News in brief

Targets and mechanisms

Stem-cell marker identified

Researchers have discovered what is believed to be the first universal stem-cell marker. The team at St Jude Children's Hospital (Memphis, TN, USA) has found that expression of the gene *Bcrp1* (also known as *ABCG2*) plays a crucial part in the identification of stem cells from various sources[1]. This will enable researchers to more accurately identify true stem cells than previously available methods. The *Bcrp1*/*ABCG2* gene was specifically expressed in stem cells in bone marrow, skeletal muscle and early mouse embryo.

It had previously been assumed that the expression of ABC transporters was responsible for the phenotype of stem cells from these tissues (known as the side-population phenotype), although specific molecules had not been identified. The group, led by Brian Sorrentino, Director of Experimental Hematology, showed that expression of the *Bcrp1*/*ABCG2* gene is the conserved feature of these stem cells. By contrast, mature cells did not show expression of the gene at all, highlighting the potential use of the gene as a stem-cell marker.

This discovery is particularly important because of the current research to find new uses for these undifferentiated cells; there are hopes for cures for Parkinson's and Alzheimer's diseases, strokes, heart attacks, liver disorders and spinal cord injuries, and the possibility of restoring function to damaged organs. In addition, the researchers found that the expression of this gene could ensure that the cells do not further differentiate, leading to the possible control of stem cell differentiation.

1 Zhou, S. *et al.* (2001) The ABC transporter Bcrp1/ABCG2 is expressed in a wide variety of stem cells and is a molecular determinant of the side-population phenotype. *Nat. Med.* 7, 1028–1034

Neuroimplantation trial shows first signs of success

Scientists have detected cell growth in nerve tissue implanted in the brains of stroke patients, a recent report claims[2]. Researchers at the University of Pittsburgh Medical Center (Pittsburgh, PA, USA) performed positron emission tomography (PET) scanning in patients a week after they had suffered strokes that resulted in persistent motor deficits. They then used PET imaging to study the brains of these patients six and twelve months after neuron implantation surgery. The metabolic response of the brain to neuron implantation was measured by the uptake of a glucose analogue, fluorodeoxyglucose (FDG).

They found that alterations in glucose metabolism correlated with measurements of motor performance (measured using the National Institutes of Health stroke scale and the European stroke scale). These preliminary studies suggest that neural implantation might improve local cellular functions in some patients. Carolyn Cidis Meltzer, lead author of the study, said, 'Although this is not direct evidence of synapse formation, it does suggest that new neurons are being wired into the brain.'

2 Meltzer, C.C. *et al.* (2001) Serial [18F] fluorodeoxyglucose positron emission tomography after human neuronal implantation in stroke. *Neurosurgery* 49, 586–591

Growth factor promotes neuron cell growth in adult rats

Scientists have reported further evidence that a growth factor can influence neuron survival and differentiation in the brain of adult rats[3]. It has previously been shown that brain-derived neurotrophic factor (BDNF) promotes the survival and/or differentiation of progenitor cells in the subventricular zone of the brain and increases the number of new neurons in the adult migratory stream and olfactory bulb[4].

More recently, researchers at the Emory University School of Medicine (Atlanta, GA, USA) have followed up this work by demonstrating that BDNF influences the proliferation and differentiation of cells in other regions of the adult rat forebrain. Sixteen days after a 12-day intraventricular infusion of BDNF and bromodeoxyuridine (BrdU; a marker of cell division), the distribution and phenotype of newly generated cells in the adult rat forebrain was determined. The infusion of BDNF resulted in many BrdU-stained cells in areas of the brain that do not usually undergo neurogenesis in adulthood, such as the thalamus and hypothalamus. These findings were correlated with the expression of the neuronal marker, microtubulin-associated protein-1, as well as with the expression of full-length TrkB, which is the high affinity receptor for BDNF. These findings suggest that the adult brain might be able to recruit and/or generate new neurons to replace those lost by disease or injury.

3 Pencea, V. *et al.* (2001) Infusion of brain-derived neurotrophic factor into the lateral ventricle of the adult rat leads to neurons in the parenchyma of the striatum, septum, thalamus and hypothalamus. *J. Neurosci.* 21, 6706–6717

4 Zigova, T. *et al.* (1998) Intraventricular administration of BDNF increases the number of newly generated neurons in the adult olfactory bulb. *Mol. Cell Neurosci.* 11, 234–245

Getting connected

Researchers are one step closer to describing how neurones in the brain and spinal cord form their connections[5]. The research describes the role of ephrins and the Eph receptor, which have been found to communicate biochemical signals between two cells.

'We found that these molecules communicate important signals that guide the growing tips of embryonic nerve fibres and, therefore, help form networks of neurones and synapses in the brain in a process called axon pathfinding,' said Mark Henkemeyer, Assistant Professor in the

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Center for Developmental Biology at the University of Texas Southwestern Medical Center (Dallas, TX, USA).

Until now, researchers thought the ephrins were ligands that bound to the Eph receptor on the axon, thereby activating the axon pathfinding signal. However, the findings suggest that the ligands are also themselves receptors. Ephrins, therefore, have the capability of 'reverse signalling' – triggering biochemical signal transduction cascades in their own cell.

The entire circuitry of the brain and nervous system is controlled by axon pathfinding. By learning how cells communicate in axon pathfinding, researchers might ultimately be able to determine how to coax nerves to regrow and regenerate.

5 Cowan, C.A. and Henkemeyer, M. (2001) The SH2/SH3 adaptor Grb4 transduces B-ephrin reverse signals. *Nature* 413, 174–179

Promising results in HIV and AIDS research

Scientists have discovered that individuals with HIV who also carry the flavivirus GB virus C (GBV-C) have a lower mortality rate than those with HIV alone[6,7]. GBV-C is related to hepatitis virus C but does not cause liver disease.

The two studies examined the effect of coinfection of GBV-C on the survival of patients with HIV infection. Those patients with the GBV-C virus survived significantly longer and had a slower progression to AIDS. Survival after the development of AIDS was also higher. It is possible that the presence of GBV-C leads to an inhibition of HIV replication, or that the virus indicates the presence of other factors that lead to a favourable HIV response.

In a separate study, researchers at the Dana-Farber Cancer Institute (Boston, CA, USA) describe how HIV functions to block the ability of the immune system to fight infection[8]. The finding, based on X-ray crystallography images, shows a meeting between T cells and HIV-1. T cells use CD4 receptors on their cell surface to recognize protein fragments cupped within class II major histocompatibility complexes (MHCs) on cells, to identify whether a cell is normal or infected.

Glycoprotein 120 (gp120) on the surface of the HIV virus latches onto the CD4 receptor enabling HIV to be transferred into the cell. The new images show a complete structural rendering of both of these interactions, and the gp120 protein is shown to cover a greater portion of the CD4 receptor than MHC. T cells thus form a stronger bond with HIV than they do with cells that help fight infections, essentially blindfolding the T cells to the presence of infections and cancer. Drugs that interfere with the ability of HIV to link with helper T-cells by binding to the gp120 protein could help prevent HIV infection while retaining the body's ability to fight disease.

6 Xiang, J. *et al.* (2001) Effect of coinfection with GB virus C on survival among patients with HIV infection. *New Engl. J. Med.* 345, 707–714

7 Tillmann, H.L. *et al.* (2001) Infection with GB virus C and reduced mortality among HIV-infected patients. *New Engl. J. Med*. 345, 715–724

8 Wang, J-H. *et al.* (2001) Crystal structure of the human CD4 N-terminal two-domain fragment complexed to a class II MHC molecule. *Proc. Natl. Acad. Sci. U. S. A.* 98, 10799–10804

Platelet-destroying antibody identified

An antibody has been identified that can destroy an essential component of the blood – the platelets. Recent research by scientists at the New York University (NYU) School of Medicine (New York, NY, USA) could open up new therapeutic avenues to treat blood platelet disorders associated with HIV infection, arterial blood clots and other types of vascular disease.

Simon Karpatkin and colleagues[9] showed that an antibody that binds to a specific protein on the platelet surface causes them to self-destruct, and that this occurs by the triggering of a particular pathway in platelets.

The researchers demonstrated that platelet lysis is caused by the antibody-induced generation of hydrogen peroxide, thus describing a novel mechanism of platelet clearance whereby anti-platelet IgG causes platelet fragmentation via the induction of reactive oxygen species.

Platelets are required for the blood to clot; when the platelet count is low, thrombocytopenia occurs and the blood cannot clot, with fatal consequences. Simon Karpatkin says, 'We believe that this discovery could be of use in the clinic. It is conceivable that this antibody could be used to dissolve clots in arteries, which can cause myocardial infarction, stroke and other conditions.' The group has developed inhibitors of the new antibody, which could potentially prevent the destruction of platelets in patients with HIV, where the platelet count is abnormally low. They are also developing monoclonal antibodies to destroy platelets, which could be useful in the treatment of arterial clots and strokes.

9 Nardi, M. *et al.* (2001) Complement-independent, peroxide-induced antibody lysis of platelets in HIV-1-related immune thrombocytopenia. *Cell* 106, 551–561

PET can predict mild cognitive impairment and Alzheimer's disease

Researchers have been able to predict the development of memory impairment in healthy elderly individuals based on scans of their brain[10].

The three-year study, conducted at the New York University (NYU) School of Medicine (New York, NY, USA), showed that metabolic changes occur in specific regions of the brain years before any clinical signs of memory loss, and that these changes can be detected using positron emission tomography (PET).

The study examined 48 healthy men and women aged 60–80 years. Although all the subjects scored within the normal range in the tests normally used to detect early memory loss, the PET scans showed a reduction in glucose metabolism in the entorhinal cortex in the brain of 12 of the individuals.

Three years later, 11 of these 12 people had experienced mild cognitive impairment (MCI) whereas the other subject had developed Alzheimer's disease. Meanwhile, the subjects with normal PET scans showed no signs of mental decline at the three-year time point.

Mony J. de Leon, Director of the Center for Brain Health and Professor of Psychiatry at NYU School of Medicine said: 'We need to confirm our results with a larger group of subjects and to identify the biological and physiological factors leading to the metabolism losses. If we can identify these factors, then we might be able to find a way to delay the onset of Alzheimer's or prevent it altogether.'

10 de Leon, M.J. *et al.* (2001) Prediction of cognitive decline in normal elderly subjects with 2-[^{18}F] fluoro-2-deoxy-D-glucose/positron-emission tomography (FDG/PET). *Proc. Natl. Acad. Sci. U. S. A.* 98, 10966–10971

Clinical trials

New highs for medication

The first safety and efficacy data from Phase I and II clinical trials on medicines derived from cannabis have been released and show promising results. The data from the studies conducted by GW Pharmaceuticals (London, UK) were presented to the *American Academy of Pain Management* conference (Arlington, VA, USA).

The placebo-controlled studies examined the effects of the sublingual administration of various cannabis-based treatments on 53 patients suffering principally from multiple sclerosis or spinal cord injury. Of this group, 41 patients showed a clinically significant benefit in terms of key outcomes (namely pain, overall symptom relief and sleep duration), enough for them to opt to continue active long-term treatment. GW also claims that any adverse events were predictable and were generally well tolerated. Furthermore, on analysis of the dosages required over an extended period of time, there was no evidence of tolerance and, therefore, no need to progressively increase the dose.

Following the presentation of these results, GW announced that the Medicines Control Agency (MCA; London, UK) has approved the extended use of its cannabis-based medicines from one to two years of treatment. The Executive Chairman or GW Pharmaceuticals, Geoffrey Guy, said that the company hopes to bring the first cannabis-based prescription medicine to the market in the year 2004.

Cancer targets and mechanisms

Kinase on the lookout for mutations

Scientists have discovered a key protein involved in regulating the expression of genetic mutations, which could lead to new therapies for genetic disorders. Researchers at the National Cancer Center Research Institute (NCCRI) in Tokyo (Japan) have identified a novel phosphatidylinositol-3-kinase-related protein kinase (hSMG-1) and have shown that it associates with components of the mRNA surveillance complex and is involved in the regulation of nonsense-mediated mRNA decay (NMD)[11].

NMD is a conserved surveillance mechanism that eliminates imperfect mRNA molecules that contain premature translation termination codons (PTCs) and encode potentially harmful or non-functional proteins. HSMG-1 is a human orthologue of a product of the *smg-1* gene in *Caenorhabditis elegans*, which is involved in NMD.

Shigeo Ohno and colleagues from the NCCRI have demonstrated that overexpression of a kinase-deficient point mutant of hSMG-1 results in a suppression of PTC-dependent β-globin mRNA degradation, whereas wildtype hSMG-1 enhances degradation. Furthermore, they report that inhibitors of hSMG-1 cause the accumulation of truncated p53 in human cancer cell lines with a p53 PTC mutation. Ohno points out, however, that the accumulation of truncated proteins is not necessarily a detrimental event, but might actually be desirable. Inhibition of NMD by hSMG-1 could, therefore, enable the accumulation of truncated proteins that can at least partially compensate for a genetic disorder. Considering that one-quarter of all mutations in human genetic disorders are of the type that could target mRNA for NMD, therapies developed from this research could have widespread use.

11 Yamashita, A. *et al*. (2001) Human SMG-1, a novel phosphatidylinositol-3-kinase-related protein kinase, associates with components of the mRNA surveillance complex and is involved in the regulation of nonsense-mediated mRNA decay. *Genes Dev.* 15, 2215–2228

Double-action antitumour vaccines

The molecular chaperone calreticulin (CRT) has been found to work as a potent antitumour vaccine by two mechanisms[12]. CRT has a variety of functions but its most interesting roles for tumour biology are its cleavage to form the antiangiogenic vasostatin, and its ability to facilitate antigen loading by associating with the Class I major histocompatibility complex (MHC).

Researchers at Johns Hopkins University School of Medicine (Baltimore, MD, USA) have engineered a fusion gene encoding a known viral tumour antigen, human papillomavirus type-16 (HPV-16) E7, linked to CRT, for potential use as an antitumour vaccine. Intradermal vaccination of mice with the fusion gene resulted in a marked increase in E7-specific $CD8^+$ T-cell precursors and a significant antitumour effect against E7-expressing tumours compared with mice vaccinated with either wildtype E7 or CRT DNA. Furthermore, vaccination of CD4/CD8 double-depleted mice and immunocompromised mice with either E7–CRT DNA or CRT DNA caused a significant decrease in lung tumour nodules. This suggests that the antiangiogenic role of CRT might have contributed to the antitumour effect. Further studies of microvessel diameter in the lungs and an *in vivo* angiogenesis assay confirmed this to be the case. This DNA vaccination protocol, therefore, provides a double-action approach to cancer therapy.

12 Cheng, W-F. *et al.* (2001) Tumor-specific immunity and antiangiogenesis generated by a DNA vaccine encoding calreticulin linked to a tumor antigen. *J. Clin. Invest.* 108, 669–678

Glutamate is key to slowing brain tumour growth

Scientists have shown that the essential and ubiquitous neurotransmitter glutamate is released by brain tumours to destroy the surrounding healthy neural tissue and provide a growth advantage to tumour cells[13]. Glutamate toxicity has already been associated with stroke, head trauma, multiple sclerosis and other neurodegenerative disorders.

Maiken Nedegaard and colleagues at the New York Medical College (Valhalla, NY, USA) studied the release of glutamate from freshly isolated brain slices using bioluminescence, and noticed that implanted glioma cells releasing high levels of glutamate grow 15-fold more rapidly than those that did not release the neurotransmitter. Treatment with N-methyl-D-aspartate (NMDA) receptor antagonists slowed the growth of the glutamate-secreting tumours *in situ*, suggesting that NMDA-receptor activation promotes tumour growth. Drugs that

suppress glutamate production or glutamate binding to receptors on healthy brain cells could, therefore, be potential treatments for brain tumours.

13 Takano, T. *et al.* (2001) Glutamate release promotes growth of malignant gliomas. *Nat. Med.* 7, 1010–1015

Structural elucidation of integrin heralds the next generation of cancer drugs

The publication of the structure of the extracellular segment of integrin $\alpha_v\beta_3$ completes the elucidation of the full structure of this protein, which has roles in cell–cell and cell–matrix adhesion and is, therefore, a key target in cancer drug discovery. A collaboration between Merck (Darmstadt, Germany) and the Massachussetts General Hospital (MGH; Boston, MA, USA) has resulted in the crystallization of the extracellular portion of integrin $\alpha_v\beta_3$ at 3.1 Å resolution[14].

The protein's 12 domains assemble into a 'head' and two 'tails', with a severe bend in the tails that reflects an unusual flexibility, possibly linked to regulation. The main inter-subunit interface greatly resembles the Gα–Gβ interface of the G-proteins. A metal-ion-dependent adhesion site (MIDAS) is positioned in a ligand-binding interface, and lies adjacent to a calcium-binding site with a potential regulatory function. M. Amin Arnaout, Director of the Structural Biology Program at MGH, said, 'Knowing the shape of this receptor will help us all develop new strategies to target many serious diseases.' Simon L. Goodman from Merck added, 'The better you know the precise shape of the binding site of the integrin, the better you are able to design specific inhibitors, which could result in novel drugs.'

14 Xiong, J-P. *et al.* (2001) Crystal structure of the extracellular segment of integrin $\alpha_v\beta_3$. *Sciencexpress* 10.1126/science.1064535 (http://www.scienceexpress.org)

Diabetes targets and mechanisms

Switching off diabetes

A molecular switch that regulates the production of glucose by the liver has been

discovered by researchers at the Dana-Farber Cancer Institute (Boston, MA, USA)[15]. Researchers have been looking for this switch for more than 20 years. The finding could lead to the design of new drug treatments for individuals with diabetes.

Gluconeogenesis, the manufacture of glucose in the liver, is regulated by insulin, which signals the liver to halt glucose production when blood sugar levels become too high. Because individuals with diabetes lack insulin, they are unable to regulate this process, thereby leading to dangerously high levels of blood sugar.

PGC-1, a protein that is active in liver cells, indirectly switches on gluconeogenesis. The study found that PGC-1 is the key regulator that insulin acts upon. Adenoviral-mediated expression of PGC-1 in hepatocytes activates an entire programme of key gluconeogenic enzymes, leading to increased glucose output. In mice, PGC-1 was found to turn on genes that triggered glucose production and caused hyperglycemia (high blood sugar). Transcription co-activators could, therefore, be specifically designed to block this protein, thus halting glucose production and providing new therapies to help diabetics.

15 Herzig, S. *et al.* (2001) Control of hepatic gluconeogenesis through transcriptional coactivator PGC-1. *Nature* 413, 131–138

Free radicals: a pain in diabetes

A treatment that could potentially combat the long-term complications of both type 1 and type 2 diabetes has been developed by researchers at the Veterans Affairs Medical Center (Jackson, MA, USA) and the University of Iowa (Iowa, IA, USA) in conjunction with MetaPhore Pharmaceuticals (St Louis, MO, USA)[16]. Complications such as neural dysfunction affect nearly 50% of all diabetics who have had the disease for 25 years or more.

Tests on diabetic rats showed that vascular abnormalities and neuropathy caused by an excess of free radicals could be significantly reduced by daily subcutaneous injections of a superoxide dismutase (SOD) mimetic (M40403). Eleven streptozotocin-induced diabetic rats were given daily injections of 10 mg kg^{-1} 'enzyme mimetic'. M40403 improved diabetes-induced vascular relaxation in arterioles around the sciatic nerve, decreased endoneurial blood flow and restored normal motor nerve conduction velocity when compared with both control and untreated diabetic rats. M40403 is a manganese (II) complex with a *bis*(cyclo-hexylpyridine)-substituted macrocyclic ligand. SOD mimetics have the advantage that they are low-molecular weight and do not appear to elicit an immune response.

The studies show that diabetes-induced oxidative stress might be one of the causes of vascular and neural complications in diabetes, and a significant new approach for treating these sufferers has been uncovered.

16 Coppey, L.J. *et al.* (2001) Effect of M40403 treatment of diabetic rats on endoneurial blood flow, motor nerve conduction velocity and vascular function of epineurial arterioles of the sciatic nerve. *Br. J. Pharmacol.* 134, 12–29

Aspirin as a possible treatment for diabetes?

Scientists have recently demonstrated that the age-old drug aspirin (a salicylate) can reverse high blood sugar, high insulin and high blood-fat levels in obese rodent models. The group at the Joslin Diabetes Center and the University of California, San Diego (CA, USA) report that high doses of salicylates influence a pathway in cells, known as the IKKβ pathway, which, in itself, appears to inhibit the effects of insulin[17].

The researchers, led by Steve Shoelson, focussed on the underlying mechanism of the blood-sugar-lowering effect of aspirin, performing their studies on genetically obese rodents, which develop diabetes in the same way as humans. They determined that these animal models had an increased sensitivity to the effects of insulin, producing less insulin, and also had low

levels of glucose, free fatty acid and triglyceraldehydes. The IκB kinase β was identified as the probable target for aspirin; activation of IκB attenuated insulin signaling in cultured cells, whereas IKKβ inhibition reversed insulin resistance.

Previously, aspirin was ignored for diabetes treatment because of the lack of understanding of its effects; high-dose aspirin therapy can cause dizziness, tinnitus and gastrointestinal disorders, including sickness and bleeding, if taken for prolonged periods of time. Shoelson says, 'If a drug could be developed with this capacity to lower blood sugar, but without high-dose aspirin's side effects, we could potentially have a potent new treatment for type-2 diabetes.'

These recent findings of the involvement of the IKKβ pathway could provide a valuable target for the discovery of new drugs to treat patients with insulin resistance and type-2 diabetes.

17 Yuan, M. *et al.* (2001) Reversal of obesity- and diet-induced insulin resistance with salicylates or targeted disruption of IKKβ. *Science* 293, 1673–1677

Miscellaneous

Biotech gains where pharma suffers

Heading your own company is tempting the pharmaceutical industry's brightest minds away from larger corporations to the biotechnology sector, revealed a recent survey by 3i Healthcare (London, UK) conducted at the recent CEO conference in Barcelona, Spain. Many delegates felt that this was resulting in a 'brain drain' in the larger pharmaceutical companies to the benefit of the biotech companies.

Of 70 European biotechnology CEOs questioned, many said that innovations and advances in the biotechnology sector have benefited from its more nimble and dynamic entrepreneurial culture. Furthermore, 74% of CEOs questioned also agreed that increasing investment over the next decade would increase the reliance of pharmaceutical and medical technology companies on the expanding biotechnology sector for future growth. In fact, they predicted that by 2010, pharmaceutical companies would double their reliance on in-licensing new products, which currently accounts for 20% of pharma's revenues.

Prostate cancer market expected to double by 2007

Revenues for the combined segments of localized, locally advanced and metastatic prostate cancer therapeutics reached US$1.2 billion in 2000 and should double by 2007, concludes a recent report from Frost & Sullivan (San Jose, CA, USA) entitled *Emerging Prostate Cancer Therapeutics Market*. Growth will be driven by demand for therapies with increased survival rates and fewer side effects. Monoclonal antibody products, bulk and patient-specific vaccines, antisense and associated gene products, angiogenesis inhibitors and other novel small-molecule immunomoderators are expected to complement standard therapies in the near future.

It is also expected, however, that new products might find it difficult to penetrate a monopolized market: TAP Pharmaceuticals (Lake Forest, IL, USA), AstraZeneca (London, UK) and Schering-Plough (Kenilworth, NJ, USA) collectively control 99% of market revenues from the hormonal treatment of prostate cancer.

Cambridge needs cash to stay in race

More cash is required by biotechnology companies clustered around Cambridge, UK, if they are to keep up with their international competitors, claimed Jef Solomon, the Chief Executive of the Eastern Region Biotech Initiative (ERBI, Cambridge, UK). 'We have all the skills and infrastructure here needed to make Cambridge a success,' he said. 'However, we could really do with some extra financial support to… rival what is happening in America.'

His comments came in response to a recent survey of 100 international experts interviewed by Boston Consulting Group (BCG; Boston, MA, USA) commissioned by the Bavarian Ministry of Economic Affairs, Transport and Technology. The survey compared four biotech clusters in Germany (Rhine-Neckar, Rheinland, Berlin and Munich) with three in the USA (Boston, the Bay area and North Carolina) and Cambridge in the UK.

The USA clusters were rated as having the best factors for success, followed by Cambridge and then the German groups. However, despite Munich having only five public biotech companies compared with the 17 in Cambridge, the Munich region was thought to have much better prospects for the development of new companies. This is because the Munich region profited from the Biotechnology 2000 program from the Federal Government and from a support program of the Bavarian Government's High-Tech offensive, which will now also provide a further DM152 million in 2000–2003 to expand the biotech cluster.

New MIND research and treatment centre at UC Davis

A new centre for the treatment of neurodevelopmental disorders is to open at the University of California, Davis (UCD; CA, USA). The Medical Investigation of Neurodevelopmental Disorders (MIND) Institute will conduct research into disorders such as autism, Asperger's syndrome, fragile X syndrome, Rett's syndrome, Tourette's syndrome, dyslexia and attention deficit disorder, and their treatment.

The Institute will consist of three buildings, which will include a 27,270-square-foot laboratory facility, a 31,979-square-foot research facility, an outpatient clinic, a resource centre and library, a research classroom and medical imaging facilities. The first phase of the building, costing US$38.8 million, which will be funded by a combination of state revenue bonds state appropriations and the UCD Health System, is expected to be completed by March 2003.

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