The role and scope of the Australian Society for Antimicrobials Newsletter were reviewed at the November 2008 ASA Committee Meeting. It was recognised that the desire to have thematic issues and, in particular, major articles was reducing the frequency of publication to 2-3 issues per year. This has been detracting from other important newsletter functions such as advertising upcoming ASA events.

It was decided that the ASA would attempt to produce a newsletter more frequently, and to drop the thematic issues. Major articles will still be accepted, but shorter mini-reviews (such as the MLST article in this edition) will also be solicited from local experts. In-depth journal reviews will still be published, but a shorter “in the journals” section is planned as well. Expect a return of the popular picture quiz in the next edition.

We hope you enjoy this first edition for 2009, and look forward to feedback on the new shorter format.

Hope to see you at Antimicrobials 2009 in Melbourne!

David Andresen,
ASA Newsletter Editor
Staphylococcal sepsis remains a significant healthcare problem worldwide, both in the community and in hospital practice. The current estimates of crude mortality are around 20%, with at least half of that considered to be attributable mortality.

To understand the local epidemiology of severe staphylococcal disease in Australia and New Zealand a local cooperative has been established; the Australian New Zealand Cooperative on Outcomes in Staphylococcal Sepsis (ANZCOSS) which operates under the auspices of the Australian Society for Antimicrobials (ASA).

Since May 2007, data has been submitted on-line on a continuous basis and this has included basic demographics, risk factors and outcome data on patients with S aureus sepsis seen at each participating site. By December 2008, over 3700 cases of staphylococcal sepsis had been collected from 26 Australian states/territories and 3 sites from New Zealand. To date the 7-day all-cause mortality is 9.7% and 30-day is 18.9%.

Data from prospective collections such as this provide essential tools for monitoring progress in the prevalence of serious staphylococcal infections and in detecting potential interventions that will result in improvements in outcomes.

ANZCOSS invites all institutions in Australia and New Zealand to participate.

For further information please contact ANZCOSS Co-ordinator John Turnidge (john.turnidge@cywhs.sa.gov.au) or the ANZCOSS Data Administrator Despina Kotsanas (despina.kotsanas@southernhealth.org.au).

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**ASANewsletter Contributions**

Submission of articles, material for the Picture Quiz, reviews for the journal club, or letters to the Editor should be sent to:

**Dr David Andresen**
ASA Newsletter Editor
email: info@asainc.net.au

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**ASA 2009 Foundation Members**

The ASA Committee wishes to acknowledge the following companies for becoming 2009 Foundation (Sustaining) Members of the Society

**PLATINUM**
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Foundation Members websites can be located in the Foundation (Sustaining) Members section of the ASA webpage
(http://www.asainc.net.au/foundation_membership/members)
1. Susceptibility Testing of VRE against pristinamycin and quinupristin/dalfopristin:

Dr. Peter Newton, a colleague in Wollongong, drew our attention to the conflicting results of CDS testing of VRE with pristinamycin (Pyostacine) and quinupristin/dalfopristin (Synercid). Both these agents are a combination of the same two streptogamins and the only difference is that in Synercid the pristinamycin components have been chemically modified to make them water soluble.

In the CDS laboratory we confirmed Peter’s observation that there were strains of VRE that yielded susceptible zones (> 4mms annular radius) with a pristinamycin 15mcg disc but appeared resistant with a quinupristin/dalfopristin 15mcg disc. We recalibrated the quinupristin/dalfopristin disc testing against the MIC and there was an excellent correlation between zone sizes and quantitative susceptibility. All susceptible strains as judged by the CDS method had a breakpoint of 2mg/L (< or = 2mg/L).

We were unsuccessful in our attempts to obtain information or pure substance from the suppliers of pristinamycin in France (of course!) and so we were unable to attempt calibration of this agent. Therefore we propose that quinupristin/dalfopristin 15mcg disc is used for surrogate testing of pristinamycin of all Gram positive organisms where it is appropriate to test.

2. CDS Workshop at ASM Annual Meeting Perth 2009

As usual there will be a CDS Workshop at ASM Scientific Meeting in Perth this year. All attendees at ASM are welcome including people from those laboratories that do not use the CDS Test. A large amount of the discussion is of relevance to susceptibility testing in general and not confined to the CDS. It would be appreciated if any prospective attendee has a particular item that they think would be of interest to others attending the workshop to get in touch with us in the CDS Laboratory (please see “Contact Us” on http://web.med.unsw.edu.au/cdstest/).

What’s New with the CDS

Professor Sydney Bell
The Antibiotic Reference Laboratory
Department of Microbiology
Prince of Wales Hospital
South Eastern Area Laboratory Services
Randwick NSW

What’s New at CLSI

Professor John Turnidge
Division of Laboratory Medicine
Women’s and Children’s Hospital
North Adelaide SA

“The only thing constant is change”

Reduced susceptibility to Vancomycin

The last few years has seen a considerable amount of work on reduced susceptibility to vancomycin in staphylococci. This is mainly a clinical problem in S. aureus. The vancomycin S breakpoint was lowered from 4 to 2 mg/L a couple of years ago, and BHI-vancomycin plates (previously recommended for VRE screening) were evaluated and recommended for staphylococci as well, knowing that the sensitivity and specificity of this screening test were suboptimal but better than nothing. There is a comment in M100 to that effect. The true recommended CLSI standard for vancomycin testing is an MIC method.

New in M100 in 2009: Removal of the vancomycin disk diffusion test for staphylococci. The rationale is that the disk test does not distinguish susceptible from intermediate strains of S. aureus and cannot distinguish S from I from R in coagulase-negative staphylococci. It will detect vanA-producing S. aureus because these give no zone of inhibition. Confusingly, teicoplanin disk testing has not been dropped; principally on the basis that there are no data to support this change. Rather it will come with a comment about the lack of recent data about its ability to differentiate between S, I and R.

Mupirocin resistance in Staphylococci

Mupirocin resistance in staphylococci is well documented and comes in two varieties: high-level (MIC >256 mg/L, encoded by the mupA gene) and low-level (MIC 4-256 mg/L). There is some evidence that strains with high-level resistance to mupirocin fail to respond to treatment. New in M100 in 2009: Introduction of methods to detect high-level resistance. No zone around a 200 µg disk or growth at 256 mg/L in a dilution test (only validated for broth microdilution). Positive control S. aureus BAA1708; negative control S. aureus ATCC 29213 or 25923

Cefoxitin for coagulase-negative staphylococci

Data on the efficacy of cefoxitin as an alternative to oxacillin for detecting mecA-positive coagulase-negative staphylococci have been reviewed. Unlike for S. aureus, cefoxitin is not superior to oxacillin and thus will not replace oxacillin in the short term.

Enterobacteriaceae

With the emergence in the US of KPC carbapenemases in Enterobacteriaceae, mainly K. pneumoniae, a considerable amount of work has gone into the development of methods to ensure that strains harbouring these enzymes are detected in the routine laboratory. This has been challenging, as some strains will test as susceptible to carbapenemases yet still contain the enzyme. Work is currently in progress to look at all the breakpoint criteria and data to see if the breakpoints for carbapenemases should be lowered. In the mean time, a reasonably robust method for detecting the presence of the enzyme in suspect cases has been developed. A so-called modified Hodge (cloverleaf) test works quite well. For interest, we don’t believe that KPC has been found in Australia yet, and is rare in the Asia-Pacific region (a few strains have been found in China in the SENTRY program).

New in M100 in 2009: Modified Hodge Test for KPC carbapenemase. A description and photos are provided in Appendix G of M100-S19 on how to perform this test to “confirm” the presence of KPC carbapenemases. The committee was on the cusp of approving the previously agreed changes in third- and fourth generation cephalosporin and aztreonam breakpoints, but chose to wait until data on disk zone diameter correlated could be reviewed (January 2009) as publication of the new breakpoints would have left some issues unresolved, especially as there is a proposal to remove cefepim from its list. Other changes in M100-S19, 2009

In the new version of M100 the MIC and zone diameter sections have been merged for the first time. In doing so, a few anomalies have been discovered, relating largely to the setting of zone diameter correlated so long ago that the breakpoints used to set them are no longer the same as the MIC correlates. These anomalies have been omitted from the new version, and will be corrected over time.

What else is new in 2009?

There are new versions of:

M2-A10 – disk diffusion test methods; mainly an update on details, not on method itself.

M7-AB – dilution test methods; mainly an update on details, not on method itself.

M39-A3 – analysis and presentation of cumulative susceptibility data (antibiograms); a new and refined version with significant improvements.

M44-A2 + S3 – disk diffusion testing method and breakpoints for yeasts; for those who wish to do this on a routine basis.

M23-A3 – development of breakpoints and QC values (released late 2008); for those who are interested, not needed in the routine lab.
Overview
There was no denying that the 2008 ICAAC/IDSA joint meeting in Washington, DC, was an impressive event, full of up-to-date clinical symposiums characteristic of IDSA meetings, blended with state of the art primary research presentations in basic and clinical science. Having not been to an IDSA meeting before, I found the joint meeting refreshing and very complimentary. Unfortunately, not all delegates were able to handle its sheer size and scope (see figure). One of the biggest highlights was the fantastic dinner organized by ASID for the Australians at ICAAC/IDSA and the great talk given by guest speaker William Hope. There is nothing like catching up with friends from home. (AP)

Opening Addresses
The landmark 2009 ICAAC/IDSA joint meeting in Washington DC was the first since the last back to back IDSA/ICAAC meeting in 2006. The context for the 2008 meeting was set by two comprehensive and inspiring opening addresses:

The Maxwell Finland Lecture was delivered by Anthony Fauci (Bethesda): “Global Health and Infectious Diseases: a look to the future”. Fauci discussed past, present and future considerations in global health where he argued that infectious diseases continue to drive global health; they are still the number one cause of death in children and account for 26% of all deaths. The complexities and disparities in disease treatment, control and impact according to a country’s level of income were discussed using HIV/AIDS as a case study. He highlighted the powerful lessons learned from the HIV epidemic (the need to garner political interest, engage new and established researchers, collaborate, engage patients etc) and the difficulties in taking these principles and practices to low and middle income countries. Fauci stepped through advances; our understanding of infection and immunity, point of care diagnoses, the contribution of new science and technologies to treatment and prevention, and vaccinology in particular our limited understanding of the principles and mechanisms that underlie microbial community structure.

The HIV story was continued in the Special Plenary Lecture delivered by Kevin De Cock “Global HIV Epidemic at the crossroads”. Whilst HIV drugs are credited with saving 300 million life years in the USA, the best estimates in sub-Saharan Africa remain much more depressing; the 2007 needs assessment in Kenya discovered that of those who were screened and found to be in need of ART, 35% were on ART, 2% were not on ART even though their status was known but, most alarmingly, 63% of those who required ART were not aware of their status and therefore were not receiving treatment. (CB)

Beta-lactams and Gram negative pathogens
A topic that is dear and near to my heart is Gram-negative pathogens. One of the most critical issues relating to these organisms is the lack of new antibiotics with novel mechanisms of action. Unfortunately, this issue does not look much brighter after this year’s ICAAC/IDSA meeting. The hot story from a not-so-hot bunch is a new monobactam called BAL30072. This monobactam has a siderophore side chain and appears to have affinity for the broadest range of penicillin-binding proteins of all monobactams (F1-1173). The excitement surrounding this drug relates to its in vitro activity toward non-fermenting Gram-negatives, especially Acinetobacter spp., Stenotrophomonas maltophilia, and Burkholderia cepacia (F1-1164, F1-1165, F1-1175). At 4 mg/L, BAL30072 had activity toward 86% of 200 carbapenem-resistant A. baumannii isolates, many carrying a range of OXA-type carbapenemases, as well as IMP-1 metallo-β-lactamases (MBL) (F1-1164). Unfortunately, the drug was not as active toward Pseudomonas spp., especially those with total derepression of an AmpC or the presence of a class A β-lactamase such as PER-1 (F1-1164). Despite this drug looking better than aztreonam toward resistant-Gram negatives, it is still a β-lactam with the same mechanism of action, and thus will likely succumb to the same resistance mechanisms. The next area that I maintain some hope for is the development of an inhibitor of MBLs. There were two poster presentations on ME-1071, a competitive MBL-inhibitor that appears to have activity toward VIM-, IMP-, GIM-, and SPM-type MBLs when combined with a range of carbapenems or ceftazidime (F1-1170, F1-1171). This is interesting as the different MBL-types have considerable differences in their active site architecture, which has been one of the hurdles in designing a pan-MBL inhibitor. These data are promising but are very preliminary and included few clinical isolates. (AP)
**Novel Gram Negative Therapeutic Classes**

Very few antibacterial agents with novel mechanisms of action toward Gram-negative pathogens are in the pipeline. However, two were presented that are worth mentioning. The first is a group of antimicrobial peptides known as protein epitope mimetics (PEMs), which have specific activity toward *P. aeruginosa* (F1-3992, F1-3995). Unlike other cationic peptides, these do not work by causing membrane lysis but rather, appear to act on membrane biogenesis. These peptides include POL7001 and POL7080, both being effective at low concentrations in a murine model of *P. aeruginosa* infection (F1-3992). Further development and studies of these compounds are clearly warranted. The second is a novel strategy to antibacterial treatment that I think we are going to see significantly more of; the targeting of virulence pathways. This late-breaker abstract presented data on a human monoclonal antibody targeting the quorum-sensing (QS) pathway of *P. aeruginosa* (B-069a). QS is an important component of virulence regulation. These investigators not only showed that the monoclonal antibody could significantly prolong the survival of mice after a lethal *P. aeruginosa* infection but also, could independently improve the efficacy of ciprofloxacin when treating an infection caused by a ciprofloxacin-resistant strain (17% survival with CIP alone vs 83% with CIP and the antibody). These data are exciting and are good examples of how we need to broaden our minds when thinking about the future management of highly drug resistant Gram-negative pathogens. (AP)

**Tuberculosis**

The few sessions on tuberculosis saw a number of interesting papers presented with promising results from phase I and II studies for new agents including the nitroimidazole PA 824. The highlight was a presentation of results from a pre-planned interim analysis for the phase II, placebo-controlled, double-blind study of the novel diaryquinolone TMC 207 in treatment of MDR-TB in combination with standard combination therapy. Results after 8 weeks of treatment showed 47.5% culture conversion in the 23 subjects in the experimental arm compared with 8.7% of the 24 subjects in the control group (P = 0.003). Despite the low rates of conversion in the control arm, the groups appeared to be well matched by demographic and disease related characteristics, and background combination therapy (modified by resistance patterns). Adverse events did not differ between groups. (KS)

**When to start HAART?**

The best time to initiate HAART remains a hot topic given the recent data from the SMART and CASCADE cohorts. Several presentations and/or posters documented better outcomes with early initiation (CD4 >350 -500) compared to deferred therapy (CD4 <350). More data are required, and several clinical trials are currently recruiting patients to address this issue. (SVH)

**New data on Integrase Inhibitors**

48 week data from the STARTMRK trial, a phase III trial comparing either EFV or raltegravir (RAL, the new integrase inhibitor) in combination with TDF/FTC were presented in one of the late breaker sessions. RAL met non-inferiority criteria with 86% of patients having an undetectable (viral load < 50copies/ml) compared to 82% of individuals in the EFV arm. CNS adverse events were more common in the EFV arm as were lipid changes (p<0.001). Malignancy rates were similar in both arms. If ongoing follow-up confirms these findings integrase inhibitors will probably become a treatment option in ART naive patients. (SVH)

**Nucleoside-sparing Regimens**

Several posters addressed nucleoside sparing regimes as alternative HIV treatment strategies. The final 96 week data (IMANI II) showed sustained virological suppression in 75% of patients (ITT analysis) taking LPV/RTV monotherapy. No failures occurred in the presence of primary PI mutations. Similar findings were reported from the OK04 trial, an induction, maintenance strategy (standard HAART with LPV/RTV, followed by PI monotherapy after viral suppression for >6months). In patients with virological failure PI resistance remained extremely rare (0.07 LPV/RTV mutations /100 patient years). In all patients with virological failure, suppression of viraemia was successfully achieved following re-initiation of standard HAART. (SVH)

**Parasitology**

Issues in parasitology included resistance testing (or the lack thereof), with the absence of validated testing methodologies highlighted in view of potential helminth mass treatment campaigns and the possible emergence of artesunate resistance in malaria. Rates of cutaneous leishmaniasis continue to increase in returning USA soldiers, with most treatment protocols using liposomal amphotericin. This is in contrast with WHO guidelines which recommend pentostam therapy. Reasons for these differences probably include lack of pentostam availability, and reluctance to use local therapy in the USA. Cure rates using very high doses of liposomal amphotericin (total doses of 18-21mg/kg) were reported, which are similar to those achievable with antimony compounds for old world disease. Failures (defined as ongoing lesions 3-6 months post initial therapy) were retreated with the same agent. Miltefosine therapy remains experimental in cutaneous disease with some preliminary data (especially in visceral disease) showing comparable response rates to antimony compounds in new world disease. (SVH)

**Clinical Mycology**

A session that I have thoroughly enjoyed in the past and that didn’t disappoint was the Top Ten Papers in Clinical Mycology. The highlight of this session is the conveners, especially John E. Bennett, MD, and the way he is able to entice the lead authors of presented papers to stand up and take questions. You also get the benefit of hearing comments from the opinion leaders in the field who can’t resist not getting up to the microphone and having their say. The stand out paper that was discussed was by Schuster MG et al. who performed a double-blind, placebo-controlled, randomized trial assessing the effect of empirical fluconazole in patients at high risk for invasive candidiasis in a surgical intensive care unit (Ann Intern Med 2008;149:93). Interestingly, the trial finished recruitment in 2000 and it has taken 8 years to publish! The authors commented that it was tough to get a negative study published. Anyway, the study included patients who were in the ICU for ≥ 96hrs, had a fever and were on antibiotics for ≥ 4 days, had a central line for ≥ 24hrs and an Apache score ≥ 16. 270 patients were randomized to receive 800mg daily of fluconazole or placebo for 2 weeks. Patients were followed for 30 days. Success was defined as a composite of; no invasive fungal infection, fever resolution, no therapy discontinuation due to toxicity, and no need for a non-study antifungal. Success was achieved in 36% of those who received fluconazole and 38% in those given placebo (P = 0.78). Documented invasive candidiasis occurred in 6 patients (5%) in the fluconazole group and 11 (9%) in the placebo group (P = 0.24). No cases of candidemia were documented in the fluconazole group and only 2 cases were observed in the placebo group. Much discussion of this study centered around the utility of a composite end point for antifungal treatment trials (ie resolution of fever probably not appropriate in this setting) and the very low number of invasive candidiasis cases observed in the study population, particularly the control group. The authors commented on using other risk factors to improve the selection of high risk patients such as candida colonization, recent abdominal surgery and renal impairment. Moreover, the authors and conveners commented on an ongoing study using β-glucan measurement in addition to clinical risk factors to try and improve selection of high risk patients who would actually benefit from empiric fluconazole in this setting. (AYP)

**Miscellaneous**

Other primary research highlights include the successful post-licensure results of the new rotavirus vaccines for children (G1-433, G1-437, G2-3748), the notable rates of oseltamivir resistance (11.3% of 913 strains) from H1N1 strains from the 2007-2008 US influenza season in patients with no previous exposure to the drug (V-918), and the possibility of artesunate resistance/tolerance in *Plasmodium falciparum* from Western Cambodia (3643). (AYP)
Multilocus sequence typing (MLST) has become the gold standard typing method for a wide range of common pathogens. The method evolved from multi-locus enzyme electrophoresis (MLEE) which measured the electrophoretic mobility of a number of housekeeping enzymes and equated mobility of variants with alleles at the underlying genetic locus. MLST determines alleles directly by nucleotide sequencing. Each allele is assigned a number and no weighting given for the number of nucleotide differences between alleles. Alleles may arise as the result of single or multiple point mutations or by recombination events. Discrimination of the method increases with the number loci interrogated. The use of seven loci has been shown to be highly discriminating.

The method involves bi-directional sequencing of 450-500 base pair internal fragments of seven housekeeping genes (essential metabolic genes from the core genome of the species in question) obtained by PCR using highly conserved primer pairs. Alleles are assigned a number by a database curator in order as they are registered and the allelic profile determines the sequence type (ST), once again assigned in numerical order (Figure 1). For example, the allelic profile 1-1-1-1-1-1-1 would be assigned ST1 and so on.

![Figure 1. Assignment of sequence types to profiles of alleles of 7 housekeeping genes (a-g)](image)

The great advantage of MLST over genetic ‘finger printing’ methods such as PFGE is the unambiguous assignment of alleles and STs, providing a universally applicable, reproducible and portable system of strain typing. New STs can be registered in Web accessible databases (http://www.mlst.net). MLST also allows inference of relatedness between STs. This is not done by proportional genetic similarity as with other typing methods but by creating a hierarchy of allelic similarity. Single locus variants (SLVs) are assumed to be most closely related followed by double locus variants (DLVs) and so on. Allelic profiles can be compared using the BURST (based upon related sequence types) program (http://www.mlst.net/BURST/burst.htm). Clusters of SLVs and DLVs are referred to as clonal complexes (CC). CCs are named for the ST with the greatest number of SLVs and this ST is assumed to be the ancestral genotype. An example of eBURST output is shown in Figure 2 which represents CC8 of *S. aureus*. The ancestral genotype or primary founder predicted by eBURST is ST8 represented by the blue circle. Subgroup founders are represented by yellow circles. The large ST239 subgroup is an important MRSA lineage (including AUS2/3).

![Figure 2. CC 8 of S. aureus as defined by eBURST](image)

MLST and eBUSRT have become powerful tools for defining bacterial populations structures. Figure 3, from the eBURST website, shows the relatively clonal *S. aureus* population structure compared with the aclonal *H. pylori* structure. As the housekeeping genes used in MLST are thought to evolve at a predictable rate relatively free from selective pressure, it is generally accepted that MLST is an excellent method for tracing the evolution of clones over extended periods. This relative stability could be a drawback for short term epidemiological studies and MLST is not quite as discriminatory as PFGE. However, this drawback can be overcome by the addition of assays interrogating markers in the variable genome.

![Figure 3. eBURST illustration of the population structures](image)

In conclusion, MLST has revolutionised bacterial typing by providing a universally applicable and unambiguous way of identifying bacterial clones. It is now deservedly the gold standard of typing methods.

**Further Reading**


Antimicrobials 2009:
ASA 10th Annual Scientific Meeting
26 – 28 February, 2009
Melbourne Hilton
www.antimicrobials2009.com

Focus on Fungal Infections
4 - 6 March, 2009
Florida, USA
http://www.imedex.com/appweb/announcements/a047-01.asp

Staphylococcus Symposium, 2009
11-14 March, 2009
Waikiki Beach, Hawaii
http://www.staph2009.com

International Forum on Quality and Safety in Health Care
17 – 20 March, 2009
Berlin, Germany
http://internationalforum.bmj.com/

7th International Symposium on Antimicrobial Agents and Resistance (ISAAR)
18 – 20 March, 2009
Bangkok, Thailand
http://www.idthai.org/conference/ISAAR2009/

American Society for Microbiology 109th General Meeting
17 – 21 May, 2009
Philadelphia, USA
www.asm.org

19th European Congress of Clinical Microbiology and Infectious Diseases (ECCMID)
16 – 19 May, 2009
Helsinki, Finland
http://www.escmid.org

Staphylococcus Symposium, 2009
11-14 March, 2009
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American Society for Microbiology 109th General Meeting
17 – 21 May, 2009
Philadelphia, USA
www.asm.org

27th Annual Meeting of the European Society for Paediatric Infectious Diseases (ESPID)
9 - 12 June, 2009
Brussels, Belgium
http://www.kenes.com/espid

26th International Congress of Chemotherapy & Infection (ICC)
18 – 21 June, 2009
Toronto, Canada
http://www.icc-09.com

FEMS 2009 – 3rd Congress of European Microbiologists
28 June – 2 July, 2009
Gothenburg, Sweden
http://www2.kenes.com/fems-microbiology

ASM 2009 – Golden Jubilee Annual Scientific Meeting
6-10 July, 2009
Perth, Western Australia

Introduction to Infectious Disease Modelling & Its Applications (Intensive Course)
6-17 July, 2009
London School of Hygiene and Tropical Medicine, UK
http://www.lshtm.ac.uk/prospectus/short/siidma.html

British Pharmaceutical Conference, 2009
6 - 9 September, 2009
Manchester, UK
http://www.bpc2009.org/
6th European Congress on Tropical Medicine & International Health
6 - 10 September, 2009
Verona, Italy
http://www.tropicalmed.eu/name/

CongressTropicalMedicine.html
49th Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC)
11 - 14 September, 2009
San Francisco, CA
http://www.asm.org

Legionella 2009
13 - 17 October, 2009
Institut Pasteur, Paris, France
http://www.pasteur.fr/infosci/conf/sb/legionella2009/

4th Trends in Medical Mycology
18 – 21 October, 2009
Athens, Greece
http://www.timm2009.org/

Infectious Diseases Society of America (IDSA) 47th Annual Meeting
29 October – 1 November, 2009
Philadelphia, PA
http://www.idsociety.org

American Society of Health-System Pharmacists: Midyear Clinical Meeting
6 – 10 December, 2009
Las Vegas, USA
http://www.ashp.org/

National Foundation for Infectious Diseases (NFID) Annual Conference on Antimicrobial Resistance
1 – 3 February, 2010
Bethesda, MD, USA
http://www.nfid.org/

14th International Congress on Infectious Diseases
9 -12 March, 2010
Miami, FL, USA
http://www.isid.org/14th_icid/index.shtml

Society for Healthcare Epidemiology of America (SHEA), 20th Annual Scientific Meeting
18 – 21 March, 2010
Atlanta, Georgia, USA
http://www.shea-online.org

20th European Congress of Clinical Microbiology and Infectious Diseases (ECCMID)
10 – 13 April, 2010
Vienna, Austria
http://www.escmid.org

16th Symposium on Infections in the Immunocompromised Host
27 – 30 June, 2010
Budapest, Hungary
http://www.ichs.org/budapest.htm

21st European Congress of Clinical Microbiology and Infectious Diseases (ECCMID)
7 - 10 May, 2011
Milan, Italy
http://www.escmid.org

Society for Hospital Pharmacists of Australia (SHPA) National Conference
5 - 8 November, 2009
Perth, WA
http://www.shpa.org.au

16th Symposium on Infections in the Immunocompromised Host
27 – 30 June, 2010
Budapest, Hungary
http://www.ichs.org/budapest.htm

ESCMID/SHEA Training Course in Hospital Epidemiology
7 - 11 November, 2009
Madrid area, Spain
http://www.escmid.org

4th Mycology Masterclass
29 October - 1 November, 2009
Hamilton Island, Queensland