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### Meeting Summaries

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#### **The First International Workshop on Molecular Epidemiology and Evolutionary Genetics of Pathogenic Microorganisms**

Under the auspices of the Centers for Disease Control and Prevention (CDC), ORSTOM (the national French agency for scientific research in developing countries), and CNRS (the national French agency for basic research), the First International Workshop on Molecular Epidemiology and Evolutionary Genetics of Pathogenic Microorganisms was held in Atlanta, from June 16 to 19, 1996. The workshop was cosponsored by the National Institutes of Health (NIH), the Burroughs Wellcome Fund, the National Foundation for CDC, Boehringer Mannheim, the French Ministry of Foreign Affairs, and Emory University. Five hundred participants (health care providers, public health professionals, and laboratory scientists) from 25 countries attended the 3-day workshop, whose purpose was to exchange information on the use of molecular tools and approaches in areas of molecular epidemiology and evolutionary genetics in studies of emerging, reemerging, and endemic diseases. The workshop provided an opportunity for CDC, NIH, the World Health Organization, the Walter Reed Army Institute of Research, the Kenya Medical Research Institute, and ORSTOM to present jointly their perspectives on meeting the challenges of emerging infectious disease.

During the workshop, public health and laboratory science-based presentations on parasitic, fungal, bacterial, and viral diseases identified information gaps in the areas of disease and pathogen detection; laboratory-based presentations focused on the use of molecular tools and approaches in pathogen identification and evolution; and other presentations focused on specialized themes, such as the definition of a strain, tools and approaches in molecular epidemiology, emerging infections, concomitant infections, insect disease vectors, opportunistic infections, and tropical parasites.

Many of the challenges of dealing with emerging and reemerging pathogens are common to parasitologists, virologists, bacteriologists, and mycologists. Many pathogens cannot be maintained or propagated in culture or in animal

models often because the biology and physiology of these pathogens are not known. This difficulty highlights the advantage of moving directly to molecular probes, polymerase chain reaction amplification, and sequence-based identification for substantiating epidemiologic relationships. Infectious disease clinicians and epidemiologists are faced with whether the disease under investigation is caused by a recently acquired infection or a recrudescing infection and whether an infection is caused by multiple species/strains. In addition, host and pathogen genetic factors that influence susceptibility and pathogenesis and environmental factors that influence transmission of pathogens are critical in the assessment of risk factors for acquiring infections. Molecular approaches to identifying emerging, reemerging, and endemic pathogens were described as most likely to yield the tools needed by epidemiologists to assess the source and risk factors, thus allowing the formulation of needed prevention and control guidelines.

Various approaches and tools now used in detecting pathogens and in studying evolution were examined, and molecular biology and evolutionary genetics applications in the following areas were discussed: 1) diagnosis of known pathogens and development of rapid means to identify unknown pathogens; 2) strain characterization for epidemiologic tracking; 3) ecologic and biologic factors that influence emergence of pathogens; 4) reassessment of taxonomy using molecular biologic data; 5) evaluation of the impact of genetic diversity of microorganisms on vaccine, drug, and insecticide efficacies; 6) gene flow in natural populations of vectors and pathogens; and 7) the role of vectors in the evolution of pathogens. Regardless of the organism under study, a unified approach in evolutionary genetics and population biology was recommended.

Two other topics related to emerging infections were also examined. The first concerned the risk for infection of human recipients of xenogeneic agents through xenotransplantation (and the subsequent transmission of these pathogens to the general population). The second concerned the role of immune activation caused by chronic infections (parasitic and bacterial) and immunization (pneumococcal and influenza) in HIV-infected persons in promoting the replication of HIV and associated progression of disease manifestations. The former is a concern in areas of sub-Saharan Africa and Asia where HIV coexists

with parasitic (e.g., malaria and schistosomiasis) and bacterial (e.g., tuberculosis) infections; the latter is a concern in the United States, where pneumococcal and influenza vaccinations are recommended for HIV-infected persons. Field-based, prospective, and longitudinal studies are needed for a complete picture of the extent of interaction between vaccination and pathogen-induced immune activation, HIV replication, and associated rapid progression to AIDS.

The need for a global partnership to facilitate a more rapid identification of infectious agents in a manner that discriminates among closely related strains and species and uses genetic information to study evolution, emergence, and dispersal of infectious agents was emphasized. To address emerging infectious disease threats, CDC has a strategic plan that emphasizes surveillance and applied research for a strong public health-based defense against infectious disease. A goal of this plan is the integration of laboratory science and epidemiology to develop and use tools to detect and promptly identify emerging and reemerging pathogens and investigate factors that influence their emergence. To promote international collaborations and interaction between clinicians, epidemiologists, and laboratory scientists, CDC, ORSTOM, and CNRS will cosponsor the 2nd International Workshop on Molecular Epidemiology and Evolutionary Genetics of Pathogenic Microorganisms at ORSTOM, Montpellier, France from May 26 to 28, 1997.

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### **Simian Virus 40 (SV40), a Possible Human Polyomavirus (Workshop Held at NIH)**

During the past 4 years, polymerase chain reaction (PCR) assays have detected DNA sequences related to SV40 (an oncogenic simian polyomavirus) in a variety of human tissues, especially choroid plexus tumors, ependymomas, mesotheliomas, and osteosarcomas (1-7). These findings were supported by the isolation of infectious SV40 from a choroid plexus tumor (8).

Although another paper reported the failure to detect SV40 DNA in mesotheliomas (9), these studies have reawakened interest in inadvertent human exposure to SV40 in the late 1950s and early 1960s when polio and adenovirus vaccines prepared in rhesus monkey cells containing SV40 were used (10,11). In response to the implications of detecting SV40 DNA in human tumors, the Food and Drug Administration, National Institutes of Health, National Vaccine Program Office, and Centers for Disease Control and Prevention sponsored a workshop on SV40 on January 27-28, 1997 at the National Institutes of Health to examine the possibility that SV40 is an infectious agent in humans.

The workshop first reviewed the biology of SV40 and the human polyomaviruses JC and BK and the data associating SV40 DNA with human tumors. In addition to tumors, SV40 DNA sequences have been detected in human pituitary gland tissue, peripheral blood mononuclear cells, and seminal fluids from healthy persons (3,5,7). Two laboratories were unable to detect SV40 DNA by PCR assays in human tissue, including mesothelioma; researchers noted the ability of the PCR primers used in these assays to amplify DNA sequences from JC and BK viruses as well as from SV40 and discussed whether each set of primers in the PCR reaction requires specific conditions to amplify virus-specific DNA. Furthermore, preliminary data suggested that primers considered to be SV40-specific could, under certain conditions, amplify what appeared to be host DNA sequences. Two laboratories demonstrated that the sensitivity of different PCR primers to detect SV40 DNA was 1-10 to 10-1,000 SV40 genomes. These discussions emphasized the need for caution in interpreting PCR data and the need for standardized, quantitative PCR assay procedures.

National Institute for Biological Standards Control scientists described the use of PCR assays to search for SV40 DNA in current and early lots of polio vaccines and concluded that polio vaccines used in the United Kingdom in 1971 to 1996 did not contain SV40 DNA, while early vaccines prepared in rhesus monkey cells contained easily detectable amounts of SV40 DNA. To evaluate the relationship between exposure to SV40 in the early polio vaccines and the development of tumors (choroid plexus tumors, ependymomas, mesotheliomas, and osteosarcomas), scientists described epidemiologic surveys

that used tumor registries in two countries (two in the United States, one in Sweden). The surveys compared tumor incidence data in persons who could have been exposed to SV40 in polio vaccines with those who, because of their date of birth, could not have been exposed directly; no discernible relationship between exposure to SV40 and development of tumors was found. The surveys also found no association between exposure to SV40-contaminated polio vaccines and the incidence of tumors of the brain and ovaries. These results support the findings of most of the earlier epidemiologic studies (10,11) and help mitigate public health concerns about the use of SV40-contaminated polio vaccines.

Whether SV40 is a human infectious agent that might play a role in human neoplastic disease was also discussed. If SV40 DNA sequences are present in choroid plexus tumors in children born many years after vaccines were SV40 free (8), the possibility that SV40 is present in the population must be considered. Researchers reviewed data on SV40 antibodies in sera taken before 1954 (12) and in sera from persons in remote regions (13,14) not exposed to SV40-contaminated polio vaccines; the data suggest that SV40 might have been present in humans before the polio vaccines were introduced in 1954 (11). Because of cross-reactivity between BK, JC, and SV40 antibodies (15) and the lack of standardized serologic assays to identify SV40 specificity of antibodies present in single samples of human serum, it is difficult to determine whether SV40 was present in humans before the population was exposed to SV40 in the early polio and adenovirus vaccines. Thus, determining whether SV40 is an infectious agent in humans and whether humans were exposed before the polio vaccine was introduced requires further study.

Two preliminary studies showed that SV40 T proteins are expressed in some cells in mesotheliomas and these proteins can bind to both the p53 and Rb cell-cycle control proteins. The SV40 T protein-p53 and SV40 T protein-RB protein interactions are thought to contribute to neoplastic transformation; however, the role of these interactions in SV40-induced neoplastic transformation is unresolved. Further attempts to assess the link between SV40 DNA sequences and neoplastic processes in humans will require more conclusive data about whether SV40 DNA sequences are present in tumors, whether this

viral DNA is integrated or extrachromosomal, and whether it is expressed. Two new experimental approaches were suggested to assess the ability of SV40 to contribute to neoplastic development in humans: prospective studies on the presence of SV40 in mothers and other family members of children with choroid plexus tumors and studies comparing the function of what may be an SV40-associated defect in the p53-independent cell-cycle control gene *SEN6* in SV40-transformed human cell with cells from tumors containing SV40 DNA sequences.

The sponsors of the workshop were reassured by the independent epidemiologic surveys in the United States and Sweden that the incidence of neoplastic diseases in persons exposed to SV40 in viral vaccines has not increased. However, the sponsors should take the following steps to resolve questions raised by human exposure to SV40 in viral vaccines prepared in rhesus monkey cells in the 1950s and 1960s: 1) Form working groups to a) analyze the sensitivity and specificity of PCR reactions for detecting SV40 DNA in human tissues and develop standardized conditions to ensure confidence in the data generated by such reactions; b) develop methods for assessing the specificity of human polyomavirus neutralizing antibodies in plaque neutralization assays and consider other assays that can measure antibodies to virus-specific epitopes on the virions of polyomavirus; and c) develop ways to search for SV40 in the environment. 2) Encourage additional attempts to isolate SV40 from human tissues and increase the number of completely sequenced SV40 chromosomes obtained from SV40 field isolates. 3) Develop standardized reagents and make them available to laboratories who wish to assess the sensitivity and reliability of their PCR assay for detecting SV40 DNA. 4) Identify reagents such as archived tumor specimens, serum specimens and databases useful for epidemiologic evaluations, and any other specimens critical for evaluating when SV40 or SV40-like viruses entered the population.

For information about transcripts and audio and video recordings of the workshop, contact the Food and Drug Administration Freedom of Information Staff HFI-35, Rm. 12A-16, 5600 Fishers Lane, Rockville, MD 20857; phone: 310-443-1813. The proceedings of the workshop will be published in *Developments in Biological Standardization*.

### References

1. Bersagel DJ, Finegold MJ, Butel J, Kupsky WJ, Garca R. DNA sequences similar to those of simian virus 40 in ependymomas and choroid plexus tumors of childhood. *N Eng J Med* 1992;326:988-93.
2. Carbone M, Pass HI, Rizzo P, Marinetti M, Di Muzio M, Mew DJY, et al. Simian virus 40-like DNA sequences in human pleural mesothelioma. *Oncogene* 1994;9:1781-90.
3. Woloschak M, Ai Qin Y, Kalmon DP. Detection of polyomaviral DNA sequences in normal and adenomatous human pituitary tissues using the polymerase chain reaction. *Cancer* 1995;76:490-6.
4. Cristaudo A, Vivaldi A, Sensales G, Guglielmi G, Ciancia E, Elisei R, et al. Molecular biology studies on mesotheliomas tumor samples: preliminary data on H-Ras, p21 and SV40. *J Environ Pathol Toxicol Oncol* 1995;14:29-34.
5. Carbone M, Rizzo P, Procopio A, Guiliano M, Pass HI, Gebhardt MC, et al. SV40-like sequences in human bone tumors. *Oncogene* 1996;13:527-35.
6. Pepper C, Jasani B, Navabi H, Wynfodr-Thomas D, Gibbs A. Simian virus 40 large T antigen (SV40LTAg) primer specific DNA amplification in human pleural mesotheliomas tissue. *Thorax* 1996;51:1074-6.
7. Martini F, Iaccheri L, Lazzarin L, Carinci P, Corallini A, Gerosa M, et al. SV40 early region and large T antigen in human brain tumors, peripheral blood cells and sperm fluids from healthy individuals. *Cancer Res* 1996;56:4820-5.
8. Lednecky JA, Garcea RL, Bergsagel DJ, Butel J. Natural simian virus 40 strains are present in human choroid plexus and ependymoma tumors. *Virology* 1995;212:710-7.
9. Strickler H, Goedert JJ, Flemming M, Travis WD, Williams AE, Rabkin CS, et al. Simian virus 40 and pleural mesothelioma in humans. *Cancer Epidemiology, Biomarkers and Prevention* 1996;5:473-5.
10. Lewis AM Jr. Experience with SV40 and adenovirus-SV40 hybrids. In: Hellman A, Oxman MN, Pollock R, editors. *Biohazards in Biological Research*. Cold Spring Harbor (NY): Cold Spring Harbor Laboratory, 1973:96-113.
11. Shah K, Nathanson N. Human exposure to SV40: review and comment. *Am J Epidemiol* 1976;103:1-12.
12. Geissler E, Konzer P, Scherneck S, Zimmerman W. Sera collected before introduction of contaminated polio vaccine contain antibodies against SV40. *Acta Virol* 1985;29:420-3.
13. Brown P, Tsai T, Gajdusek C. Seroepidemiology of human papovaviruses. Discovery of virgin populations and some unusual patterns of antibody prevalence among remote peoples of the world. *Am J Epidemiol* 1975;102:331-40.
14. Brown P, Morris JA. Serologic response to BK virus following human infection with SV40. *Proc Soc Exp Biol Med* 1976;152:130-1.
15. Penny JB, Narayan O. Studies of the antigenic relationships of the new human papovaviruses by electron microscopy agglutination. *Infect Immun* 1973;8:299-300.

### Conference on Foodborne Pathogens: Implications and Control

More than 400 food protection and public health professionals from 18 countries, including microbiologists, epidemiologists, physicians, and health policy makers in industry, academia, and government, attended the Conference on Foodborne Pathogens: Implications and Control. The conference participants examined the response of the food industry and its related public health/food safety regulatory agencies to the emergence of new microbiologic threats and to the reemergence of known pathogens in previously unimplicated foods. The 3-day conference was held in Alexandria, Virginia, USA, March 24-26, 1997. It was organized by the International Life Sciences Institute North American (ILSI N.A.), the Centers for Disease Control and Prevention, the U.S. Department of Agriculture, and the U.S. Food and Drug Administration, in cooperation with the Food and Agriculture Organization and the Pan American Health Organization.

The specific goals of the conference were to identify factors that foster the emergence/reemergence and dissemination of foodborne microbial hazards, explore scientific and food safety strategies to identify and address these hazards, determine future research needs, and review the lessons learned and knowledge gained concerning the emergence and dissemination of food-related microbial threats to health.

The rapid emergence and dissemination of microbial foodborne pathogens and human diseases is affected by factors related to the pathogens themselves, their hosts, and the food production and consumption environment. The conference explored the role of the rapid mutation of foodborne pathogens such as *Escherichia* and *Salmonella*; the increasing numbers of susceptible persons; the effect of current livestock production practices, produce handling and food processing practices, and aquaculture; and changes in consumer lifestyles and food preferences.

Identifying and anticipating new foodborne microbial hazards require concerted efforts. The changing epidemiology of foodborne disease calls for improved surveillance including rapid subtyping methods, cluster identification, and collaborative epidemiologic investigation (including case-control studies). Also examined was the

need for better integrated, coordinated, and standardized animal disease surveillance and health monitoring programs. Several speakers stressed the importance of risk assessment (a component of overall risk analysis that combines science and policy) as a decision support tool and the need to effectively communicate risk to consumers. Because microbes do not respect national borders, they need to be addressed at a global level through strengthened infrastructure and standardized trade that will ensure the health of consumers.

The new problems of foodborne disease require new control and prevention strategies (as well as further research) to ensure that food in both domestic and international trade is safe. The development of the Hazard Analysis Critical Control Point (HACCP) process was presented as a first step toward an analytic process for identifying hazards and their points of control. Other research needs in the area of foodborne pathogen control were also examined. Topics included a need for multidisciplinary teams that can provide "just in time" research; for basic research to explain factors associated with food production and processing that contribute to new foodborne microbial threats; for prompt evaluation and implementation of innovative preservation methods (e.g., food irradiation) to meet consumer demand for fresh foods; for a centralized system accessible electronically, with information on pathogenic organisms in a standardized format; for the use of emerging molecular methods (e.g., DNA hybridization and polymerase chain reaction) to examine emerging viral and parasitic foodborne disease organisms; and for models to predict the probability of a particular microbial event (e.g., growth and death), which may be useful in the design of HACCP programs and in defining processes, formulations, and storage conditions to yield foods with acceptable shelf life and safety characteristics.

Lessons learned from outbreaks in the last 15 years contribute to developing strategies for the mobilization of resources to respond rapidly to emerging foodborne microbial hazards. Retrospective analyses of data from cases of *E. coli* O157 infections identified risk factors, variations in treatment, and estimates of the incidence of hemolytic uremic syndrome. Unusual foods have been associated with outbreaks of *Clostridium botulinum*, including potatoes baked in

aluminum foil, bean dip, cheese sauce, and mascarpone cheese; nontoxigenic clostridia could emerge as a new pathogen with the transfer of botulism toxin genes. The multistate outbreak of *Salmonella* serotype *enteritidis* underscored the value of molecular subtyping and public health action based on epidemiologic data in identifying outbreak cases when dispersed in a larger group of unrelated infections. Finally, epidemiologic data were presented from a multistate outbreak of *Cyclospora* infection associated with consumption of raspberries from Guatemala. These examples emphasized that the future of foodborne disease epidemiology will involve new technology and greater coordination among local, state, and federal public health and regulatory agencies.

Papers from this conference will be published in the Emerging Infectious Diseases journal.

### International Conference on Emerging Infectious Diseases in the Pacific Rim, Bangkok, Thailand

Approximately 200 participants gathered in Bangkok, Thailand, March 6-8, 1997, to discuss issues related to emerging infectious diseases in the Pacific Rim. The meeting was organized under the auspices of the U.S.-Japan Cooperative Medical Science Program. Scientists from the United States, Japan, the host country, and 15 other nations of the region, as well as from the World Health Organization (WHO) attended. The meeting focused on research topics relevant to emerging diseases and discussed surveillance and disease prevention. Formal presentations focused on themes of special interest to the region: enterohemorrhagic *Escherichia coli* (EHEC), dengue and dengue hemorrhagic fever (DHF), and the growing problem of antimicrobial resistance. Summaries of the WHO global and regional plans to address emerging infectious diseases were presented along with summaries from participating countries of their national plans and problems relevant to these diseases.

The session on EHEC included presentations on the status of this important pathogen in the United States, Japan, Australia, and Thailand, as well as a summary of recent efforts to develop better strategies to detect, treat, and prevent EHEC illness. Presentations on dengue included

a discussion of atypical infections and brief mention of new results regarding the stability of dried whole blood samples for serologic examination and the use of insecticide-impregnated screens to control vector mosquitoes; the prospective clinical study of DHF under way in Bangkok, carried out as a multicenter collaborative study involving Thai, United States, and Japanese scientists, was described. Presentations on nosocomial and community-acquired resistant infections, acute respiratory infections, and tuberculosis underlined the growing problem of antimicrobial resistance.

Several themes emerged from country reports: the growing importance of dengue fever/DHF and Japanese encephalitis in many countries of the region; increasing problems with diarrheal diseases and other food or waterborne diseases, including cholera; antimicrobial resistance and the need for assistance in laboratory culturing and sensitivity testing; the need for regional surveillance to better define the current patterns of antimicrobial resistance and for the establishment of regional quality control and proficiency testing as one aspect of the regional response; frustration with existing surveillance systems and need for assistance in developing improved surveillance tools and easier information sharing; the need for improved laboratory support, especially the regional availability of high quality diagnostic reagents and development of regional reference facilities; and the desire for a regional approach to addressing emerging infectious diseases.

The meeting concluded with recognition of the need for both greater research in the areas of the epidemiology, diagnosis, treatment, and prevention of EHEC and other Shiga-toxin producing organisms; further studies on DHF, including pathogenesis, clinical intervention, viral genetic variability, and genomic analysis; vaccine development; and improved vector control and fundamental strengthening of public health practices to address emerging infectious diseases including improved laboratory capacity, better surveillance programs, easier and more open communications and information sharing, and assistance in outbreak responses. Participants highlighted the need for greater training opportunities for scientists of the region and for development of regional reference facilities and

centers of excellence. The meeting did not cover human immunodeficiency virus and AIDS, although there was clear recognition of its importance within the region.

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### **International Conference on Emerging Zoonotic Infectious Diseases, Taipei, Taiwan**

The International Conference on Emerging Zoonotic Infectious Diseases, cosponsored by the Taiwan Departments of Health and Defense and the Centers for Disease Control and Prevention, was held March 1-4, 1997 in Taipei, Taiwan. The conference brought together scientists from Australia, France, the United States, and Taiwan and highlighted local work on dengue, Japanese encephalitis, plague, and rodentborne hantaviral infections.

The opening session outlined current efforts in the United States and internationally to improve and coordinate surveillance, laboratory diagnosis, and research of emerging infectious diseases. An example of a disease (yellow fever) whose threat has not been realized was described and reassessed in the context of globalization and other factors favoring and mitigating against the virus' dissemination. Although the possibility of epidemic yellow fever in Asia is small, it is important to reduce the disease at its sources in Africa and South America to further minimize this possibility. Ongoing efforts to elucidate the pathogenesis of dengue hemorrhagic fever, a growing problem in Taiwan and a leading cause of childhood illness and death in Asia and the tropics were summarized. Recent studies in Thai children have defined early clinical immunologic markers that differentiate febrile patients who contract dengue hemorrhagic fever from those with self-limited dengue fever; these findings suggest potential approaches to early recognition and specific intervention.

A session on viral hemorrhagic fevers reviewed recent Ebola virus outbreaks and the

discovery of a rapidly growing number of arenavirus and hantaviruses, their phylogeny and associations, and their specific rodent hosts. The virtual explosion of viruses identified in rodent reservoirs has left studies of their biologic, clinical, and epidemiologic correlates lagging; many of the newly discovered agents are orphan viruses. A report of local rodent surveys showed the presence of several hantaviruses in numerous species in Taiwan; human disease has not been recognized but epidemiologic studies are planned to define the spectrum and incidence of human infection. Approaches toward producing recombinant hantavirus vaccines and efforts to produce naked DNA vaccines for related vectorborne infections were reviewed.

Summaries of the recent emergence of dengue and dengue hemorrhagic fever globally and on Taiwan led to a series of talks on dengue vaccine development. Various approaches were discussed, including candidate live attenuated vaccines, purified inactivated and recombinant subunit antigens, and infectious clone-derived viruses and their engineered chimeras. A similar session focused on Japanese encephalitis (JE), its changing ecology and epidemiology on Taiwan and regionally in Australia, the molecular taxonomy of JE viruses, and recent developments in producing much needed rapid diagnostic kits. The cellular and molecular basis of JE pathogenesis was addressed in a series of reports on the protective role of *bcl-2* in viral-induced apoptotic death, viral inhibitory activity of cell derived NO<sub>2</sub>, and viral genetic determinants of virulence and attenuation. Alternatives to the only internationally accepted JE vaccine, the relatively reactogenic and expensive inactivated mouse brain-derived vaccine, were discussed, including the live-attenuated SA14-14-2 vaccine produced in China, a Vero cell-derived inactivated vaccine under development in Taiwan, and a chimeric JE vaccine engineered upon a yellow fever 17D virus infectious clone.

The final session concerned plague; it described the history and current status of plague globally and on Taiwan; reviewed new developments in the molecular taxonomy of *Yersinia pestis*; compared the performance characteristics of various serologic and PCR-based diagnostic tests; and described plague pathogenesis and vaccine development. F1 and V antigens were defined as important virulence factors in mouse and primate parenteral and

aerosol challenge models. Preliminary studies indicate their promise as constituents of a recombinant subunit vaccine.

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### The 4th International Conference on Hantaviruses, Atlanta, Georgia March 5-7, 1998

The Centers for Disease Control and Prevention in Atlanta and cosponsors will host the 4th International Conference on Hantaviruses to allow exchange of scientific information on hantaviruses in the areas of epidemiology, clinical management, ecology, molecular biology, laboratory diagnostics, pathogenesis, drugs, and vaccine development.

The meeting will host plenary sessions with invited speakers as well as oral and poster sessions based on accepted abstracts.

Deadline for abstract submission is October 31, 1997. For more information, call 404-639-1510.

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### International Conference on Emerging Infectious Diseases, Atlanta, Georgia, March 8-12, 1998

#### Preliminary Information and Call for Abstracts

The Centers for Disease Control and Prevention (and other cosponsors) will convene a conference to 1) encourage the exchange of scientific and public health information on global emerging infectious disease issues, 2) highlight programs and activities that address emerging infectious disease threats, 3) identify program gaps, 4) increase emerging infectious disease awareness in the public health and scientific communities, and 5) enhance partnerships in addressing emerging infectious diseases.

The meeting will host plenary sessions and symposia with invited speakers as well as oral and poster sessions based on accepted abstracts. Major topics will include current work on the surveillance, epidemiology, research, and prevention of emerging infectious diseases as well as on emergency preparedness and response. Abstracts should address new, reemerging, or drug resistant infectious diseases that affect human health, e.g., foodborne, tropical, sexually transmitted, and respiratory diseases; diseases transmitted by animals and arthropods or acquired in health care settings; diseases in infants and children, immunodeficient persons, and minority and other populations at risk; and diseases related to blood safety and xenotransplantation.

Conference attendance will be limited to 2,500 participants. Deadline for abstract submission is October 31, 1997. Proceedings of the conference will be published in the *Emerging Infectious Diseases* journal.

For additional information on registration and abstract submission send an e-mail message to [meetinginfo@asmusa.org](mailto:meetinginfo@asmusa.org) or call 202-942-9248.

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### **Emerging Infectious Diseases Laboratory Fellowship Program: Recruitment Begins for a Third Class of Fellows**

The Emerging Infectious Diseases Advanced Laboratory Training Fellowship, a 1-year program designed for bachelors or master level scientists, emphasizes the practical application of technologies, methods, and practices related to emerging infectious diseases. Fellows participate in a core curriculum session at the Centers for Disease Control and Prevention (CDC)/Atlanta to gain a general understanding of the public health laboratory system and how it relates to infectious disease surveillance, prevention, research, and control. Fellows are placed within federal and state public health laboratories to receive advanced infectious disease laboratory-related training tailored to each fellow's areas of interest, high-priority laboratory personnel needs, and host laboratory capabilities.

The Emerging Infectious Diseases Post-Doctoral Laboratory Research Fellowship, a 2-year program designed for doctoral level (Ph.D., M.D., D.V.M.) scientists, awards fellowships for the conduct of research or development in infectious diseases areas relevant to public health. This program's fellows also participate in the core curriculum session at CDC/Atlanta and are then placed within federal and state public health laboratories to conduct approved research.

For further information and application materials, contact EID Laboratory Fellowship Program, ASTPHLD, 1211 Connecticut Avenue, N.W., Suite 608, Washington, D.C. 20036, phone: 202-822-5227, fax: 202-887-5098. Fellowship application deadline: June 12, 1997.

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### **Emerging Infections: Clinical and Pathologic Update II**

This year, the Armed Forces Institute of Pathology course on infectious disease (November 8-11, 1997) will be held in collaboration with Emory University School of Medicine and the Centers for Disease Control and Prevention. The course, which will be held at the Emory University Conference Center, will be directed by Drs. Ann Marie Nelson and C. Robert Horsburgh, Jr. and will focus on newly emerging and reemerging diseases (including yellow fever, dengue hemorrhagic fever, leptospirosis, AIDS, bovine spongiform encephalopathy, cholera, diphtheria, tuberculosis, *Mycobacterium avium* complex, chancroid, meningitis, *Escherichia coli* O157:H7, fungal infections, malaria, babesiosis, filariasis, and emerging infections in captive wildlife). The course will also cover antibiotic resistance and the role of zoonotic infections and will feature a roundtable discussion of emerging infectious disease issues. The epidemiology and clinical features, as well as the pathology and pathogenesis of each disease, will be presented by experts in emerging infectious disease. An optional slide review session of 10 hours is available the day following the lecture series. (Approximately 30 CME credits)

For further information, contact the Department of Education Services, 14th and Alaska Ave., NW, Washington D.C. 20306-6000;

telephone (202) 782-5021, toll free (800) 577-3749 (U.S. only); fax (202) 782-7164 DSN 662-5021.

### Rabies Conference

The 8th Annual Rabies Conference in the Americas will take place in Kingston, Ontario, Canada, November 2-6, 1997. For more information, contact Dr. Christopher Nunan, Ontario Ministry of Natural Resources, 300 Water Street, P.O. Box 7000, Peterborough, Ontario, Canada, K9J8M5; telephone (705) 775-1554; e-mail: nunanc@gov.on.ca or Dr. Rolly Tinline, Director, GIS Center, Queens University, Kingston, Ontario, Canada, K7L3N6; telephone (613) 545-6039; e-mail: tinliner@qucdn.queensu.ca.

### Erratum: Vol. 3, No. 1

In the article "Exudative Pharyngitis Possibly Due to *Corynebacterium pseudodiphtheriticum*, a New Challenge in the Differential Diagnosis of Diphtheria," by H.S. Izurieta, P.M. Strebel, T. Youngblood, D.G. Hollis, and T. Popovic on page 68, the following were omitted from the list of references:

45. Feery BJ, Forsell P, Gulasekharam J. Streptococcal sore throat in general practice—a controlled study. *Med J Aust* 1976;1:898-91.
46. Clarridge JE. When, why and how far should coryneforms be identified? *Clin Microbiol Newslett* 1986;8:32-4.

We apologize to our readers for this error.