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Medical - 440 words

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Medical - 500 words

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Research – 600 words

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Leukaemia Virus Discovery May Help Treat HIV

By David Welsh, LPS Special Correspondent

Medical - 440 words

RESEARCHERS in England and Japan have discovered the mechanism by which human T-lymphotropic virus type one - the virus which causes adult T-cell leukaemia - spreads through the body.

Previously, it was not understood how HTLV-1 (human T-lymphotropic virus type one) was able to spread between cells and pass between individuals. According to the joint research published recently in the journal *Science*, the virus spreads by subverting normal T-cell (a type of immune cell) behaviour, without needing to release virus particles. The research is being done by Imperial College London and Oxford University, working with teams at the universities of Kagoshima and Ryukyus in Japan.

Professor Charles Bangham from Imperial College London at St Mary's Hospital is the senior author of the report. He said: "The HTLV-1 virus affects between 10 and 20 million people worldwide with around two to three per cent developing leukaemia. Although this research is not a cure, it does show how the virus is able to spread through the body and infect other people. From this we hope to be able to develop more effective treatments for this fatal disease."

The researchers used confocal microscopes to examine the distribution of HTLV-1 proteins and the genome in blood cells from HTLV-1-infected individuals, discovering that the HTLV-1 proteins and genome gather at the cell-cell junction and are then transferred to the uninfected cell. This method of transfer is not only very efficient but also helps the virus to avoid the immune system.

Most viruses spread through the body when an infected cell releases thousands of virus particles that travel in the blood or other body fluids to infect other cells. HTLV-1 has evolved a different strategy of spreading: instead of releasing viruses, the infected cell moves round the body and transfers the virus to other cells when they make contact.

This new finding could also have implications for the development of novel HIV/Aids treatments, said the scientists. Because the HIV-1 virus is similar to HTLV-1, it may be able to subvert immune cell physiology allowing the virus to spread between cells.

Professor Bangham added: "HTLV-1 is a very similar virus to HIV. It is spread through the same methods and, if we can use this research to develop a way of treating HTLV-1, this research could be a crucial first step towards developing new and more effective treatments to combat the global Aids epidemic." The research in both countries was supported by the UK's Wellcome Trust.

Tony Stephenson, Imperial College London Press Office, Sherfield Building, London, United Kingdom, SW7 2AZ. Telephone: +44 20 7594 6712

E-mail: at.stephenson@ic.ac.uk Website: www.imperial.ac.uk/

Probing The Genetics Of Brain Vulnerability

By David Welsh, LPS Special Correspondent
Medical - 500 words

WHY are people with a specific genetic make-up more likely to develop brain disease and less likely to make a good recovery from head injury? A study by researchers at Edinburgh University, Scotland, has important implications for those with the particular brain protein and who choose to take part in potentially dangerous contact sports such as boxing, ice hockey, football and rugby.

One third of the population carries the specific form of brain protein (APOE-4), which is known to be associated with higher danger both of developing dementia and also of recovering less well from a head injury.

At present there are few effective treatments for brain injury and damage and there is poor understanding of the underlying mechanisms involved. The Edinburgh scientists aim to understand why people with APOE-4 protein are more vulnerable to the effects of brain injury and diseases.

Dr Karen Horsburgh, of the Centre for Neuroscience Research, Edinburgh, said: "In the United Kingdom more than 100,000 people are admitted to hospital each year with head injury, and there are an estimated 100,000 head injury survivors with persistent handicap - many of these are young people.

"A history of previous head injury is also a major risk factor in developing Alzheimer's disease, the cause of most cases of dementia. Alzheimer's affects five per cent of the population over the age of 65 and 20 per cent of those aged over 80, so it is seen as a growing problem in an ageing society.

"Brain cells slowly deteriorate and die as part of normal ageing but this process is accelerated in Alzheimer's disease and also as a result of a brain injury such as stroke or head injury. Those with APOE-4 have a greater risk than normal of developing dementia.

"We also believe that young people carrying APOE-4 will make a worse recovery from head injury, perhaps because of a reduced capacity for brain repair. This research therefore has implications for doctors treating patients with brain injury, stroke or dementia and strategies for patient care," added Dr Horsburgh.

The Edinburgh team is taking part in a major joint initiative to discover how gene mutations can influence a broad range of behaviour, especially intellectual and memory functions.

The researchers are working with scientists from the Wellcome Trust Sanger Institute near Cambridge, England, on a five-year research programme costing six million pounds to try to find new ways of treating a range of diseases, including inherited learning disabilities and psychiatric disorders. The programme draws together experts in human

genetics, psychology, psychiatry and neuroscience to better understand normal brain function.

As well as basic molecular and genetic research it includes human clinical studies of mental health, intellectual function and learning disorders. The programme of research aims to create a general model for tackling diseases and brain function and establish the basis of memory and intellectual disorders and diseases, including common inherited learning disabilities.

Dr Horsburgh and her colleague Professor James McCulloch's study of the links between APOE-4 and brain injury and disease is supported by the Wellcome Trust.

Linda Menzies, Communications and Public Affairs, University of Edinburgh, 7
Nicolson Street, Edinburgh, United Kingdom, EH8 9BE. Telephone: +44 131 650 6382.
Fax: +44 131 650 2253
E-mail: Linda.Menzies@ed.ac.uk Website: www.ed.ac.uk/

Investing In Tomorrow's Technologies

By David Welsh, LPS Special Correspondent

Research – 600 words

IN THE latest round of funding under the United Kingdom government's Basic Technology Programme, 21 million pounds sterling is allocated to seven research projects, ranging from computer codes that defy the fraudster, to more efficient ways of diagnosing and treating disease and new techniques in oil exploration.

The programme aims to support the development of fundamental new technology that "will apply to the entire scope of scientific, engineering and technology endeavour in the next 10 to 20 years", and underpin major new industries of the future.

Research which will benefit from this round of funding includes a project to develop a fundamentally new method of sequencing, screening and synthesising DNA (deoxyribonucleic acid) at a rate that is thousands of times faster than existing methods.

This is led by Southampton University, southern England, and if successful it will allow four billion bases to be sequenced in less than a day on one instrument – an advance with the potential to change the landscape of biological and medicinal science, leading to huge changes in personalised medicines and healthcare.

Also with applications in the medical field as well as in materials science is a collaboration led by Nottingham University, English Midlands, to develop and enhance technologies that increase dramatically the magnetic alignment of materials (known as hyperpolarisation).

This will improve the sensitivity of magnetic resonance imaging (MRI) and spectroscopy up to a million fold, enabling new applications in cancer and other fields of medical research, and in industrial applications ranging from oil exploration to pharmaceuticals.

Knowledge of materials will also be boosted by attosecond technology, enabling the very fast motion of atoms and electrons, occurring on a time scale known as attoseconds (one attosecond equals one billion-billionth of a second) - to be studied for the first time by using a light pulse of attosecond duration to probe these super-fast processes.

The technology will, it is predicted, provide many new opportunities in understanding electron motion in molecular, nano-scale and, ultimately, even biological structures. It is being carried out by a team at London's Imperial College.

The use of light in analysing biological samples is undergoing explosive growth. Another of the seven projects, based at the University of Wales College of Medicine in Cardiff, aims to bring down to a micro-scale the optical tools of a modern life-sciences laboratory so that they are contained in a portable microchip, including lasers the size of a single human cell.

Samples, such as live cells, will be moved around this “optical biochip” for processing and analysis. A big social and medical benefit would arise from being able to place the power of a sophisticated, intelligent laboratory at the “point of care”, helping to diagnose disease and select treatment, whether in the field, a clinic or the home.

A project to develop quantum computing for a variety of specialised applications including security is based at University College London. Such devices, the researchers believe, could “catalyse many new applications, just as the laser surprised its inventors”.

Although room-temperature quantum computing is a long-term aim, many of the most exciting developments in quantum devices need a cryogenic environment and must be supported by several stages of electronics operating at successively lower temperatures. A project led by Oxford University scientists aims to help integrate appropriate technologies to make full-scale quantum computers.

Finally, in this round of funding, is a scheme to develop technologies to predict all the possible crystal structures (polymorphs) of organic materials, again based at University College London. All industries involved in the manufacture of organic materials, including the pharmaceutical and speciality chemical companies, would benefit from such an advance.

Alasdair Rose, Basic Technology Programme Manager, Research Councils UK, Polaris House, North Star Avenue, Swindon, United Kingdom, SN2 1ET. Telephone: +44 1793 444225.

E-mail: alasdair.rose@epsrc.ac.uk/ Website: www.research-councils.ac.uk/basictech/

Smile And The World Does Smile With You

By Dr Penny Lewis, LPS Special Correspondent

Medical – 680 words

IF YOU are depressed but think everyone looks more approachable and happier, then you may mimic their behaviour, thus eventually overcoming depression. That is a simple summary of new research suggesting that the ability of antidepressants to improve state of mind may be due to the influence of these drugs on social interaction.

Volunteers in the United Kingdom who took antidepressants for a week were asked to identify facial expressions presented on a computer screen. The expressions showed six emotions: happiness, sadness, anger, fear, disgust and surprise. Volunteers on antidepressants identified more of the presented faces as happy and fewer as fearful or disgusted than volunteers taking a placebo pill.

Prior work has shown that depressed patients tend to focus excessively on negative social cues, ignoring the more positive information in their environment. Dr Catherine Harmer of Oxford University, the author of the present study, therefore suggests that the shift towards positive interpretation of facial expressions might lead to improved social interactions and eventually to a better state of mind.

“When people are depressed they often report that everyone is looking at them in a negative way, as if they are disgusted with them or angry,” explained Dr Harmer. “When they are on antidepressants, they often report that people seem to like them more. But our work suggests that this might be because their perception is different. If you think everyone looks more approachable and happier, then you are going to behave differently - maybe interacting with them more and thus eventually overcoming depression.”

Two chemical messengers in the brain, called serotonin and norepinephrine, are known to be linked to depression and anxiety. Increasing the levels of these substances counteracts depression, high levels of anxiety, and feelings of worthlessness or guilt. Decreasing their levels leads to an opposite swing in mood.

“If you reduce serotonin in the brains of people who have been depressed in the past, and are therefore vulnerable to relapse, they will become depressed again,” said Dr Harmer.

This reduction can be accomplished by asking patients to drink a milkshake containing a high proportion of amino acids, the building blocks for proteins. The specific amino acid which is needed to construct serotonin is excluded from this drink.

Because all of the building blocks present in the blood compete to cross into the brain, decreasing the proportion of this one means that less of it gets in, and less serotonin can be made in the brain.

Most antidepressant drugs work by increasing levels of serotonin and norepinephrine, but the improvement they provide comes only after a long delay. Patients typically

experience an initial decrease in anxiety during the first few days of drug taking, followed by decrease in anxiety and a gradual, increase in happiness which does not peak until the drug has been taken for a few weeks.

“The critical issue with antidepressants is why they take so long to work. They can take a month to kick in and, if they start increasing these chemicals (serotonin and norepinephrine) straightaway, it is not clear why this delay occurs,” added Dr Harmer.

There are various hypotheses around, such as autoreceptor desensitisation (the idea that antidepressants do not immediately increase levels of serotonin in the brain because the brain initially compensates for the increase by activating a special “negative feedback” receptor which temporarily prevents it).

“After a few weeks, this negative feedback process becomes less effective and levels of serotonin are able to increase, but even given that, you do still see effects on chemical messengers before you get a clinical response,” said Dr Harmer.

If the effect of antidepressants is modulated by improved social interaction then an alternate explanation for their delayed effects becomes apparent, because the changes in behaviour which might lead to improved mood and outlook would likely evolve over several weeks.

“It might just take time for the effects of feeling better and interacting with the people around you to build up,” added Dr Harmer.

The new findings could represent a break-through in our understanding of how antidepressants function. Future investigations will determine whether the alterations in recognition of facial expressions seen in normal volunteers are also found in depressed patients who take the drugs. Confirmation of this hypothesis might lead to new therapies specifically targeting improved social interaction as a way of fighting depression.

Press Office, University of Oxford, University Offices, Wellington Square, Oxford, United Kingdom, OX1 2JD. Telephone: +44 1865 280531.

Website: www.ox.ac.uk

Dr Catherine Harmer e-mail: Catherine.harmer@psychology.ox.ac.uk