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Report on the NIH SARS meeting, 30th May 2003

Dr. Kirsten St. George, University of Pittsburgh Medical Center

In November of 2002 a severe respiratory disease now known as severe acute respiratory syndrome (SARS) emerged in the Guangdong Province of China. By March 2003 it had developed into a world-wide outbreak and on 30th May 2003 the National Institutes of Health (NIH) hosted a meeting on this new disease at the NIH Bethesda campus.

In his brief opening address, Tony Fauci (NIH) commented on the then-latest statistics of the epidemic. At the time of this writing, these have increased by only a few percent to 8,439 cases in 30 countries with 812 deaths. The epidemic is now considered "contained" world-wide, with no new cases reported to the World Health Organization since June 15. Fauci went on to list the objectives of the meeting, specifically: 1) to develop a national research agenda for SARS, 2) to identify gaps in the scientific understanding of the disease and its causative agent, and 3) to identify logistical and scientific barriers to the development of an effective intervention.

Epidemiology

The first plenary speaker, Klaus Stöhr (WHO), reviewed the current state of the knowledge of the epidemiology of SARS. Serological surveys in Hong Kong had found 340 sera prior to the SARS epidemic and 200 sera collected in April of 2003, to all be negative for SARS antibodies. Likewise 400 pre-SARS sera from blood donors in the USA were seronegative for SARS antibodies.

Additionally, the testing of 365 stored nasopharyngeal swabs from symptomatic patients in Canada prior to the SARS outbreak all tested negative for the SARS coronavirus (CoV). Moreover, a retrospective analysis of 800 atypical pneumonia cases in Guangdong province from 1999 to 2002, revealed no temporal or spatial clustering; a finding consistent with these being caused by endemic agents and not SARS. Collectively, all of the above findings strongly support the belief that SARS is a newly emerged, rather than a newly recognized, disease.

Klaus Stöhr went on to report that the **fatality rate for SARS was running at about 15% with the highest rate (more than 50%) in patients over 60 years of age.** The incubation period usually ranged from two to ten days, with isolated reports as long as 14 days, and a median of five days. It is not yet known if the route of infection affects the incubation period. Respiratory specimens have been found to remain positive by PCR for as long as 32 days post onset of symptoms, while stool specimens had been reported to remain positive for more than 40 days. The spectrum of disease is still incompletely understood but there have been cases of seroconversion, SARS CoV isolation, and RNA detection, in the absence of disease. There have also been a few reported cases of RNA detection without seroconversion.

There is no strong evidence of significant transmission prior to symptomatic disease or after

resolution of clinical symptoms, and few cases of transmission during the early prodrome. **In general SARS infection is most communicable during the very ill symptomatic phase of disease** and even then, while transmission has certainly been reported following casual social contact, it generally requires close physical contact. There have also been no reports of vertical transmission at this time. The primary mode of transmission is by mucous membrane contact with infected respiratory droplets and contamination of materials with secretions of body fluids is a risk. Despite the well publicized outbreak at the Amoy Gardens apartment complex in Hong Kong, the role of fecal/oral transmission is still uncertain and there is no evidence of food or water borne transmission.

The SARS CoV has been isolated from civets, raccoon dogs and badgers in animal markets in the Guangdong Province of China, and this is believed to be the source of the virus. However, while numerous animals had been tested as potential animal models for disease studies the only one that is working thus far is non-human primates.

Etiology

Malik Peiris (Hong Kong) then followed with a presentation on the etiology of SARS. While consensus PCR, low-stringency PCR and various other molecular tools were tried, the virus was in fact found by classic isolation in cell lines. The virus was then inoculated into Macaques which subsequently developed pneumonia, and the SARS CoV was detected in the animals' lung tissues, satisfying Koch's postulates as the causative agent of disease. In early laboratory studies with diagnostic tests, 92% of designated SARS cases and 3% of "other respiratory non-SARS" were found to show evidence of seroconversion for SARS antibodies, while only 63% of SARS cases were positive for SARS RNA by PCR. However, it should be remembered that this was using the very early PCR assays and there is no strong data on time of collection, type of specimen collected, storage procedures or extraction method used.

Peiris noted that the viral load is lower in the first five days of symptomatic disease, peaking in the stool for example at about day 10 to 15 post-onset of symptoms. This is markedly different to most other severe acute respiratory infections such as influenza where the virus titer in respiratory samples is very high in the early days of disease. The unusual pattern presents

additional challenges for the development of sensitive diagnostic assays. The virus itself is more stable in the environment than most other respiratory viruses, surviving on dry surfaces for one to two days at room temperature. In common with most viruses, the stability and survival can be greatly increased by the presence of organic matter, and is in keeping with findings from studies of animal CoVs which remain infectious for long periods of time in the environment. Peiris further commented that molecular epidemiological studies on various available isolates had indicated that the outbreak originated from a single viral clone.

Clinical Experience

Allison McGeer reported on the SARS clinical experience in Toronto, initially commenting that it was nice to come to work that day without having to wear a mask. SARS infections had been transmitted in Toronto in hospitals to patients, visitors and health care workers (HCW), in physician offices, within households and between family members, but uncommonly in "community settings". **Approximately 2% of cases had been contracted during travel, 24% in the home, and 77% to HCW.** The median age of cases was 42 with a slightly higher incidence among females. Patients presenting to their primary care physician (PCP) generally presented at about day three to four post onset of symptoms, while those presenting at the Emergency Room (ER) were likely to be suffering from more severe symptoms and to be five to ten days post-onset.

She gave graphic examples of the downstream cases from some patients, which had resulted in the theory of super spreaders. **As she progressed through the slides pertaining to this part of the presentation, and stick figures representing cases started to jump exponentially, there were audible gasps from the auditorium.** It quickly became clear why the outbreak in Toronto had caused such drastic action as the closure of hospitals. McGeer also reported on the very large variation in disease symptomatology as seen in the outbreaks in Toronto compared with Hong Kong. While fever for example was an almost universal symptom at both locations, the incidence of cough varied from 29% to 57%, headaches from 15% to 56%, dizziness from 4% to 43% and coryza from 3% to 22%. Thus it was difficult to come up with a good case definition of the disease.

Among standard laboratory tests, approximately 70% of patients showed lymphopenia, 78% elevated LDH, and 45% were

found to be thrombocytopenic. Less frequent were low white cell count (33%), elevated creatinine kinase (32%) and elevated liver function tests (23%). Of those patients presenting to an emergency room 20 to 25% were ultimately admitted to an ICU and 12 to 15% required ventilation. The disease severity peaks at about 13 to 16 days after onset of symptoms – a much more slowly progressive illness than most other severe respiratory viruses – and in fatal cases it may be four, five or even six weeks before the patient dies. **Notably, infected young children show much milder disease symptoms while teenagers exhibit more severe adult-like manifestations.** By the time of this meeting they had already discontinued using Ribavirin for SARS in Toronto and were noticing late post-recovery sequelae. These included neurological symptoms such as memory loss but it was unclear as to whether this was caused by virus in the central nervous system or steroid therapy.

Viral Characteristics

Kathryn Holmes reviewed the basic virology and pathogenesis of CoVs noting some unusual differences between SARS and other members of this virus family. For example, other human CoVs do not replicate in vitro as well as SARS, the SARS CoV has not yet been successfully cultured in human cells in vitro whereas non-SARS human respiratory coronaviruses 229E and OC43 only grow in primary human cells, and animal CoVs usually infect only the cells of the affected organ and do not go systemic. With a positive sense ssRNA genome of approximately 30,000 bases, CoVs are the largest of the RNA viruses but considerably smaller than many DNA viruses.

The enveloped CoV particle contains a number of proteins including the spike (S), transmembrane glycoprotein (M), and scaffolding glycoprotein (E), all embedded in the envelope. Inside the envelope the nucleocapsid (N) protects the genomic RNA in a helical structure. The envelope of CoVs may or may not also contain a hemagglutinin-esterase protein (HE); in the case of the SARS CoV this is not present.

The replication cycle of the virus causes the production of multiple copies of subgenomic positive sense RNA strands with a common 3' end. Since the N gene is located at this 3' end the replication results in the presence of many more copies of this gene inside an infected cell than any other of the viral genes. The genome organization of the SARS CoV has been described in detail by Paul Rota et al in Science (300, 30 May 2003).

There are three main groups of coronaviruses: the non-SARS human respiratory CoVs 229E and OC43 are members of groups one and two respectively. Due to the extensive sequence variation in every gene in the viral genome, it has been suggested that the SARS CoV should be placed in a new fourth group. The phylogenetic analysis of these gene sequences is also described in the above article by Rota et al. Holmes listed the potential targets for SARS treatment as: vaccines against the S protein, receptor blocking agents, fusion inhibitors, protease inhibitors, and inhibitors of the assembly of the polymerase complex or the assembly or budding of the viral particle. She also cautioned that there had been very variable experiences across different animal CoVs with vaccines: some protect well, some protect poorly, while others result in exacerbation of disease severity.

Molecular Biology

Mark Denison opened his presentation on the molecular biology and genetics of coronaviruses with the comment that the **total cost of all NIH-funded research on CoVs in the last 20 years was approximately the same as what the SARS epidemic was now costing on a daily basis.** His purpose was to highlight the importance of basic research and brought many smiles and a small round of applause from the audience. He went on to explain that the genomic structure of the SARS CoV is very close to that of the mouse hepatitis CoV (MHV) – a virus that he has spent about 20 years studying – and argues that it is possibly better placed as a variant group 2 rather than creating the new group 4 for its taxonomic assignment.

He reviewed some of the general themes of CoV infections that are seen in animal CoV diseases, including a high rate of recurrent or repeated infections, prolonged periods of viral shedding from infected hosts, a high mutation rate in the CoV polymerase (1/10,000), the ability to undergo homologous recombination, rapid adaptation, recovery and virulence, and a high tolerance in the genome for deletions, mutations and substitutions. While all this would predict a dramatically variable genome in the SARS virus, the isolates that have been studied thus far in fact do not show particularly extensive genetic differences. So while there is a theoretical ability for this to occur, it appears that the SARS virus may in fact have a relatively low tolerance for the in vivo survival of substantial numbers of mutations.

During the panel discussion that followed the morning sessions, it was noted that many animal CoVs are super spreaders all of the time, so in fact the human race were rather fortunate that this did not turn out to be the case with the SARS virus. At this stage there is no equivalent in the animal CoVs that have been studied, to explain the “occasional super spreader” phenomenon that seems to occur with SARS in humans. It was also noted that SARS seems to behave more like a systemic disease with a later severe respiratory manifestation, similar to Q fever and measles, rather than a respiratory disease. Also, in animal models again, live attenuated vaccines have generally been found to be better than killed vaccines so presumable T cell immunity is important for protection.

Diagnosis

The afternoon breakout session on SARS diagnostics opened with a presentation by William Bellini (CDC). He outlined the methods that have been used to detect the SARS virus including electron microscopy on sections of bronchoalveolar lavage cells, where SARS CoV was seen inside the cells, and viral isolation on Vero cells where SARS produces small plaques with refractile cells at the edge. They had also performed immunohistochemistry for the detection of viral antigens in infected tissues and were hoping to improve the sensitivity of IFA and EIA methods for the detection of viral antigen in respiratory secretions.

SARS CoV RNA had been detected by reverse transcription PCR assays in respiratory secretions, stool specimens, blood, urine and tissue samples including lung and kidney. For the detection of viral nucleic acids with molecular amplification assays, the pol region is the most highly conserved and at the time of the meeting there was already one commercially available kit based on the RT-Taqman PCR assay developed at the Bernhard Nocht Institute in Hamburg, Germany. However, the N gene was present in greatest abundance in infected cells because of the subgenomic transcripts, although pol region assays must always be used for viral load studies.

Most of the assays developed were able to detect down to 10 to 100 copies, and the CDC at this time had numerous primer/probe sets for the pol, M, N, and non-coding 3' regions of the SARS genome. Bellini noted that convalescent serum should be collected more than 21 days post-onset of symptoms (95% of patients seroconvert by 3 weeks) and up to 30 days. Other human CoV

antisera do not cross react with SARS in either IFA or ELISA assays and tests of normal populations have not revealed any positives. The sensitivity of serological assays may be improved with better antigen preparations and in urgent need of development are IgM capture assays, monoclonal antibodies for antigen detection in respiratory secretions and tissues, and recombinant proteins for serology assays.

A representative from the FDA who was present at the diagnostics breakout session commented that ASRs are not required by the FDA to have sensitivity and specificity data – this is the responsibility of the testing laboratory that is using them. There are more than 1,000 human gene tests available and only six of them have ever been submitted to the FDA.

The serology test for SARS antibodies is IDV-exempt because it requires a convalescent specimen and therefore is considered to be being performed retrospectively. However, the current molecular test-of-choice from the CDC is the three-target Taqman assay, in which two of the three targets are required to give a positive signal before the test is considered to be positive. The performance of this test requires prospective informed consent of the patient.

A number of laboratories, including the CDC, are using PCR to confirm culture positivity and the comment was made that it would be highly beneficial to have an antibody available that would enable the confirmation in a shell vial type system. However, the culture would still have to be performed in a BSL3 facility. It was also noted that the collection of nasopharyngeal aspirates is now considered far too dangerous and they have been banned in Hong Kong. For maximum sensitivity a nasopharyngeal and an oropharyngeal swab should be collected, and both swabs placed in the same vial of viral transport medium.

Conclusions

The meeting concluded with a plenary wrap-up session chaired by John La Montagne in which the breakout session co-chairs reviewed the discussions and conclusions that had been reached during the individual concurrent sessions that afternoon. This resulted in extensive lists of needed research and development in all areas, and highlighted the enormous lack of knowledge on human coronaviruses in general, and SARS in particular.

The recommendations for research in each area are too extensive to list here, and are

available on the meeting web site. The final unanswered question now that the epidemic has been declared over, is whether or not the disease will prove to be seasonal and return next winter, or whether it has been truly eliminated by the

stringent procedures put in place to contain it. In the opinion of the animal CoV experts, it will probably emerge again towards the end of this year.



Monkeypox Business in a Diagnostic Laboratory

Dr. Sue Kehl, Medical College of Wisconsin

On May 28, 2003, I was consulted by an Infectious Disease physician for a patient with fever >40C for 2 days duration, chills, sweats, tender lymph nodes and history of exposure to sick prairie dogs as well as other exotic animals. His wife had similar clinical symptoms and animal exposure. The patient had a nodular lesion on his right wrist near the site of a prairie dog scratch. Blood cultures and routine bacterial cultures of the lesion were performed. Antibody tests for *Francisella tularensis*, *Bartonella* sp., and *Yersinia pestis* were also ordered. Within three days, he developed worsening sweats, chills, and sore throat. He noted multiple, new lesions on his face and trunk. He was hospitalized and eventually transferred to Froedtert Memorial Lutheran Hospital.

May 30. A second Infectious Disease physician consulted me for a patient with tender lymph nodes and a vesicular lesion on his hand. He also gave a history of exposure to sick prairie dogs. Blood cultures as well as antibody tests for *Francisella tularensis*, *Bartonella* sp., and *Yersinia pestis* were also ordered. He returned for biopsy and bacterial cultures of the hand lesion and repeat blood cultures on June 2, 2003.

After several days, the I.D. physician and myself were commiserating over the negative culture and antibody findings. We had been hopeful that the cultures would have been positive for *Yersinia*, as their disease presentation fit well, except for the lesions. We joked that it was probably nothing more interesting than a *Staphylococcus* infection. Be careful what you wish for, because you just may get it...

June 4. The State Laboratory notified us that a Poxvirus, probably Orthopox, had been identified in skin lesions taken from a child in North Central Wisconsin who was exposed to sick prairie dogs. On the same day, another patient presented to our Emergency Department and was admitted with

fever, sore throat and widespread distribution of papules on the trunk and extremities. A fifth patient was also admitted that day with fever and several small erythematous papules. Both of these patients gave a history of exposure to prairie dogs. All hospitalized patients with skin rash and history of exposure to prairie dogs were placed in contact and airborne isolation.

June 5. We collected specimens from all hospitalized patients for submission to the CDC. We utilized our smallpox specimen collection kit and followed the CDC protocol for smallpox specimen collection guidelines (Guide D of the Smallpox Response Plan and Guidelines v3.0). We collected multiple specimens, including touch prep slides, swabs of derroofed vesicles in viral transport medium, vesicle roofs in sterile containers and in formalin, and punch biopsies in formalin, viral transport media and glutaraldehyde. In addition, the biopsy specimens were sent for histological examination, bacterial and viral culture. Two patients had produced sputum specimens and these were submitted for bacterial and viral cultures. At the time, identification of the potential orthopox virus was unknown; so all specimens were processed employing Biosafety Level 3 practices and facilities.

June 6. PCR testing performed at the CDC on these specimens revealed "monkeypox-like" orthopox virus DNA sequences. Monkeypox was first described in humans after the eradication of smallpox when it was discovered in Africa. It is a zoonotic disease with a clinical presentation very similar to smallpox. Laboratory testing is required to differentiate the diseases.

Monkeypox is a relatively large, brick-shaped virus in the orthopoxvirus genus. Orthopox viruses cannot be differentiated based on their electron microscopic appearance. Other members of the genus include Variola, Vaccinia, Cowpox, Buffalopox and Whitepox. PCR testing performed

at the CDC can differentiate among the viruses of the Orthopox genus. Orthopox viruses can be isolated in human and nonhuman primate cells. Human embryonic diploid cells, primary rhesus monkey kidney, and Vero cells are recommended.

Once the confirmation of monkeypox was received from the CDC, specimens were handled employing biosafety level 2 practices and facilities, as recommended in the HHS Publication Biosafety in Microbiological and Biomedical Laboratories (1). It is also recommended that all personnel working with monkeypox have documented evidence of smallpox vaccination within the preceding ten years. Since we had no lab personnel that had recently been vaccinated against smallpox, once CPE was demonstrated specimens were handled employing biosafety level 3 practices and facilities.

Viral cultures of the skin lesions were inoculated to Rhesus monkey kidney cells, and MRC-5 cells. The respiratory specimens were inoculated to RMK, MRC-5, and Hep-2 cells. Cells demonstrated CPE 48 – 72 hours after inoculation. (Figure 1 and 2). Cell fusion was seen with large plaque formation and cytoplasmic bridging. Because we were already aware that monkeypox had been identified by PCR, the cells from the skin lesions were scraped and sedimented and sent for electron microscopy. Electron microscopy demonstrated viral particles consistent with orthopox virus.

In a report of a monkeypox outbreak in the Democratic Republic of Congo (2), person-to-person transmission was reported among household members. Due to concerns regarding person-to-person transmission, a sign was posted at the entrance to the emergency department notifying individuals with fever, sweats, chills, and rash to wear a mask upon entry to the institution. Many of the exposed individuals seen at our institution had recovered from their illness, were not ill enough to require hospitalization or were being referred for specimen collection by the local health department. To facilitate specimen

collection while alleviating safety concerns from hospital employees, a “pox clinic” was held on multiple occasions.

A multi-room negative pressure area within the emergency department was utilized as a waiting room where patients were interviewed prior to specimen collection. An infectious disease physician, a dermatologist, an infection control practitioner, and laboratory staff were all present to facilitate patient registration and interview, and to obtain all of the appropriate specimens in proper transport media. If skin lesions were present, biopsy was performed. Biopsy specimens were placed in formalin, viral transport media and 2% glutaraldehyde. Whole blood and serum and throat swabs were also collected. Specimens were sent via courier to our State Laboratory of Hygiene for transport to the CDC.

A total of 32 patients were seen with the peak from June 12 through June 17. The connection between the patients, the pet prairie dogs and an exotic animal distributor was made early in the investigation (3). During the early part of the outbreak, daily conferences were held with individuals from the Wisconsin State Laboratory of Hygiene and the Wisconsin Division of Public Health to keep everyone informed on the outbreak. Public health issues as well as laboratory issues were discussed. Communication among physicians, laboratorians and public health officials was key to smooth handling, calm heads and timely dissemination of information.

- 1) Centers for Disease Control and Prevention and National Institutes of Health. 1999. Biosafety in Microbiological and Biomedical Laboratories. U.S. Government Printing Office, Washington D.C.
- 2) Hutin, Y.J.F., et al. 2001. Outbreak of human monkeypox, Democratic Republic of Congo, 1996-1997. *Emerging Infectious Diseases* 7(3):434-438.
- 3) Centers for Disease Control and Prevention. 2003. Multistate outbreak of Monkeypox – Illinois, Indiana and Wisconsin, 2003. *MMWR* 52(23):537-540.

Clinical Evaluation Of The OraQuick Rapid HIV-1 Antibody Test A.

Roberto, C. Starkey, S. Schindler, and B. Yen-Lieberman, Cleveland Clinic Foundation

Rapid HIV testing is becoming essential to help guide treatment decisions after needle sticks and other occupational exposures. Occasionally stat HIV testing is necessary for liver transplants and high risk pregnant women in labor, when

prompt antiretroviral therapy is needed. We currently use the Murex SUDS HIV-1 Test for rapid screening but find it to be very subjective in interpretation. We evaluated the OraQuick (OQ) Rapid HIV- 1 Antibody Test (OraSure

Technologies, Inc.) as a rapid test for post-exposure screening in our laboratory.

Methods: Forty-five serum samples previously characterized by our routine HIV-1 EIA test (Vironostika HIV-1 Microelisa System), and HIV-1 Western Blot (Organon Teknika) WB were selected. A collection loop is filled with serum and transferred to a vial of developer solution. The sample is stirred using the loop. A paddle like testing device is inserted into the vial of developer solution/serum mixture. Results are read from the paddle after 20 minutes. The device has a control area and a test area. The presence of a red line in the control area indicates a valid test result. A red line in the test area is read as a positive result and the absence of a line is negative. All discrepant results were repeated using our routine HIV-1 EIA. All HIV-1 WB Indeterminate results were repeated with the HIV-1 EIA test.

Results: There was 96% agreement between OQ and SUDS (43/45). There were 21 NEG, 22 POS and 2 discrepant by both methods. Of the 2 discrepant results Sample #1 was POS by OQ, WB, and EIA but NEG by SUDS. Sample #8 was NEG by WB, OQ and EIA but POS by SUDS. Thus, SUDS had 1 false NEG and 1 false POS in our study. Seven samples with Indeterminate WB results were NEG by OQ and SUDS. Repeat EIA on these samples were also NEG. All WB NEG were NEG by OQ and all WB POS were POS by OQ.

Conclusion: The OraQuick results concurred with standard HIV testing by EIA and WB. The OraQuick test was simple and quick with minimal hands on time of 2-3 minutes per sample. No equipment was needed. The results were easy to interpret.



John Alvin Stewart, M.D.
1934-2003

We are deeply saddened by the death of Dr. John A. Stewart on Friday, June 20. Dr. Stewart received his MD from the University of Rochester in 1961 and completed an internship and residency in pediatrics in Cleveland. He began his career at CDC in 1964 and was a public servant in the highest sense of the term. John Stewart lived an extraordinary and an extraordinarily well-lived life that touched many people directly, and many more indirectly. Dr. Stewart has been a member of the Pan-American Society for Clinical Virology since 1991, and was a frequent attendee and active participant at the Clinical Virology Symposium and Molecular Virology Workshop in Clearwater.

John was Chief of the Clinical Virology Section, Viral Exanthems and Herpesvirus Branch, Division of Viral and Rickettsial Diseases, NCID for 20 years of his tenure at CDC. His many accomplishments include a long and productive career studying diseases of childhood, including measles, rubella, chickenpox, cytomegalovirus

(CMV), and roseola. He coined the acronym TORCH (toxoplasmosis, rubella, CMV, and herpes), an expression recognized by virtually every pediatrician and family practitioner. TORCH represents a panel of neonatal infections that are difficult to discriminate on purely clinical grounds, especially in the days when diagnostic assays were scarce and crude. Dr. Stewart was a major contributor to the development and deployment of diagnostic methods for the identification of TORCH agents. He also expended considerable effort studying other herpesvirus infections, such as those caused by herpes simplex and Epstein-Barr viruses. He worked tirelessly for more than a decade to raise awareness both within and outside CDC for congenital CMV infection, a costly and ultimately preventable disease. More recently, he worked on improving our understanding of chronic fatigue syndrome. In addition, during his long career, John served as an author of more than 100

articles in the peer-reviewed literature and as an author of numerous book chapters.

That is a good, if cursory, summary of John's professional accomplishments, but it is not what his colleagues, friends, and family will be dwelling on over the days and weeks ahead. What most of us will be talking about is that John Stewart was one of the kindest, warmest, and most selfless human beings we have ever had the privilege and pleasure to know. He was a treasured friend who invariably joined his words with action. John had a wonderful sense of humor and a hearty laugh. John was also a man of deep faith who lived his beliefs with neither judgmentalism nor

proselytization. He was deeply and actively committed to his church and played a significant role in establishing a new church in Gwinnett County. So John's professional accomplishments were considerable, but of at least equal importance was the way in which he performed them—ethically, and always with the interests of others ahead of his own.

Dr. Stewart is survived by his wife, Madeline, three sons and a daughter, and fifteen grandchildren. He will be greatly missed. Submitted by Scott Schmid and Phil Pellet

Report on the 19th Annual Clinical Virology Symposium

Attendance - The 19th Annual Clinical Symposium was held in Clearwater from April 28 – May 1. This meeting was the most successful to date, with the highest number of attendees, abstracts, and exhibits. There were 721 registrants for the meeting. This included participants from 43 different states, 6 Canadian Provinces and 10 other countries. There were 80 non-US participants of which 39 were Canadian. The symposium had 166 abstracts and 61 commercial exhibitors, and 2 additional companies providing financial support. The Molecular Workshop had 265 participants.

Awards - Two of our colleagues were recognized at the awards banquet for their life-long work in virology. Dr. Robert Belshe was the recipient of the 2003 PASCV Clinical Virology Award. Dr. Belshe is a Professor of Infectious Diseases and Immunology at Saint Louis University. He is the Director of the NIAID funded Vaccine and Treatment Evaluation Unit at Saint Louis University. He chaired the NIH study evaluating the live attenuated influenza vaccine in young children, and his recent work includes re-evaluating the safety and immunogenicity of vaccinia and dilutions of vaccinia. Currently, he is study chair for the ongoing Herpevac Trial for Women, a gender specific HSV-2 vaccine designed to prevent genital herpes in women.

Dr Giuseppe Gerna graduated as a Medical Doctor in 1963 at the University of Pavia, Italy. He served there as a Professor of Virology from 1971 to 1986, then Professor of Clinical Virology until 1996. He was the Director of the Virus Laboratory, Institute of Infectious Diseases, University of Pavia till 1994, when he became the

Director of the Virology Diagnostic Service, IRCCS Policlinico San Matteo, Pavia. His scientific interests have included development of new methodologies for the diagnosis of viral infections, definition of new serotypes of human rotavirus and respiratory and enteric coronaviruses, monitoring HSZ infections and antiviral treatment in immunosuppressed patients, prenatal diagnosis of CMV and rubella virus infections, and the pathogenesis of CMV infection in infected fetuses and immunocompromised patients.

The Annual PASCV business meeting was held, and among the numerous items on the agenda was the presentation of seven Travel Awards for \$600, and three awards for \$1,000. Two of these were sponsored by Diagnostic Hybrids, Inc in memory of Dr. Edwin Lennette, and in Honor of Dr. Edith Hsuing. The final award was presented in memory of Dr. Mario Escobar to a recipient from Latin America.

Treasurer's Report - Dr. Ella Swierkosz presented the Secretary-Treasurer report. The PASCV balance as of December 31, 2001 was \$41,973. As of December 2003, this had increased to \$47,099. Most of the Society's income was generated from membership dues and the Molecular Virology Workshop. There are 369 PASCV members, of whom most are from the United States.

Website and Listserv Dr. Dave Myerson gave an update on the Website and Listserv. He has reserved the name www.pascv.org, which links automatically to www.virology.org. The Listserv has 187 individuals on it. All problems should be directed to dmyerson@fhcrc.org.

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Future Virology Meetings of Interest

20TH ANNUAL CLINICAL VIROLOGY SYMPOSIUM

The 20th Annual Clinical Virology Symposium and Annual Meeting of the Pan American Society for Clinical Virology will be held April 25-28, 2004 at the Hilton Clearwater Beach Resort, Clearwater Beach, FL. The Symposium will feature three plenary sessions, 3 poster presentations of submitted abstracts, two panel discussions, case presentations, informal sessions, exhibits and banquet presentations of the Diagnostic Virology Award (Sponsored by Becton Dickinson) and Clinical Virology Award (Sponsored by Bion Enterprises). A limited number of Travel Awards are available from the PASCV for students, fellows and technologists. Further information and registration materials are posted on the symposium website www.hsc.usf.edu/MEDMICRO/virology and PASCV website at www.virology.org. Information may also be obtained by calling 813 974-0897.

MOLECULAR VIROLOGY WORKSHOP

WHEN: April 23-24, 2004

WHERE: 20th Annual Clinical Virology Symposium and Annual Meeting of the PASCV; Hilton Clearwater Beach Resort, Clearwater, Florida 33767 Tel: (727) 461-3222.

COST: PASCV Members: \$110.00; PASCV Non-Members: \$125.00

ENROLLMENT: Enrollment is limited. Register early by mail or through the internet (<http://www.virology.org>)

FURTHER INFORMATION: Contact **Richard L. Hodinka, Ph.D.** (215) 590-2028, E-mail: hodinka@email.chop.edu, **Danny L. Wiedbrauk, Ph.D.** (734) 665-8300, E-mail: wiedbraukd@wardelab.com or **Curt Gleaves, M.S.** Tel: (503) 215 -6194, E-mail: curt_gleaves@phsor.org

THE FIRST INTERNATIONAL SYMPOSIUM ON CLINICAL VIROLOGY IN ARGENTINA

WHEN: November 5-8, 2003

WHERE: CEMIC Hospital Universitario, Buenos Aires, Argentina

COST: \$200 US

FURTHER INFORMATION: www.cemic.edu.ar

Contact Mrs. Paola e-mail - iuc-cursosposgrado@emic.edu.ar; Tel (5411) 4546-8272
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Scientific Program

SARS

Influenza

Congenital and Perinatal Infections

New Advances in Molecular Diagnosis

Viral Infections Transmitted by Food

Clinical Case Presentations

Epidemiology and Diagnosis of Respiratory Viruses

Viral Infections in Immunocompromised Patients

Viral Hepatitis

Emergent and Reemergent Viruses

HIV

Confirmed Speakers

From USA: Christine Ginocchio, Director Microbiology, North Shore LIJ Health System Labs; Richard Hodinka, Director Virology Laboratory, Children's Hospital of Philadelphia; Gregory Storch, St. Louis Children's Hospital; Steve Young, Technical Director, Tricore Reference Laboratories

From Canada: James Mahony, Director, Regional Virology Lab, St. Joseph's Hospital, Hamilton

From Latin America: Rita Nogueira, Fernando Motta (Brasil); Marcela Ferres, Luis Avendano, Pablo Vial (Chile); Guadalupe Guzman (Cuba); Vilma Savy (Argentina) and many other regional experts.

PAN AMERICAN SOCIETY FOR CLINICAL VIROLOGY MEMBERSHIP FORM

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