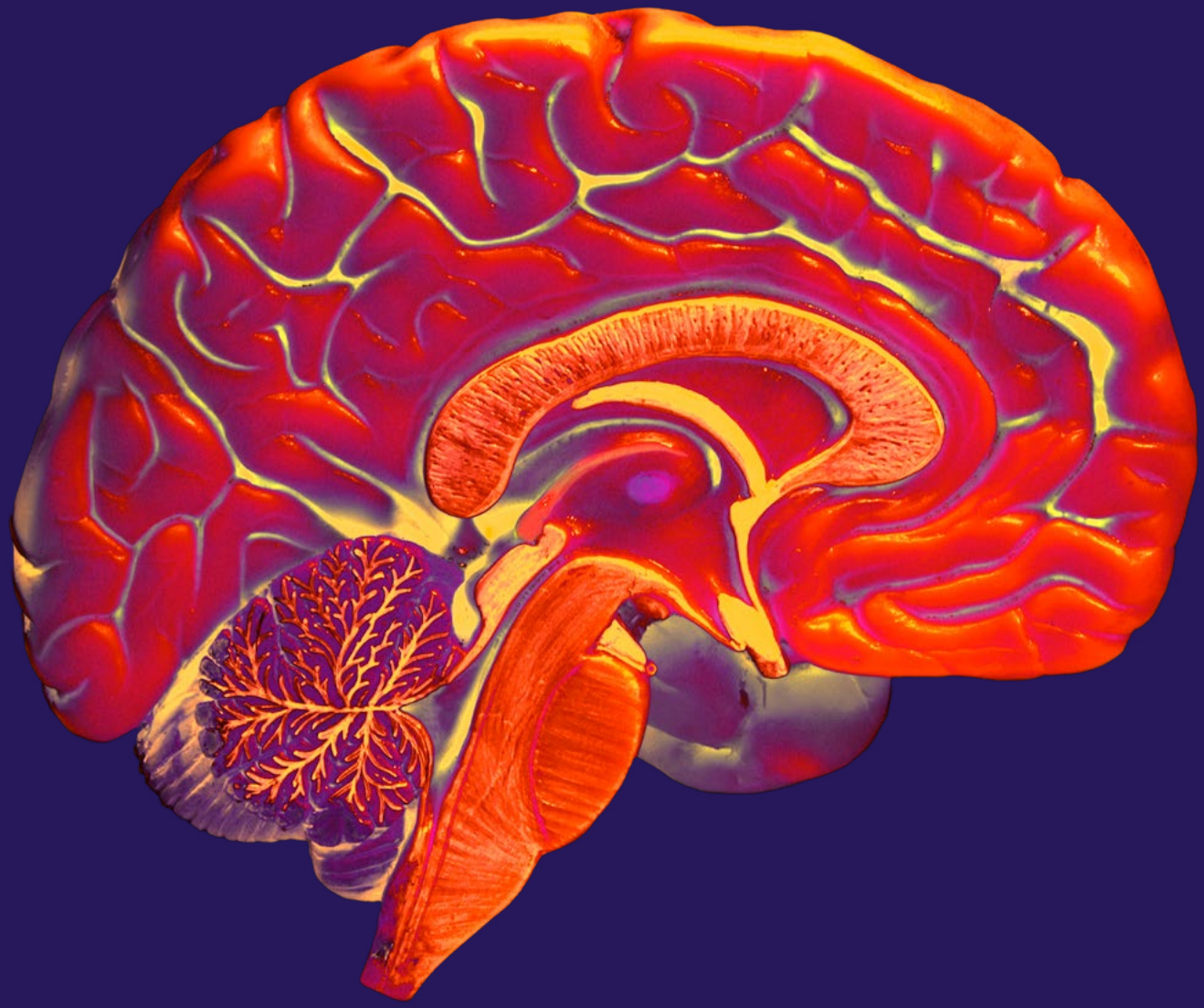
Brain Research UK



Brain Research UK
Impact Report 2026

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Introduction

Brain Research UK is the working name of Brain Research Trust. Brain Research Trust was founded in 1971 as an independent medical charity with the aim of supporting research into diseases of the brain and nervous system at the Institute of Neurology, London (now known as UCL Queen Square Institute of Neurology).

Until December 2015, we provided funding exclusively to the Institute of Neurology. During this time, thanks to the generosity of our supporters, we provided more than £40 million to support a wide range of initiatives, including infrastructure projects, academic chairs, fellowships, projects and PhD studentships. This funding continues: we have provided a further £6.3 million to UCL Queen Square Institute of Neurology between 2016 and March 2026. A summary of the use and impact of this funding can be found in this report.

In 2016, we took the decision to focus our funding on three priority areas where research investment is most urgently needed.

Having broadened our charitable objects in 2015 to enable us to fund nationally through open, competitive funding schemes, in 2016, we took the decision to focus our funding on three priority areas where research investment is most urgently needed: **neuro-oncology, acquired brain and spinal cord injury,** and **headache and facial pain**.

From 2016 to March 2026, we have provided research funding of £12.7 million in these priority areas through our national funding schemes: **PhD studentships, post-doctoral fellowships** and **project grants**.

We last provided an overview on the impact of this funding in our Impact Report 2024; this report provides an overview of progress since then.

Impact highlights

Our recently finished awards have:

Led to clinical trials monitoring the benefits of adding a new drug to radiotherapy to protect the healthy brains of **brain tumour** patients.



Demonstrated that combining a protein-targeted therapy with current standard treatments may offer **a new strategy** for improving the treatment of **glioblastoma**.



Developed powerful new tools and insights that increase understanding of how **childhood brain tumours** start, grow, and might be treated more effectively in the future.



Produced a deeper understanding of how to target the immune system with drugs that could prevent the development of autoimmunity and promote its healing role in patients with **traumatic brain injury.**



Created a roadmap for future clinical trials that can potentially provide more effective brain-targeted treatments and improve outcomes for **stroke survivors**.



Provided a new understanding of **cluster headache** and evidence of specific ways in which **cluster headache** could be treated in the future.



Produced new insights into how pain signals are amplified in the head, face and neck that could lead to more effective treatments for **migraine**.



Identified potential therapeutic targets that could enable a precision approach to treating **amyotrophic lateral sclerosis** and **motor neuron disease.**



Paved the way for a clinical trial of an already approved drug compound in patients with **multisystem proteinopathy**.



Context

There are more than 600 neurological conditions.

They affect one in six of us.

Neurological conditions include everything from brain tumours and sudden onset conditions such as stroke or traumatic brain injury to progressive, degenerative conditions such as Alzheimer's disease and motor neuron disease, as well as intermittent conditions such as epilepsy and headache disorders.

Some conditions are rapidly fatal; others are chronic and debilitating. Together, they account for c.140,000 deaths in the UK every year – one in five of all deaths[1].

Over the last 55 years, great progress has been made in many areas of neurological research. Advances in imaging technology have enabled us to obtain detailed images of the brain, facilitating diagnosis, treatment-planning and surgery.

Advances in gene-sequencing technology have transformed our knowledge of the genetics of neurological disease, opening up an understanding of why people contract disease, the basic mechanisms behind disease and how we might ultimately treat it.

Advances in computational and data processing power have enabled the analysis of huge amounts of complex data in very short timeframes. Artificial Intelligence looks likely to accelerate these advances further and faster.

As a result, some progress has been made in relation to the specific neurological conditions that provide the focus for our three priority areas. However, as the following pages show, there is still much to be done.



1 in 6

of us has a neurological condition



12,500

people are diagnosed with a primary brain tumour every year



2.6m

people live with the effects of traumatic brain injury or stroke



65,000

people suffer from cluster headache, the 'suicide headache'



Neuro-oncology

In 2000, 33% of adults diagnosed with a primary tumour of the brain or spinal cord survived for one year after diagnosis and only 15% survived for five years; now 48% survive for one year and 25% survive for five years.[2] This increase is encouraging. However, survival rates for some kinds of brain tumour remain shockingly low: for glioblastoma, only 25% of patients survive more than one year and only 5% survive for five years or more.[3] Moreover, advances in the understanding and treatment of other types of cancer mean that more than 95% of prostate and breast cancer patients now survive for one year after diagnosis and more than 85% survive for more than five years.[4, 5] More research on the understanding, diagnosis and treatment of brain cancer is therefore still urgently needed to improve survival rates to the levels of other major cancers.



Acquired brain and spinal cord injury

In the UK, c.335,000 people are admitted to hospital every year with an acquired brain injury,[6] including more than 143,000 for stroke.[6] However, advances in emergency care mean that many more people are now surviving such injuries: at least 1.5 million people in the UK have survived a stroke or transient ischaemic attack,[7] and more than 2.6 million people now live with the effects of stroke or traumatic brain injury.[7, 8] Unfortunately, this success also comes with a cost: many of those who survive are left dependent on others for many of their everyday activities while the burden on the individual, their families or other carers, the NHS and society more broadly is huge. More research on how to improve recovery from stroke and other traumatic brain injuries is therefore urgently required.



Headache and facial pain

In the UK, more than 20 million people – two in five adults – are affected by headache or facial pain disorder and, every day, 190,000 people suffer from migraine. The associated cost of migraine to the NHS is £1 billion per year; the cost to the wider economy is £10 billion per year.[9]

Current acute migraine treatments rely on triptans and non-steroidal anti-inflammatory drugs, and the development and use of these drugs has been transformative for many people. However, these drugs are often ineffective unless taken early in a migraine attack and fail to provide adequate relief for up to 40% of patients.[10] Similarly, the more recent development of calcitonin gene-related peptide (CGRP) therapies represents a notable advance in migraine prevention and treatment, but these drugs also reduce migraine in only about 50% of patients.[11, 12] There are also c.65,000 people in the UK who suffer from cluster headache, known as the 'suicide headache'[13] due to its severity, for which CGRP treatments may be ineffective.[19] The need to identify additional causes of headache disorders and potential treatments therefore remains acute.

Conclusion

Overall, the last 55 years have seen much progress in the understanding, diagnosis and treatment of multiple neurological conditions, and the pace of such developments looks set to accelerate. However, there remains much more that still needs to be done if the devastating impacts of these conditions are to change.

This report shows how the generosity of our supporters is enabling and delivering vital research and how funding from Brain Research UK continues to help improve the lives of those affected by neurological conditions to help people live better, longer.

Funding history

Since our inception in 1971, we have invested over £60 million towards our vision of a world where everyone with a neurological condition lives better, longer.

Capacity and infrastructure at the Institute of Neurology

Brain Research Trust was established to counter the limited funding that was available to support neurological research at that time. The Institute of Neurology's 1970/71 Annual Report refers to 'an era of deprivations' limiting neurological research.

There was a major drive underway to promote neurology as an academic subject and entrench research into clinical training programmes. This was coupled with a move away from silo-based research towards collaborative, multi-disciplinary basic research programmes to complement and underpin the clinical research being carried out at the two Institute hospitals, the National Hospital for Neurology and Neurosurgery and University College London Hospital.

It was anticipated that this would attract scientists from all over the world and encourage cross fertilisation of ideas.

Funding from Brain Research Trust was instrumental in helping to achieve this, providing funding for research facilities, new academic posts and research fellowships. Some major benefactions to the new charity were particularly important, establishing endowments that have provided long-term support for specific research centres and academic posts within what is now UCL Queen Square Institute of Neurology.

Funding from Brain Research Trust was instrumental in promoting neurology as an academic subject and in encouraging cross fertilisation of ideas.

These endowments include:

- **A donation of £250,000 from the Miriam Marks Charitable Trust in 1973 in memory of Mrs Miriam Marks, who had suffered from Alzheimer's disease.**
- **A donation of £250,000 from Sir Michael Sobell in 1975 to establish a Chair of Neurophysiology.**
- **A gift of £855,000 from the Will of Miss Eileen Watts in 1994 in memory of her late brother Graeme Watts, who had suffered from motor neuron disease. This was later supplemented with a gift from the Will of Countess de L'Armee.**
- **A donation of £3.6 million, our largest ever single donation, from the Leopold Muller Trust in 1996 to support the Functional Imaging Laboratory.**

Endowment funding impact

The Miriam Marks endowment is now used to fund early career fellowships focused on neurodegeneration. Four fellowships were awarded between 2020 and March 2026, and further fellowships will continue to be funded from the endowment for many more years.

The Sobell endowment now funds the Sobell Chair of Neuroscience, and will be fully expended once the current funding award ends in 2027. In recognition of this longstanding support, a suite of laboratories in UCL's new Gray's Inn Road building will be named the Sobell Neurophysiology Laboratories.



The Graeme Watts endowment was used to establish the Graeme Watts Laboratories in 1999, and now provides funding for a research programme grant and the post of Laboratory Manager within the Graeme Watts Laboratories. The programme grant was renewed in March 2026. Results from the recently completed programme grant can be found on page 34 of this report. The Laboratory Manager post will also be renewed in 2026/27. These two awards will fully expend the Graeme Watts endowment.

The Muller endowment was fully expended in 2019, when we applied the remaining funds to support the purchase and installation of a new, state-of-the-art 7T scanner. This cutting-edge technology transformed the Functional Imaging Laboratory's studies of detailed brain function and significantly advanced the mechanistic understanding of neurological and psychiatric disorders. Using 7T imaging to study the brains of patients with neurodegenerative diseases has provided unprecedented new insights into the very earliest stages in the development and progression of these diseases, and has paved the way for pre-symptomatic treatments and other improvements in patient care.



Brain Research UK Chair of Neurosurgery
In June 2013, Brain Research UK awarded funding of c.£1.7 million to UCL Queen Square Institute of Neurology to support the establishment of a new Chair of Neurosurgery. Our funds were combined with funds of c.£1.1m from the Gough Cooper Fund, held by UCL, to create an endowment intended to fund this new Chair for at least 10 years. Professor Robert Brownstone was appointed to the new Chair in 2015. A summary of Professor Brownstone's achievements since taking up this Chair can be found on page 35.

National funding

In 2015, we broadened our charitable objects to enable us to fund nationally through open, competitive funding calls.

This broadening of our objects enables us to fund ground-breaking peer-reviewed research anywhere in the UK, increasing the impact of our research funding for those affected by neurological conditions.

Then, in 2016, we took the decision to focus our funding on three priority areas: neuro-oncology, acquired brain and spinal cord injury, and headache and facial pain. Our aim was to make a more significant impact than could be achieved by spreading our funding over the full range of neurological conditions. These three disease areas were selected on the basis that they were areas of high patient need, but low historic research investment.

These priority areas have been the focus of our three national funding schemes: PhD studentships, post-doctoral fellowships and project grants.

Funded researchers have delivered important advances in knowledge, each contributing pieces of a wider puzzle.

National funding schemes

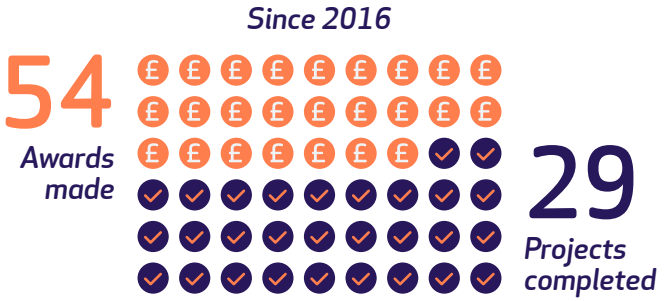
In 2016, we issued our first call for applications for **PhD studentships**, making our first awards in March 2017. This scheme ran annually until 2023, with seven funding rounds completed.

In 2023, we made the decision to replace our PhD studentships with a **post-doctoral fellowship scheme** intended to build research capacity by supporting talented non-clinical researchers at a crucial stage of career development and qualified clinicians wishing to undertake a PhD. We issued our first call for fellowships in 2024 and completed the first round of fellowship funding in February 2025.

We awarded our first **project grants** in September 2017 and completed our ninth round of project grant funding in November 2025.

Overall, since 2016, we have made **a total of 54 awards** through our three national funding schemes to researchers in 20 different research institutes across the UK, representing a commitment of £12.7 million.

Twenty-nine of these awards are now complete, including 16 that have concluded since our Impact Report 2024. A brief overview of these recently completed awards is included in this report. The majority of the research we fund is early-stage discovery science research into the fundamental causes and mechanisms of neurological conditions. As a result, much of this work has yet to deliver measurable patient impact. However, the funded researchers have delivered important advances in knowledge, each contributing pieces of a wider puzzle that will underpin continuing efforts in their respective fields.



Research spotlights

One of the main barriers to successful treatment of brain cancer is that tumours do not always have a clean boundary; they grow into the surrounding healthy brain. However, unlike surgery for cancers in other parts of the body, brain surgeons cannot remove extra tissue to be sure of capturing all the cancer cells – the risk of damaging vital brain tissue is too high. Some cancer cells are therefore almost always left behind after surgery, which causes tumours to regrow.

To address this issue, **Mr Ryan Mathew and Dr Heiko Wurdak from University of Leeds** have undertaken pioneering collaborative research in rodents demonstrating that the cavity in the brain created by the removal of the tumour can be used to deliver a paste that releases drugs intended to prevent the return of the tumour, and that physical devices can also be implanted into the cavity after surgery that can be used both for treating the residual cells left behind after surgery and for monitoring the leftover cancer cells around the cavity. These implants have now proceeded to large animal studies and are on track for first-in-human trials.

In another study, **Clarissa Rocca from University College London** researched families with cluster headache and discovered new genetic variants in a specific calcium channel gene called CACNA1G. Calcium channels act like small gates, allowing calcium to move into brain cells. This process is essential for normal brain activity, but when it goes wrong, it can trigger disease.

Clarissa tested these new CACNA1G variants in the laboratory and showed that the variants reduce the normal activity of the calcium channel, which helps explain how these genes could contribute to cluster headache. This is the first strong evidence that CACNA1G is involved in cluster headache, and it also highlights calcium channels as a critical pathway in the disease, offering a potential new target for diagnosis and treatment of this debilitating condition.

Building research workforce and capacity

Overall, since 2016, funding awarded through our national funding schemes has supported 41 research salaries, more than 100 full-time equivalent research years, and 17 PhD students. This is directly helping to build and maintain a research workforce in our priority areas, and will help to increase the pace of change so that we can make a difference to those living with neurological conditions sooner.



41 ***Research salaries***

17 ***PhD students***

100 ***Full-time research years equivalent***

...to increase the pace of change for those living with neurological conditions.

By targeting our funding at specific research areas and by investing in individual researchers, we are also building capacity to compensate for the historic lack of funding and to help develop these fields for future research. This is especially true of clinical research, where the number of clinical research staff is rapidly declining in both absolute numbers and in proportion to the NHS workforce.[14] We have helped address this through our funding of post-doctoral fellowships, which offer PhD funding for medical doctors, and a focus on translational research in our project grant calls.

We have also collaborated with the Royal College of Surgeons of England to co-fund four **"Brain Research UK – Royal College of Surgeons of England Joint Research Fellowships"**.

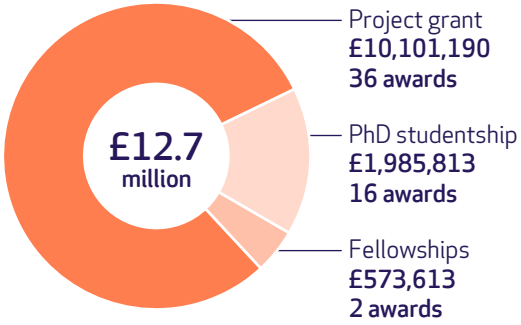
These one-year fellowships provide opportunity and mentorship to surgeons who develop a research interest later in their career, with the aim of investing in these individuals to kick-start new talent and to build the future of surgical research in the UK. All four joint BRUK-RCSEng fellowships are now complete, and an overview of two of these awards can be found on pages 21-22.

Analysis of awards, 2016 to 2026

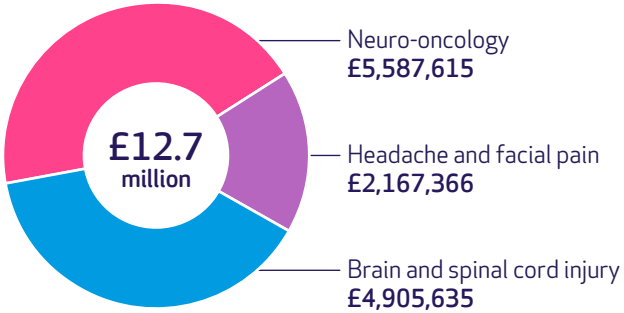
Since 2016, we have awarded funds of £12.7 million through our national calls for applications in our three priority areas. The awards are listed in the Appendices, and those awards that have recently completed are outlined in the pages that follow.

Overall funding awarded

by funding type



by priority area



to these locations



1 University of Dundee
2 University of Glasgow
3 University of Strathclyde
4 University of Edinburgh
5 Newcastle University
6 The Walton Centre
7 University of Manchester
8 University of Leeds
9 University of Hull
10 Nottingham Trent University
11 University of Nottingham
12 University of Birmingham
13 University of Oxford
14 University of Cambridge
15 Cardiff University
16 Imperial College London
17 King's College London
18 University College London
19 The Institute of Cancer Research
20 University of Plymouth

PhD studentships

In 2016, we issued our first national call for applications for PhD studentships, making the first awards in March 2017. The call ran annually until 2023, with seven funding rounds completed. Our funding covered PhD fees at the UK rate, a competitive stipend and research costs.

Through these seven rounds of applications, we awarded 16 studentships across the three priority areas – at a total cost of £2 million.

Twelve of our PhD students had successfully completed their PhDs by March 2026, and one more is currently in the final stages of submission. Information about six of the complete awards is included in the relevant disease sections in this report.

Between them, the 16 PhD students funded through our studentships have authored or co-authored 64 peer-reviewed research articles (to March 2026), of which 22 are first author publications.

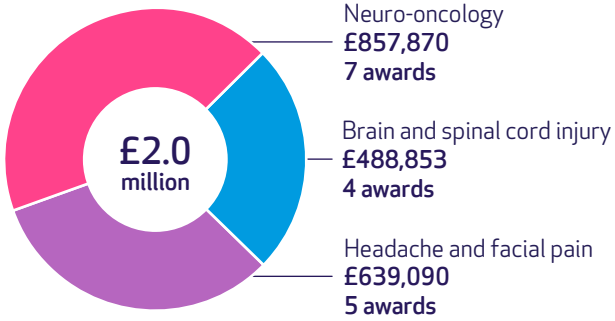
Post-doctoral fellowships

In 2023, we replaced our PhD studentships with a new post-doctoral fellowship scheme, inviting applications from junior candidates working in our three priority areas. These fellowships enable individuals to apply for their own salary (unlike our project grants, which require applicants to have their salary already in place). The aim of these fellowships is to build capacity in our priority research areas by supporting talented individuals at a crucial stage of career development where they often encounter funding uncertainty. This scheme also provides funding for medical doctors wishing to undertake a PhD, to encourage and enhance clinical research.

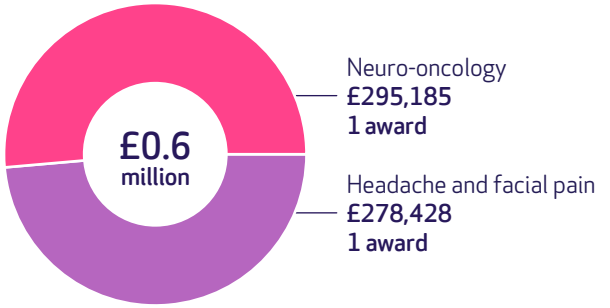
We completed our first round of fellowship funding in February 2025, awarding two fellowships: one in neuro-oncology and the other in headache and facial pain.

Supporting talented individuals at a crucial stage of career development where they often encounter funding uncertainty.

PhD studentships awarded by priority area



Fellowships awarded by priority area



Project grants

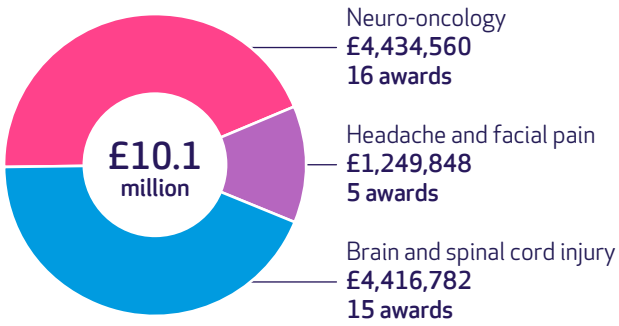
Our first project grant call was issued in 2017, and the ninth funding round concluded in November 2025. We have awarded 36 project grants across our three priority areas – a total commitment of £10.1 million.

The profile of these awards is different to those for our PhD studentships and fellowships. The priority area of headache and facial pain has received a much smaller share of the funding relative to the other two areas, accounting for only 5 of the 36 awards. This is largely a reflection of the number of applications received in each priority area in each funding round, and we have recently taken various steps to encourage and increase applications for headache and facial pain research.

Seventeen of these project grants have so far completed. Information about 10 of the projects that have completed since our Impact Report 2024 is included under the research priority sections below.

Work funded by these project grants has so far contributed to 73 peer-reviewed research articles (to March 2026), several of which are in publications with a top 3 impact factor in their respective research areas.

Project grants awarded by priority area



Research priority:

Neuro-oncology

Every year in the UK, c.12,500 people are diagnosed with a primary tumour of the brain or spinal cord and c.5,600 lives are lost to such tumours.[2] This is 15 deaths every day.

Despite decades of progress and improving survival in many other types of cancer, much less progress has been made in relation to brain and spinal cord tumours. More people under the age of 40 still die from brain tumours than from any other cancer,[15] and glioblastoma – the most common primary brain cancer – still has no effective curative treatment. The average survival for a glioblastoma patient is just 12-18 months after diagnosis, and only 5% of glioblastoma patients survive 5 years or more.[3]

However, historically, research into brain and spinal cord tumours has not attracted the same level of attention and funding as research into other cancers.

This is why we identified neuro-oncology as one of our three priority research areas. We want to improve the outlook for people with brain and spinal cord tumours by funding research that takes forward our understanding of the mechanisms underlying tumour development and helps develop better ways to diagnose and treat these tumours.

Over the past 10 years, through our national calls, we have invested funds of £5.6 million in research into neuro-oncology – through 16 project grants, seven PhD studentships and one fellowship, listed in Appendix 1.

Eleven of these awards have so far completed, seven of which finished since our Impact Report 2024 and are outlined on the following pages.

Understanding the margin zone in brain cancer

Project grant

£296,086

Apr 2021 – Mar 2026

University of Leeds

Lead researchers:
Mr Ryan Mathew and Dr Heiko Wurdak





Impact: **The understanding and models generated by this project can now be used to trial new drugs and devices to tackle brain cancer.**

One of the main barriers to successful treatment of brain cancer is that the tumours do not have a clean boundary; they grow into the surrounding healthy brain. This means that while surgery is often effective at removing most of the brain cancer 'bulk', cancer cells are almost always left behind in a 'margin zone' that comprises both cancer cells and healthy brain tissue. These remaining cells are targeted with radiation and chemotherapy but, despite this, the tumours almost always regrow.

In this project, Mr Mathew, Dr Wurdak and their colleagues recreated human brain cancer surgery in mice and rats, to enable the team to study the cancer cells in the margin zone; work on better ways to eliminate these cells; and test treatments that might be given after surgery.

One way in which new treatments can be given after surgery is by using the cavity created by the removal of the tumour bulk to deliver gels or pastes that can release drugs intended to prevent the return of the tumour.

During this project, the team identified a drug that could be steadily released from a paste that was suitable for testing within the cavity. These tests showed increased survival of animals that received delivery of the paste after surgery when compared with control animals that received surgery alone.

The cavity created by the removal of a tumour can be used to deliver gels or pastes that release targeted drugs.

In a second study, the team also established that physical devices can be implanted into the cavity after surgery. These devices can be used both for treating the residual cells left behind after surgery, and for monitoring the leftover cancer cells around the cavity. This proof-of-feasibility has contributed to the ongoing development of specialised, miniaturised brain-computer interface implants, which have proceeded to large animal studies and are on track for first-in-human trials.

Finally, the team compared the brain tumour bulk removed by surgery with the edge of the remaining tissue, and with the tumour when it returned. The results show exciting and significant changes over time in the area around the cavity that might explain why the body's natural defence system cannot target and eliminate the leftover cancer cells.

Reducing the damage caused by radiotherapy in brain tumour patients

Project grant

£298,251

Dec 2022 – Nov 2025

University of Glasgow

Lead researchers:
Professor Anthony Chalmers and Professor Kaye Williams





Impact: The results from this project suggest that adding AZD1390 to radiotherapy for patients with brain tumours might protect the healthy brain from the side effects of radiotherapy. This possibility is now being monitored in ongoing clinical trials.

Radiotherapy is a vital tool in the treatment of brain tumours. However, it can also damage the healthy brain, so there is an urgent need to find drugs that can reduce the side-effects of radiotherapy without interfering with its beneficial effects.

We know that radiotherapy damages the normal brain by causing inflammation that continues even after the radiotherapy has finished. This project set out to understand whether a new drug, AZD1390, could help protect the healthy brain from the damaging effects of radiotherapy.

AZD1390 blocks a molecule that plays a key role in how cells respond to radiation, and is already being tested in patients to see whether it can make radiotherapy more effective against brain tumours. However, the effects of AZD1390 on healthy brain tissue are not known.

This new drug suppresses the ability of radiotherapy to cause inflammation in the brain.

As part of this project, Professor Williams' research showed that treating mice with radiotherapy caused a progressive deterioration in their neurological function over a six-month period. However, treating mice with AZD1390 for one week at around the same time as radiotherapy significantly reduced this deterioration.

Then, in experiments aimed at understanding what causes these protective effects, Professor Chalmers and his team observed that AZD1390 suppresses the ability of radiotherapy to cause inflammation in the brain.

Brain tumours: types and treatments

There are over 100 different types of brain tumour. Some grow slowly and can often be successfully treated or monitored for many years. Others are aggressive, difficult to treat and can have a devastating impact on patients and their families. Standard treatments involve a combination of surgery, radiotherapy and chemotherapy, but many tumours return after treatment. More research is needed to improve our understanding of these tumours, develop kinder and more effective treatments, and ultimately give more people the chance to live longer, better lives.

Neuro-oncology

How do brain tumour cells persuade immune cells to promote their growth?

Project grant

£298,427

Feb 2022 – Jun 2025

University of Edinburgh

Lead researcher:
Dr Dirk Sieger



Impact: **Findings from this project have opened up promising avenues for research that could ultimately be translated into clinical applications, with the potential to provide significant benefits for patients who have one of the deadliest forms of cancer.**

Glioblastomas are recognised by the immune cells of the brain, the *microglia*. Unfortunately, however, rather than fighting the brain tumours, the microglia have been shown to actively promote their growth. However, we don't understand why this happens.

Dr Sieger's previous research had shown that microglia acquire this tumour promoting activity at the earliest stages of glioblastoma formation. It is not known exactly how this is happening, but Dr Sieger and his colleague Dr Mazzolini hypothesised that very small particles called extracellular vesicles (EVs) are released by the cells that initiate glioblastoma. These EVs are then taken up by microglia and change their activity.

To test this hypothesis, the team used a combination of different laboratory models to understand if and how these EVs impact on the function of microglia. Specifically, the team used a high-resolution microscope to look inside zebrafish brains to visualise the release and uptake of different types of EVs during glioblastoma initiation stages. What the team observed was that EVs from both zebrafish and human glioblastoma initiating cells are all engulfed by zebrafish microglia and affect their function in the brain.

These results then allowed the team to use another zebrafish model to understand the role of EVs in the communication between glioblastoma initiating cells and microglia during the early stages of tumour growth.

Interfering with cellular communication in the brain can enable natural immune cells to attack glioblastoma cells and impair early tumour growth.

Finally, the team tested different drug compounds known to interfere with EV release and assessed their efficiency and impact on the microglial population in living brains. Importantly, the team showed that inhibiting EV release in the brain resulted in microglia engulfing tumour cells and impaired tumour growth. These results encouraged the team to perform further experiments to understand in detail how EVs from human glioblastoma cells change the gene expression and function of human microglia cells.

Dr Sieger's project has provided evidence that extracellular vesicles play an important role during glioblastoma initiation by impacting on microglia and subsequently on glioblastoma growth. The project has also laid the groundwork for future research that can provide detailed insights on how these changes impact on glioblastoma growth.

Neuro-oncology

Blocking progression of low-grade brain tumours

Project grant

£299,590

Nov 2022 – Nov 2025

University of Cambridge

Lead researcher:
Mr Richard Mair



Impact: **This project is a key step on the pathway towards the identification and development of drugs that could block the progression from low-grade to high-grade glioma.**

Low-grade glioma (LGG) is a rare brain tumour that predominantly affects young adults. There is no cure and all patients ultimately progress to high-grade glioma, at which point their prognosis is around two years.

LGG is commonly associated with a mutation to a gene called IDH within a patient's brain cells. This mutation affects the way that a cancer cell makes energy – its *metabolism*. We know that this altered metabolism causes many changes to the way that genes are controlled within the tumour cells. We also know that these cancers become starved of oxygen as they grow. However, investigation into the cause, growth and response to treatment of these tumours has been limited by a lack of biologically accurate models that correctly reproduce the characteristics of these slow growing tumours.

In this project, Mr Mair and his colleagues characterised a novel animal model of IDH mutant astrocytoma (a type of brain tumour) that faithfully reproduces the molecular underpinnings of this disease. Moreover, they used novel cell culture techniques to derive additional models from patient material donated during surgery. By using these model systems, the team have further developed a method to explore how interactions between tumour cells and those of the immune system might regulate or promote tumour behaviour.

Finally, the team used their experience of tumours grown in low oxygen environments – similar to those identified within patient tumours – to show that certain molecular characteristics of brain tumours are enriched in these circumstances.

The ability of cells to amplify abnormal signals has been demonstrated to occur via several mechanisms within the cell, and has been implicated as a method by which cancer cells might escape therapies targeted to inhibit those specific signals. This project has demonstrated how this signal responds to the low oxygen environment within a tumour, and that this low oxygen environment further amplifies the signal, making these cells even harder to treat with targeted therapies. This understanding of amplification, together with the models that Mr Mair has generated, can now be taken forward to explore therapeutic opportunities that try to inhibit this amplification itself.

Understanding how low-oxygen environments amplify tumour signals can now be taken forward to explore new therapeutic opportunities.

Towards precision therapy for diffuse midline gliomas

Project grant

£289,874

Nov 2020 – Sep 2025

University of Cambridge

Lead researcher:
Dr Manav Pathania



Impact: **These three studies offer powerful new tools and insights that increase understanding of how paediatric brain tumours start, grow and might be treated more effectively in the future, offering new hope to those affected by this devastating condition.**

Diffuse midline glioma (DMG) is an aggressive and incurable brain tumour that most commonly presents in children. It has a dismal prognosis – most children die within 18 months.

Genetic sequencing has revealed that DMGs represent several distinct tumour types, based on the different mutations they carry and where they occur in the brain. However, the relevance of many of these extra mutations is unknown: they may be present in tumours, but what do they do? Are they required for tumour growth? And how important are they for treatment? Dr Pathania and his team explored these questions via three interlinked studies.

In the first study, the team created 16 new mouse models of paediatric high-grade gliomas that accurately mimic the diversity seen in human tumours – appearing in different brain regions, at different ages, and growing at different speeds. The team then tested various treatments, and found that different tumour subtypes responded to different drugs. This work shows that treatments could be customised based on a tumour's specific mutations, helping move toward more personalised and effective care for children.

The second study focused on the immune environment around these brain tumours – an area that hasn't been well studied in paediatric cancers. Dr Pathania and his team found that these tumours have lots of myeloid immune cells but very few T cells, which are key fighters in the immune system. The team then applied a combination of drugs that target both the myeloid immune cells and the proteins that block immune activity, and found that tumour growth slowed down as a result. This opens the door to new immunotherapy strategies for treating childhood brain tumours.

Mapping the unique biology of childhood brain tumours lays the foundation for targeted, effective treatments for aggressive paediatric cancers.

Finally, the team looked at a rare and aggressive tumour called NB FOXR2, which is difficult to diagnose. By comparing tumour cells to normal brain development, the team discovered that this tumour likely comes from a very specific type of early brain cell marked by the genes LHX6 and DLX. This discovery helps explain where these tumours come from and provides a valuable model for studying the disease and testing treatments.

Telomere biology in glioblastoma

PhD studentship

£122,541

Dec 2022 – Dec 2025

Cardiff University

Lead researcher:
Mr Harsh Bhatt



Impact: **This research demonstrates that knowing individual patients' tumour telomere lengths and underlying faulty DNA repair mechanisms could help clinicians predict who will benefit from which targeted treatments, and reduce the potential harm caused by other treatments.**

Mr Bhatt was awarded a PhD studentship to enable him to pursue research into glioblastoma. As an academic neurosurgery trainee, Mr Bhatt has treated many patients with glioblastoma and is passionate about improving understanding of their disease and helping improve treatments.

Analysing telomere length and chromosome changes reveals how glioblastomas evolve over time and respond to treatment.

Mr Bhatt's research focuses on *telomeres*, sections of DNA that cap and protect the ends of our chromosomes. Telomeres shorten each time a cell divides, including during the formation of cancer cells – a process that involves rapid cell division. This shortening can cause chromosomes to fuse together and is a key step in the formation and growth of many cancers.

For this project, Mr Bhatt and his colleagues studied 146 patients with glioblastoma (GBM) and analysed their tumours for precise telomere length measurements. This showed that knowing a tumour's telomere length can add powerful prognostic value to current biomarker tests for predicting patient survival.

The team also learnt that there might be subgroups of patients with GBM in whom shorter telomeres predict much better survival, compared to other subgroups for whom shorter telomeres were much more detrimental. This difference might depend on the tumour's abilities to faithfully repair damaged DNA after, for example, the patient has had radiation and chemotherapy treatments.

The team also found that in patients with GBM, telomeres are generally much shorter when compared to those in a normal brain, and that chromosomal fusions and rearrangements of genetic material seem widespread. These fusions showed signs of relying on specific repair mechanisms for damaged DNA, which may make such tumours suitable for specific targeted treatments.

Finally, in a handful of patients, the team were able to compare the same tumour at first surgery and when it grew back after treatment, to assess differences in their telomere length and fusions over time and following treatment. The team learnt that telomere length may not remain the same in a tumour over time, and that a less diverse spectrum of chromosomal fusion types is seen when the cancer came back.

Neuro-oncology

CBX2 as a new therapeutic target in glioblastoma

PhD studentship

£106,581

Oct 2022 – Sep 2025

University of Hull

Lead researcher:
Danielle Marsh



Impact: **Danielle's findings provide important new insight into the biological role of CBX2 in glioblastoma, and suggest that combining CBX2-targeted therapies with current standard treatments may offer a new strategy for improving treatment effectiveness.**

Danielle was awarded her PhD in June 2026 and is now a postdoctoral researcher.

Epigenetics is a biological process that controls the way our genes behave by turning them on and off. In healthy cells, epigenetics is tightly controlled. In cancerous cells, however, the epigenetic control is faulty and can lead to uncontrolled cell growth.

For her PhD, Danielle studied a protein called CBX2, which is known to be involved in epigenetic control, and which is found at higher levels in glioblastoma than in healthy brain tissue. Danielle's aim was to determine whether CBX2 is necessary for glioblastoma growth and whether it could therefore be a therapeutic target, through which tumour cell growth could be brought under control.

To do this, Danielle treated glioblastoma cells with a specific compound that blocks CBX2 from binding to DNA and carrying out its normal function, and she then examined the effects of this treatment on tumour cell growth, survival, and gene activity.

Danielle's study showed that blocking CBX2 from binding to DNA reduced glioblastoma cell growth and triggered cell death. Further analysis indicated that CBX2 also regulates genes involved in the cell cycle, the process through which cells copy their DNA and divide to produce new cells.

Inhibiting CBX2 caused tumour cells to accumulate DNA damage and allowed them to continue dividing before this damage could be repaired. This appeared to force the tumour cells into a fatal process known as *mitotic catastrophe*, whereby cell death is triggered through defective cell division.

Targeting CBX2 in patient tissue produced clear anti-tumour effects, strengthening confidence that it is a clinically relevant target.

Importantly, the study also assessed the effects of targeting CBX2 in living glioblastoma tumour tissue from patient donors, which was maintained in the laboratory using a specialised 'chip' device developed at the University of Hull. This chip allowed Danielle and her colleagues to closely monitor how glioblastoma tumour tissue reacts to different treatments. Encouragingly, targeting CBX2 in patient tissue produced similar anti-tumour effects to those observed in the glioblastoma cell models, strengthening the confidence that CBX2 is a clinically relevant target.

Transcranial magnetic stimulation for cognitive rehabilitation in brain tumour patients

Brain Research UK – Royal College of Surgeons of England Joint Research Fellowship

£36,500

Feb 2024 – Jan 2025

The Walton Centre

Lead researcher:
Mr Ahmad Ali



Impact: **This work has created the evidence base for a non-invasive form of brain stimulation that could improve cognitive abilities for patients and has enabled the careful planning of a study that will begin to establish the potential benefits of TMS for patients with LGG.**

This fellowship enabled Mr Ali to secure a three-year NIHR Doctoral Fellowship to deliver the proof-of-concept study evaluating the cognitive effects of TMS.

Neurosurgical trainee Mr Ali was awarded his joint research fellowship to take forward research that aims to improve quality of life for patients with low-grade gliomas.

Whilst neurosurgery is central to the treatment of brain tumours, the surgery can also cause other problems – including problems with memory, attention and other cognitive functions. Mr Ali was keen to find ways to reduce the risk of these cognitive issues and to improve quality of life for his patients. He focussed specifically on patients with low-grade gliomas (LGG), a group that he feels is hit particularly hard by cognitive impairment.

Mr Ali's research used a technique called Transcranial Magnetic Stimulation (TMS), in which magnetic fields are applied non-invasively to stimulate a part of the brain. If done repetitively to the same part of the brain, TMS can induce long-lasting changes to the wiring of the targeted area. The benefits of TMS have been demonstrated in patients with a range of conditions: TMS is now used routinely to help patients with depression and chronic pain, and trials have demonstrated improved cognition in patients with Alzheimer's disease, and improvements in upper limb function in glioma patients.

Analysing MRI scans can create personalised brain stimulation targets, paving the way for more precise and effective cognitive therapies.

There are many parameters that affect whether TMS works. It is paramount, therefore, that these parameters are carefully controlled in any trials of the technique.

During this project Mr Ali achieved three key aims:

1. He established a database of patients with LGG that can be used to identify patients that are eligible for a proof-of-concept trial and he met with these patients to design the details of this trial;

2. He undertook a comprehensive meta-analysis of protocols used in previous TMS studies, which can be used to guide the selection of the optimal TMS parameters in the trial.

3. He established a pipeline for analysing the Magnetic Resonance Imaging (MRI) scans of patients with brain tumours to create personalised stimulation targets for TMS.

Neuro-oncology

Developing MRI-active drugs to optimise chemotherapy for brain tumours

Brain Research UK – Royal College of Surgeons of England Joint Research Fellowship

£36,500

Feb 2023 – Feb 2024

University of Nottingham

Lead researcher:
Mr Milo Hollingworth



Impact: **The team's 'proof of principle' preclinical work demonstrates for the first time that a drug can be directly labelled in such a way that it can be visualised non-invasively without affecting its biological function. This next-level technique could allow researchers and clinicians to reignite and repurpose off-patent drugs and fine-tune drug dosing and delivery techniques to target brain tumours and other difficult to treat diseases.**

When administering drugs for the treatment of brain diseases it is important to know where the drugs are going and in what concentration. However, while we can put drugs into the brain and we can measure outcomes (such as changes to a brain tumour, and length of patient survival), we can't see what is happening within the brain. If there is no effect, we don't know why. Is it that the drug itself isn't effective? Is it because the drug isn't getting to the right target, or in a high enough concentration?

Mr Hollingworth and his colleagues have begun addressing these questions by developing drugs that are magnetic resonance imaging (MRI) active – that is, drugs that are modified with molecules that are visible when using MRI. When injected into the brain, their location, concentration and activity can therefore be visualised and monitored. In particular, the team has developed an MRI-active version of Olaparib, a targeted drug that is already used in the treatment of several cancers, and which has shown some effectiveness against certain brain tumours in laboratory testing.

Developing MRI-active drugs allows researchers to directly visualise drug location, concentration, and activity within the brain in real time.

As part of this project, the team:

1. established a clinical trial aiming to visualise glucose metabolism in glioblastoma multiforme (the most common primary brain cancer in adults);

2. developed an MRI-active drug that could accurately visualise drug concentrations directly in time and space, and kill cancer cells in-vitro; and

3. developed a simulation platform to allow troubleshooting of clinically relevant problems to aid translation of this technology without the need for animal research.

The team were able to demonstrate that their imaging technique is feasible and comfortable for patients and, with increasing patient numbers, are hopeful that they can identify unique metabolic signatures in brain tumours that will avoid the need for biopsy in certain patients and can predict treatment response more accurately than conventional imaging techniques.

Research priority:

Acquired brain and spinal cord injury

Every 90 seconds someone is admitted to hospital with a brain injury. This includes injuries caused by accidents, assaults and falls, as well as stroke and other non-traumatic causes.

However, advances in emergency care mean that many more people are now surviving such injuries: more than 2.6 million people now live with the effects of traumatic brain injury or stroke. This also means that more people are living with the long-term effects of these injuries:

- 1.3 million people are living with traumatic brain injury related disabilities[8]
- 1.5 million people have survived a stroke or transient ischaemic attack[7]
- 105,000 people are living with a spinal cord injury[16]

The impact of these injuries varies enormously from person to person, depending on the nature, severity and location of the injury, as well as the treatment and rehabilitation undertaken. Many people experience a reduced quality of life due to problems with speech, thinking and basic bodily functions, so more research is urgently needed to help them regain as much independence as possible.

We aim to improve quality of life after injury by funding research that helps us understand what causes non-traumatic brain injury and how the brain and spinal cord can be repaired following all kinds of injury. This will help people recover as fully as possible.

Over the past 10 years, we have invested £4.9 million in research to improve understanding of brain injury and to explore ways to repair the brain and spinal cord – through 15 project grants and four PhD studentships, listed in Appendix 2.

The research funded has been wide-ranging, including both clinical activities and discovery science. Ten of these awards have so far completed, five of which finished since our Impact Report 2024 and are outlined on the following pages.

Understanding differences in recovery from stroke

Project grant

£339,589

Oct 2019 – Mar 2025

University College London

Lead researcher:
Professor Nick Ward



Impact: The results of this project have significant consequences. Being able to predict outcomes for individual patients can help guide treatment and rehabilitation, enabling personalisation of therapy and determining 'who' and 'when' to treat with current therapies. This points to possible new targets for drugs that can be tested in future clinical trials aimed at promoting recovery after stroke.

In this project, Professor Ward set out to ascertain why some stroke patients recover function more easily than others, to understand what factors are important in the recovery process itself, and to unveil opportunities for – and barriers to – better outcomes.

Brain function – rather than physical damage – is the primary predictor of how much function a patient recovers following a stroke.

The team hypothesised that structural damage to the brain would be the main determinant of the immediate severity of stroke symptoms, but that brain function would be the main determinant of how much recovery occurs in subsequent months. The team therefore set out to compare the two groups of patients – good early recovery vs poor early recovery – using advanced brain imaging techniques to study differences in the pattern of anatomical brain damage and differences in brain repair mechanisms.

Magnetic resonance imaging (MRI) was used to assess structural brain damage whilst an imaging technique called magnetoencephalography (MEG), which maps electrical activity in the brain, was used to assess brain function. The team collected neuroimaging and clinical data from acute (first three months post-stroke) and chronic (beyond six months) patients as well as healthy controls. They also took advantage of some previously collected datasets to carry out additional analyses.

Their hypotheses proved largely correct, with their findings suggesting that in the early post-stroke phase, structural brain damage is associated with initial severity of impairment, whereas measures of brain function are more predictive of subsequent recovery.

Stroke: the leading cause of severe disability

A stroke occurs when blood supply to part of the brain is cut off. This starves the brain of oxygen, with devastating consequences. Better awareness and improved treatment have both contributed to a reduction in mortality from stroke, with the number of deaths from stroke now less than half what it was 30 years ago.[17] However, the lower mortality rates mean that more people are surviving stroke than ever before, and many of these survivors are left with disabilities that limit their ability to live productive, independent lives. Improving stroke recovery to enable stroke survivors to live independent, productive lives is a key goal.

Using immune cells to enhance brain repair after stroke

Project grant

£271,931

Sep 2021 – Dec 2024

University of Edinburgh

Lead researcher:
Dr Barry McColl



Impact: **This project is a crucial step in the development of a potential new treatment to boost brain repair in stroke survivors and enhance recovery of function.**

Existing treatments for stroke work by unblocking the damaged artery, which needs to be done very quickly after a stroke happens in order to limit the damage. There is currently no approved treatment to help heal the damaged brain beyond this initial emergency treatment.

In this project, Dr McColl and his colleagues built on the relatively new understanding of the importance of the immune response in brain recovery and repair. The team tested the effects of a modified version of a naturally occurring protein in the body called CSF1-Fc, using an experimental type of stroke in mice that mimics a blood clot forming in an artery as happens in humans.

The results showed that CSF1-Fc treatment caused a change in the blood immune cell count, confirming that the intended immune cells were being targeted. Treatment with CSF1-Fc also accelerated recovery of forelimb function, with most mice showing function similar to pre-stroke levels by one week. Furthermore, brain scanning showed that CSF1-Fc did not change the amount of early brain damage or brain swelling after stroke, which is important as increased brain swelling might have been a risk of this approach.

Finally, the team found that CSF1-Fc also did not change the number of immune cells, called *macrophages*, accumulating around the damaged brain area, but that it did change the amount of a protein produced by the macrophages that is linked to repair of tissues. Some additional analysis of brain scans also suggested that the nerve wiring of the brain that connects different brain regions may also be altered by CSF1-Fc treatment.

Harnessing the body's immune response opens new pathways to repair damaged brain tissue and restore function after stroke.

This project showed several positive effects of CSF1-Fc treatment, including enhanced functional recovery and an increase in the amount of protein linked to brain tissue repair. The team now need to conduct further trials in different stroke models and across multiple laboratories, before being able to progress to human studies. However, their findings support the general idea that targeting immune cells like macrophages can support recovery of function after stroke.

Understanding the immune response to traumatic brain injury

Project grant

£280,848

Aug 2021 – Aug 2024

University of Cambridge

Lead researchers:
Professor David Menon and Dr Ed Needham





Impact: As a result of this project, we now have a deeper understanding of how to target the immune system with drugs that could prevent the development of autoimmunity and promote its healing role in patients with TBI.

Traumatic brain injury (TBI) is a leading cause of death and disability of young adults in the developed world. There is evidence that the body's immune response to a severe traumatic brain injury compounds the initial damage in some patients, leading to a worse outcome. In this project, Professor Menon, Dr Needham and their team set out to improve understanding of this immune response, how it might impact recovery, and how it could be prevented or treated to improve outcomes for patients.

In a previous pilot study, the team had found evidence that some TBI patients develop an autoimmune response against brain cells, producing 'autoantibodies' that attacked the brain cells. The patients who developed these autoantibodies appeared to have a worse-than-predicted outcome. This suggested that the autoantibodies might be contributing to brain damage.

In this project, the team carried out a larger study to confirm and further explore these results. Using a cohort of 470 patients with TBI, the team used blood samples, brain scans and memory tests to find out more about the autoantibody response, and how it relates to outcomes after TBI over time.

Traumatic brain injury triggers a significant autoantibody response, revealing distinct immunological processes that could predict patient outcomes.

Their results confirmed the findings of their pilot study: that TBI triggers a significant autoantibody response, and this was found to be associated with an increase in the levels of certain immune cells in the first week after injury. The study also showed that there was no relationship between this autoimmune response and the severity of injury, but that there was a clear association with age, whereby younger patients had more marked responses.

In addition, patients with high levels of a certain type of autoantibody were found to have worse than expected outcomes six months later. The team therefore looked to see whether the autoantibodies related to markers of ongoing brain damage, or to other later consequences of TBI. The autoantibodies did not appear to be associated with late brain damage but, interestingly, patients who went on to develop epilepsy after their head injury had lower levels of certain autoantibodies, despite having higher general levels of inflammation in their blood. This suggests that the autoantibodies might play a useful role later after the injury.

This research was the most comprehensive study of the inflammatory response in human TBI at that time, and confirmed that there are distinct immunological processes that predict outcome after TBI, shedding light on the underlying biological mechanisms.

The role of multimodal cues in aphasia

PhD studentship

£95,303

Oct 2021 – Jun 2025

University College London

Lead researcher:
Isobel Chick



Impact: These four studies highlight how important it is for clinicians to consider interactions between communication and other cognitive systems when planning aphasia therapy. Isobel's work has identified key cognitive factors that clinicians should consider when planning and delivering aphasia therapy.

Isobel was awarded her PhD in October 2025, and is now the Adult Speech and Language Therapy Lead at University Hospitals Coventry and Warwickshire (UHCW) NHS Trust.

Isobel was awarded a PhD studentship to enable her to pursue research into how one specific visual aspect of communication – gestures – are affected in aphasia.

First, Isobel explored whether seeing and using gestures helped people with aphasia (PWA) to understand and use single words during conversations. She found that when PWA saw gestures it helped them to understand single words, but only if they were able to draw meaning from the gestures. PWA who could not do this – a condition known as 'ideational apraxia', which often occurs alongside aphasia – did not benefit from seeing gestures. Isobel also found that using gestures generally did not help PWA to produce words more easily, but seeing gestures did help PWA with poorer working memory to produce words.

Second, Isobel explored whether seeing and using gestures helped PWA understand longer phrases in conversations. She found that PWA with poorer visual attention abilities did not find gestures helpful for understanding, which may be because they struggled to retain visual information encoded in gestures long enough to integrate it with speech.

Third, because conversations always include at least two people, Isobel wanted to explore whether the gestures used by PWA helped other people to understand them. She videoed PWA describing everyday objects and actions: in half the videos the PWA used gestures, and in half they did not. Isobel then asked neurologically healthy adults to watch the videos and guess what each PWA was describing. She found that neurologically healthy adults rated the descriptions easier to understand when PWA used gestures, but the effect was moderated by whether PWA had 'ideomotor apraxia', a condition which affects planning and production of skilled motor movements. PWA with more severe ideomotor apraxia made gestures which were less intelligible, which did not help watchers understand them.

Finally, Isobel wanted to explore whether combining gestures with speech in a therapy context could help PWA to relearn words. She ran an intensive online therapy programme with four PWA with significant word-finding difficulties, and found that gestures did support word relearning, but only if they were obviously visually closely related in meaning to the words.

Aphasia: a severely disabling disorder

Aphasia is one of the three main communication problems that can be caused by stroke, and around a third of people who have a stroke will experience aphasia.[18] It is a severely disabling disorder, causing profound frustration for those affected, and often leading to social withdrawal and depression. Many people with aphasia are unable to return to work, and relationships can suffer, so effective rehabilitation is a healthcare priority.

Enhancing the effectiveness of brain stimulation for stroke recovery

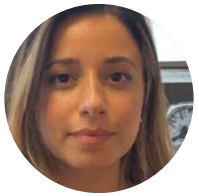
PhD studentship

£120,505

Sep 2019 – Mar 2024

University College London

Lead researcher:
Haya Akkad



Impact: Haya's research provides a roadmap for future clinical trials that can potentially provide more effective brain-targeted treatments and improve outcomes for stroke survivors.

Haya was awarded her PhD in 2024, and is now a postdoctoral research fellow in the Department of Clinical and Experimental Epilepsy at UCL Queen Square Institute of Neurology.

Haya was awarded a PhD studentship to enable her to pursue research aimed at improving recovery from stroke.

Stroke and other brain injuries can disrupt vital connections between different parts of the brain. These connections, or *neural networks*, usually interact to give rise to particular functions and their disruption underlies some of the disabilities seen in stroke.

Non-invasive brain stimulation (NIBS) is becoming widely used as a tool to promote brain recovery after injury. It involves the delivery of electrical current to the brain via electrodes or a magnetic coil placed on the head, with the aim of 'resetting' the disrupted neural networks. The use of NIBS has shown some promising results in stroke patients, but a lack of understanding of precisely how and why it works has meant that effects have been inconsistent across studies and between patients, hindering its wider application. A better understanding of the mechanisms involved will inform better decisions about precisely which part of the brain to target, what dose is required, and how to deliver that dose to the target area.

Non-invasive brain stimulation aims to 'reset' disrupted neural networks, but a lack of understanding of precisely how and why it works has hindered wider application.

Haya set out to address these issues by focussing on the two most common post-stroke symptoms – severe word-retrieval deficits (anomia) and one-sided upper limb weakness (hemiparesis) – and by collating evidence about behaviour, brain structure and brain function to provide new insight as to why current NIBS approaches can fail, and how they can be improved.

Starting with an extensive literature review and progressing to deliver three major experimental studies, Haya demonstrated that the most common brain regions targeted with NIBS for post-stroke anomia and upper limb hemiparesis are not the regions that most commonly drive these deficits, and thus may not be the most suitable targets. This sheds light on the low effectiveness of current NIBS interventions for post-stroke recovery.

From this, Haya proposed revised, evidence-based approaches for consideration in future clinical trials, and designed detailed brain stimulation protocols, tailored to the relevant brain network.

Research priority:

Headache and facial pain

Headaches are very common and most people experience them from time to time. When they happen often, they may be part of a headache disorder. In the UK, more than 20 million people – around two in five adults – live with headache or facial pain disorders.[9]

Due to their prevalence, headaches are one of the leading burdens on society. Migraine costs the NHS around £1 billion per year, and it costs the UK economy around £10 billion per year.[9] New treatment approaches are desperately needed if we are to lift this burden.

Research into headache and facial pain has been limited in the past, which has slowed progress in developing better treatments. This is why we are prioritising this area. We fund research to improve understanding of what causes these conditions, as well as to improve diagnosis and treatment.

Over the past 10 years, we have invested £2.2 million in research to improve understanding and care in this area – through five project grants, five PhD studentships and one fellowship, listed in Appendix 3.

These projects cover cluster headache, migraine, idiopathic intracranial hypertension, and orofacial pain. They are a mix of clinical and discovery research projects, which attempt to elucidate the pathophysiology of different headache and facial pain disorders, identify possible treatment targets, and develop potential therapies.

Four of these awards have completed since our Impact Report 2024 and are outlined on the following pages.



Improving sleep in patients with chronic migraine

Project grant

£191,510

Sep 2021 – Jun 2025

University of Strathclyde

Lead researcher:
Dr Megan Crawford



Impact: **This project found that using a digital CBT-I intervention can reduce insomnia in some cases of migraine. The project also provided valuable insight into how best to design a larger clinical trial which, if successful, could support the use of digital CBT-I in patients with migraine with the aims of improving their sleep and quality of life.**

People with migraine often suffer from insomnia, a lack of sleep, which can, in turn, trigger a migraine. Treating the insomnia can break this cycle, and one of the best treatments is cognitive behaviour therapy for insomnia (CBT-I), which teaches people to change their sleep behaviours and thoughts/ worries about sleep loss.

Addressing sleep behaviours through digital cognitive behavioural therapy opens new pathways for breaking the cycle of migraine and insomnia.

In a previous small pilot study, Dr Crawford and her team found that people with migraine liked digital CBT-I, and that it improved both their insomnia and their migraines. This project built on the pilot study to develop a feasibility randomised controled trial (RCT) to see whether digital CBT-I could offer a better treatment than another option, and to refine the study methodology prior to progressing to full-scale clinical trial.

For this feasibility RCT, the team recruited 90 participants through several neurology services, and randomised participants into either a digital CBT-I group or a digital 'sleep hygiene education' control group.

Participants in both groups were asked to undertake various matched tasks, including completing baseline questionnaires, engaging with online materials, keeping a 30-day 'sleep and headache diary', wearing an actigraphy monitor (a device that tracks sleep and activity) for seven days to record their sleep and light exposure, and completing follow-up assessmentsat specific time points. In addition, a subset of participants and providers were interviewed to determine potential barriers to participation in this study and uptake of digital CBT-I.

The project found that insomnia and migraine symptoms improved post-treatment and at six months in both groups. More specifically, the digital CBT-I group improved more in insomnia symptoms at post-treatment than the control group, but there was no significant group difference at six months. There were also no differences between groups in improvement of migraine symptoms.

In addition, the study found that participants were motivated to take part in the trial and had high expectations of changes, but were concerned about the commitment involved, particularly relating to the need to keep the 30-day 'sleep and headache diary' and to wear an actigraphy device.

Understanding the role of inflammation in cluster headache

Project grant

£262,812

Jan 2022 – Jun 2025

King's College London

Lead researcher:
Dr Anna Andreou



Impact: **This project has shed new light on the processes underlying cluster headache, and has provided evidence of a specific way in which cluster headache could be treated in the future.**

Cluster headache is a rare and debilitating headache disorder, described as one of the most painful conditions known to man. The causes and underlying mechanisms of the disorder are not understood, limiting the development of effective treatments.

Aspects of cluster headache suggest that an area of the brain called the *hypothalamus* is involved in active headache clusters and attacks.

In this project, Dr Andreou studied the role of inflammatory and immune reactions in the hypothalamus, to delineate their role in cluster headache, and to explore possible routes to treatment. Specifically, Dr Andreou and her team investigated the role of a complex of proteins known as NF-kB, which are involved in several inflammatory processes.

What Dr Andreou and her team showed was that NF-kB is indeed a key mediator of the inflammatory responses in their model of headache, and that it is also very likely to play a role in cluster headache. The team also demonstrated that targeting the NF-kB pathway can be a future therapeutic avenue for the treatment of cluster headache.

Uncovering the role of inflammatory NF-kB protein pathways in the hypothalamus brings us closer to targeted treatments for cluster headache.

In addition, the team found evidence that calcitonin gene-related peptide (CGRP) treatments may not be effective for cluster headaches, despite being an effective treatment for migraine in some cases.

Cluster headache: 'the suicide headache'

Cluster headache is a rare headache disorder characterised by recurring bouts of excruciating pain on one side of the head. It affects one in 1,000 people, around 65,000 people in the UK, and affects six men to every woman.[13]
The intensity of the pain is often reported to give rise to suicidal thoughts. The causes of cluster headache are unknown, which severely limits our understanding of the disease. In order to work towards a cure, we first need to understand more about the underlying causes.

The genetic mechanisms of cluster headache

PhD studentship

£121,464

Aug 2021 – Jul 2025

University College London

Lead researcher:
Clarissa Rocca



Impact: **Clarissa's PhD demonstrated that calcium channel dysfunction in CACNA1G is linked to cluster headache, providing a new understanding of cluster headache and pointing to better ways of diagnosing and eventually treating this debilitating condition.**

Clarissa was awarded her PhD in July 2025, and has since secured a prestigious computational postdoctoral position at the National Institutes of Health (NIH), where she is continuing her work combining genetics, computation, and neurological disease.

People with close relatives who experience cluster headache have an increased risk of developing it themselves, which suggests an underlying genetic cause. During her MSc, Clarissa was involved in a study that identified two promising cluster headache genes. For her PhD, Clarissa built on this work – drawing on large national and international genetic databases to understand the roles of these two genes in cluster headache, working out how they cause the symptoms associated with the disorder.

Clarissa's PhD focused specifically on genes that control calcium flow in brain cells, particularly CACNA1G, a calcium channel gene. These channels act like small gates, allowing calcium to move into the cells. This process is essential for normal brain signalling, and when it goes wrong, it can trigger disease.

Discovering genetic variants in CACNA1G provides the first strong evidence that calcium channels are a critical pathway in cluster headache.

Clarissa studied families with cluster headache and discovered new genetic variants in the CACNA1G gene. In the laboratory, she tested these variants by introducing them into cells and recording how the calcium channels worked. These experiments showed that the variants reduce the normal activity of the channel, which helps explain how they could contribute to cluster headache. This is the first strong evidence that CACNA1G is involved in cluster headache. It also highlights calcium channels as a critical pathway in the disease and offers a potential new target for diagnosis and treatment in the future.

While working on CACNA1G, Clarissa also wanted to test whether another type of genetic change – called short tandem repeats (STRs) – might be linked to cluster headache. STRs are stretches of DNA where a sequence of letters is repeated many times. When they become too long, they can disrupt genes and cause disease. To study this, Clarissa generated data on repeats across many neurological genes. Although she did not find STRs directly linked to cluster headache, the analysis nonetheless showed the potential value of looking at repeat expansions in unexplained neurological cases.

Pain signalling in migraine

PhD studentship

£117,739

Oct 2020 – Feb 2024

University of Leeds

Lead researcher:
Char Palfrey



Impact: **The findings from Char's research provide new insights into how pain signals are amplified in the head, face and neck, which could lead to more effective treatments for migraine.**

Char submitted their PhD thesis in December 2024, and is now a Postdoctoral Research Associate at the University of Sheffield.

During a migraine, activated nerve fibres in the head send pain signals to the brain and release calcitonin gene-related peptides (CGRP), an inflammatory substance that sensitises nerves, increasing pain signalling. In groups of nerve cells further down the spine, previous research has suggested that a particular protein, ANO1, has a role in increasing activity of pain nerves, increasing the perception of pain.

Char's PhD research applied this previous research to groups of nerve fibres in the head that are activated during migraine, to investigate whether ANO1 plays a role in migraine, and whether it could be targeted by drugs to reduce pain signalling and migraine pain.

What Char discovered was that ANO1 forms dynamic molecular complexes in response to pain-inducing stimuli, which suggests that ANO1 may play a critical role in amplifying pain signals in the nerve cells that specifically detect painful stimuli in the head, face and neck. In particular, novel evidence from Char's PhD suggests that ANO1 function could significantly increase release of CGRP. This finding highlights ANO1 signalling mechanisms as an important novel therapeutic target for the development of new anti-migraine medications.

In addition, Char's project discovered that a protein called Esyt1 helps regulate the contact between different membranes within the neurons that are responsible for detecting and transmitting pain signals in the body and in the head, face and neck, and that Esyt1 also plays a role in modulating the ANO1 pain signalling processes, suggesting that it too could be a useful therapeutic target to disrupt pain signalling mechanisms.

Char's research shows that targeting the molecular interactions between ANO1 and other important pain signalling proteins could offer a novel way to interfere with pain pathways. In addition, understanding how proteins like Esyt1 regulate the formation of dynamic molecular complexes opens up new possibilities for therapeutic interventions aimed at reducing both chronic and acute pain.

Understanding how proteins regulate dynamic molecular complexes opens up new possibilities for reducing both chronic and acute pain.

Endowment funding

Targeting the heat shock response in ALS

Graeme Watts programme grant

£610,820

Jan 2020 – Dec 2025

UCL Queen Square Institute of Neurology

Lead researcher:
Professor Linda Greensmith



Impact: **This new understanding of how the heat shock pathway changes during disease progression in ALS has allowed the team to identify various potential therapeutic targets that could enable a precision approach to treating ALS and MND. The results of the second study suggest that Arimoclomol may have therapeutic potential in MSP, paving the way for a clinical trial of Arimoclomol in patients with MSP.**

Amyotrophic lateral sclerosis (ALS) is the most common form of adult motor neuron disease (MND). It is a rapidly progressing neurodegenerative disease in which motor neurons progressively die, resulting in muscle paralysis. There is no cure, and patients with ALS typically survive for only two to five years from diagnosis.

One of the key features of MND is *protein aggregation* – the abnormal clumping of proteins within motor neurons. In some earlier research undertaken in the Graeme Watts Laboratories, Professor Greensmith's team demonstrated that strategies that target protein aggregation in ALS may be a possible therapeutic strategy for ALS. Their results showed that treatment with a new compound called Arimoclomol prevented protein aggregation in neurons and as a result improved the disease in their mouse model of ALS. More specifically, Arimoclomol was shown to act by boosting a mechanism present in all cells called the *heat shock response*, which normally protects cells under stress but was less effective than normal in ALS.

Boosting the body's heat shock response helps prevent abnormal protein clumping, offering a promising therapeutic strategy for ALS.

As part of this programme grant, Professor Greensmith and her team worked on two key projects: (1) Characterisation of the heat shock response under ALS conditions to identify the key components of this pathway and to determine which cell populations could be targeted to rescue motor neurons in ALS, and (2) Investigating the therapeutic potential of Arimoclomol in another neurodegenerative disorder, multisystem proteinopathy (MSP), which is caused by mutations in a single gene, meaning that it is much easier to test and monitor the effects of Arimoclomol in patients.

In the first project, the team found that heat shock pathways are activated very early in ALS before symptoms appear, but initially fail to translate into effective protein-level protection until the symptomatic stages of disease.

In the second project, the team showed that treatment with Arimoclomol reduced pathology in the muscle, spinal cord and brain in their mouse model, as well as in human neurons developed from patient cells and grown in a dish, and improved muscle strength, motor neuron and cortical neuronal survival in mice.

Brain Research UK Chair of Neurosurgery

£1,684,556

Since Aug 2015

UCL Queen Square Institute of Neurology

Professor Robert Brownstone



Professor Brownstone took up the new role of Brain Research UK Chair of Neurosurgery in 2015. Here, he reports on the progress made over the past 10 years.

The Division of Neurosurgery at UCL Queen Square Institute of Neurology works closely with the National Hospital for Neurology and Neurosurgery to improve neurosurgical care. Their goal is to benefit both current and future patients through world class research, excellent training and education, and top-quality clinical care.

During Professor Brownstone's time as Brain Research UK Chair of Neurosurgery, the Division has been transformed into a world-leading centre of excellence that is at the forefront of developments in both clinical neurosurgery and neurosurgical research.

Professor Brownstone says:

"I would like to thank Brain Research UK for its support... I would also like to think that we are witnessing the rebirth of academic neurosurgery in the UK, and I hope that I have contributed at least a little to that."

Key achievements include:

- **More neurosurgeons** at all levels of seniority are now **involved in research**, from early-career doctors to senior professors.
- An **increase** in peer-reviewed **research publications** and in the value of **research funding** (reaching c.£6 million in 2025).
- **Regular** national and international **lectures**, including the annual Victor Horsley Lecture.
- An **annual Neurosurgery Research Day**, where trainees present their work and compete for the Lindsay Symon Research Award.
- A successful **Master's-level** (MRes) **Neurosurgery programme**, now in its fourth year.
- **Greater participation in clinical trials** to turn research discoveries into real treatments.
- **Progress on the development of the UCL Interdisciplinary Brain Cancer Centre**, in collaboration with other UCL departments and external partners.

References

1. Nomis. *Official census and labour market statistics.* nomisweb.co.uk
2. Cancer Research UK. *Brain, other CNS and intracranial tumours statistics.* cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/brain-other-cns-and-intracranial-tumours
3. The Brain Tumour Charity. *Glioblastoma prognosis.* thebraintumourcharity.org/brain-tumour-diagnosis-treatment/types-of-brain-tumour-adult/glioblastoma/glioblastoma-prognosis
4. Cancer Research UK. *Breast cancer statistics.* cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/breast-cancer
5. Cancer Research UK. *Prostate cancer statistics.* cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/prostate-cancer
6. Headway. *Brain injury statistics.* headway.org.uk/about-brain-injury/further-information/statistics
7. British Heart Foundation. *Heart statistics publications.* bhf.org.uk/what-we-do/our-research/heart-statistics/heart-statistics-publications
8. United Kingdom Acquired Brain Injury Forum (UKABIF). *Acquired brain injury and neurorehabilitation: Time for change (PDF).* cdn.ymaws.com/ukabif.org.uk/resource/resmgr/campaigns/appg-abi_report_time-for-cha.pdf
9. The Migraine Trust. *Rapid research review: What is the impact of migraine in the UK? (PDF).* migrainetrust.org/wp-content/uploads/2021/08/State-of-the-Migraine-Nation-impact-rapid-review.pdf
10. Bentivegna E, Galastri S, Onan D, Martelletti P. Unmet needs in the acute treatment of migraine. *Adv Ther* 2024;41:1–13.
11. Wang Y-F, Wang S-J. CGRP targeting therapy for chronic migraine – evidence from clinical trials and real-world studies. *Curr Pain Headache Rep* 2022;26:543–554.
12. Kuburas A, Russo AF. Shared and independent roles of CGRP and PACAP in migraine pathophysiology. *J Headache Pain* 2023;24:34.
13. World Health Organization (WHO). *Fact sheet: Migraine and other headache disorders.* who.int/news-room/fact-sheets/detail/headache-disorders
14. Medical Research Council. *Clinical researchers in the United Kingdom: Reversing the decline to improve population health and promote economic growth.* 2025.
15. The Brain Tumour Charity. *Brain tumour data: statistics that shock, but can also bring hope.* thebraintumourcharity.org/news/charity-news/brain-tumour-data-statistics-that-shock-but-can-also-bring-hope
16. Spinal Injuries Association. *What we do.* spinal.co.uk/about-us/what-we-do
17. Seminog OO, Scarborough P, Wright FL, Rayner M, Goldacre MJ. Determinants of the decline in mortality from acute stroke in England: linked national database study of 795,869 adults. *The BMJ* 2019;365:l1778.
18. Stroke Association. *Aphasia and its effects.* stroke.org.uk/stroke/effects/aphasia/effects
19. Chan C, Goadsby PJ. CGRP pathway monoclonal antibodies for cluster headache. *Expert Opin Biol Ther* 2020;20(8):947–953.

Appendix 1. Funded neuro-oncology projects, 2016 to 2026

Award year	Recipient	Project title	Award amount*
2025	Dr Ali Gooya University of Glasgow	**Project grant**: BIOGRAPH – Biophysical Tumour Growth Modelling for Novel GBM Treatment Response Assessment: Harnessing Physics-Informed Deep Learning	**£195,384**
2025	Dr Lucy Brookes University of Oxford	**Project grant:** Investigating vulnerabilities of polyploid tumour cells to disrupt therapy resistance in glioblastoma	**£299,872**
2025	Dr Elizabeth Cooper University of Cambridge	**Project grant:** Adaptive immune responses in establishing immune privilege in the normal and malignant developing brain	**£298,753**
2025	Professor Ester Hammond University of Oxford	**Project grant:** Improving radiation response in paediatric High-Grade Glioma through modulation of reactive oxygen species	**£226,513**
2025	Dr Hannah Jackson University of Nottingham	**Fellowship:** Unlocking the potential of circulating extracellular vesicles as biomarkers and therapeutic agents for paediatric ependymoma	**£295,185**
2024	Dr Harry Bulstrode University of Cambridge	**Project grant:** Gene and mRNA therapy approaches to tumour associated macrophages in glioblastoma	**£232,729**
2024	Professor Petra Hamerlik University of Manchester	**Project grant:** Development of D-2-hydroxyglutarate biosensor device for non-invasive and early detection of IDH1/2-mutant gliomas	**£299,916**
2023	Dr Daniel Williamson Newcastle University	**Project grant:** A mouse single cell atlas modelling radiotherapy-induced brain injury and late-effects in children treated for brain tumours	**£299,014**
2023	Dr Natividad Gomez-Roman University of Strathclyde	**Project grant:** Characterising cholesterol trafficking in glioblastoma and brain cells, and its contribution to tumour development, progression and treatment resistance	**£298,409**
2023	Febe Ferro University of Dundee	**PhD studentship:** Understanding the role of a secreted kinase in promoting myeloid signature of glioblastoma	**£124,989**
2023	Dr Kate Atkinson University of Birmingham	**PhD studentship:** Understanding the developmental origins of a childhood brain cancer	**£124,932**
2022	Professor Anthony Chalmers University of Glasgow	**Project grant – completed:** Investigating the radioprotective effects of ATM inhibition on the healthy brain	**£298,251**
2022	Mr Harsh Bhatt Cardiff University	**PhD studentship – completed:** A study of telomere biology in gliomas and drivers of genomic instability	**£122,541**
2022	Danielle Marsh University of Hull	**PhD studentship – completed:** The epigenetic regulatory protein CBX2 as a novel therapeutic target for glioblastoma	**£106,581**
2021	Mr Richard Mair University of Cambridge	**Project grant – completed:** Using a cross-species genomics approach to reveal the interplay between hypoxia and epigenomic deregulation in the transformation from low grade glioma to secondary glioblastoma	**£299,590**
2021	Dr Dirk Sieger University of Edinburgh	**Project grant – completed:** Analysing the impact of extracellular vesicles on the establishment of glioma promoting functions in microglia	**£298,427**

Continued over

* Final cost for completed projects

Appendix 1. Funded neuro-oncology projects, 2016 to 2026 (continued)

Award year	Recipient	Project title	Award amount*
2021	Lauren Gay Imperial College London	**PhD studentship:** Phage-guided CRISPR-Cas9 deletion of the mTOR therapeutic target in paediatric medulloblastoma	£120,000
2020	Professor Khuloud Al-Jamal King's College London	**Project grant:** Can immunogenic cell death be exploited in brain cancer?	£299,346
2020	Dr Manav Pathania University of Cambridge	**Project grant – completed:** The role of ATRX loss and G-quadruplexes in the pathogenesis and treatment of Diffuse Midline Gliomas	£289,874
2020	Mr Ryan Mathew University of Leeds	**Project grant – completed:** Characterising and therapeutically targeting the margin zone of infiltrative glioma using new in vivo patient-derived craniotomy orthotopic models	£296,086
2018	Professor Chris Jones The Institute of Cancer Research, London	**Project grant – completed:** The role and therapeutic targeting of ACVR1 in DIPG	£306,494
2018	Rhiannon Barrow University of Leeds	**PhD studentship – completed:** Investigating the role of TauT in conferring resistance to standard treatment in glioblastoma and its potential as a therapeutic target	£133,000
2017	Dr Claudia Barros University of Plymouth	**Project grant – completed:** Identification of novel genes controlling human glioblastoma initiation and growth mechanisms	£171,237
2017	Richard Baugh University of Oxford	**PhD studentship – completed:** Enhancing oncolytic virotherapy of glioblastoma	£128,750

* Final cost for completed projects

Appendix 2. Funded acquired brain and spinal cord injury projects, 2016 to 2026

Award year	Recipient	Project title	Award amount*
2025	Dr Stephen Ray University of Oxford	**Project grant:** Identifying the mechanisms, biomarkers and pathogenic causes of brain injury in febrile comatose Malawian children	£68,371
2025	Dr Aida Rodrigo-Albors University of Edinburgh	**Project grant:** Deconstructing the neural stem cell niche in a mammalian model of brain regeneration	£300,000
2024	Professor Stuart Allan University of Manchester	**Project grant:** Targeting interleukin-1 for the treatment of acute brain injury	£282,136
2023	Professor Rob Brownstone University College London	**Project grant:** Plasticity of neural circuits for micturition following spinal cord injury	£294,575
2023	Giada Vanacore University of Cambridge	**PhD studentship:** Facilitating regeneration in the brain via stimulation of neural stem cells	£125,000
2022	Dr Lynsey Duffell University College London	**Project grant:** Epidural spinal cord stimulation for restoration of uro-genital function after SCI	£294,732
2022	Dr Philippa Warren King's College London	**Project grant:** Recovering locomotion after acute ischemic spinal cord injury	£299,999
2022	Dr Virginia Newcombe University of Cambridge	**Project grant:** Understanding the pathogenesis of traumatic vascular injury	£292,069
2021	Dr Zahraa Al-Ahmady Nottingham Trent University	**Project grant:** A targeted approach to inhibit ferroptosis in a rodent model of intracerebral haemorrhage	£272,138
2021	Isobel Chick University College London	**PhD studentship – completed:** Face-to-face comprehension and production in aphasia: do multimodal non-arbitrary cues play a role?	£95,303
2019	Dr Kimberley Whitehead University College London	**Project grant – completed:** Brain injuries acquired during the neonatal period: investigating mechanisms which can correct depressed cortical electrical activity	£279,582
2019	Dr Barry McColl University of Edinburgh	**Project grant – completed:** Manipulating microglial/macrophage function via CSF1R to improve functional recovery and neuroplasticity after stroke	£271,931
2019	Professor David Menon University of Cambridge	**Project grant – completed:** Adaptive immune responses in traumatic brain injury	£280,848
2019	Haya Akkad University College London	**PhD studentship – completed:** Modulating speech recovery after stroke	£120,505
2018	Professor Simone Di Giovanni Imperial College London	**Project grant – completed:** Targeted multimodal modulation of neuronal activity for regeneration and recovery after spinal cord injury	£302,500
2018	Professor Nick Ward University College London	**Project grant – completed:** Investigating spontaneous early recovery after stroke	£339,589

Continued over

* Final cost for completed projects

Appendix 2. Funded acquired brain and spinal cord injury projects, 2016 to 2026 (continued)

Award year	Recipient	Project title	Award amount*
2018	Franziska Mueller Imperial College London	**PhD studentship – completed:** Combinatorial HDAC3 inhibition and neurorehabilitation for functional recovery following spinal cord injury	**£129,922**
2017	Dr Lawrence Moon King's College London	**Project grant – completed:** Improving grasping after brain injury (stroke) by enhancing spinal neuroplasticity combined with automated, intensive rehabilitation	**£317,575**
2017	Professor Sven Bestmann University College London	**Project grant – completed:** Re-opening the critical period for plasticity after stroke with dose-controlled non-invasive brain stimulation	**£421,105**

* Final cost for completed projects

Appendix 3. Funded headache and facial pain projects, 2016 to 2026

Award year	Recipient	Project title	Award amount*
2025	Dr Alejandro Labastida-Ramirez University of Manchester	**Fellowship:** Modulation of cortical spreading depolarisations by amylin in awake mice using functional imaging and electrophysiology	**£278,428**
2024	Dr Philip Holland King's College London	**Project grant:** Exploring the neural mechanism of fatigue in migraine: novel therapeutic avenues	**£299,851**
2023	Professor Zameel Cader University of Oxford	**Project grant:** Identifying a novel circadian coupled target for treating headache and facial pain	**£298,805**
2021	Dr Anna Andreou King's College London	**Project grant – completed:** Hypothalamic and immune interactions in cluster headache	**£262,812**
2021	Clarissa Rocca University College London	**PhD studentship – completed:** Investigating channel and pump genes in cluster headache	**£121,464**
2020	Dr Megan Crawford University of Strathclyde	**Project grant – completed:** Digital Cognitive Behaviour Therapy for Insomnia in Chronic Migraines: Feasibility of a Randomised Controlled Trial	**£191,510**
2020	Char Palfrey University of Leeds	**PhD studentship – completed**: Junctional multiprotein signalling complexes in trigeminal ganglion sensory neurones and their possible role in headache	**£117,739**
2019	Olivia Grech University of Birmingham	**PhD studentship – completed**: Defining mechanisms and new treatments for headache and raised intracranial pressure	**£92,744**
2018	Professor Peter McNaughton King's College London	**Project grant – completed**: HCN2 channels: a novel target for treatment of migraine	**£190,058**
2018	Oakley Morgan University College London	**PhD studentship – completed**: Stress, orofacial pain and headache disorders	**£135,000**
2017	Dr Emer O'Connor University College London	**PhD studentship – completed**: The genetics and pathophysiology of cluster headache	**£131,069**

* Final cost for completed projects

Together we can accelerate the progress of brain research.
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Published in November 2026