Glenn Morris has long been a frontline researcher in antibiotic resistance, and an eloquent commentator on this growing threat. Now chairman of the department of epidemiology and preventive medicine at the University of Maryland School of Medicine, he joined the school in 1982, after having served in the Centers for Disease Control’s enteric diseases branch. But his interest in emerging pathogens reaches back even further – to his childhood in Thailand, where his parents were missionaries. “I lived in Bangkok when the seventh pandemic of cholera was moving rapidly through Asia,” Morris says. “I became fascinated by the questions: Why do new pathogens emerge? What’s behind the dynamism of infectious diseases?”

Those questions became central in July 2002, when the CDC published the first documented report of Staphylococcus aureus resistant to the last line of defense antibiotic vancomycin. The organism was isolated from the dialysis catheter exit site of a Michigan man hospitalized with diabetes, peripheral vascular disease, and chronic renal failure. Perhaps of most interest to scientists, the isolate contained the vanA vancomycin resistance gene from enterococci, which had not previously been found in clinical isolates of S. aureus. Morris discussed the report on August 7, 2002 with Madeline Drexler, APUA newsletter associate editor.

Q: It seems we’ve waited a long time for the vanA gene to jump from VRE to staph aureus. What took so long?
A: It actually took longer for the first one to be identified than many of us had anticipated – which is encouraging, because it suggests that it’s a rare event. It’s been a decade since scientists were able to demonstrate it in the test tube.

Q: Around the same time that conjugal gene transfer was reported in the lab, weren’t your colleagues demonstrating that many hospital patients were co-colonized with VRE and MRSA?
A: Our major paper was in 1995, which was a summary of five years worth of work. It outlined the initial appearance of VRE, and emphasized that this was not just a simple nosocomial pathogen that was transiently introduced in the hospital, but that it was a very complex system and it was endemic. Much to our shock, we found that 20 percent of our hospitalized patients – all comers – had intestinal colonization with VRE.

One of the things we did early on was try to monitor the number of patients who had both MRSA and VRE. What we found was that there are an awful lot of patients who are dually colonized – so many that we stopped keeping track of them as a distinct group. We sensed that this was where we would start seeing VRSA strains. But interestingly enough, we didn’t – which suggested that in nature, while conjugation may occur, there was a sufficient reduction in fitness that it was not something that was selected for or that easily arose.
Q: Some people see this as a harbinger for a return to the preantibiotic age. Do you agree?
A: The emergence of vanco-resistant staph is clearly a major concern. On a national level, there are major warning signs that the Gram-positives are again becoming major players in resistance. At the same time, for many clinicians, on a day to day level, it’s the Gram-negatives that we lose sleep over. Some of the Pseudomonas strains are resistant to virtually everything, as are the E. coli, the Kbsiella, and other extended-spectrum beta-lactamase-producing strains.

Q: What’s the solution?
A: We’re going to have to start thinking beyond simple antimicrobial use. We need to look at vaccines, immunotherapy, phage therapy. Probiotics may have a great deal of promise, in terms of recolonizing patients with low-virulence strains.

Within the hospital we need to work on infection control, but the problem is that a lot of infection control does not have outstanding science behind it. We don’t understand what works and what doesn’t. And infection control measures, when practiced aggressively, tend to be very expensive.

Q: Will the appearance of VRSA spur action?
A: For scientists, it provides even stronger motivation to understand how resistance strains are transferred in the hospital, how they move around, how resistance strains are transferred in the national level, there are major warning signs that the Gram-positives are again becoming major players in resistance. At the same time, for many clinicians, on a day to day level, it’s the Gram-negatives that we lose sleep over. Some of the Pseudomonas strains are resistant to virtually everything, as are the E. coli, the Kbsiella, and other extended-spectrum beta-lactamase-producing strains.

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hospitals due to MRSA that exhibit susceptibility to all antistaphylococcal non-β-lactam antibiotics.

From January 1999 to August 2000, 211 MRSA isolates (51.2% of S. aureus isolates) were recovered from clinical infections of separate patients in Hippokration General Hospital, Thessaloniki, Greece. Sixty-five (30.8%) of the MRSA were susceptible to all antistaphylococcal non-β-lactam antibiotics (erythromycin, ciprofloxacin, gentamicin, tobramycin, tetracycline, clindamycin, rifampin, co-trimoxazole, nitrofurantoin, fusidic acid and vancomycin) (Table 1). An additional 47 (22.3%) of the MRSA isolates were susceptible to all the above antibiotics except the tested aminoglycosides. These 112 multi-susceptible isolates were recovered from sterile sites, judged to be clinically significant by microbiology staff. Patients were hospitalized in nine different departments, including special ICUs, medical, pediatric and surgery units.

A PCR test for the mecA gene was positive in all cases. The isolates had MICs for oxacillin ≥ 16 mg/l and were characterized by disk diffusion and population analysis as heterogeneously resistant. The ermA and aadD genes that are usually conserved within mec DNA and which encode resistance to macrolides and tobramycin, respectively, failed to be amplified in all cases. A PCR product for gene aacA-aphD was amplified only in the aminoglycoside-resistant isolates. Pulsed-field gel electrophoresis (PFGE) analysis showed that all isolates belonged to a unique genotype, which included two subtypes differing from each other by two bands. The prevalent subtype corresponded to the MRSA strain that was susceptible to all non-β-lactam antibiotics, while the other subtype included the aminoglycoside-resistant isolates.

MRSA susceptible to all non-β-lactam antibiotics have also been detected during the last two years in two other tertiary hospitals in northern Greece; they showed a clonal identity with those recovered in Hippokration Hospital, indicating the geographical spread of this clone. The PFGE pattern of the Greek genotype was distinct from those of the tobramycin-susceptible 'epidemic' MRSA ('EMRSA') strains isolated in the UK.9 The latter isolates generally retained resistance to some non-β-lactam antibiotics. The Greek strain differed also from other multi-susceptible MRSA clones that have appeared in other European regions,4,5,9 as well as from the previously reported major heterogeneous Greek MRSA clones.10

The strains of reported here had spread into different departments of a general hospital and infected several patients, the majority of whom were immunocompetent. This observation suggests a high degree of virulence in these strains, despite their susceptibility to a wide range of antimicrobials. It should be noted that the antibiotics commonly used in this as well as other Greek hospitals are β-lactam/β-lactamase inhibitor combinations, expanded spectrum cephalosporins, monobactams, carbapenems and glycopeptides (Table 2). The overuse of most of these drugs might have facilitated the spread of this MRSA strain. Also, the high consumption of aminoglycoside antibiotics in the above setting might have facilitated the appearance of the aminoglycoside-resistant subtype.

Multi-resistant MRSA have become a major nosocomial pathogen and usually their management requires the use of a glycopeptide.12 However, it is believed that the widespread use of vancomycin in regions with high proportions of MRSA may hasten the emergence of resistance in staphylococci and may also select for vancomycin-resistant enterococci. In view of the intra-hospital spread of 'new' multi-susceptible MRSA clones, we feel that additional therapeutic options are presented and vancomycin use could be considerably reduced. Antibiotics such as aminoglycosides, macrolides, lincosamides, fluoroquinolones or co-trimoxazole (Table 3), could be used to a greater extent against such strains, even for the treatment of severe infections.

References

Table 1: Non-β-lactam antibiotics effective against one of the MRSA strains of this study:
<table>
<thead>
<tr>
<th>generic name</th>
<th>group name</th>
</tr>
</thead>
<tbody>
<tr>
<td>erythromycin</td>
<td>macrolide</td>
</tr>
<tr>
<td>ciprofloxacin</td>
<td>quinolone</td>
</tr>
<tr>
<td>gentamicin</td>
<td>aminoglycoside</td>
</tr>
<tr>
<td>tobramycin</td>
<td>aminoglycoside</td>
</tr>
<tr>
<td>tetracycline</td>
<td>tetracycline</td>
</tr>
<tr>
<td>clindamycin</td>
<td>lincomamide</td>
</tr>
<tr>
<td>rifampin</td>
<td>rifampin</td>
</tr>
<tr>
<td>co-trimoxazole</td>
<td>sulphonamide</td>
</tr>
<tr>
<td>nitrofurantoin</td>
<td>miscellaneous</td>
</tr>
<tr>
<td>fusidic acid</td>
<td>miscellaneous</td>
</tr>
<tr>
<td>vancomycin</td>
<td>glycopeptide</td>
</tr>
</tbody>
</table>

Table 2: Commonly used antibiotics in Greek hospitals:
<table>
<thead>
<tr>
<th>group name</th>
</tr>
</thead>
<tbody>
<tr>
<td>β-lactam/β-lactamase inhibitors</td>
</tr>
<tr>
<td>(includes penicillins and methicillin)</td>
</tr>
<tr>
<td>cephalosporins</td>
</tr>
<tr>
<td>carbapenems</td>
</tr>
<tr>
<td>monobactams</td>
</tr>
<tr>
<td>glycopeptides</td>
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</tbody>
</table>

Table 3: Suggested antibiotics to use for MRSA infections:
<table>
<thead>
<tr>
<th>generic name</th>
</tr>
</thead>
<tbody>
<tr>
<td>aminoglycosides</td>
</tr>
<tr>
<td>macrolides</td>
</tr>
<tr>
<td>lincosamides</td>
</tr>
<tr>
<td>fluoroquinolones</td>
</tr>
<tr>
<td>co-trimoxazole</td>
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</tbody>
</table>
APUA Asks the Expert Clinician: What Constitutes Prudent Antibiotic Use in Burn Patients

Robert Sheridan MD, Chief of Burn Surgery, Shriners Hospital for Children, Co-Director, Adult Burn Unit, Massachusetts General Hospital, Clinical Director of Trauma, Massachusetts General Hospital, Associate Professor of Surgery, Harvard Medical School

Despite the wide availability of increasingly potent antibiotics, systemic infection remains the leading killer of those burn patients who survive burn shock but then die anyway. The relationship between host resistance and threatening organisms is a complex and dynamic one. Although antibiotics are important adjuncts with specific indications, they are not the principal tools to keep lethal systemic infection at bay, and their inappropriate use can be harmful. As patients with larger injuries are surviving with greater frequency, increased accuracy and prudence with antibiotic use is essential.

Burn Physiology
After an initial period of reduced perfusion (the “ebb phase”), successfully resuscitated burn patients enter a protracted period of hypermetabolism (the “flow phase”). This physiology is characterized by fever, increased catabolism, and a hyperdynamic circulation. These changes are also characteristic of sepsis and can make the uninfected burn patient appear septic, when in fact they may not be actually infected.

Another relevant aspect of burn physiology is immunosuppression that makes these patients more prone to infectious complications. This immune compromise is related to both local factors, such as the absence of skin and presence of invasive devices, as well as a poorly understood reduction in white cell function. This enhanced susceptibility to infection is an important consideration when managing patients with serious burns.

Infection Control
Prior to discussing indications for antibiotics in burn care, it is important to emphasize the essential role of infection control policies and universal precautions in this setting. Burn patients, especially those transferred between units, are often colonized or infected with resistant organisms that must be prevented from cross colonizing other patients. Only through rigid attention to infection control policies is this possible.

Prophylactic Antibiotics
Burn wound cellulitis is a virulent infection with Group A Streptococcus (GAS) and tends to occur in the first days after injury. Historically, prior to the era of early identification and excision of extensive deep wounds, GAS cellulitis was a major cause of death in burn patients. Consequently, for many years, prophylactic antibiotic treatment was routine, especially in children. However, a small study in adult patients suggested that such treatment might not decrease the incidence of burn wound cellulitis. This conclusion was supported by a much larger study in children which demonstrated prophylactic antibiotic treatment did not further reduce a low baseline incidence of GAS burn wound cellulitis. An interesting finding in this study was that children who were GAS carriers, or who had GAS recovered from wound cultures, had a substantial risk of developing subsequent GAS wound infections, supporting prophylactic treatment for this subgroup. Other than this, data do not support prophylactic systemic antibiotic treatment during burn resuscitation.

Prophylactic Antifungals
The incidence of systemic Candida infections has in the past prompted prophylactic administration of enteral antifungal agents, as the source of the infections was generally felt to be endogenous enteral organisms. However, this high incidence of Candida infection may have been at least in part an artifact of relatively uncontrolled use of systemic antibiotics. Other data do not support the use of prophylactic enteral antifungal agents.

Perioperative Antibiotics
Several studies have demonstrated that up to 40% of major burn wound manipulations are associated with transient bacteremia. Most operative events include major wound manipulations, and therefore prophylactic antibiotics targeted to the known wound flora seem reasonable, if only to reduce the incidence of secondary infection of intravascular devices. Interestingly, there is no data to support this practice, but short course (single dose on induction up to 24 hours of treatment) prophylactic antibiotic treatment for major burn excisions is common practice in burn programs. In small acute burn operations and reconstructive burn surgery, cases are generally considered clean-contaminated or clean and antibiotic prophylaxis, although common, is not routine in all programs. Limited data support the use of short course perioperative coverage for these cases.
Empiric Coverage
Burn patients are often febrile and hyperdynamic. They often develop various degrees of organ failures. It is frequently difficult to know if these signs are secondary to the physiologic changes associated with the wound, or to the development of a new infectious focus, to which burn patients are especially prone. The temptation is to treat liberally with broad spectrum antibiotics, but this is not in the patient’s best interest.

This common conundrum is best managed through a familiarity with burn patients in general and the individual patient in particular. If the team is familiar with burn physiology and has been closely following a particular individual, they will develop a sense of when changes are atypical for the individual, more suggestive of infection than of the usual hyperdynamic state. Thrombocytopenia, extreme leukocytosis or a new neutropenia, hypotension, or hypothermia raise concerns for a new infection. Other clinical signs suggesting infection are a new volume requirement, ileus, purulent sputum, or physical signs of wound sepsis. In these circumstances, it may be prudent to begin broad spectrum antibiotic treatment after obtaining cultures. After 72 hours, if cultures are not suspicious for infection, antibiotic therapy can be stopped. If infection is supported by cultures, broad spectrum antibiotics can be narrowed to recovered organisms and continued for a short therapeutic course.

It is important to ensure that all necessary surgical maneuvers have been effected. Infected wounds and necrotic tissue should be excised. Potentially infected vascular access devices should be changed. Purulent collections should be drained, as antibiotic therapies are generally ineffective in these circumstances if surgery has not been done.

Treatment of Specific Infections
Burn patients are prone to a host of unusual infections secondary to their immunocompromised state. It has been said that successful treatment of large burns requires prompt recognition and successful treatment of a series of infections while the wound is progressively closed. The most common of these are pulmonary (tracheobronchitis or pneumonia), intravascular (central venous catheter sepsis or suppurative thrombophlebitis), wound (invasive or non-invasive infection), or intraabdominal (most commonly cholecystitis).

Simple measures can markedly reduce the incidence of these infections. Tracheobronchitis and pneumonia can be reduced by aggressive pulmonary toilet. Central venous catheter sepsis and suppurative thrombophlebitis can be minimized by routine line changes and use of minimum caliber catheters. Wound sepsis should be infrequently seen if deep wounds are identified, excised and closed early and other wounds are treated with appropriate topicals and closely monitored. Cholecystitis can be minimized by early administration of enteral feedings.

However, when these and other infections occur, prompt treatment is essential and is greatly aided by focused use of appropriate antibiotics. Regular reassessment of the patient and a sensitivity to deviation from the expected clinical course will aid early identification of septic foci. Most of these infections can be successfully treated with a combination of surgical maneuvers and systemic antibiotics administered in a focused and specific way.

Conclusion
The use of nearly continuous antibiotics because of fear of infection, in the absence of clear signs of infection, is clearly detrimental to the patient, as it fosters the development of resistant bacterial strains that can be very difficult to treat if actual infection does eventually occur. In the current era, antibiotic use in burns should be thoughtful and prudent, and based on a clear idea of what is being treated.

Chicago Tribune Series
Hospital Infections
A three-part investigation of hospital infections ran in the Chicago Tribune (www.chicagotribune.com) July 21-23, shining light on what the report called “a hidden epidemic.” Written by staffer Michael J. Berens, the widely-cited series detailed the causes and costs of nosocomial infections. According to the Tribune, nearly three-quarters of hospital infections in the year 2000 – or about 75,000 cases in the U.S. – were preventable, “the result of unsanitary facilities, germ-laden instruments, unwashed hands and other lapses.” The series also traced the spread of antibiotic-resistant staph and other bacteria from hospitals into the community, noting that in 2001 at least 200 people in Illinois died after becoming infected with these drug-resistant microbes at home, at work, or during leisure activities. To calculate the rising rate of infection-related deaths, the newspaper analyzed records fragmented among 75 federal and state agencies, as well as internal hospital files, patient databases, court cases, and other public documents. The series noted that U.S. hospitals have cut infection control staffs by 20 percent in the past three years, and that cost-cutting has forced facilities to ignore the CDC’s recommendation of at least one infection-control professional for every 250 beds.

References
Q: Shouldn’t our government be bolder in tackling the resistance problem?
A: Government is going to have to take a much more aggressive role. NIH acts as if antimicrobial resistance is something the CDC should be taking care of. CDC doesn’t really fund much extramural research. The big drug companies have not been interested in looking at these issues because they’re interested in selling drugs. There’s a lot of lip service and government commission type stuff, but the reality is that not much is being done on a large-scale government level.

Q: How can APUA make a difference?
A: Keep doing what you’re doing. It’s a superb organization to provide and advocate for focused research and education, and funding for new interventions.

Q: After nearly 30 years in the field, how has your thinking about antibiotic resistance evolved?
A: When I started out as a physician, resistance was an irritant. When you talked about epidemics, you meant things like cholera or typhoid. Today, antibiotic resistance is, in many ways, a “quiet” epidemic; we do not see people dying in the streets, as occurred during the cholera epidemics of my childhood in Bangkok. But it is still very real, and there is an urgent need for us as physicians, scientists, and consumers to work together to slow the emergence and spread of these pathogens. Just as cholera was epidemic, so today is the explosion of new resistance types.

APUA News

Madeline Drexler has joined APUA as new associate editor of the APUA newsletter. Ms. Drexler is a science editor and medical journalist based in Boston. Her new book -- Secret Agents: The Menace of Emerging Infections -- was published to widespread praise in 2002, and will be issued in paperback in March 2003. A former medical columnist for The Boston Globe Magazine, she was a 1996-1997 Knight Science Journalism Fellow at the Massachusetts Institute of Technology. Her articles have appeared in The New York Times, The American Prospect, Self, Good Housekeeping, and many other national publications.

From August 12 to August 20, 2002, APUA was pleased to have as our guest, and consultant on the establishment of a chapter in sub-Saharan Africa, Iruka Okeke, Ph.D. An expert in the field of antibiotic resistance (esp. pathogenicity, epidemiology and antibiotic resistance of faecal Escherichia coli), Dr. Okeke received her Doctorate from Obafemi Awolowo University, Ile-Ife, Nigeria in 1998 and has since that time received numerous awards and honors. She is currently Assistant Professor of Biology at Haverford College, Pennsylvania, USA.

APUA Provides Testimony & Expertise

Recent Events
APUA has sponsored and provided speakers at a number of events since Vol 20 No 2 of the APUA newsletter, including:
- A meeting of project FAAIR Stakeholders held in Salt Lake City, UT on May 21, 2002.
- A provider lecture at the Nurse Practitioner Associates for Continuing Education (NPACE) Conference of the Older Adult in Falmouth, MA on July 11, 2002. David C. Hooper, MD gave the Presentation.
- The Third LIBRA Summit, July 14-16 in Germany. LIBRA is a research-based initiative developed by Bayer AG to foster the worldwide, responsible and appropriate use of antibiotics. Barbara A. Souder, PhD, Director of Public Policy, Domestic Programs and Public Relations, represented APUA.
- The National Foundation for Infectious Disease conference. APUA President, Stuart B. Levy, M.D. made two presentations; one, given on June 27, 2002, was entitled “Appropriate Antibacterial Drug Use: Current problems and future prospects”; the other, given on July 18, 2002, was a discussion of the community problem of antibiotic resistance: from threat to reality.
- A symposium, convened by APUA, at the American Veterinary Medical Association on the Global Consequences of Antibiotic Resistance. APUA Ecology Director, Stephen DeVincent, DVM, moderated and Stuart B. Levy, M.D. presented on the “Ecology of Antibiotic Resistance”.
- A symposium on “Strategies to Contain Antimicrobial Resistance” organized by the Government of Venezuela Ministry of Health officials, Dr. Esperanza Briceño and Dr. Adelaida Matos, with the assistance of the APUA-Venezuela chapter coordinator, Dr. Manuel Guzman. Dr. Anibal Sosa, Director of International Programs at APUA, presented on “Strategies to control AMR” and “Non-human use of antibiotics”.

Upcoming Events
APUA will exhibit jointly with Centers for Disease Control and Prevention at the 42nd Annual Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC) in San Diego from September 27-29, 2002. On the 28th, join other APUA friends and supporters at APUA’s Annual Reception, to be held at the San Diego Marriott Hotel & Marina (Headquarters Hotel for
ICAAC) from 6:30pm-8pm. On the 29th, the Steering Committee for the Global Advisory on Antibiotic Resistance Data will meet to discuss current GAARD surveillance projects and the preparation for future ones.

APUA will hold its annual Health Provider Evaluation Meeting during the meeting of the Infectious Disease Society of America (IDSA) in Chicago to analyze and refine APUA’s U.S. Health Provider Education Series. APUA and CDC will be exhibiting jointly at this meeting as well as at two events taking place in Boston: the American Academy of Pediatrics (October 18-22, 2002) and the Nurse Practitioner Associates for Continuing Education (NPACE) national conference (November 13-16, 2002). Richard Ellison, MD will lecture at the NPACE conference in Boston on November 15 at 10:30am on “Antibiotics for Community Acquired and Emergency Infections.”

Antibiotics and Agriculture: FAAIR

The APUA “FAAIR Report,” entitled “The Need to Improve Antimicrobial Use in Agriculture: Ecological and Human Health Consequences,” was published in full as Vol 34. Supplement 3, June 1, 2002 of the journal Clinical Infectious Diseases.

Selected conclusions were:
- All uses of antimicrobials in animals, agriculture and humans contribute to the global pool of antimicrobial resistance genes in the environment.
- Use of antimicrobials in food animals contributes to the growing problem of antimicrobial resistance in human infections. Transfer of bacteria from food animals to humans is a common occurrence.
- The amount of antimicrobials administered to animals is comparable to that used in humans. Unlike use in humans, however, much of the antimicrobial administration to food animals is to large groups at low doses, for non-therapeutic purposes such as growth promotion and disease prevention.
- The elimination of non-therapeutic use of antimicrobials in food animals and agriculture will lower the burden of antimicrobial resistance in the environment with consequent benefits to human and animal health.

Specific changes called for include:
- Antimicrobial agents should not be used in agriculture in the absence of disease.
- Use of antimicrobials for economic purposes such as growth promotion or to enhance feed efficiency should be discontinued (with the exception of ionophores and coccidiostats, because current evidence indicates that use of these antimicrobials does not affect resistance in human pathogens).
- Because of their critical importance to treat human disease, fluoroquinolones and third generation (or higher) cephalosporins should not be used in agriculture except to treat refractory infections in individual animals.

For more information see www.apua.org.

Grant for ROAR 2

APUA has received an award from NIH/NIAID to conduct the 5 year ROAR 2 project. This project is intended to investigate how commensal organisms can serve as reservoirs of resistance in pathogenic organisms. This is the first direct federal grant to APUA.

APUA International and Chapter News

APUA is pleased to welcome APUA-Taiwan as a new chapter.

APUA in Lusaka, Zambia

From April 8 to 12, 2002, as part of a collaborative effort on AMR with the Rational Pharmaceutical Management program/Management Science for Health, APUA went to Zambia to provide technical assistance to Lusaka Urban District Health Management Team on the development of strategies to deal with AMR.

These strategies consisted of a series of interventions including better use of standard treatment guidelines, improving surveillance efforts, design research plans, developing antibiotic national policy, usefulness of drug therapeutic committees and hospital antibiotic formulary, and training for medical practitioners.

APUA in the United Kingdom

Dr. Anibal Sosa, Director of the International Program at APUA, attended the Symposium Anti-infectives: the way forward at The Royal Pharmaceutical Society of Great Britain headquarters in London from Monday, July 8th to Tuesday July 9th, 2002. A new chapter is being formed under the auspices of Dr. Peter Davey, along with other key colleagues. More information will be available in the next edition of the APUA newsletter.
If you are concerned about the public health threat of antibiotic resistance, become part of the solution. Make a tax-deductible contribution and join our global network of citizens, clinicians, researchers and policy makers. For further information and to find out how, Visit www.APUA.org today!

The APUA Newsletter welcomes your feedback!
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- or -
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Founded in 1981 as a nonprofit organization, APUA is the only organization in the world solely dedicated to strengthening society’s defenses against infectious diseases through research and education on antibiotic use and antibiotic resistance. APUA’s mission is to improve infectious disease treatment and control worldwide through promoting appropriate antibiotic access and use and reducing antibiotic resistance. With members in over 100 countries and numerous foreign affiliated chapters, APUA provides a unique network to support country-based activities and facilitate international planning and communication. APUA’s resources include an international scientific advisory board with members of national academies of medicine and science and a professional staff with specialized expertise. APUA’s global network of affiliated chapters serves to tailor interventions to local customs and practices.