News in brief

Targets and mechanisms

How viruses penetrate cells

The recent discovery of how the bacteriophage T4 attacks its host could lead to a novel class of antibiotics. The research at Purdue University (West Lafayette, IN, USA), which was funded by the National Science Foundation, discovered the needle-like device that enables the phage to penetrate its host [1]. Scientists have elucidated the crystal structure of the device, which consists of a baseplate located at the end of the phage tail: this regulates the interaction of the tail fibres and the DNA injection machinery.

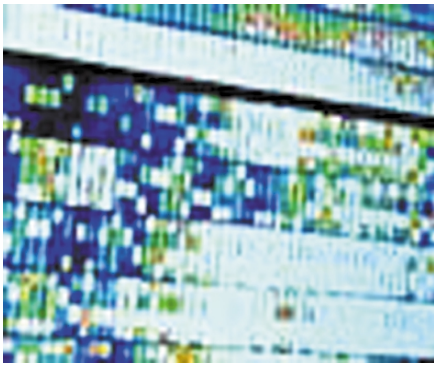
Lead researcher Michael Rossmann says, 'We show, in its entirety, a complex machine that allows a virus to efficiently infect its unfortunate host cell…*Escherichia coli.* The baseplate portion of the virus tail is essential in this process.' This baseplate transmits a message to the T4 phage tail when its fibres attach to a bacterium. The tail contracts and a cutting enzyme makes a nanometer-sized hole through the *E. coli* cell wall. Viral DNA is then injected into the host cell and the bacterium reproduces the phage particle, which results in host cell death.

The actual structure of the needle-like device could hold potential for developments in nanotechnology, such as microscope probes, adds Rossmann. The structure was solved by X-ray crystallography at Purdue and showed the complex of the baseplate gene products gp5 and gp27, determined to 2.9 Å resolution and fitted onto a cryoelectron map at 17 Å. The C-terminal domain of gp5 is a triple-stranded β-helix, which forms a triangular prism and acts as the puncturing device. The N-terminus of gp5 is inserted into a cylinder formed by three gp27 monomers, which could serve as the DNA injection channel.

Kamal Shukla, the National Science Foundation project officer for this research, said: 'Knowing the exact mechanism of T4 bacteriophage infectivity is a significant breakthrough. This information could eventually help in creating designer viruses that could be the next class of antibiotics.'

1 Kanamaru, S. *et al.* (2002) Structure of the cell-puncturing device of the bacteriophage T4. *Nature* 415, 553–557

Microarrays for better vaccines



The way that microbes communicate can be used as cues to produce safer, more effective vaccines, according to scientists at The Whitehead Institute of Biomedical Research (Cambridge, MA, USA) [2]. Richard Young, whose laboratory has employed DNA microarrays to explore the responses of human macrophages to a variety of bacteria, said: 'We are in the midst of a revolution in the way researchers study infectious diseases – instead of depending on culture dishes as the only way to observe the behaviour of pathogens, scientists are able to eavesdrop on the crosstalk between invading microbes and the immune cells of our body.'

Macrophages are immune cells that recognize and engulf microbes as a first line of defence against infection. These cells elicit a response when bacteria are detected so that the rest of the immune system cells are prepared for attack. The researchers discovered that the macrophages only had to detect certain sugars or proteins present within the attacking organism to be able to respond to the specific bacterial infection.

The research focussed on the analysis of responses that are specific to *Mycobacterium tuberculosis*, and revealed the inhibition of interleukin-12 (IL-12) and IL-15 production, which suggests this is how this organism survives host defences. This supports the use of both IL-12 and IL-15 in clinical tuberculosis therapies. Also, the activation of immune responses by the presence of heat-shock proteins (among others) supports their use in preclinical and clinical trials.

'These findings will help researchers design therapeutics that will stimulate the immune system in a targeted manner, perhaps with fewer side effects,' adds Young.

2 Nau, G.J. *et al.* (2002) Human macrophage activation programs induced by bacterial pathogens. *Proc. Natl. Acad. Sci. U. S. A.* 99, 1503–1508

Hunting for Huntington's cure

Researchers have discovered a potential treatment for Huntington's disease that enhances the brain's natural protective response to the disorder [3]. Researchers have found a compound that successfully alleviated the uncontrollable tremors and prolonged the lives of mice with a neurological disease that mimics Huntington's.

Huntington's disease is a hereditary disorder affecting 1 in 10,000 people, and is characterized by memory loss, abnormal movement and premature death. A mutant form of the *huntingtin* gene is responsible for the disease, although the function of the protein encoded by the normal gene remains unknown. When the abnormal gene form is present, the brain becomes clogged with protein aggregates. The aggregates are formed from abnormal huntingtin proteins, hooked together by cross-linking transglutaminase enzymes.

A compound called cystamine is known to inhibit the transglutaminase enzyme. Lawrence Steinman (Stanford University, CA, USA) and Marcela Karpuj (University of California, San Francisco; UCSF, CA, USA) treated mice that had a condition similar to Huntington's disease with cystamine injections. Although the mice showed marked signs of improvement, the researchers were surprised to find that the aggregates remained unchanged. After this unexpected result, the Stanford team began screening the brains of cystamine-treated and untreated mice for differences in gene expression. Mice treated with cystamine had elevated levels of three proteins known to have protective roles in the brain. Similar elevated levels of these proteins were found in the brains of humans with Huntington's disease, implying that the brain makes an unsuccessful attempt to protect itself against the disease.

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Although these findings suggest that cystamine could some day offer hope to patients with Huntington's disease, more effective and specific compounds will be sought. 'Perhaps multiple treatments in combination would have even greater benefits,' said Steinman.

3 Karpuj, M.V. *et al.* (2002) Prolonged survival and decreased abnormal movements in transgenic model of Huntington disease, with administration of the transglutaminase inhibitor cystamine. *Nat. Med.* 8, 143–149

Hearing loss hope

Scientists have demonstrated the restoration of hearing in deafened guinea pigs [4], providing hope for a viable treatment for deafness in humans.

Hearing loss is the most common disability in the industrialized world, affecting one in seven people. It usually results from progressive irreversible degeneration of sensory hair cells in the cochlea of the inner ear, followed by degeneration of the auditory neurons that innervate those hair cells. There is a 'window of opportunity', however: degeneration of the nerves takes time and there is a period when cochlear hair cells have degenerated but the innervating neurons are still viable. During this time, hearing can be partially restored by electrical stimulation via a cochlear implant, currently the most successful treatment in the profoundly deaf. However, the effectiveness of these implants is dependent on the number of excitable auditory neurons.

As a way to prevent the secondary loss of neurons, researchers from the Karolinska Institute (Stockholm, Sweden), the University of Michigan (Ann Arbor, MI, USA) and the Ehime University School of Medicine (Ehime, Japan) have used infusions of neurotrophic factors to preserve the auditory nerve after peripheral damage and restore auditory function in the cochlea of deafened guinea pigs.

Cochlear hair cells were destroyed in guinea pigs by infusing neomycin directly into the cochlea. The cochlea was then infused with a solution of a ciliary neurotrophic factor (CNTF) analogue and brain-derived neurotrophic factor (BDNF) for four weeks. Electrical tests of the auditory nerve and histological examination after this time showed significant restoration of hearing function.

A number of questions remain unanswered regarding the exact molecular basis for the effect – particularly as to whether the restoration is simply a reflection of a delay in neuronal loss. However, these studies provide a rationale for human clinical trials involving focal infusion of the neurotrophic factors for the treatment of deafness.

4 Shinohara, T. *et al.* (2002) Neurotrophic factor intervention restores auditory function in deafened animals. *Proc. Natl. Acad. Sci. U. S. A.* 99, 1657–1660

Cancer targets and mechanisms

Making BCR-Abl unable

Scientists at the European Molecular Biology Laboratory (EMBL; Heidelberg, Germany) have produced a technical diagram of a molecule that causes cancer, giving them a good starting point from which to begin designing cancer-preventing drugs [5]. The molecule, c-Abl, is produced in all human cells. However, some people acquire a genetic defect in c-Abl resulting in the production of the malformed BCR-Abl, which is linked to forms of leukaemia.

The research focussed on the thus far elusive molecular mechanism that is responsible for the regulation of the c-Abl tyrosine kinase and showed that the N-terminal 80 residues of the protein are essential to achieve and maintain inhibition. The loss of this portion of the protein makes c-Abl oncogenic and contributes to the deregulation of BCR-Abl. Abl autoregulates the normal division processes of cells and, when it malfunctions, cells divide rapidly, which can lead to cancer.

Guilio Superti-Furga, a researcher at EMBL, said: 'Abl needs to be switched off and one of the chief questions…is whether other molecules are needed to throw the switch, or whether Abl can turn itself off.' He added, 'We've now discovered that there is an internal switch that allows it to shut itself down. BCR-Abl is missing an important structural piece of the protein…and the molecule can't stop sending signals.'

Superti-Furga and colleagues discovered that the 'internal switch' lies in the N-terminal region of the protein, distant from the machinery that transmits the signals. By showing that this cap is essential in Abl's switch, the researchers have provided a starting place for the design of novel anticancer drugs.

5 Pluk, H. *et al.* (2002) Autoinhibition of c-Abl. *Cell* 108, 247–259

Hrs involved in endocytosis

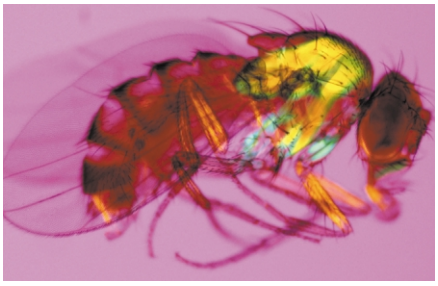
Researchers at Baylor College of Medicine (Houston, TX, USA) have identified a potential new target for anticancer drugs [6]. The hepatocyte growth-factor regulated tyrosine kinase substrate, Hrs, which oversees the transport of receptors from the cell surface to its interior, has been discovered to also tag receptors for degradation. The research elucidated the role of Hrs in endocytosis – the regulation of the cell-surface expression of many receptors – during development. During endocytosis, some of the cell membrane containing the receptor is internalized by the cell and forms small vesicles, which subsequently fuse to form the endosome. Electron microscopy studies of Hrs mutant fruitfly larvae revealed that mutants had enlarged endosomes, a result of the inability to form multi-vesicular bodies.

Hugo Bellen, co-author of the study and an investigator at the Howard Hughes Medical Institute, said: 'What's exciting in this finding is that we now understand one reason why multi-vesicular bodies form…these bodies are needed to turn off signals from key receptors involved in cellular communication.'

The study showed that the epidermal growth factor receptor (EGFR) and Torso tyrosine kinase remained switched on in the Hrs deficient flies because they depend on Hrs for activation. Receptors such as EGFR control cell proliferation; therefore, overactivity as a result of a mutation in Hrs leading to the EGFR not being guided to the lysosyme for degradation could be an underlying factor in many cancers. 'We know that Hrs is involved in regulating key signalling proteins that have been implicated in numerous cancers because they control cell proliferation and cell differentiation,' says Bellen. 'One could potentially use drugs to affect this protein's activity – either to eliminate it or to overexpress it – to modify a signalling pathway.'

6 Lloyd, T.E. *et al.* (2002) Hrs regulates endosome membrane invagination and tyrosine kinase receptor signaling in *Drosophila. Cell* 108, 261–269

Fruitful search for cancer metastasis



Fruit flies have provided a clue as to how stationary cells mobilize [7]. The research, performed at the Johns Hopkins School of Medicine (Baltimore, MD, USA), shows that a key signal enabling normally stationary cells in the ovary to travel could help to clarify how human cancer cells invade distant tissues.

Denise Montell, Associate Professor of Biochemistry in the school's Institute for Basic Biomedical Sciences, says: 'Cells usually hold on to their neighbours, so a lot of things have to change for a cell to become migratory. We've now found the first signal that seems to be sufficient to get cells moving.'

With funding from the American Cancer Society and the National Institutes of Health, graduate student Debra Silver identified a protein called 'Unpaired' (UPD), which activates stationary cells. At a certain stage in development, a small cluster of epithelial cells detaches and comes to rest at the edge of the oocyte. This cluster consists of polar cells, which recruit border cells as their vehicle. Montell's laboratory used mosaic flies, which contain knockout patches of cells. This led to the discovery that polar cells make and release UNP, the only known trigger in the fruitfly of the JAK–STAT pathway, which was already known to aid cell division regulation and survival in fruitflies and humans. UNP leaves the polar cells and attaches to its receptor on nearby epithelial cells, turning on JAK (Janus kinase), which builds a docking point for STAT (signal transducer and activator of transcription). STAT then enters the nucleus to activate certain genes.

'Releasing UNP and turning on the JAK–STAT pathway is all that's needed for the cells to start moving,' says Montell. The

Miscellaneous

Bureaucracy and unfairness rife in Europe

Top decision makers have decided that it is unnecessary bureaucracy, such as protracted decision-making and the provision of translations, that is causing the licensing of new medicines in the European Union to be much slower than they are in the US.

Regulatory processes, the issuing of market authorizations, price negotiation and reimbursement systems, were all named as reasons for delay by G10 Medicines (http://pharmacos.eudra.org), a task force set up to look at the problem. 'One of the recommendations includes introducing greater commercial flexibility into both the centralized and mutual recognition systems,' said Tomas Salmonson of Medical Product Agency (MPA; Uppsala, Sweden). 'Obtaining multiple registrations and trade marks, and managing co-promotion, [will have] benefits for public health,' he said.

G10 will submit a report in April 2002, which will outline practical recommendations to improve the competitiveness of the EU pharmaceutical industry. Also included in the report will be an analysis of the level of equality of access to medicines between European member states. At present, the time difference between the introduction of a medicine to the EU and when it reaches the last member state can be up to four years. Much of this delay is caused by different procedures for price-setting in different countries.

'There is no economic justification for direct product price control; the issue is political,' said Bengt-Jonnson, Professor at the Centre for Health Economics (Stockholm, Sweden). 'Price control…has a number of well-documented negative effects and few, if any, positive outcomes.'

UK researchers gain access to Celera products

UK-based academic researchers will gain access to Celera Genomics (Rockville, MD, USA) database products through a new multi-year agreement signed with the Medical Research Council (MRC, London, UK). All UK researchers, whether MRC funded or not, will be able to access Celera's database information and research tools, in addition to the publicly-funded genomic data that is currently available.

Study results end Santen/Iomed collaboration

Santen Pharmaceuticals (Osaka, Japan) and Iomed (Salt Lake City, UT, USA) are to cease joint development of a novel treatment for age-related macular degeneration (AMD) after Santen deemed feasibility study results unsatisfactory. The treatment, which involves the non-invasive delivery of an anti-angiogenic drug to the retina and choroid, demonstrated proof-of-concept in an animal model but Santen, the makers of the drug, decided that its therapeutic index was too low to warrant further collaboration.

'We were well aware of the systemic toxicity of the drug, but its pharmacodynamics combined with a local ocular delivery system make it an ideal candidate for the treatment of multiple angiogenic-based diseases of the eye,' said Robert J. Lollini, Iomed's Chief Operating Officer. 'The concept remains a good one [but]…without an alternative drug candidate to take into development it is in the best interests of both companies to pursue separate interests,' he said.

US$2.5 million funds new Thomas Jefferson heart unit

The Thomas Jefferson University (Philadelphia, PA, USA) is to receive US$2.5 million over the next 10 years from the Jones Apparel Group (New York, NY, USA) to fund a new cardiovascular disease research and teaching unit. The donation to the Sidney Kimmel Laboratory for Preventive Cardiology, named after the Group's founder and chairman, will enable the University to pursue its research into the disease, as well as maintain its patient-risk-assessment programs.

'We are developing new and improved tests to identify and measure particles in the blood that will help predict those at risk of heart disease and stroke,' said David Capuzzi, Director of the University's Cardiovascular Disease Prevention Center. 'These additional resources will greatly accelerate the progress of our research.'

next step is to examine ovarian cancer tissue to see if this pathway regulates human cancer cell mobility.

7 Silver, D.L. and Montell, D.J. (2001) Paracrine signaling through the JAK–STAT pathway activates invasive behavior of ovarian epithelial cells in *Drosophila*. *Cell* 107, 831–841

Immune disorders

How cells fight infection

Researchers have discovered a way to control a protein involved in helping the body to fight infection [8]. The discovery could lead to new treatments in immune-system-related diseases.

Amy Andreotti, Assistant Professor of Biochemistry at Iowa State University (Ames, IA, USA), together with Kristine Brazin and co-workers, focused on the regulation of the non-receptor protein tyrosine kinase of the Tec family, interleukin-2 tyrosine kinase (Ikt), which is present in T cells. They discovered an on/off switch (known as proline *cis*/*trans*-isomerization) that controls the activity of Ikt. Using NMR, the researchers showed that cyclophilin A (a type of peptidyl-prolyl *cis*/*trans*-isomerase; PPIase), which is found abundantly in T cells, can regulate this on/off switch in Ikt. Until this work, regulation of Ikt and other related proteins was poorly understood and a function for cyclophilin A had not been identified.

Although this mode of regulation is considered novel, Andreotti believes that it is possible that cyclophilin A could act as a general switch operator. Indeed, the switch mechanism might be present in other proteins. Moreover, the researchers suggest that this work will advance understanding of T-cell signalling and could help to elucidate the mode of action of immunosuppressive agents, such as cyclosporin A and FK506, which has foiled researchers for the past decade. Although these agents target cyclophilin and FK506-binding proteins (another type of PPIase), the fact that they inhibit PPIase activity is apparently irrelevant to their immunosuppressive effects.

The researchers also comment that these findings could help explain why the use of cyclosporin in organ transplant patients is often accompanied by a high incidence of cancer. Hyperactivation of protein tyrosine kinases, such as Itk, during the use of PPIase inhibitors might contribute to unregulated cellular proliferation and ultimately formation of malignancies in immunosuppressed patients.

8 Brazin, K.N. *et al.* (2002) Regulation of the tyrosine kinase Ikt by the peptidyl-propyl isomerase cyclophilin A. *Proc. Natl. Acad. Sci. U. S. A.* 99, 1899–1904

Switching off the inflammatory response

An off-switch for the systemic inflammatory response has been found in mice by researchers at Johns Hopkins University School of Medicine (Baltimore, MD, USA) and the Howard Hughes Medical Institute (Baltimore, MD, USA) [9].

The acute phase response is a systemic inflammatory reaction triggered by a range of factors, including infection and trauma. At a site of inflammation, cytokines such as interleukin 6 (IL-6) are produced, which act on hepatocytes to either suppress or stimulate acute phase protein synthesis. The effect of IL-6 is mediated mainly via the signal transducer and activator of transcription 3 (Stat3).

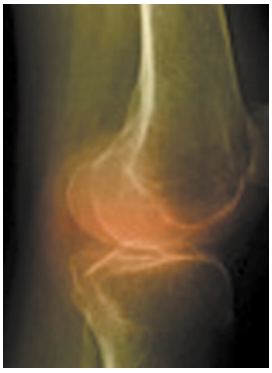
The researchers spliced the gene encoding Stat3 to produce two isoforms, *Stat3α* and *Stat3β*, and then genetically engineered *Stat3β*-deficient mice. When challenged with bacterial endotoxin, the *Stat3β*-deficient mice showed marked mortality compared with wild-type mice, including general signs of septic shock, thymic and hepatic necrosis, and acute kidney failure, resulting from massive inflammation.

The researchers concluded that the mutant mice were unable to turn off the initial inflammatory response to the endotoxin, and that the inflammatory reaction caused irreversible damage to the animals' own tissues. They concluded that, although a range of molecules respond to the initial infection or injury, *Stat3β* is responsible for shutting off that response.

Although the details of the action of *Stat3β* in humans are likely to be different from those in mice, its role in humans is likely to be similar. These results could have significant implications for the treatment of inflammation-related disorders in humans, such as autoimmune disorders and atherosclerosis.

9 Yoo, J-Y. *et al.* (2002) Specific ablation of Stat3β distorts the pattern of Stat3-responsive gene expression and impairs recovery from endotoxic shock. *Cell* 108, 331–344

Improving safety of arthritis drug



A new anti-inflammatory drug with reduced side effects has shown positive preclinical [10] and clinical Phase I results. NCX1015, developed by NicOx (Sophia Antipolis, France), is a nitric-oxide-releasing derivative of prednisolone. NicOx has now expanded development of the compound, which should have greater patient compliance for the treatment of inflammatory bowel disease and arthritis.

The Phase I study, prior to this paper, was designed to evaluate the general safety, local tolerability and pharmacokinetics of NCX1015. In a randomized, double-blind, placebo-controlled, parallel group study, 32 healthy male volunteers were given four escalating doses of the drug by enema. The overall safety and tolerability were satisfactory throughout the study and the pharmacokinetic data showed no systemic absorption.

New preclinical data on NCX1015 shows anti-inflammatory activity comparable to that of traditional drugs of this class but without the usual side effects [10]. The study was headed by Roderick J. Flower (Head of the Division of Pharmacology, St Bartholomew's and Royal London School of Medicine and Dentistry, London, UK) and Mauro Perretti (William Harvey Research Institute, London, UK). They demonstrated that, in a rat model, NCX1015 has potent anti-inflammatory activity. NCX1015 is a glucocorticoid, a class of compounds that are widely used in the treatment of chronic inflammatory pathologies. Glucocorticoid therapy is often accompanied by side effects, especially osteoporosis. For this reason, Flower predicts that the drug could 'become a treatment of choice for arthritis.' Michele Garufi, Chairman and CEO of NicOx commented, 'The success of the Phase I clinical trial and these new preclinical results for NCX1015 are highly encouraging and demonstrate that NCX1015 has the potential to be developed in both inflammatory bowel disease and arthritis.'

10 Paul-Clark, M.J. *et al.* (2002) Potent antiarthritic properties of a glucocorticoid derivative, NCX-1015, in an experimental model of arthritis. *Proc. Natl. Acad. Sci. U. S. A.* 99, 1677–1682

An immunological plus for GH therapy

Growth hormone (GH) therapy in elderly patients might increase the production of cells involved in fighting disease, suggest researchers from the University of Illinois (IL, USA) [11]. GH therapy is already known to reduce body fat and increase lean body mass in elderly patients, and some patients are now prescribed GH to counteract the effects of somatopause.

Somatopause is the aging-associated reduction in levels of plasma insulin-like growth hormone 1 (IGF-1), often to levels associated with GH-deficient children. IGF-1 is released from the liver and local tissues following stimulation by GH, and is thought to be a key regulator of body growth, including muscle synthesis. The researchers either implanted synthetic GH-secreting pituitary epithelial cells or injected GH into aging rats and found that both approaches resulted in the stimulation of production of immunity-promoting haematopoietic cells in bone marrow, adrenal glands, liver and spleen. Production was accompanied by a complete reversal of fat-cell accumulation in the bone marrow, which usually fills the gaps left by the declining number of haematopoietic cells. The levels of production of these cells were threefold higher than those of similarly aged untreated rats, and 80% of those in the fitter, younger rats in the control group.

The results suggest that IGF-1 might be the crucial molecule that acts directly on progenitor cells to promote haematopoiesis. Keith W. Kelley, lead investigator and Professor of Animal Science at the University of Illinois Laboratory of Immunophysiology also commented that: 'These results establish that a classic hormone, GH, is a potent stimulator of the production of blood cells', which is usually reduced dramatically in the elderly.

11 French, R.A. *et al.* (2002) Age-associated loss of bone marrow hematopoietic cells is reversed by GH and accompanies thymic reconstitution. *Endocrinology* 143, 690–699

News in Brief was written by *Matt Brown, Joanne Clough, Lisa Deakin, Ben Ramster* and *Linsey Stapley*

People

Two key appointments at Johnson & Johnson

William C. Weldon has been appointed as the new Chairman and CEO of Johnson & Johnson, while James T. Lenehan will be President of the company, in addition to his responsibilities as Vice-Chairman. They take over these roles from Ralph S. Larsen, who is currently Chairman and CEO, and Robert N. Wilson, who is currently Senior Vice-Chairman of the Board and Vice-Chairman of the Executive Committee. These appointments are effective from 25 April 2002, the date of the next annual shareholders' meeting.

Both Weldon and Lenehan currently serve as Vice-Chairmen of the Board. Weldon leads the Pharmaceuticals Group and the Consumer Pharmaceuticals & Nutritionals Group, while Lenehan leads the Medical Devices & Diagnostics Group and the Consumer & Personal Care Group.

Weldon joined the company in 1971 as a sales representative at McNeil Pharmaceutical. He eventually became Executive Vice-President and Managing Director of McNeil, and then Managing Director of Ortho-Cilag Pharmaceutical. He then took up the post of Vice-President, Sales & Marketing for Janssen Pharmaceutica and later became President of Ethicon Endo-Surgery where he then served as their Group Chairman and Worldwide Franchise Chairman. After this, he moved to being Executive Committee Member and Worldwide Chairman of the Pharmaceuticals Group and joined the company's Board of Directors in February 2001.

Lenehan joined the company in 1976 as Assistant Product Director in marketing at McNeil Consumer Healthcare Co. In 1990, he became President of McNeil and then was named Company Group Chairman and Worldwide Franchise Chairman for Consumer Pharmaceuticals and a member of the Consumer Group Operating Committee. He was later promoted to Executive Committee Member and Worldwide Chairman, Consumer Pharmaceuticals & Medical Devices Group and, in 1999, was named Worldwide Chairman, Medical Devices & Diagnostics Group. He joined the company's Board of Directors in February 2001.

Larsen will not be standing for re-election onto the Company's Board in April and will retire on 1 July 2002. Meanwhile, Wilson will stand for re-election in April 2001 but plans to retire from the company and its Board of Directors in April 2003.

John Pople chairs the Scientific Advisory Board of eXegenics

John A. Pople, 1998 Nobel Laureate in Chemistry, has accepted the position of Chairman of eXegenics' (Dallas, TX, USA) Scientific Advisory Board. Pople is Trustees Professor of Chemistry at Northwestern University and was awarded the Nobel Prize for his pioneering work in the development and application of computational methods in quantum chemistry. This work is the foundation of eXegenics' proprietary drug creation methodology.

Prior to joining Northwestern University, Pople was a Professor of Chemical Physics at Carnegie-Mellon University. He has received several awards including the Pauling Award, the Wolf Prize, the Marlow Medal from the Faraday Society, the Davy Medal (from the Royal Society, UK) and the Award for Computers in Chemistry (from the American Chemical Society). He is a Fellow of the Royal Society and a Foreign Associate of the US National Academy of Science.

Tracy Tsuetaki leaves Kaiser Permanente for Quintiles Transnational

Quintiles Transnational Corporation (Research Triangle Park, NC, USA) has announced the appointment of Tracy K.

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