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# News in brief

## Targets and mechanisms

### Glowing worms as possible models of Parkinson's disease

*Caenorhabditis elegans* could become a powerful model for studying the effects of Parkinson's disease (PD), according to new research by scientists at the Vanderbilt University School of Medicine (Nashville, TN, USA) and the Albert Einstein College of Medicine (New York, NY, USA) [1].

PD is a neurodegenerative disorder that is characterized by uncontrollable tremor and an inability to initiate movement. It results from the progressive, irreversible loss of dopamine neurons in the brain. Although, in most cases, it is unclear what triggers this loss, possibilities include exposure to environmental toxins, inhibition of mitochondrial electron transport, and an increase in the generation of reactive oxygen species. However, such hypotheses are difficult to test in mammalian models of PD.

The researchers focused their work on *C. elegans* because this worm has dopamine neurons (eight out of the ~300 neurons in its nervous system) that are genetically and structurally essentially the same as those in mammals. The worm is also much easier to work with, compared with mammalian models. Using green fluorescent protein, the researchers developed worms in which the dopamine neurons glowed green when viewed using confocal epifluorescence.

The glowing worms were exposed to 6-hydroxydopamine (6-ODHA), a neurotoxin used in mammalian studies of PD. 6-ODHA is particularly interesting because it is found in brain and urine samples from patients with PD. Exposure to 6-ODHA caused selective degeneration of the dopamine neurons, and the green glow faded 1 h post-exposure, and neuron death was complete after 24–48 h. When dopamine transporter function was blocked, by using drugs or by genetic disruption, 6-ODHA had no effect and the green glow remained.

The researchers suggest that *C. elegans* could, therefore, become a powerful tool in PD research, and could be used to screen chemical libraries to identify possible targets for drug development for treatment of PD. The model could also be used to identify genes that are required for toxin-induced degeneration of these neurons.

1 Nass, R. *et al.* (2002) Neurotoxin-induced degeneration of dopamine neurons in *Caenorhabditis elegans*. *Proc. Natl. Acad. Sci. U. S. A.* 99, 3264–3269

### Miniature pigs could aid organ transplantation without risk of PERV

Scientists at Immerge Biotherapeutics (Charlestown, MA, USA) have identified miniature pigs that do not transfer the porcine endogenous retrovirus (PERV) to humans. This could be a breakthrough in the success and safety of transplantation of pig tissues into humans. More than 79,000 patients in the USA are on a waiting list for organ transplantation (and there is potentially a further 79,000 patients that were unable to make this waiting list because of the severe shortage of human organ donors); xenotransplantation would offer a greater hope for these patients.

The use of porcine tissues and organs for xenotransplantation to treat human disease has been hindered by the transmission of PERV which, unlike other viruses, cannot be eliminated from the transplanted tissue by breeding or laboratory cleanliness. This is because multiple copies of PERV exist within the pig genome and, therefore, are passed from one generation to the next.

In this latest study [2], scientists have characterized three subgroups (A, B and C) of PERV that are found in the herd of inbred miniature swine and have shown that families of animals can be identified that possess a consistent non-transmitting phenotype. Furthermore, results suggest that it is the replication-competent subtype, PERV-A, that might be missing from the miniature swine genome, because in human infected cells, all of the viruses were recombinants of the receptor-binding domain of PERV-A- and PERV-C-related sequences.

2 Oldmixon, B. *et al.* (2002) Porcine endogenous retrovirus transmission characteristics of an inbred herd of miniature swine. *J. Virol.* 76, 3045–3048

### Antioxidant enzyme prolongs life

The antioxidant peptide methionine sulphoxide reductase A (MSRA) has been shown to slow the ageing process in *Drosophila* [3]. Cumulative oxidative damage by reactive oxygen species is considered a factor in ageing and age-related diseases. The oxidation of proteins might be particularly important with claims that up to 50% of proteins are oxidized in an 80-year-old human.

MSRA catalyses the repair of oxidised methionine (Met) residues by converting Met sulphoxide to Met. MSRA expression and activity in rats decrease with age, and it is also less active in the brains of Alzheimer's patients.

A collaborative group of researchers from the USA and Germany confirmed the hypothesis that overexpression of MSRA limits age-related events and increases lifespan. They designed transgenic flies overexpressing *msrA* predominantly in their nervous systems.

The team found that these transgenic flies live significantly longer: median lifespan of 95 days versus 58 days in controls (females); 80 days versus 45 days (males). They are also more resistant to oxidative stress induced by the herbicide paraquat – at 30 days, only 10% of transgenic males had died compared with 60–70% of controls. Thus, lifespan extension appears to be achieved by conferring greater protection against oxidative stress.

The decline in the levels of reproductive capacity and general activity with age was also delayed significantly compared with controls. Reproductive capacity was preserved at all stages and sexual activity was more frequent and vigorous.

The authors argue that in these transgenic flies, overexpression of MSRA increases lifespan while maintaining quality of life – they retain a normal food intake and bodyweight, they are more physically active, and they preserve high physical and reproductive activity. The flies' rate of development, behaviour and appearance are also normal. These effects could extend to mammals, including humans. The next challenge is to test this hypothesis and to elucidate the underlying mechanisms involved.

3 Ruan, H. *et al.* (2002) High-quality extension by the enzyme peptide methionine sulfoxide reductase. *Proc. Natl. Acad. Sci. U. S. A.* 99, 2748–2753



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## DNA detection breakthrough

Scientists at Northwestern University (Evanston, IL, USA) have described a new, conductivity-based DNA detection method using gold-bound oligonucleotide nanoparticle probes [4]. A current challenge in the DNA-detection field is to develop methods that do not rely on PCR or similar target-amplification systems as these techniques require additional equipment not ideal for point-of-care or field use. The scientists claim that this new method is straightforward, and is tenfold more sensitive and 100,000-fold more specific than current genome-based detection systems.

A short 'capture' oligonucleotide is placed between two fixed microelectrodes while a longer 'target' oligonucleotide remains in a silver enhancer solution. One end of the target oligonucleotide is complementary to the capture oligonucleotide, the other end to the gold-bound oligonucleotide probe, allowing selective binding. When the device with the electrode pair is immersed in the silver solution containing a particular probe and target oligonucleotide, target-bound gold probes fill the electrode gap. Silver deposition facilitated by these nanoparticles bridge the electrode gap and create measurable conductivity changes, indicative of the number of probe–target molecules in the gap.

The system is highly specific: complementary oligonucleotides bound to probes in the gap exhibit lower resistance and hence a higher signal (>105) than those comprising mismatched strands. The system is also highly sensitive: denaturation of the capture strand–target oligonucleotide–probe complex is salt-concentration-dependent, and this can be exploited to distinguish between complexes containing complementary versus mismatched targets. Thus, there is no need for the thermal denaturation step used in current detection methods. In theory, therefore, probes sensitive to heat denaturation can be used in this system. Furthermore, the researchers have been able to detect target DNA at concentrations down to 500 fm.

The researchers suggest that this nanoparticle probe detection system will lower the cost, improve the quality, and decrease the time-to-market for hand-held molecular testing devices, and its potential will be further improved by the use of much larger arrays of electrode pairs. 'This research represents a revolutionary advance and paradigm shift in the evolution of genome-based point-of-care diagnostics,' said Chad Mirkin, Professor of Chemistry, Northwestern University and Director of the Northwestern University Institute for Nanotechnology, as well as a co-founder of Nanosphere, the company that will be commercializing this technology.

**4** Park, S-J. *et al.* (2002) Array-based electrical detection of DNA with nanoparticle probes. *Science* 295, 1503–1506

## Protein folding: short and sweet or long and winding

New research offers the strongest evidence yet that proteins fold into three-dimensional shapes via individualistic routes, challenging the notion that protein folding takes a single, prescribed course [5]. Feng Gai, Assistant Professor of Chemistry at the Department of Chemistry and his colleagues at the University of Pennsylvania (Philadelphia, PA, USA), found that the time it takes for a protein to fold varies enormously, offering compelling, if indirect, support for a more heterogeneous model of protein folding.

'The traditional view has been that a protein passes through a series of fixed reactions to reach its folded state,' said Gai. 'Our work suggests quite strongly that folding is a far richer phenomenon…some proteins rocket down an energy gradient to their destination while others take their time, meandering indiscriminately.'

Protein folding has broad implications for human diseases, such as Alzheimer's disease and Parkinson's disease, believed to be the result of misfolded proteins. Abnormal proteins are believed to pool in the brain plaques characteristic of these neurodegenerative disorders.

Gai's research could explain how molecular chaperones – a group of promising compounds that might provide a remedy for these diseases – work. The compounds appear to inhibit the progression of neurodegenerative diseases by rescuing misfolded proteins, and the new research indicates that they might return misfolded proteins to an unfolded state so that they can begin folding again.

This could lead to drugs that mimic the ability of chaperones to avert neuro-degenerative disease, or to the design of artificial proteins that are engineered to precisely fold into biologically active, three-dimensional structures.

**5** Huang, C-Y. *et al.* (2002) Helix formation via conformation diffusion search. *Proc. Natl. Acad. Sci. U. S. A.* 99, 2788–2793

## Proteasome inhibitors could treat psoriasis

Scientists have discovered a proteasome inhibitor that reduces inflammation and has been therapeutically effective in a mouse model of psoriasis [6]. PS519 is a potent and selective proteasome inhibitor based on the naturally occurring compound, lactacystin, and was discovered by researchers at the Goethe University of Frankfurt (Germany) and Millennium Pharmaceuticals (Cambridge, MA, USA).

The proteasome is responsible for regulating the turnover of IκB, a transcription factor that downregulates nuclear factor-κB (NF-κB) by binding to it. Thus, it is thought that PS519 mediates its anti-inflammatory effects by blocking the NF-κB-mediated activation of T-cell genes, a well-known mechanism for dexamethasone, a standard treatment for psoriasis.

Proteasome inhibitors have already been effective in animal models of rheumatoid arthritis and multiple sclerosis, both of which are also thought to result from T-cell exposure to bacterial antigens. In this latest study, researchers showed that PS519 reduced superantigen-mediated T-cell activation and blocked the expression of T-cell activation molecules such as CD69, CD25 and HLA-DR both *in vitro* in cultured T-cells and *in vivo* in the SCID-hu xenogeneic psoriasis transplantation model. Furthermore, the expression of cell-surface ligands such as E-selectin, which are responsible for T-cell homing, was reduced. The authors concluded that blocking the proteasome is a promising means to treat psoriasis and other T-cell mediated disorders.

**6** Zollner, T.M. *et al.* (2002) Proteasome inhibition reduces superantigen-mediated T-cell activation and the severity of psoriasis in a SCID-hu model. *J. Clin. Invest.* 109, 671–679

## Scientists link common virus to brain tumours

A common virus has been linked to the development of the most common malignant brain tumours in children. Scientists at Temple University

(Philadelphia, PA, USA) studied the presence of human polyomavirus JCV gene sequences and the late-gene product, agnoprotein, in paediatric medulloblastomas [7]. The *Agno* gene (which encodes agnoprotein) was detected in 69% of samples, and further analysis by immunohistochemistry revealed the localization of agnoprotein in the cytoplasm of neoplastic cells in 55% of the samples tested.

An oncogenic viral gene product, viral T-antigen, was also detected in the nucleus of some, but not all, neoplastic cells. This is significant because the T-antigen is thought to cause tumours by blocking tumour suppressor genes such as p53 and interfering with cell growth. Interestingly, it has been recently suggested that agnoprotein might interact with the T-antigen and affect its ability to control cell growth. However, in this study, some cancer cells expressed only agnoprotein and no T-antigen, and only six of the 11 tumours expressing agnoprotein also expressed p53, which suggests that agnoprotein could have an independent role in the development of JCV-associated medulloblastomas.

JCV infects 70% of the human population worldwide during early childhood and remains in a latent stage in most healthy individuals. However, in immunocompromised people, such as transplant patients or those suffering from AIDS, JCV can become active and lead to the fatal demyelinating disease, Progressive Multifocal Leukoencephalopathy (PML). Kamel Khalili, senior author of the study, says: 'We are trying to show that the JC virus has the ability to do more than just cause PML…we have a virus in our body that has the potential to cause tumours.' The group now need to determine whether: (1) the virus actually causes cancer, (2) the tumours formed because of other elements and the virus helped as a cofactor, or (3) the presence of the virus is merely coincidental. Khalili says that if they can establish an association between JCV and tumour development, then there is the potential to start developing anti-cancer vaccines.

7 Del Valle, L. *et al.* (2002) Expression of human neurotropic polyomavirus JCV late gene product agnoprotein in human medulloblastoma. *J. Natl. Cancer Inst.* 94, 267–273

## Clinical trials

### NIAID Phase III HIV vaccine trial cancelled

The National Institute of Allergy and Infectious Diseases (NIAID; Bethesda, MD, USA) will not be proceeding with Phase III clinical trials (HVTN 501) of their 'prime-boost' HIV vaccine combination after unsatisfactory Phase II results. The decision was made in conjunction with the vaccine manufacturers, Aventis Pasteur and VaxGen.

Preliminary analysis of the Phase II (HVTN 203) trial that assessed the safety and immunogenicity of a combination of two experimental HIV vaccines, canarypox-virus-based primer vaccine (ALVAC-HIV) followed by a gp120 subunit booster vaccine (AIDSVAX B/B2), showed that the percentage of volunteers with a detectable CD8 cellular immune response was likely to be too low to provide a valid immune correlates analysis. The decision not to proceed with HVTN 501 might not mean a lack of efficacy of the vaccines because it is not known which immune assay might eventually be shown to correlate with protection in humans.

Evaluation of a similar vaccine combination, ALVAC-HIV (vCP1521) and AIDSVAX B/E, by US Army Medical Research and Materiel Command of the Department of Defence (Pentagon; Washington, DC, USA) has been given support by the NIAID. The project, to be conducted in Thailand, will incorporate envelope antigens from the predominant circulating strain of HIV in the country.

### Pharmacia ends colorectal cancer clinical trial

Pharmacia Corporation (Peapack, NJ, USA) has ended its Phase III clinical trial of the angiogenesis signalling inhibitor SU5416 after it was shown to have inadequate clinical benefit when used in patients with advanced stage colorectal cancer.

Angiogenesis signalling inhibitors have been shown to play a role in blocking vascular endothelial growth factor-receptors (VEGF-R), thereby inhibiting the blood supply to the tumour. Pharmacia, who conducted the trial through their subsidiary company Sugen, will now work to bring all remaining trials of the drug to an appropriate conclusion. 'It is critical to recognize that the results…do not invalidate the VEGF-R target or the entire angiogenesis field. We will take what we have learned…in order to develop the next generation of compounds and studies,' said Lee Rosen, of the Jonsson Cancer Center, University of California (Los Angeles, CA, USA) and lead investigator in the trial.

### New initiatives improve access to clinical trials in USA

A new national resource launched by CenterWatch (Boston, MA, USA) and an initiative in the State of Michigan (USA) could make it easier in future for patients to access active clinical trials. The CenterWatch directory, produced in association with the Physicians Desk Reference (Medical Economics Company, Montvale, NJ, USA), will provide information on medical treatments in development that have not yet been approved by the Food and Drug Administration (FDA; Rockville, MD, USA). The publication, which includes background information on the clinical trials process, a glossary of terms and listings of more than 7500 clinical trials, has been created because of 'overwhelming interest from patients and professionals,' said Dan McDonald, Director of CenterWatch.

Meanwhile in Michigan, a newly formed coalition of patient advocate groups, employers and insurance companies has formed to cover the costs of cancer clinical trials. The Michigan Cancer Consortium (Lansing, MI, USA) makes the State one of only five in the USA to cover routine patient costs associated with clinical trials and is unique because some of the 25 or more groups it includes normally oppose each other legislatively.

'We hope this change will improve recruitment to cancer clinical trials and ultimately help determine more quickly whether a new cancer treatment works or is no better than standard care,' said Thomas Simmer, Vice-President of Blue Cross Blue Shield of Michigan (Lansing), which underwrites coverage for approximately two million members. 'A critical part of this agreement spells out clearly what sorts of clinical trials will be considered for reimbursement,' said Eugene B. Farnum, Executive Director of the Michigan Association of Health Plans (Lansing, MI, USA).

## Infectious diseases

### New cancer drug inhibits HIV

A new cancer treatment currently undergoing clinical trials could inhibit HIV-1 replication, according to scientists at Pharmacyclics (Sunnyvale, CA, USA), and their collaborators at Stanford University Medical Center (Stanford, CA, USA) [8]. The researchers report that Xcytrin® (also known as motexafin gadolinum, gadolinium texaphyrin, or Gd-Tex) selectively acts on HIV-1-infected $CD4^+$ T lymphocytes and induces them to commit cell suicide.

Xcytrin is the first of a new class of drugs called texaphyrins and, according to the researchers, appears to inhibit HIV replication by a mechanism of action similar to the one it uses against cancer cells. Xcytrin works by reacting with, and diminishing, intracellular reducing metabolites. Cancer and HIV-infected cells are under oxidative stress and these metabolites keep these cells alive. Hence, the drug appears to deplete these reducing metabolites in HIV-infected cells as well as in cancer cells, resulting in the death of HIV-infected cells and, therefore, inhibition of virus production.

The scientists concluded that Xcytrin might have therapeutic potential as an anti-HIV agent that selectively targets and removes HIV-infected cells, while leaving uninfected cells untouched. 'Xcytrin's unique mechanism of action attracted our interest and we reasoned that it might specifically attack the HIV-infected cells in a similar way as it does cancer cells,' said Leonard Herzenberg, Professor Emeritus of Genetics at Stanford University and senior author of the study [8]. 'We are excited by our results...[and] we are eager to test the activity of this drug in AIDS patients.'

**8** Perez, O.D. *et al.* (2002) Motexafin gadolinum (Gd-Tex) selectively induces apoptosis in HIV-1 infected $CD4^+$ T helper cells. *Proc. Natl. Acad. Sci. U. S. A.* 99, 2270–2274

### Ebola and Marburg port of entry

A recent study has shown that two of the most pathogenic human viruses are dependent on cell membrane lipid rafts for entry into the cell, viral assembly and budding [9]. The research, at the US Army Medical Research Institute of Infectious Diseases (Frederick, MD, USA), demonstrated that the Ebola and Marburg viruses, which have mortality rates from infection as high as 80%, could enter and exit cells via lipid rafts that exist on the outer cell membrane. In addition, the group, led by M. Javad Aman, showed that virus-like particles (VLPs) that do not contain viral genome (and are therefore of potential therapeutic use) can be generated.

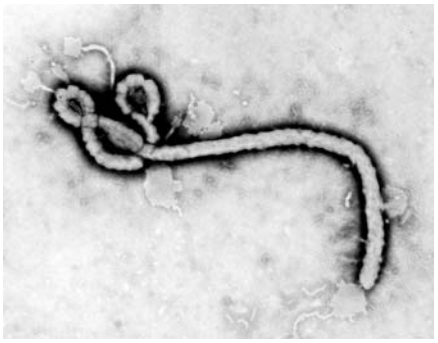

Image of the Ebola virus courtesy of James D. Gathany, CDC.

The Ebola and Marburg viruses belong to the family of filoviruses, which, when released from infected cells, incorporate raft-associated molecules suggesting that viral exit occurs via these rafts. The ectopic expression of the Ebola matrix protein and the raft-dependent release of filamentous VLPs was similar to live virus, as determined by electron microscopy.

The research also revealed that the entry of these viruses required functional rafts, identifying them as the gateway for the entry and exit of virus and the raft-dependent generation of VLPs. These findings have important implications for the development of therapeutics and vaccination strategies against Ebola and Marburg infection.

**9** Bavari, S. *et al.* (2002) Lipid raft microdomains: a gateway for compartmentalized trafficking of Ebola and Marburg viruses. *J. Exp. Med.* 195, 593–602

### Anti-malarial agent

Scientists have demonstrated that a glycolipid can serve as an adjuvant against malaria when used in conjunction with *Plasmodium* vaccines [10]. The team, at the New York University School of Medicine (New York, NY, USA), showed that the natural killer (NK) T-cell ligand, α-galactosylceramide (α-GalCer), can induce an immune activation to promote cytotoxic T cells (CTLs), which has been shown to be necessary for the effective prevention of malaria.

Moriya Tsuji and colleagues showed that α-GalCer interacts with a receptor on a specialized lymphocyte (NK T-cell), which bridges two arms of the immune system: innate and adaptive immunity. α-GalCer was co-injected along with various malaria vaccines, such as irradiated sporozoites and recombinant adenoviruses expressing a malaria antigen. This enhanced long-lasting CTL-mediated immunity against a mouse model of malaria infection.

$CD8^+$ T lymphocytes play a major role in the control of parasitic and viral infections, as well as in tumour development, which raised the need for developing adjuvants that are capable of increasing cell-mediated immunity. Activation of NK T-cells by α-GalCer caused activation of NK, B, $CD4^+$ and $CD8^+$ T cells. α-GalCer stimulates both human and murine NK T-cells, so this finding should contribute to the design of effective vaccines against malaria, and also a variety of other intracellular pathogens, including HIV.

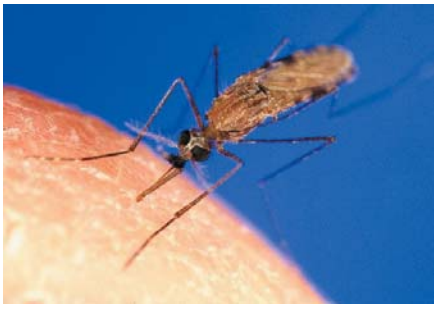

Image of the *Anopheles gambiae* mosquito, one of the predominant malaria vectors in the world courtesy of James D. Gathany, CDC.

**10** Gonzalez-Aseguinolaza, G. *et al.* (2002) Natural killer T-cell ligand α-galactosylceramide enhances protective immunity induced by malaria vaccines. *J. Exp. Med.* 195, 617–624

## Miscellaneous

### Roche to protest awards made to Igen

Roche Diagnostics (Basel, Switzerland) are to appeal damages and licensing rulings awarded by the US District Court for the

District of Maryland to Igen International (Gaithersburg, MD, USA) over their disputed electrochemiluminescence (ECL) agreement. Roche has been banned from marketing, selling, placing or distributing outside of its licensed field any products based on Igen's Origen technology and ordered to pay the company US$505 million.

A licence for the disputed technology, including the Elecsys diagnostics product line, and the right to terminate their licensing agreement with Roche, will be granted to Igen once all appeal proceedings have been completed.

'This judgement confirms the recent jury verdict and properly awards to Igen the relief that we have been seeking over many years,' said Samuel J. Wohlstadter, Chairman and CEO of Igen. 'The company is now able to complete business arrangements with one or more prospective new partners to fill the markets currently being served by Roche.'

Roche feel that it was their highly successful development and marketing campaign that allowed Igen to realize the full potential of its technology. 'Roche has invested a great deal of money, time and resources alongside Igen to commercialize this technology,' said Manfred Baier, Head of Lab Network, a business area of Roche Diagnostics. 'Igen's management and shareholders have benefited financially despite difficulties in the administration on the contract by Roche,' he said.

### Model proposed to determine new product development

An answer to the so-called pipeline problem, of how many approaches to take to each stage of R&D, could have been found in a new model published in the report *Structuring the new product development pipeline* [11]. The study found that many pharmaceutical companies actually underspent on R&D. The model, which takes into account the cost of a development approach, its probability of survival, and the expected profitability, was applied to several real-world scenarios in the pharmaceutical industry.

'Our results suggest, in general, that the pharmaceutical firms we studied employ narrower pipelines than they should for developing their new drugs,' said Min Ding, co-author and Assistant Professor of Marketing at Penn State's Smeal College of Business (University Park, PA, USA). 'This indicates that the current development costs for new medicines are very well justified,' he said.

11 Ding, M. and Eliashberg, J. (2002) Structuring the new product development pipeline. *Management Sci.* February

### AIIRBs set to dominate anti-hypertensives market until 2008

Angiotensin II receptor blockers (AIIRBs) will continue to drive growth in the global anti-hypertensives market until 2008, claims two reports by Datamonitor, entitled *Market dynamics 2001: anti-hypertensives* and *Strategic perspectives 2001: diabetic hypertension*. Sales of US$5.6 billion in 2001 (16.5% of the anti-hypertensive market) are predicted to reach US$24.3 billion (45.5%) in 2008 because of an increase in the population of individuals intolerant to angiotensin converting enzyme (ACE)-inhibitors and diversification in the application of AIIRBs to treat other complaints such as diabetic hypertension, diabetic retinopathy, heart failure, post-myocardial infarction and isolated systolic hypertension.

Despite such sales potential, many pharmaceutical companies are reluctant to trial the drugs head-to-head against ACE inhibitors. Boehinger Ingelheim's ONTARGET trial bucks the trend comparing Micardis (telmisartan) with the ACE inhibitor ramipril both as a monotherapy and in combination. This is worth the risk, say report authors Datamonitor (London, UK), because the potential rewards are great. Even if AIIRBs do fare relatively poorly, the drugs have a guaranteed market in treating the ACE-intolerant population. Final results of this trial are expected in 2007.

News in Brief was written by *Daphne Chung, Joanne Clough, Lisa Deakin, Joanna Owens, Ben Ramster and Linsey Stapley*

# People

### American Cancer Society appoints new Vice-President for National Research Endeavors

Jerome W. Yates has been appointed as Vice-President for National Research Endeavors by the American Cancer Society (ACS; Atlanta, GA, USA). Yates will be responsible for the organization's surveillance of worldwide scientific investigation in the field of oncology and will represent the society at national and international research meetings. In addition, as a member of the ACS's management team, he will coordinate research initiatives with other strategic programs of the ACS.

Yates has previously been Senior Vice-President for Population Sciences and Senior Vice-President for Clinical Affairs at Roswell Park Cancer Center. Before this, he served as the Associate Director for Centers and Community Oncology at the National Cancer Institute, where he was involved in the generation and subsequent evaluation of the Community Clinical Oncology Program (CCOP).

Harmon J. Eyre, Chief Medical Officer of the ACS said, 'Yates brings a wealth of experience and innovation to his position here at the Society. He will enable us to reach new heights in cancer research, building on his vast experience and adding to the role we have played in the field of cancer research since 1946.'

### Exelixis appoints Executive Vice-President

Robert M. Myers has joined Exelixis (South San Francisco, CA, USA) as Executive Vice-President, where he will be responsible for building the pharmaceutical business of the company and expanding its corporate and commercial development activities. Myers will report to the President and CEO of Exelixis, George A. Scangos, who said: 'Bob's broad experience in business and corporate development, new product

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