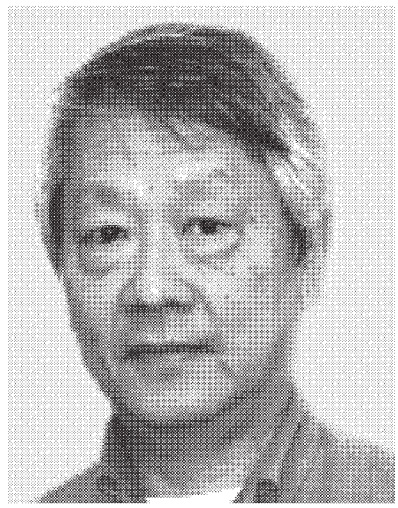
2006 SCBA KEYNOTE SPEAKERS

2006 SCBA Presidential Awardee

Yuh-Nung Jan, Ph.D.
Investigator, Howard Hughes
Medical Institute
Jack and DeLoris Lange Professor
Departments of Physiology and
Biochemistry & Biophysics
University of California, San Francisco



Yuh-Nung Jan was born in Shanghai and grew up in Taipei. He studied physics as an undergraduate at National Taiwan University. In 1968, he entered Caltech as a graduate student in physics. Two years later, he switched to biology. After completing his Ph.D. with Max Delbrück in 1974, he joined Seymour Benzer's lab as a postdoctoral fellow, began working on *Drosophila* neurobiology, and started a long-term collaboration with his wife Lily Jan, a collaboration that continues to date. In Benzer's lab, they began their work on the *Shaker* gene, which eventually led to the cloning of the first potassium channel. From 1977 to 1979, they worked in Steve Kuffler's lab at Harvard Medical School and found that peptides can function as neurotransmitters. They started their own lab at UCSF in 1979 and have happily settled there ever since. At UCSF, besides continuing their work on potassium channels, they started working on neural development to understand how different neuronal cell fates are specified. Past highlights include the discovery of *atonal*, a founding member of a large family of proneural genes and *numb*, the first asymmetrically localized cell fate determinant identified in neural development. More recently, they have been focusing on how diversity in neuronal morphology (especially dendritic morphology) is elaborated during development. Yuh-Nung Jan is currently Lange Professor of Physiology and Biochemistry & Biophysics at UCSF and an investigator of the Howard Hughes Medical Institute. Among other honors and awards, he was elected to the U. S. National Academy of Sciences and the Academia Sinica.

ABSTRACT

Roles of Tumor Suppressor Genes in Dendrite Development.

The nervous system is composed of a vast number of neurons with strikingly different dendritic morphology. The control of dendrite branching morphogenesis is an interesting and relatively understudied problem. A few years ago we established the dendritic arborization (da) neurons of the Drosophila peripheral nervous system as a useful model system for a genetic dissection of dendrite development. We have been using this system to address the question of how

neurons establish and maintain their type-specific dendritic fields. One contributing mechanism is known as dendritic tiling, for the complete but non-overlapping coverage of the dendritic fields of certain functionally homologous neurons. Tiling of Drosophila class IV da neurons is established through a like-repels-like behavior of dendrite terminals mediated by Tricornered (Trc) (Grueber et al., Curr. Biol., 2003; Emoto et al., Cell, 2004), one of the two NDR-family of kinases in Drosophila. Recently, we found that the other NDR family kinase, the tumor suppressor Warts (Wts), regulates aspects of dendrite development distinct from that regulated by Trc. In wts mutants, dendrites initially tile the body wall properly, but progressively lose branches at later larval stages leading to severe coverage defects in the dendritic field. Thus Wts is required for the maintenance of tiling of sensory neuron dendrites. Furthermore, we found that the tumor suppressor kinase Hippo (Hpo) functions as an upstream regulator of both Wts and Trc: it acts together with Wts to regulate dendrite maintenance but with Trc to control dendritic tiling. Hippo kinase is known for its role in coordinating cell proliferation and apoptosis through its regulation of Wts and Sav. Our study reveals previously unknown and essential roles of tumor suppressor genes of the Hippo signaling pathway in regulating dendritic branching morphogenesis in post-mitotic neurons. Given that the defect in dendrite stability is a likely cause in some mental disorders, our findings have implications in both normal neural development as well as neurological diseases.

2006 SCBA Presidential Awardee

Lily Jan, Ph.D.
Investigator, Howard Hughes
Medical Institute
Jack and DeLoris Lange Professor
Departments of Physiology and
Biochemistry & Biophysics
University of California, San Francisco

Lily Jan graduated from National Taiwan University before entering Caltech in 1968 for graduate study, initially in physics and then in biology under the guidance of Max Delbrück and Jean-Paul Revel. During postdoctoral training with Seymour Benzer, she began her long-term collaboration with Yuh Nung Jan on studies that uncovered potassium channel abnormalities as the basis for the neurological phenotypes of *Shaker* mutant flies. Similar neurological syndromes of patients with episodic ataxia type I, which was linked to mutations of the human homolog of Shaker potassium channel a decade after their cloning in the Jan lab at UCSF, attest to the evolutionary conservation of potassium channel functions. The Jan lab has followed up on the studies of the Shaker family of voltage-gated potassium channels with cloning of some of the founding members of another large family of potassium channels, inwardly rectifying potassium channels that control heart rate and neuronal excitability, and with molecular and cell biological studies of how these ion channels work, and how they contribute to neuronal signaling and protect neurons and muscles under metabolic stress. Lily Jan is currently Lange Professor of Physiology and Biochemistry & Biophysics at UCSF and an investigator of the Howard Hughes Medical Institute. She has received numerous awards and honors, including election to the U. S. National Academy of Sciences and the Academia Sinica.

ABSTRACT

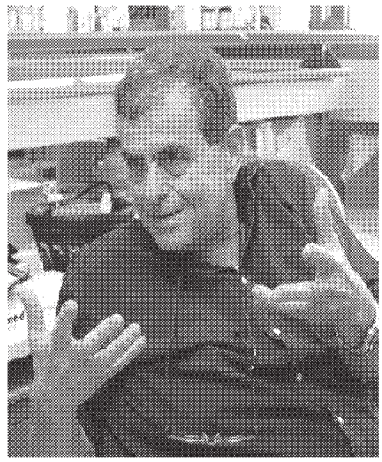
A Novel Mechanism for Trafficking Gap Junction Proteins to the Cell-Cell Border.

To understand how ion channels serve their physiological functions, it is important to learn how the channel density and subcellular localization may be regulated in cells. For this reason, we have been pursuing cell biological studies on the trafficking and targeting of ion channels, such as potassium channels and gap junctions. Gap junctions are intercellular channels that connect the cytoplasms of adjacent cells. For gap junctions to properly control organ formation and electrical synchronization in the heart and brain, connexin-based hemichannels must be correctly targeted to cell-cell borders. While it is generally accepted that gap junctions form via lateral diffusion of hemichannels following microtubule-mediated delivery to the plasma membrane, we have obtained evidence, based on siRNA knockdown and recovery of fluorescence after photobleaching (FRAP), for direct targeting of hemichannels to cell-cell junctions through a pathway dependent on microtubule dynamics, microtubule plus-end tracking proteins and adherens junctions.

Keynote Speakers

Aaron Ciechanover, M.D., D.Sc.

Distinguished Research Professor
Department of Biochemistry
Faculty of Medicine
Technion-Israel Institute of Technology



Aaron Ciechanover was born in Haifa, Israel, in 1947. He is currently on the academic staff of the Faculty of Medicine of the Technion in Haifa, Israel. He received his M.Sc. (1971) and M.D. (1975) from the Hebrew University in Jerusalem, Israel, and his D.Sc. (1982) from the Technion. There, as a graduate student with Dr. Avram Hershko and in collaboration with Dr. Irwin A. Rose from the Fox Chase Cancer Center in Philadelphia, Pennsylvania, USA, they discovered that covalent attachment of ubiquitin to the target substrate signals it for degradation. They deciphered the mechanism of conjugation in a cell-free system, described the general proteolytic function of the system in cells, and proposed a model according to which this modification serves as a recognition signal for a specific downstream protease. As a post doctoral fellow with Dr. Harvey Lodish at M.I.T., he collaborated with Drs. Alexander Varshavsky and Daniel Finley, and described the first mutant cell of the system, further corroborating the role of ubiquitin modification as a proteolytic signal in intact cells. Among the many prizes that Dr. Ciechanover has received are the 2000 Albert Lasker Award for Basic Medical Research, the 2003 Israel Prize in Biology and the 2004 Nobel Prize in Chemistry.

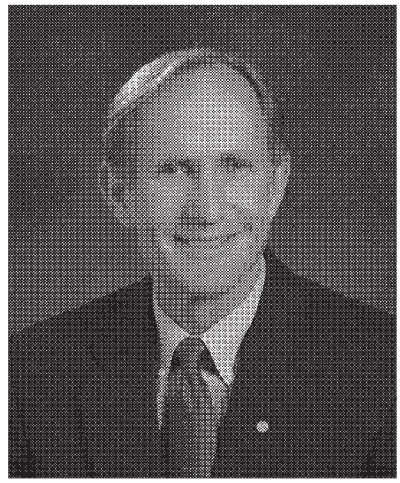
ABSTRACT

The Ubiquitin Proteolytic System: From Basic Mechanisms through Human Diseases and onto Drug Targeting.

Between the sixties and eighties, most life scientists focused their attention on studies of nucleic acids and the translation of the coded information. Protein degradation was a neglected area, considered to be a non-specific, dead-end process. While it was known that proteins do turn over, the large extent and high specificity of the process — whereby distinct proteins have half-lives that range from a few minutes to several days — was not appreciated. The discovery of the lysosome by Christian de Duve did not significantly change this view, as it was clear that this organelle is involved mostly in the degradation of extracellular proteins, and their proteases cannot be substrate-specific. The discovery of the complex cascade of the ubiquitin pathway revolutionized the field. It is clear now that degradation of cellular proteins is a highly complex, temporally controlled, and tightly regulated process that plays major roles in a variety of basic pathways during cell life and death, and in health and disease. With the multitude of substrates targeted, and the myriad processes involved, it is not surprising that aberrations in the pathway are implicated in the pathogenesis of many diseases, certain malignancies and neurodegeneration among them. Degradation of a protein via the ubiquitin/proteasome pathway involves two successive steps: (a) conjugation of multiple ubiquitin moieties to the substrate, and (b) degradation of the tagged protein by the downstream 26S proteasome complex. Despite intensive research, the unknown still exceeds what we currently know on intracellular protein degradation, and major key questions remain unsolved. Among these are the modes of specific and timed recognition for the degradation of the many substrates, and the mechanisms that underlie aberrations in the system that lead to pathogenesis of diseases.

Peter Courtland Agre, M.D.

Vice Chancellor for Science and Technology
Professor of Cell Biology
Professor of Medicine
Duke University School of Medicine



Peter Agre's early life reads like a broadcast of the popular radio program A Prairie Home Companion. Son of a St. Olaf College professor, Agre was raised in a small Minnesota farming community made festive by annual visits of the King of Norway. During the summer, the Agre brothers worked on their cousin's dairy farm, and as Eagle Scouts they explored the Boundary Water Canoe Wilderness at the US-Canada border. In winter they traversed the landscape on cross-country skis. The family moved to Minneapolis where Agre attended high school and studied chemistry at Augsburg College (BA 1970).

It was while attending medical school at Johns Hopkins (MD 1974) that Agre discovered a love for biomedical research while working in the laboratory of eminent membrane biologist, Pedro Cuatrecasas. Following an Internal Medicine Residency at Case Western Reserve University Hospitals of Cleveland and a Hematology-Oncology Fellowship at the University of North Carolina at Chapel Hill, Agre returned to Johns Hopkins as a Postdoctoral Fellow in the Department of Cell Biology in the laboratory of his medical school room-mate, Vann Bennett.

Agre joined the faculty at Johns Hopkins School of Medicine in 1984 and rose through the ranks to Professor of Biological Chemistry and Professor of Medicine. After nearly three decades at Johns Hopkins, Agre moved to Duke University School of Medicine in 2005 where he is Vice Chancellor for Science and Technology and also Professor of Cell Biology and Professor of Medicine.

Agre's research led to the first known membrane defects in congenital hemolytic anemias (spherocytosis) and produced the first isolation of the Rh blood group antigens. In 1992, Agre's lab became widely recognized for discovering the aquaporins, a family of water channel proteins found throughout nature and responsible for numerous physiological processes in humans — including kidney concentration, as well as secretion of spinal fluid, aqueous humor, tears, sweat, and release of glycerol from fat. Aquaporins have been implicated in multiple clinical disorders — including fluid retention, bedwetting, brain edema, cataracts, heat prostration, and obesity. Water transport in lower organisms, microbes, and plants also depend upon aquaporins. For this work, Agre shared the 2003 Nobel Prize in Chemistry with Roderick MacKinnon of Rockefeller University.

Among his awards, Agre received the 1999 Homer Smith Award from the American Society of Nephrology and the 2005 Karl Landsteiner Award from the American Association of Blood Banks. Agre was elected to the National Academy of Sciences in 2000 and the Institute of Medicine in 2005. He was also elected to the American Academy of Arts and Sciences in 2003 and the American Philosophical Society in 2004. In 2005, Agre received the Distinguished Eagle Scout Award from the Boy Scouts of America. Agre has received honorary doctorates from universities in Denmark, Japan, Norway, Greece, Mexico, and Hungary as well as his alma mater, Augsburg College.

Agre always devoted major efforts to medical school activities. He served as Co-Founder and Director of the Johns Hopkins Graduate Program in Cellular and Molecular Medicine — the first NIH funded program in molecular medicine. In addition Agre served as Chairman of the Young Investigators' Day Student Research Awards Program at Johns Hopkins. He now chairs the Advisory Board of the MD PhD Program [Medical Scientist Training Program] at the Duke University School of Medicine. Agre also serves his country as Chairman of the Committee for Human Rights for the National Academies.

ABSTRACT

Aquaporin Water Channels: From Atomic Structure to Clinical Medicine

The high water permeability of certain biological membranes is due to the presence of aquaporin water channel proteins. AQP1 was discovered in human red cells. AQP1 has been thoroughly characterized biophysically, and the atomic structure of AQP1 has been elucidated. Thirteen homologs have been identified in humans. These are selectively permeated by water (aquaporins) or water plus glycerol (aquaglyceroporins). The sites of expression predict the clinical phenotypes in humans. Individuals lacking Colton blood group antigens have mutations in the AQP1 gene. When deprived of water, AQP1-null individuals exhibit a defect in urine concentration and a marked reduction in fluid exchange between capillary and interstitium in lung. AQP1 is expressed in multiple tissues where physiologically important fluid secretion is known to occur including choroid plexus and anterior chamber of eye. AQP0 is expressed in lens fiber cells and mutations result in familial cataracts. AQP2 is expressed in renal collecting duct principal cells where membrane trafficking is regulated by vasopressin. Mutations in the human AQP2 gene result in nephrogenic diabetes insipidus, but underexpression is found in clinical disorders with reduced urinary concentration (e.g., lithium therapy and nocturnal enuresis) and overexpression is found in disorders with fluid retention (e.g., congestive heart failure and pregnancy). AQP5 is expressed in the apical membranes of salivary and lacrimal gland acini, and mistargetting has been identified in some patients with Sjogren's syndrome. Involvement of aquaporins is expected in other human clinical disorders such as brain edema and muscular dystrophy (AQP4), anhidrosis (AQP5) renal tubular acidosis (AQP6), obesity (AQP7) conversion of glycerol to glucose during starvation (AQP9), and cystic fibrosis (several aquaporins). Aquaporins are known to protect micro-organisms from freezing and osmotic shock. Plant aquaporins are involved in numerous processes including the uptake of water by rootlets and carbon dioxide by leaves. The physiological roles of aquaporin homologs are being pursued by multiple laboratories worldwide.

2006 SCBA Junior Award Winner

Yi Zhang, Ph.D.
Investigator, Howard Hughes
Medical Institute Professor
Department of Biochemistry and Biophysics
University of North Carolina
Chapel Hill

Yi Zhang earned an undergraduate degree and a M.S. at the College of Biological Sciences, Beijing Agricultural University, China. He received a Ph.D. at The Institute of Molecular Biophysics of Florida State University in Tallahassee in 1995. He did his postdoctoral training with Danny Reinberg at The Howard Hughes Medical Institute and The Robert Wood Johnson Medical School. He became an independent investigator in 1999 at the University of North Carolina at Chapel Hill. He was named an Investigator of The Howard Hughes Medical Institute in 2005 and is currently a Professor of the Department of Biochemistry and Biophysics and the Lineberger Comprehensive Cancer Center. He has won the Gertrude B. Elion Cancer Research Award from the American Association for Cancer Research and the Sidney Kimmel Foundation for Cancer Research Scholar Award.

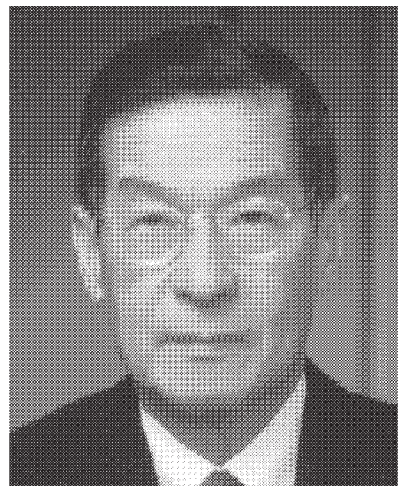
Research in the Zhang lab has been centered on identification and characterization of enzymes that regulate chromatin dynamics and gene expression. The long term goal of the lab is to understand the role of epigenetic modifications in cell lineage determination, maintenance, stem cell pluripotency, and cancer biology.

ABSTRACT

Histone Modifications: From Basic Biology to Therapeutic Intervention.

Dynamic changes in chromatin structure play an important role in regulating many biological processes ranging from gene transcription, heterochromatin formation, to X-chromosome inactivation and cancer. As an integral component of chromatin, histones are subject to a variety of covalent modifications including acetylation, methylation, phosphorylation, and ubiquitylation. Characterization of the enzymes that mediate these modifications, particularly methylation and ubiquitylation, revealed important functions of these modifications in Polycomb gene silencing and cancer development, raising the possibility that these enzymes may serve as targets for therapeutic intervention.

2006 Lifetime Achievement Award



Yuet Wai Kan,
M.B.B.S. (Hons), D.Sc.
Louis K. Diamond Professor of Hematology
Department of Laboratory Medicine
University of California, San Francisco

Dr. Y. W. Kan received his M.B., B.S. (with honors) from the University of Hong Kong Medical School in 1958, and D.Sc. from the University of Hong Kong in 1980. He received clinical training at the Queen Mary Hospital, Hong Kong. In 1960, he went to the United States and obtained additional clinical, hematology and research training in Boston, Pittsburgh, Philadelphia and in Montreal, Canada. In 1970, he joined the faculty of Harvard Medical School and Children' Hospital, Boston, and in 1972 moved to the University of California at San Francisco (UCSF) where he is currently the Louis K. Diamond Professor of Hematology.

Dr. Kan has served on numerous national committees and on the editorial boards of many scientific journals. He was President of the American Society of Hematology (1990) and a former member of the President's Committee on the National Medal of Science (1988–90), a member of the Advisory Council of the National Institute of Diabetes, Digestive and Kidney Diseases of NIH (1991–95) and served on the Visiting Committee to advise the Board of Overseers of Harvard University on Harvard Medical School and School of Dental Medicine (1992–1997). He was also on the Scientific Advisory Board of St. Jude Children's Research Hospital, Memphis, Tennessee (1993–1997), and the President of the Society of Chinese Bioscientists in America (1998–1999). He is currently a member of the Committee on Human Rights and the Executive Council of the National Academy of Sciences (2000–).

Dr. Kan has participated in a number of medical and educational projects in Hong Kong. He was a member of the Research Grants Committee of the University & Polytechnic Grants Committee, Hong Kong (1991–1995) and the Director of the Institute of Molecular Biology of the University of Hong Kong (1990–1994). He is currently the Chairman of the Croucher Foundation of Hong Kong which supports science in Hong Kong and a member of the Scientific Advisory Board and Executive Committee of the Qiu Shi Foundation on Science and Technology of Hong Kong which supports science in China.

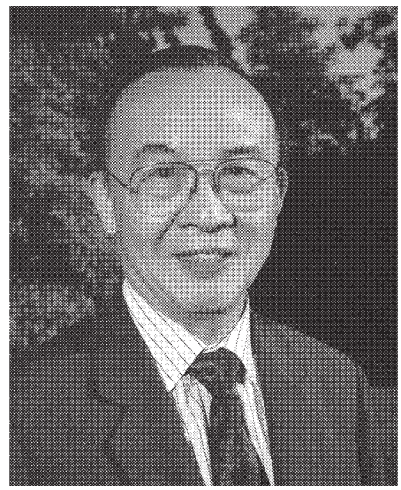
Dr. Kan's contributions are in the fields of hematology and genetics. He was the first investigator who demonstrated the phenomenon of restriction fragment length polymorphism and its linkage to a genetic disease. His work led to the innovation of DNA diagnosis that has found

wide application in many diseases. In recognition of his achievements, Dr. Kan has received many national and international awards. Notable national awards include Dameshek Award, American Society of Hematology (1979); Stratton Award, International Society of Hematology (1980); George Thorn Award, Howard Hughes Medical Institute (1980); Allan Award, American Society of Human Genetics (1984); Lita Annenberg Hazen Award for Excellence in Clinical Research (1984); Waterford Award in Biomedical Science (1987); American College of Physicians Research Award (1988); Warren Alpert Foundation Prize, Harvard Medical School (1989); Albert Lasker Clinical Medical Research Award (1991); City of Medicine Award (1992); Christopher Columbus Discovery Award in Biomedical Research, given one time on the 500th anniversary of Christopher Columbus' discovery of America (1992); Excellence 2000 Award (1993). International awards include the Gairdner Foundation International Award, Canada (1984); Seremo International Award for Genetic Research, Italy (1989); the Federation of Canadian Chinese Professionals Award (1994); the Helmut Horten Foundation Research Award, Switzerland (1995) and the Shaw Prize, Hong Kong (2004).

Dr. Kan is a member of many learned societies among which are the Royal Society, London; National Academy of Sciences, USA; Academia Sinica, Taiwan; and Chinese Academy of Sciences, Beijing. He was the first person of Chinese origin to be elected to the Royal Society of London (1981).

Dr. Kan has received honorary degrees from the University of Cagliari, Sardinia, Italy (M.D.1981), the Chinese University of Hong Kong (D.Sc.1981), the University of Hong Kong (D.Sc.1987) and the Open University of Hong Kong (D.Sc.1998).

Shu Chien, M.D., Ph.D.
University Professor of Bioengineering
and Medicine
University of California, San Diego



Dr. Shu Chien is University Professor of Bioengineering and Medicine of the entire University of California system. Currently, among approximately 9,000 professors in the UC system, there are only twenty University Professors, with about half of them being active. At UCSD Dr. Chien is also Director of the Whitaker Institute of Biomedical Engineering, which he founded in 1991, Chairman of the Department of Bioengineering, which he founded in 1994, and Director of the University of California Systemwide Bioengineering Institute in California, which he founded in 2003.

Dr. Chien received his medical degree from National Taiwan University and his Ph.D. in Physiology from Columbia University, where he was Professor of Physiology and Director of Circulatory Physiology and Biophysics from 1969 to 1988. In 1988 he came to UCSD as Professor of Bioengineering and Medicine. He led the efforts for the UCSD Bioengineering Department to win the Development Award and Leadership Award from the Whitaker Foundation and obtain the first Program Project Grant from the NIH.

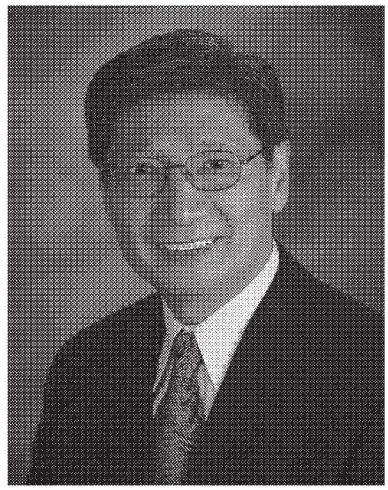
Dr. Chien's research focuses on molecular, cellular and integrative studies on bioengineering and physiology in health and disease, especially in relation to cardiovascular dynamics and mechanotransduction in endothelial cells. Dr. Chien is the author of over 400 peer-reviewed journal articles and editor of nine books. He has received many awards and honors, including the Fahraeus Medal, Landis Award, Melville Medal (twice), Zweifach Award, ALZA Award, Joseph Mather Smith Prize, Ray Daggs Award, Poiseuille Gold Medal, and Galletti Award.

Dr. Chien is a member of the U.S. *Institute of Medicine*, *National Academy of Engineering*, and *National Academy of Sciences*. There are only nine scientists who are members of all three U.S. Academies, and only three of them are active faculty members. Dr. Chien is also a member of the Academia Sinica in Taiwan and a Fellow of the American Institute for Medical and Biological Engineering (AIMBE). He was President of the Microcirculatory Society, American Physiological Society, Federation of American Societies for Experimental Biology, AIMBE, and International Society of Biorheology. He was Co-Chair of the World Congress of Biomechanics in 1990, Chair of the National Organizing Committee of the 2005 Congress of the International Union of Physiological Sciences, and Chair of the International Congress of Biorheology in 2005.

2006 SCBA Lifetime Achievement in Biopharmaceutical Science Award

Allen Chao, Ph.D.

Watson Pharmaceuticals, Inc.



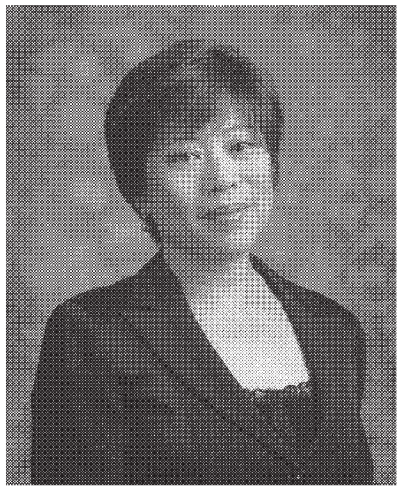
Allen Chao, age 60, is a co-founder of Watson Pharmaceuticals, Inc. (NYSE: WPI), a member of S&P 500. Founded in 1984, Watson develops and markets a broad line of generic and brand pharmaceuticals products and reported 2005 revenues of $1.65 billion. Dr. Chao has been Chief Executive Officer of the Company since August 1985 and Chairman of the Company since May 1996 and he served as President from 1998 until October 2002. Dr. Chao served as Director of Pharmaceutical Technology and Packaging Development at Searle Laboratories, Inc. from September 1979 to August 1983, where he had overall responsibility for new product implementation and new pharmaceutical technology development.

Dr. Chao earned his B.S. in Pharmacy from Taipei Medical College, his master's in pharmaceutics from West Virginia University, and received a Ph.D. in industrial and physical pharmacy from Purdue University in 1973. In May 2000, Dr. Chao received the degree of Doctor of Science from Purdue University in recognition of his leadership and vision for the marketing and production of pharmaceutical products for human healthcare.

2006 SCBA Biopharm Pioneer Award

Nancy T. Chang

Co-founder and Chairman
Board of Directors, Tanox, Inc.



As Co-founder and Chairman of the Board of Directors of Tanox, Inc., Dr. Nancy Chang draws on a quarter century of drug-development experience to guide the company's corporate governance. She was President and Chief Executive Officer from June 1990 to January 2006 and also served as Chairman from 1986 to 2004.

After she received her doctorate in biological chemistry from Harvard University, Dr. Chang was Director of the molecular biology group, for Centocor, Inc., where she contributed to the development of several therapeutic and diagnostic breakthroughs. She also served as an associate professor at Baylor College of Medicine in the division of molecular virology.

It was Dr. Chang's entrepreneurial spirit and scientific vision that led to the creation of Tanox to address significant medical needs in the areas of asthma, allergy, inflammation and diseases that affect the human immune system. She led the company to new heights by completing what was, at the time, the largest biotech initial public offering. The IPO in April of 2000 on the NASDAQ Stock Exchange raised a record $244 million.

Dr. Chang has received numerous academic, national and international awards for her leadership and contributions to the biopharmaceutical industry. She was named a 2005 Most Respected Woman in biotechnology by *MedAd News*, a leading pharmaceutical trade journal. In addition, Dr. Chang received the Global Business Achievement Hall of Fame Governor's Award in 2005 from the Global Federation of Chinese Business Women in the Southern U.S. She was inducted into the Texas Science Hall of Fame in 2001 for exemplary achievement in science and is the recipient of several additional awards, such as the Association of Women in Computing: Top 20 Houston Women in Technology and Houston Entrepreneur of the Year.

As an active member in the biopharmaceutical industry, Dr. Chang has published more than 35 papers on topics ranging from monoclonal antibodies to HIV and holds seven patents. She currently serves on the board of directors of the Federal Reserve Bank in Houston, the Houston Technology Center, the Alliance for Medical Research, BioHouston and the Greater Houston Partnership. She also had served previously on the board of the Biotechnology Industry Organization, the CSIS Council on Biotechnology Research, the Texas Science and Technology Council, the Governor's Advisory Committee on Biopharmaceuticals, and the Center for Houston's Future.