Evolution, Health & Disease

20th Annual International Symposium

Center for Study of Gene Structure & Function
Hunter College, City University of New York

Friday, January 19th, 2007
Room 714 Hunter West Building, 68th Street & Lexington Avenue
West Building, Room 714 HW, New York City

SYMPOSIUM PROGRAM

The 20th annual international symposium of the Center for the Study of Gene Structure & Function (Gene Center) is supported by the Research Centers in Minority Institutions Program (RCMI) of the Division of Research Infrastructure of the National Center for Research Resources (NCRR) of the National Institutes of Health (NIH) - Grant Number G12 RR-03037

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The RCMI Program enhances the research capacity and infrastructure at minority colleges and universities that offer doctorates in health sciences. http://www.ncrr.nih.gov/resinfra/ri_rcmi.asp

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Center for Study of Gene Structure and Function

The Gene Center is a consortium of researchers within Hunter College of The City University of New York — one of the largest public universities in the nation. At the heart of the Gene Center's mission is an imperative to build unique collaborations among biologists, chemists, biopsychologists, biophysicists, and bioanthropologists; to recruit and equip outstanding faculty; to develop and share core research facilities; and to implement strategies for scientific networking.

Since the Center's inception in 1985, the growing number of papers published in peer-reviewed journals and the number and amount of grants obtained by the faculty have been the most visible hallmarks of the Center's success. The Gene Center provides a vibrant research environment marked by workshops on cutting-edge research techniques; frequent research colloquia by guest scientists; and an annual international symposium, which is a major event on the New York scientific calendar.

The Gene Center encourages bright undergraduates to make a career of scientific research by hosting a Summer Program for Undergraduate Research (SPUR). Established in 1994, SPUR was formed to prepare and mentor qualified American undergraduate students who would like to pursue graduate biomedical research. SPUR was especially developed to recruit and nurture minority talent. Hunter College is a leader in academic diversity, with an undergraduate student population that reflects the demographics of New York City. Dr. Robert Dottin, Director of the Gene Center, has successfully diversified the faculty and graduate student bodies, providing role models for excellence in Science. The Gene Center is proud to be a sponsor of the Diversity Supplement of The Scientist.

Visit the Gene Center website: http://genecenter.hunter.cuny.edu

8:00am Science Poster Set-Up

MORNING SESSION: EVOLUTION OF MOLECULES AND PATHOGENS

9:00 Michael Steiper – Session Chair

9:15 Paul Ewald – Keynote Speaker:
Gene/Environment Interaction and the Causes of Atherosclerosis

9:45 Holly Wichman
Experimental Evolution in a Virus Model System

10:15 M. Cristina Gutierrez, Pasteur Institute
Parallel Origin and Diversity of TB Agents and Humans

10:45 BREAK

11:00 Steve Mack
Using the Human Major Histocompatibility Complex to Study Disease, Natural Selection and Human Evolution

11:30 Questions to Panel of Speakers

12:00 – 1:15 LUNCH & SCIENCE POSTER SESSION

AFTERNOON SESSION: HEALTH EVOLVING – PEOPLE AND POLICY

1:15 Remarks from sponsors

1:30 Christopher Braun – Session Chair

1:35 Randolph Nesse – Keynote Speaker:
Darwinian Medicine: Why has Natural Selection Left us so Vulnerable to Disease?

2:05 Arata Kochi
Malaria Control: Why It Has Failed and How to Fix It? Public Health Approaches and International Politics

2:35 BREAK

2:50 Stuart B. Levy, M.D.
The Ecology of Antibiotic Resistance

3:20 Paul Sherman
Allergies and Cancers: Are The Complex Relationships Comprehensible?

3:50 Stephen Bezruchka
Targeting Childhood Development to Make the Nation Healthy Again

4:20 Questions to Panel of Speakers

4:40 Awards for 3 Best Posters
Closing Remarks

Stephen Bezruchka
University of Washington
Targeting Childhood Development to Make the Nation Healthy Again

Abstract: The US is less healthy by any measure than pretty well all the other rich countries and a few poorer ones despite spending half of the world's health care bill. Our health continues to decine compared to other nations and this year there are at least 28 healthier countries while 55 yeras ago the US could boast being in the top five or ten. Even if we eradicated heart disease, our leading cause of death, we would still not be the healthiest. Research on the determinants of health of populations over the last 25 years points to structural factors impacting economic justice as the root cause of our poor health as a population. More egalitarian societies have better average health thus implicating our increasing economic inequality as the primordial cause. This factor may have its greatest impact during early life. If this nation is to regain its health status compared to other countries, just political policies that promote advantaged early life for all will need to stay in place for decades . Enacting and maintaining these policies for a generation or two will have major health impacts, far beyond those of any other 'medicine.'

Bio: Stephen Bezruchka graduated from Stanford Medical School in 1973 after getting a master's in mathematics at Harvard. He also received a Master's in Public Health from Johns Hopkins University in 1993. He continues to practice medicine in the US as an emergency physician. He has also worked in Nepal for over ten years in community health projects, setting up remote district hospitals as teaching institution for Nepali family practice doctors and supervising them in their rotations there. He currently works with Nepali doctors there to improve surgical services in remote hospitals. He is a Senior Lecturer in the International Health Program of the Department of Health Services at the School of Public Health and Community Medicine in the University of Washington. He set up and facilitated the introductory component of their Community Oriented Public Health Practice MPH curriculum that creates learning opportunities by group study of problems rather than by faculty giving lectures. He received the 2002 Outstanding Teacher Award in the School of Public Health. He worked as the assistant health officer for Kittitas County in Washington State in the mid 1990s. His main interest is in disseminating information about population health and for this purposes he maintains a web site: (http://depts.washington.edu/eqhlth/) where the science is presented. He runs a Population Health Forum at the University of Washington where people try to advance understanding of these concepts. The most exciting developments at present revolve around developing curriculums on population health for middle and high schools. He speaks widely to groups from the homeless, to teachers conferences, to student groups, church organizations, unions and political action networks.

Paul Ewald
University of Kansas
Gene/Environment Interaction and the Causes of Atherosclerosis

Abstract: Gene/environment interactions are broadly considered to be important for understanding biomedical phenomena. But the value of considering such interactions depends on the breadth of environmental variables that are considered and the how the

environmental variables are integrated into alternative causal hypotheses. In medical research on chronic diseases, environmental variables are often considered descriptively as risk factors, but the spectrum of known or suspected risk factors are rarely cast integratively into alternative hypotheses of causation. An evolutionary perspective is central to this process because evolutionary considerations aid in the generation of alternative hypotheses of causation, and help distinguish reasonable hypotheses of from infeasible ones. My presentation will apply such an integrative approach to the causes of atherosclerosis as a model for investigating of the causes of chronic disease in general.

Bio: Paul W. Ewald is a Professor of Biology and Director of the Program on Disease Evolution at University of Louisville. He holds appointment in the Department of Biology at the Academic campus and the Department of Microbiology and Immunology at the School of Medicine. Professor Ewald received his B.Sc. in Biological Sciences from the University of California and his Ph.D. from the University of Washington with a specialization in evolutionary biology. He was the first recipient of the Smithsonian Institution's George E. Burch Fellowship in Theoretic Medicine and Affiliated Sciences, which was established in honor of the renowned cardiologist to foster pioneering advancements in the health sciences. Prior to joining University of Louisville, Professor Ewald was on the Faculty at Amherst College, where he held Assistant, Associate and Full Professorships and was the Dominic Paino Professor of Global Environmental Studies. During this time he also held and adjunct appointment at the University of Massachusetts. Professor Ewald was a principle founder of the discipline evolutionary medicine, by virtue of the papers and books he has published from 1980 onwards. He is the author of Evolution of Infectious Disease (Oxford) which is widely acknowledged as the watershed event for the emergence of this discipline (awarded Honorable Mention from the Association of American Publishers, 1994 Annual Award Program for Excellence in Professional and Scholarly Publishing). The book summarized the conceptual framework for understanding the evolution of acute infectious diseases, which he developed during the 1980s. It also laid down its application to the threat posed by influenza, which has been proven accurate by the dozen years that have elapsed since its publication. His second book Plague Time (Free Press & Anchor) integrated many of these ideas with our emerging understanding of the broad role of germs as causes of chronic diseases.
He has written many articles for scientific journals on topics ranging from territorial behavior to new strategies for designing vaccines. He also has written numerous articles for popular magazines such as Natural History, National Geographic, and Scientific American, and Op Ed pieces for the New York Times and the London Times. His work has been featured in publications such as Science, Newsweek, The New York Times, Omni, Scientific American, Forbes, Fortune, US News & World Report, The Atlantic Monthly, and Esquire's Best and Brightest issue for 2005, often as cover stories. He has lectured extensively at college campuses and symposia around the world and has made approximately 200 appearances on television (PBS, Learning Channel, Discovery Channel, NBC, and Canadian Broadcast Company, Australian Broadcast Company, etc.) and radio (NPR's Science Friday, Soundprint and The Connection, CBC's Quirks & Quarks and Sunday Edition, the Newsweek Radio Show, etc.).

M. Cristina Gutierrez
Pasteur Institute
Parallel Origin and Diversity of TB Agents and Humans

Abstract: The agent of tuberculosis is inextricably intertwined with humankind. Every second someone somewhere in the world gets infected, and each year TB kills about 2 million people. How long these bacteria have been co-living with our ancestors? Recent

studies indicate that the ancestor of contemporary *Mycobacterium tuberculosis* originated from a 3 million years old species. Extant *Mycobacterium prototuberculosis* are found in patients from East Africa, not far from some of the richest lodes of hominid fossils in Ethiopia, and the genetic diversity of these bacteria reflects the genetic diversity of living Africans. One branch of this species expanded about 35,000 years ago and spread around the world, today infecting one-third of humankind. *Mycobacterium tuberculosis* had to spread with the waves of human migration, because these bacteria are not found in the environment and humankind is virtually its only reservoir. The worldwide contemporary tuberculosis bacilli show a high genetic diversity, with a global population structure and geographical distribution comparable to those of modern human population. Almost certainly, tuberculosis has impacted on humankind through pre-history. *M.tuberculosis* and its earlier relatives have probably been co-evolving with *Homo sapiens* and its earlier relatives for hundred of thousand of years.

Bio: Cristina Gutierrez was born in 1962 in Vigo (Spain). She obtained her medical degree in 1986, and her Ph.D. in Molecular Biology in 1990, both from the University of Santiago de Compostela, Spain. In 1996, she received her Specialty in Medical Microbiology from the Spanish Ministry of Education. She is presently a senior researcher at the Institut Pasteur, Paris. She has been working at the Institut Pasteur since 1997. First she focused her work on developing molecular methods for characterization of the agents of tuberculosis and other mycobacteria, and on their application on molecular epidemiological studies of tuberculosis. Then she focused on the molecular evolution of the tuberculosis agents and their diversity on different human populations around the world. From 2000 to 2005, she worked as well as deputy director at the French National Reference Center for Mycobacteria. She was a founder-member of the European Network for surveillance of multidrug resistant tuberculosis. Since 2000, she teaches at the Institut Pasteur School for Post-graduate Training where she heads a course on molecular tools and epidemiology of tuberculosis. She is active in national and international Mycobacteriology Societies, and she is a scientific advisor for the French High Council of Public Health. Recently, on the basis of her experience with mycobacteria, she and her collaborators have challenged the hypothesis of a recent origin for human tuberculosis. Working with bacteria from patients in eastern Africa, she has instead proposed the hypothesis that the agents of tuberculosis originated millions years ago in eastern Africa, where they have been co-evolving with humans since the origin of the humankind. She has authored or coauthored over sixty scientific publications and book chapters, and has been a referee for various scientific journals. Articles about her work have appeared in newspapers and magazines around the world, including the BBC News, The New York Times, The Washington Post, The Spiegel and Le Figaro.

Arata Kochi
Director, World Health Organization's
Global Malaria Program (WHO/GMP)
Malaria Control: Why It Has Failed and How to Fix It?
Public Health Approaches and International Politics

Abstract: Through comparative analysis of 'successful' global efforts to combat infectious diseases, including immunization, TB, and past malaria control efforts, the speaker will describe the history of global malaria control and why past efforts have 'failed' from both scientific and political perspectives. The analysis will include, inter alia, epidemiology, available tools, mindset/culture of the malaria community, as well as interactions between research and control efforts, and global partners. New strategic

approaches --both technical and political, adopted and espoused by the World Health Organization's Global Malaria Programme beginning in 2006, will be presented.

Bio: In October 2005, Dr Arata Kochi, one of the most senior public health experts at WHO, was appointed by WHO's Director-General, Dr LEE Jong-Wook, to revitalize the Organization's malaria control efforts. Prior to his appointment as Director of the Global Malaria Programme, Dr Kochi served in several key leadership posts at WHO. In 1989, Dr. Kochi was charged by WHO to revise and strengthen its programs to control the global tuberculosis (TB) epidemic. At that time, only 2% of the world's TB patients were receiving proper TB treatment recommended by WHO. Because of new control practices initiated by Dr Kochi, nearly 60% of TB patients now receive this quality treatment and care. In 2001, Dr Kochi became the Director of WHO's HIV Department and initiated AIDS treatment in poor countries which lead to the "3 by 5" initiative.
As a medical doctor, Dr Kochi was trained at the Tohoku University Medical School in Japan where he also obtained a PhD in Social Medicine. He also holds a Master of Public Health and a Master of Science in Food Policy and Nutrition degrees from the Harvard School of Public Health. Dr. Kochi also has extensive field experience, having worked for UNICEF as a health and nutrition expert in Myanmar and Afghanistan.

Stuart B. Levy, M.D.
Tufts University
The Ecology of Antibiotic Resistance

Abstract: Antibiotic resistance is a major public health problem in countries throughout the world. Bacteria know no boundaries: resistant, like susceptible strains can move from person to person, community to community, city to city and country to country. Resistant forms are selected by antibiotic use in various locations, e.g. people, animals, agriculture, and become prominent in these environments. All bacteria are subject to resistance selection and these resistances are transferable among bacteria of different types. Thus while it is important to be aware of resistance in bacteria which are of clinical importance, we must also maintain awareness of resistance among the commensal flora. Both clinical and commensal strains make up the pool of resistant bacteria that share resistance determinants among themselves in broad ecologic and environmental areas. Since bacteria and resistance determinants move, the selection of drug resistance anywhere in the world can eventually be a problem somewhere else. This phenomenon highlights the concept that antibiotics are unique therapeutics – they are societal drugs. The emergence of resistant bacteria in an individual taking an antibiotic becomes a problem for all members of society since these bacteria are easily shared. The frequency of resistance emergence can be seen as a result of "selection density." The greater numbers of individuals being treated with antibiotics in one location, the greater rate and frequency of resistant bacteria there. To control the spread of resistance, we must improve our understanding of how to use these valuable therapeutics and increase our awareness of the broad ecologic presence of drug resistance in bacteria.

Bio: Stuart B. Levy, M.D., Professor of Medicine and of Molecular Biology and Microbiology, is the Director of the Center for Adaptation Genetics and Drug Resistance at Tufts University School of Medicine and Staff Physician at the New England Medical Center. He also serves as President of the *Alliance for the Prudent Use of Antibiotics*, an international organization with members in over 100 countries of the world. He is a past President of the 42,000 member *American Society for Microbiology*. He is co-founder

and Chief Scientific Officer of Paratek Pharmaceuticals, Inc.

A magna cum laude, Phi Beta Kappa graduate of Williams College, Dr. Levy received his medical degree from the University of Pennsylvania. He did his residency at Mt. Sinai Hospital in New York and postdoctoral research at the National Institutes of Health, Bethesda. Dr. Levy is a Fellow of the American College of Physicians, Infectious Disease Society of America, American Association for the Advancement of Science, and the American Academy of Microbiology.

Dr. Levy is well known for his contributions to the antibiotic resistance field. He has published over 250 papers on antibiotic use and resistance. He authored *The Antibiotic Paradox*, now in its second edition, and has edited four books and two special journal editions devoted to the subject and has organized numerous international symposia and meetings on antibiotic resistance. Dr. Levy has been featured and quoted for his work on antibiotic use and resistance in major national and international newspapers and magazines and on all major U.S. television network news shows, including the *CBS Evening News, ABC World News* Tonight and the *NBC Today* shows. He has appeared on many National Public Radio programs including *Fresh Air, The Connection, Science Friday*, and *All Things Considered*.

Steve Mack
Roche Institute

Using the Human Major Histocompatibility Complex to Study Disease, Natural Selection and Human Evolution

Abstract: The Human Leukocyte Antigen (HLA) genes are the most polymorphic loci in the human genome. The products of these genes present endogenous and exogenous peptides for inspection by T-cells, which recognize and respond to HLA-presented antigens by initiating specific immune responses, and as such are central to immune function. Susceptible and protective associations have been observed between alleles at specific HLA loci and a variety of autoimmune diseases (e.g., Type-1-Diabetes and Rheumatoid Arthritis) and cancers (e.g. Nasopharyngial Carcinoma), and for many diseases, the HLA region appears to be the major genetic determinant of disease. These associations of disease with HLA diversity are observed to be population-specific, and are likely the result of the genetic diversification of the human population in its spread across the globe.

The central immune role played by the HLA loci results in strong selection for population-level diversity (balancing selection), a phenomenon that, when considered with the tremendous diversity at these loci, makes them suitable for studies of population history, and the testing of specific anthropological hypotheses. For example, phylogenetic analysis of HLA allele and haplotype frequencies in Oceanian and Pacific populations indicates that the Highland populations of Papua New Guinea (PNG) are more closely related to Aboriginal Australian populations than to coastal and Lowland PNG populations, a finding that supports the so-called "Sahul Hypothesis" of the colonization of Australia and New Guinea.

The strong balancing selection observed at the locus level for HLA loci can be dissected at the amino-acid level by exploring the selective forces shaping diversity within specific functional groups of peptides and at individual peptide residues. For example, the strong balancing selection seen worldwide at the HLA-C locus may result from the functional constraints placed on this molecule by interaction with both T-cells and Natural Killer cells. In this way, the fundamental biochemistry of these molecules, which is the same

in all populations, serves as the basis for understanding the tremendous molecular diversification and diversity of disease-associations seen around the world.

Bio: Steven J. Mack earned his Ph.D. in Molecular and Cell Biology in Allan Wilson's laboratory at the University of California at Berkeley in 1996, studying the molecular evolution of mitochondrial DNA and MHC genes in Native American populations. He then post-docced with Henry Erlich at Roche Molecular Systems, studying the population genetics and molecular evolution of MHC in human populations. From 1999 to 2005, he co-chaired the Anthropology / Human Genetic Diversity components of the 13th and 14th international Histocompatibility workshops, and served on the Histocompatibility Committee of the National Marrow Donor Program. Dr. Mack is now an assistant staff scientist at the Children's Hospital Oakland Research Institute, but is a native of the Bronx, and still roots for the Yankees.

Randolph Nesse
University of Michigan

Darwinian Medicine: Why has Natural Selection Left Us So Vulnerable to Disease?

Abstract: Darwinian medicine is the enterprise of using the principles of evolutionary biology to address the problems of medicine. This lecture begins with data showing evolutionary biology nearly absent from medical curricula. Even the fundamental distinction between evolutionary and proximate questions remains poorly understood in medicine. Asking evolutionary questions is crucial to understand why natural selection has left the body so vulnerable to so many diseases. The possible explanations fit nicely into six categories:

1. The mismatch between our bodies and novel aspects of the modern environment gives rise to much chronic disease.
2. Pathogens evolve so quickly that we cannot keep up
3. Constraints such as path dependence limit the perfection of traits shaped by selection.
4. Tradeoffs leave every trait in the body imperfect and vulnerable to disease
5. Selection shapes organisms for maximal reproductive success, even if that compromises individual health and longevity.
6. Defenses such as pain, cough and fever are not diseases, but responses shaped by selection and regulated so they are expressed when they are useful.

The regulation of defensive responses like pain fever and anxiety is addressed in detail. Following the principles of signal detection theory, normal defense regulation systems are shaped by selection to express many painful defenses in situations where they are not actually necessary. This has major implications for everyday medical practice, and research. The overall conclusion is that evolutionary biology is an invaluable basic science for medicine, one whose applications are just now being explored.

Bio: Randolph M Nesse is Professor of Psychiatry, Professor of Psychology and Research Professor at the Research Center for Group Dynamics at the Institute for Social Research at the University of Michigan where he directs the Evolution and Human Adaptation Program. He is a founder of the field of Darwinian Medicine. His early research was on the neuroendocrinology of anxiety and the treatment of anxiety disorders. Currently he is dedicated to advancing the field of Darwinian medicine, with special attention to implications for psychiatry. His specific research now focuses on the evolutionary origins of emotions, especially mood and moral emotions that make committed relationships possible.

Paul Sherman
Cornell University

Allergies and Cancers: Are the Complex Relationships Comprehensible?

Abstract: Are allergies and cancers related and, if so, why? To address these questions we comprehensively reviewed all published information. We located 118 papers (1955-2005), which reported results of 278 studies of individual types of allergies and 136 studies of multiple allergies combined in relation to cancers of 18 specific tissues and organ systems or multiple cancers combined. We used these data to test three alternative hypotheses: (1) antigenic stimulation (allergy symptoms increase cancer risk because chronic inflammation and stimulation of cell growth provides frequent opportunities for mutations and malignant proliferation of actively-dividing stem cells), (2) prophylaxis (allergy symptoms reduce cancer risk by binding heterospecific cells, toxins, and foreign particles with mucous and expelling them before they, and any contained or adhering mutagens, can trigger carcinogenesis), and (3) immunosurveillance (allergy symptoms do not directly affect cancer risk, but allergies and cancers are inversely related because immune systems that are especially capable of detecting and attacking pre-malignant autogenic cells also are adept at recognizing and eliminating heterospecific cells, toxins, and foreign particles). Overall, 0.44 of studies of specific allergies and 0.44 studies of multiple allergies combined reported that people diagnosed with a cancer were less likely to have expressed allergy symptoms – coughing, itching, tearing, diarrhea, etc. - prior to their diagnosis than matched comparison groups of non-cancer patients. By contrast, only 0.18 of studies of specific allergies and 0.16 of studies of multiple allergies combined reported that cancer patients were more likely to have expressed allergy symptoms prior to diagnosis than non-cancer comparison groups. The remaining studies of specific allergies (0.37) and of multiple allergies combined (0.40) found no relationships with cancers. Contrary to antigenic stimulation, there was a significant excess of inverse allergy-cancer relationships over positive and null relationships. Allergy-cancer relationships also differed among types/sites of cancers: inverse relationships were significantly more frequent for cancers of seven tissues and organ systems of epithelial embryonic origins that can interface with the external environment than for cancers of five tissues and organ systems of endothelial origins that interface only with the body's internal milieu. These results suggest that normal (sub-lethal) allergy symptoms help protect certain tissues frfom cancer, especially tissues that are routinely exposed to environmental toxins, microorganisms, and particulate matter such as dust, pollen, smoke, and mold to which mutagenic chemicals may be adhering.

Bio: Prof. Sherman was an undergraduate at Stanford, a graduate student at Michigan, and a Miller Postdoctoral Fellow at Berkeley. He joined the Cornell faculty in 1981, was awarded tenure in 1985, and was promoted to Full Professor in 1991.
Prof. Sherman's research has contributed to scientific understanding in six general areas: altruism, kin recognition, eusociality, sexual selection, conservation biology, and Darwinian Medicine. He has studied birds, insects, and mammals, including an insect-like mammal, the naked mole-rat. He has published almost 200 papers and books.
Prof. Sherman teaches courses and seminars in Behavioral Ecology, Animal Behavior, and Darwinian Medicine. In 2005 he was appointed an S. H. Weiss Presidential Fellow in recognition of "effective, inspiring, and distinguished teaching." He was a Sigma Xi National Lecturer in 2004-06, and was elected a Fellow of the Animal Behavior Society in 2004.

Holly Wichman
University of Idaho
Experimental Evolution in a Virus Model System

Abstract: Most pathogens evolve at a rate that is very rapid relative to human life span. Thus pathogens can adapt to new hosts, evolve resistance to drugs and vaccines, and evade immune response. Population genetics has given us a large body of theory relevant to these issues, and now experimental evolution puts us in the position of testing some of these theories. We use experimental evolution of icosahedral phages as well as phenotypic and sequence analysis of wild phages to explore the rules of viral evolution. The strengths of the system include: the well-described biology and known structure of the prototype icosahedral phage, X174; the small genome size, which allows for extensive whole genome characterization over the course of evolution; and several methods of propagation, which allow many different problems to be addressed.

Bio: Dr Wichman is a Professor in the Department of Biological Sciences, University of Idaho. She obtained her Ph.D. in Biology from Wesleyan University, Connecticut in 1983. Her laboratory conducts research in two areas – the molecular basis of adaptive evolution and the genomic impacts of mammalian transposable elements. Her studies of adaptive evolution take an experimental approach, using bacteriophages as a model system to study patterns and processes of evolution in real time. Dr.Wichman has published extensively in both these fields, and is also interested in the applications of evolutionary biology to societal issues in health, agriculture and industry. She is the associate Editor of the journal "Genetica" and has served as an editorial board member of the journal "American Naturalist". In 2005 she co-organized an NIH workshop on Evolution of Infectious Disease.

Thank you for attending the 20th Annual International Symposium of the Center for Study of Gene Structure & Function at Hunter College.

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