Keeping MRSA in Check

Francis S. Mah, MD

Methicillin-resistant Staphylococcus aureus is a serious threat, but one that we are learning how to handle.

As physicians, we sometimes find ourselves in the position of needing to raise public awareness about important clinical threats and health hazards. That does not seem to be the case with methicillin-resistant Staphylococcus aureus (MRSA), however (Figure 1). Not only is the medical community keenly aware of its steady emergence, the press—in conjunction with doctors and other medical authorities—has done a commendable job of make the public aware of MRSA and other “superbugs.”

As a result, patients with MRSA infections, in my experience, tend to honor the seriousness of their infection and are appropriately compliant with treatment and follow up. However, clinicians still need to remain vigilant for MRSA as a potential cause of ocular infections and act accordingly.

MRSA Rates: What’s Happening?

Just glancing at the latest ocular infection surveillance data, one might be tempted to conclude that in recent years the rise of MRSA among ocular isolates has leveled off, following several decades of steady increase. Until recently, methicillin resistance patterns seemed to be (and may still be) echoing that of penicillin resistance in S. aureus in the 1950s through 1970s. Penicillin resistance arose in a series of stages: emergence of resistant strains shortly after the introduction of the antibiotic; rapid, steady rise in resistance with increased antibiotic use; hospital-acquired strains predominating initially, followed by the emergence and growth of community-acquired strains; and urgent calls to identify alternatives for treatment.1

Penicillin resistance, conferred by a penicillinase enzyme, became so widespread that today, penicillin and related beta-lactams such as ampicillin are considered universally inactive against staphylococci. It is too early to judge, but the latest Antibiotic Resistance Monitoring in Ocular Microorganisms (ARMOR) surveillance data suggest that, despite initial similarities, methicillin resistance rates may be behaving slightly differently: between 2009-2011...
and 2012, rates plateaued at around 40% of *S. aureus* and 45% of *S. epidermidis*.2

**Statistical Artifact?**

This interpretation may be a mirage, however. We should remember that, while ARMOR is a helpful surveillance tool, there are limits to its generalizability. First, ARMOR data reflect mean rates at participating sites; no matter how diverse the geography and type of institution sampled, there is no guarantee that rates are reflective of other hospitals and clinics. It is always advisable to follow local microbiologic trends and antibiotic patterns rather than rely on nationwide data. Secondly, since sample collection with ARMOR is not active surveillance, there is an opportunity for bias, for example toward samples that are easier to collect and transport. Thirdly, ARMOR was not designed to follow year-to-year trends in methicillin resistance; it was designed to follow susceptibility of ocular pathogens to ophthalmic agents. So we must resist any urge to overinterpret the ARMOR data.

That said, it is conceivable that some of our anti-MRSA strategies are working. One thing that may be helping is greater use of antibiotics that cover MRSA in appropriate circumstances, including later generation fluoroquinolones (specifically besifloxacin, gatifloxacin, and moxifloxacin) and certain generics (eg, sulfacetamide, bacitracin, and gentamicin). Reliance on older fluoroquinolones (including levofloxacin and ciprofloxacin), which are more active against methicillin susceptible *S. aureus* (MSSA) than MRSA) may in fact have been a driver of increasing MRSA emergence in the past.3 One study has shown that actively seeking to reduce use of early generation fluoroquinolones in hospitals lowers MRSA prevalence.4 I believe, too, that healthcare providers are practicing simple universal precautions with greater consistency out of concern for MRSA and other resistant pathogens. Although further improvements are warranted, programs to promote hand hygiene in hospitals

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**STATEMENT OF NEED**

Ophthalmologists face numerous challenges in optimizing their competencies and clinical practices in the realm of preventing, diagnosing, and treating ocular infections and their sequelae; these challenges include:

- The widespread "off-label" use of topical ocular antibiotic biotic to prevent and treat serious and sight-threatening infections—given the reality that the most widely used topical antibiotics in ophthalmology have FDA approvals restricted to bacterial conjunctivitis.
- The escalating levels of multi-drug resistance in common ocular pathogens.1
- The emergence and increasing prevalence of once-atypical infections that may require diagnostic and treatment techniques relatively unfamiliar to comprehensive ophthalmologists.1
- The introduction of new and potentially more efficacious and/or safe topical anti-infectives.1
- The introduction of new and potentially more accurate diagnostic techniques for ocular infections.4
- Widespread discussion over the efficacy and safety of novel or alternative delivery techniques and vehicles for prophylactic ophthalmic antibiotics (including but not limited to intracameral injection and topical mucosubstances).1,6
- Increased understanding of the inflammatory damage caused by ocular infections and the best ways to prevent/ alleviate the problem without fueling the growth of pathogenic organisms.2

Given the continually evolving challenges described above, *Topics in Ocular Antinfectives* aims to help ophthalmologists update outdated competencies and narrow gaps between actual and optimal clinical practices. As an ongoing resource, this series will support evidence-based and rational antinfective choices across a range of ophthalmic clinical situations.

**REFERENCES**


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**OFF-LABEL USE STATEMENT**

This work discusses off-label uses of anti-infective medications.

**GENERAL INFORMATION**

This CME activity is sponsored by the University of Florida College of Medicine and is supported by an unrestricted educational grant from Bausch + Lomb, Inc.

**Directions:** Select one answer to each question in the exam (questions 1–3) and in the evaluation (questions 11–16). The University of Florida College of Medicine designates this enduring material for a maximum of 1.0 AMA PRA Category 1 Credit™. There is no fee to participate in this activity. In order to receive CME credit, participants should read the report, and then take the posttest. A score of 80% is required to qualify for CME credit. Estimated time to complete the activity is 50 minutes. On completion, tear out or photocopy the answer sheet and send it to: University of Florida CME Office PO Box 100233, Gainesville, FL 32610-0233 Phone: 352-735-0064 Fax: 352-735-1007 Or you can take the test online at [http://cme.ufl.edu/ocular](http://cme.ufl.edu/ocular)

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**FACULTY AND DISCLOSURE STATEMENTS**

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**DISCLAIMER**

Participants have an implied responsibility to use the newly acquired information to enhance patient outcomes and professional development. The information presented in this activity is not meant to serve as a guideline for patient care. Procedures, medications, and other courses of diagnosis and treatment discussed or suggested in this activity should not be used by clinicians without evaluation of the patient's condition, contraindications, and other dangers in use, applicable manufacturer's product information, and comparison with recommendations of other authorities.

**COMMERCIAL SUPPORTERS**

This activity is sup-ported by an unrestricted educational grant from Bausch + Lomb, Inc.
have succeeded in reducing MRSA rates (Figure 2).³

Marking the charts of patients colonized with MRSA helps remind healthcare practitioners to take extra precautions to prevent spread within the clinic or hospital. Wearing gloves as a contact barrier when examining MRSA-colonized patients and cleaning rooms with a bactericidal agent such as bleach are advisable extra steps that may limit transmission.

FIGURE 2 Handwashing remains one of the key elements in the prevention of bacterial spread.

MRSA and Surgery

The relationship between MRSA colonization (on the eye or in the nares) and risk for infectious complications of ocular surgery is complex. A study from California identified factors associated with an increased probability of finding multidrug-resistant periocular flora among patients preparing for routine cataract surgery; these included diabetes, asthma, chronic blepharitis, active conjunctivitis, ocular discharge, immune, skin, or autoimmune disease; and taking immunosuppressant medication. In this series, the presence of drug-resistant flora was not associated with an increased risk for infectious complications of surgery, although the number of patients studied might have been too low to demonstrate a correlation.⁶

A separate multicenter study looked at a similar population—patients planning routine cataract surgery—and showed that risk for MRSA colonization of the ocular surface increased with advancing age.⁷ The authors speculated that increasing rates of methicillin-resistant commensals in older eyes might be one of the factors behind something that has been demonstrated in other studies: the risk of postoperative endophthalmitis increases with age.

Preventing Spread of MRSA Among Commensals

It is reasonable to hypothesize that identifying and eradicating MRSA carriage might prevent intrahospital MRSA spread and reduce the incidence of drug-resistant infections. However, questions related to this have not been definitively answered. These include: how widely to screen, what constitutes an effective decolonization method, what to do when decolonization efforts fail, and whether or not any of the recommended decolonization steps actually prevents disease.

While system-wide screening for MRSA upon hospital entry, as has been required by the United Kingdom Department of Health since 2006, is commendable in its ambition, it presents a unique constellation of dilemmas. First, surveys suggest that policy comprehension and conformity of application is mixed.⁸ Secondly, screening can result in treatment delays, causing significant detriment to care and outcomes.⁹ Thirdly, emerging mupirocin resistance among MRSA skin flora can interfere with decolonization.¹⁰ Lastly, it is not clear that the additional time and expense involved in MRSA screening and decolonization has the intended effect of curbing the incidence of MRSA-related disease. Thus, universal screening appears not to be the most efficient or effective means of targeting MRSA and cannot be recommended.

Preventing MRSA Infection

As mentioned, consistent hand washing between patients and opting for the most recent generation fluoroquinolone or other agent with acceptable MRSA coverage (eg, tetracycline, bacitracin, trimethoprim, or an aminoglycoside) over earlier generation fluoroquinolones for the empiric treatment of ocular infections are two mainstays of preventing MRSA spread and infection.¹¹ Local antibiograms should inform the choice of empiric therapy. Furthermore, prescribing antibiotic treatment for an adequate length of time is an important means for preventing the emergence of resistance.

Presurgical antisepsis with povidone-iodine is a critical and undisputed step for preventing endophthalmitis following intraocular surgery.¹² Concerns have been raised about the potential for transmission of MRSA and other pathogens on the felt-tip pens used for marking patients (on the eye or on skin) prior to surgery. There is in vitro evidence that MRSA does survive on a pen tip inoculated with the organism; in theory, pens could act as a fomite and source of infection for patients, although the clinical implications are not clear.¹³,¹⁴ Until further information is available, using a fresh pen with each patient, especially ones who are immune-suppressed, is probably prudent.¹⁵

There has been much discussion within the global ophthalmology community about the role of intracameral prophylaxis at the end of intraocular surgery, with European voices prominent among those arguing in favor of intracameral prophylaxis. In the context of MRSA, I think it is worth pointing out that, of the three agents most commonly used (in all cases off-label) for intracameral prophylaxis—vancomycin, moxifloxacin, and cefuroxime—only vancomycin and moxifloxacin show good activity against MRSA strains.

Cefuroxime, the only agent supported by very large multicenter prospective studies and the agent marketed in Europe specifically for intracameral use,
Exotic Ocular Infections: Beyond the Tropics

Sivakumar R. Rathinam, PhD

As climate change and global trade, travel, and migration help pathogens and their vectors find their way to supportive niches outside the tropics, US ophthalmologists have to become increasingly adept at diagnosing and managing exotic infections.

Infections due to exotic pathogens cause high levels of ocular morbidity worldwide, with prevalence increasing in formerly less affected regions. While most exotic pathogens prefer tropical and subtropical habitats, outbreaks of infections that can cause ocular disease—including West Nile virus (WNV), dengue fever, and others—now occur with increasing frequency in the US and Europe.

Globalization of trade, travel, and migration and a changing climate all contribute to the emergence of so-called tropical pathogens and their vectors in many nontropical regions. Other trends—including intensified farming, changes in landscape, and human encroachment on formerly untraveled areas—intensify disease emergence in wildlife and set the stage for spillover of animal pathogens into human populations.1

Travel and Transmission

Human travel allows pathogens endemic to one region to find their way to another. For example, WNV, a Flavivirus endemic in the Middle East, Asia, Africa, and Australia, is capable of causing systemic disease, neurologic impairment, and ocular complications, including chorioretinitis. The emergence of WNV in the US in 1999 is thought to have been the consequence of air

does not provide good coverage against MRSA.15 Nor have either vancomycin or moxifloxacin been proven to reduce endophthalmitis rates in prospective trials. Also, vancomycin, which provides excellent coverage against MRSA, cannot be recommended for prophylactic use, as it should be reserved for difficult-to-treat infections due to resistant organisms per the AAO and CDC.

One reasonable approach would be to use a 500 µg dose of intracameral moxifloxacin (specifically, the nonpreserved formulation), which would likely eradicate a reasonably high proportion (approximately 70%-80%) of MRSA strains. Vancomycin could then be used to treat endophthalmitis that might break through moxifloxacin prophylaxis.

Conclusion

MRSA remains among the most important ocular pathogens of our time, capable of causing superficial and deep infections. Keeping MRSA top of mind in the differential diagnosis of ocular infections and working to control its spread—via handwashing, antisepsis, and proper antimicrobial use—remain central to efforts to control it.

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REFERENCES

transport of an infected human (host), mosquito (vector), or bird (reservoir). Since 1999, over 41,000 cases of human WNV have been reported in the US, including over 18,000 cases of neuroinvasive disease and 1765 deaths.

What began as a regional outbreak of WNV (nearly all CDC-reported cases between 1999 and 2001 were in New York, New Jersey, or Connecticut) has since then surfaced in every state in the continental US.

When one considers the life cycle of WNV, it is easy to see how its widespread emergence is not only possible but likely. Humans with WNV may experience mild to severe symptoms or none at all. Thus, subclinically infected individuals who travel can serve as a source of infection to naive mosquitoes in a distant, previously uninfested area. Mosquitoes, in turn, can infect local birds. Similarly, infected mosquitoes or birds that migrate or are transported to a new region can also spread the disease. Thus, the zoonotic loop of viral transmission—from host to vector to reservoir and back to host—is perpetuated, and the virus may establish a new geographic niche.

**Climate Change**

Outbreaks of vector-borne infection occur when three critical factors converge: a parasite or pathogen, an arthropod vector (eg, a mosquito or a tick), and an animal reservoir or host. The host/reservoir may be a wild or domesticated animal—or even humans. Importantly, an environment suited to the pathogen and vector is also required. Weather can profoundly influence vector abundance, vector competence, and the efficiency of viral replication within the vector. Climate change sets the stage for a variety of complex environmental changes, including increased geographic distribution of pathogens and vectors previously confined to tropical and subtropical regions.

Dengue fever—the most common mosquito-borne viral infection in the world—has caused three outbreaks in the US since 2001, including a 2009-2011 outbreak in the Florida Keys. For the prior 60 years, dengue fever had not been seen in the continental US outside the Texas/Mexico border. Environmental factors conducive to proliferation of *Aedes aegypti* mosquitoes—likely related to climatic and other changes—are thought to have played a role. Index cases had not traveled outside the US. Individuals who reported mosquito bites, not using mosquito repellent, or who had plant-filled yards, birdbaths, or kept windows open rather than using air conditioning were more likely to become infected. In the Texas outbreak, standing water in abandoned manmade materials (tires or buckets) likely contributed to vector breeding.

Climate change has also been implicated in the emergence of ocular dirofilariasis, a helminthic disease transmitted by mosquitoes or black flies that can cause subconjunctival, eyelid, anterior chamber, and orbital disease. Treatment depends on removal of the parasite. Ocular dirofilariasis is being reported with increasing frequency in endemic countries—including Italy, France, Greece, and Croatia—but is also being reported in nonendemic countries, including India. Nonocular dirofilariasis has been reported in the US and Canada.

**Global Warming**

Predicting global warming’s influence on vector-borne infectious disease is a complex undertaking. Researchers have attempted to anticipate global distribution shifts in vector-borne viruses by correlating primary occurrence and climate data with future climate and ecological niche models. It is believed, for example, that two mosquito vectors—*Aedes aegypti* (which carries dengue fever, yellow fever, and Chikungunya) and *Aedes albopictus* (which carries Chikungunya virus)—will colonize new geographic niches, including broadened inhabitation of North America. Recent phylogenetic evidence suggests that US-established *Aedes* mosquito populations are capable of rapid evolutionary change (within 10 years) in the face of temperature-related selective pressure.

In addition to gradual shifts in temperature, climate change may cause increased levels of severe storms that can abruptly alter local ecosystems and contribute to the emergence of disease. This can be seen, for example, in the increasing emergence of leptospirosis in flood zones following typhoons and hurricanes.

With disease patterns gradually changing and the potential for outbreaks ever-present, eyecare providers will be helped by a working knowledge of key exotic pathogens that can infect the eye. Four such—WNV, chikungunya, dengue fever, and leptospirosis—are particularly relevant inside and outside the tropics.

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**CORE CONCEPTS**

- Human and animal migration, travel, and international transport of foods and other goods contribute to the spread of vectors and pathogens that cause eye disease
- Vector-borne diseases involve a pathogen that is passed back and forth among hosts (human and/or animal) by a vector (a tick or a mosquito)
- Climate plays a significant role in vector-borne disease transmission and establishment
- Human behaviors—including land use, animal contact, letting standing water accumulate, fresh water swimming, and pollution—influence disease risk
- Dengue fever, chikungunya, WNV, and other pathogens are carried by mosquitoes; they may cause retinitis or other ocular syndromes
- Leptospirosis may cause uveitis and cataracts
- Serologic and molecular testing can identify most pathogens
**West Nile Virus**

WNV is a member of the Japanese encephalitis complex and the most widely distributed of the encephalitic Flaviviruses. It is now found throughout Africa, Asia, Europe, and Australia and is emerging in locations in the Western hemisphere. WNV first came to the US in 1999 and continues to be reported. WNV is amplified and transmitted via virus-infected Culex mosquitoes (and other species) to birds, horses, humans, and other mammals in both urban and rural settings, typically in summer to early fall.

A majority of individuals who acquire WNV from a mosquito bite experience no symptoms. Approximately 20% experience a mild febrile illness. Perhaps one in 150 will experience neuroinvasive symptoms; among those, approximately 80% will experience ocular manifestations. Patients with WNV infection may complain of floaters or visual loss. The most typical ocular presentation is that of bilateral multifocal chorioretinitis; people over 50 and diabetics are at increased risk. WNV infection may also be associated with macular edema, optic neuritis, retinal hemorrhage, retinal pigment epithelium atrophy, or related findings.

In a more recent WNV outbreak, a large number of young, nondiabetic south Indian patients presented with acute febrile illness with severe arthralgia. Multifocal retinitis and neuroretinitis were more predominant than choroidal lesions. In these patients, the vitreous was clear without inflammatory cells, and the retinitis healed without scarring. Patients with retinal vascular occlusions had poor visual prognosis (Figure 1).

**Chikungunya**

An Alphavirus disease, chikungunya is one of three arthropod-borne viral pathogens that has managed to free itself from the typical zoonotic cycle involving reservoir vertebrates (eg, bats, birds, and dogs), establishing humans as its primary host. (Yellow fever and dengue fever are also nearly if not entirely exclusive to humans.) Chikungunya’s vector is the *Aedes* mosquito, widely distributed throughout the world, including most of the US. Over the centuries, chikungunya has tended to cause major outbreaks and then recede. Its recent reemergence in the Caribbean in 2013 affected over 300,000 individuals; spread to the Americas quickly followed. In 2014, 232 US cases of imported chikungunya were reported.

“Chikungunya” means to become contorted, which characterizes its typical presentation of fever, chills, and severe arthralgias that can persist well past the acute phase. Patients may also experience rash; epistaxis; headache; low back pain; hemorrhagic, neurologic, and ocular complications; and organ failure.

**Leptospirosis**

Leptospirosis, caused by a waterborne spirochete *Leptospira*, is among the most widespread zoonoses in the world; and its incidence is increasing. Proximity to its reservoirs—cattle, pigs, dogs, rodents—and soil and water contaminated by urine from these animals increases risk for leptospirosis infection. Leptospirosis rates tend to increase following hurricanes, typhoons, and earthquakes. Several hundred emergent cases in the US resulted in a return of leptospirosis to the Center for Disease Control’s reportable diseases list in 2013.

Leptospirosis is a nonspecific febrile illness characterized by headache, arthralgias, and fatigue. The spectrum of severity is wide: some cases are subclinical or mild, while others are complicated by multiorgan failure and are life-threatening. Nongranulomatous anterior or pan uveitis occurs in 40% of systemic leptospirosis patients and may present up to a year after acute illness. Hypopyon, optic disc edema, retinal vasculitis, and membranous vitreous opacities are important diagnostic indicators (Figures 3 and 4). Unlike the viral illnesses discussed above, leptospirosis can be treated with antibiotics (intravenous penicillin or oral doxycycline).
Diagnosis and Management

In all these conditions, identifying the causative organism is critical for managing patients, controlling outbreaks, and preventing further spread. If an outbreak is detected, public health measures may include identifying the specific reservoir (e.g., WNV in birds), controlling the vector (e.g., reducing mosquito populations), protecting at-risk populations (e.g., providing rubber shoes to farmers to prevent leptospirosis), vaccinating livestock (e.g., against leptospirosis), and cautioning the public.

Exotic infection should be considered among patients presenting with eye complaints and/or findings following mild to severe systemic illness. (There may be a longer gap in the case of leptospirosis.) History of travel, outdoor activities (water sports, swimming in natural bodies of water, hiking in forested areas, camping), animal exposure, exposure to stagnant water, mosquito and tick bites, and use of insect repellents and mosquito nets should be elicited. Vaccination, occupational, and past medical history may also provide clues.

Serologic and molecular tests for viral and bacterial pathogens are widely available in the US; both may be ordered to make the diagnosis. Virus isolation may also be useful. Have a low threshold for testing, since presentations are highly variable and signs may be vague and overlap with more common conditions. Consultation with infectious disease specialists should be considered when unusual and/or reportable pathogens are suspected.

Patients with viral infections such as dengue, chikungunya, or WNV require supportive care and antiinflammatory agents to reduce the risk for inflammation-induced scarring. Patients with leptospirosis will require antimicrobial treatment. Knowing the pathogen also helps us look for and treat disease sequelae, such as cataract development following WNV infection.

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REFERENCES
1. Which of the following has/have been implicated in the emergence of exotic infections outside of endemic regions?
   A. Climate change
   B. International travel
   C. Global trade
   D. All of the above

2. Which of the following topical antibiotics is active against MRSA?
   A. Ciprofloxacin
   B. Trimethoprim
   C. Vancomycin
   D. Both B and C are true

3. In the Dengue virus outbreak of 2009–2011 in southern Florida, these people were more likely to have acquired the disease:
   A. Those with a birdbath and lush vegetation in the yard
   B. Those who used air conditioning
   C. Those who were never bitten by a mosquito
   D. Those who used insect repellent

4. Which of the following has NOT been shown to be a risk factor for ocular colonization with drug-resistant organisms?
   A. Asthma
   B. Parenthood
   C. Periocular skin disease
   D. Advanced age

5. Which of the following exotic infections is NOT transmitted by mosquitoes?
   A. Dengue fever
   B. Leptospirosis
   C. Dirofilariasis
   D. WNV

6. In which of the following ways has the emergence of S. aureus resistance to methicillin paralleled the emergence of S. aureus penicillin resistance?
   A. Resistance increased as selective pressure increased
   B. Resistance to both agents is encoded on the mecA gene
   C. Community-acquired resistance preceded hospital acquired
   D. All of the above are true

7. Nongranulomatous anterior or pan uveitis occurs in 40% of patients with which systemic infection?
   A. Leptospirosis
   B. Dengue fever
   C. WNV
   D. Chikungunya

8. Which of the following strategies can be expected to reduce MRSA emergence and/or spread?
   A. Handwashing between patients
   B. Treating ocular infections with agents with MRSA coverage for an adequate length of time
   C. Consistent disinfection of clinical surfaces
   D. All of the above are true

9. What proportion of patients with WNV experience neuroinvasive (including ocular) symptoms?
   A. 10%
   B. 5%
   C. Less than 1%
   D. Almost 100%

10. Which of the following is true of the ARMOR study?
    A. It was designed to track rates of MRSA and MRSE
    B. It uses active surveillance methodology
    C. It tracks susceptibility rates among ocular pathogens across multiple US centers
    D. None of the above is true

EVALUATION:

11. Extent to which the activity met the identified objectives:
    Objective 1: 1 2 3 4 5
    Objective 2: 1 2 3 4 5
    Objective 3: 1 2 3 4 5
    Objective 4: 1 2 3 4 5

12. Rate the overall effectiveness of how the activity:
    Related to my practice: 1 2 3 4 5
    Will influence how I practice: 1 2 3 4 5
    Will help me improve patient care: 1 2 3 4 5
    Stimulated my intellectual curiosity: 1 2 3 4 5
    Overall quality of material: 1 2 3 4 5
    Overall met my expectations: 1 2 3 4 5
    Avoided commercial bias/influence: 1 2 3 4 5

13. Will the information presented cause you to make any changes in your practice? Yes No

14. If yes, please describe:

15. How committed are you to making these changes? 1 2 3 4 5

16. Are future activities on this topic important to you? Yes No

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