

1st Quarter 2016

# SCRIPTS

A JOURNAL OF THE COBB COUNTY MEDICAL SOCIETY

## Bacterial Vaginosis

By: Mary Pitcher, MD

## BEHIND THE WHITE COAT

WITH ALLAN CAMPBELL PURDIE, MD

## Gut Bacteria & Heart Disease

By: Nydia M. Bladuell, MD F.A.C.C.

## The Skin Microbiome

By: Elizabeth (Betsy) Richwine, MD

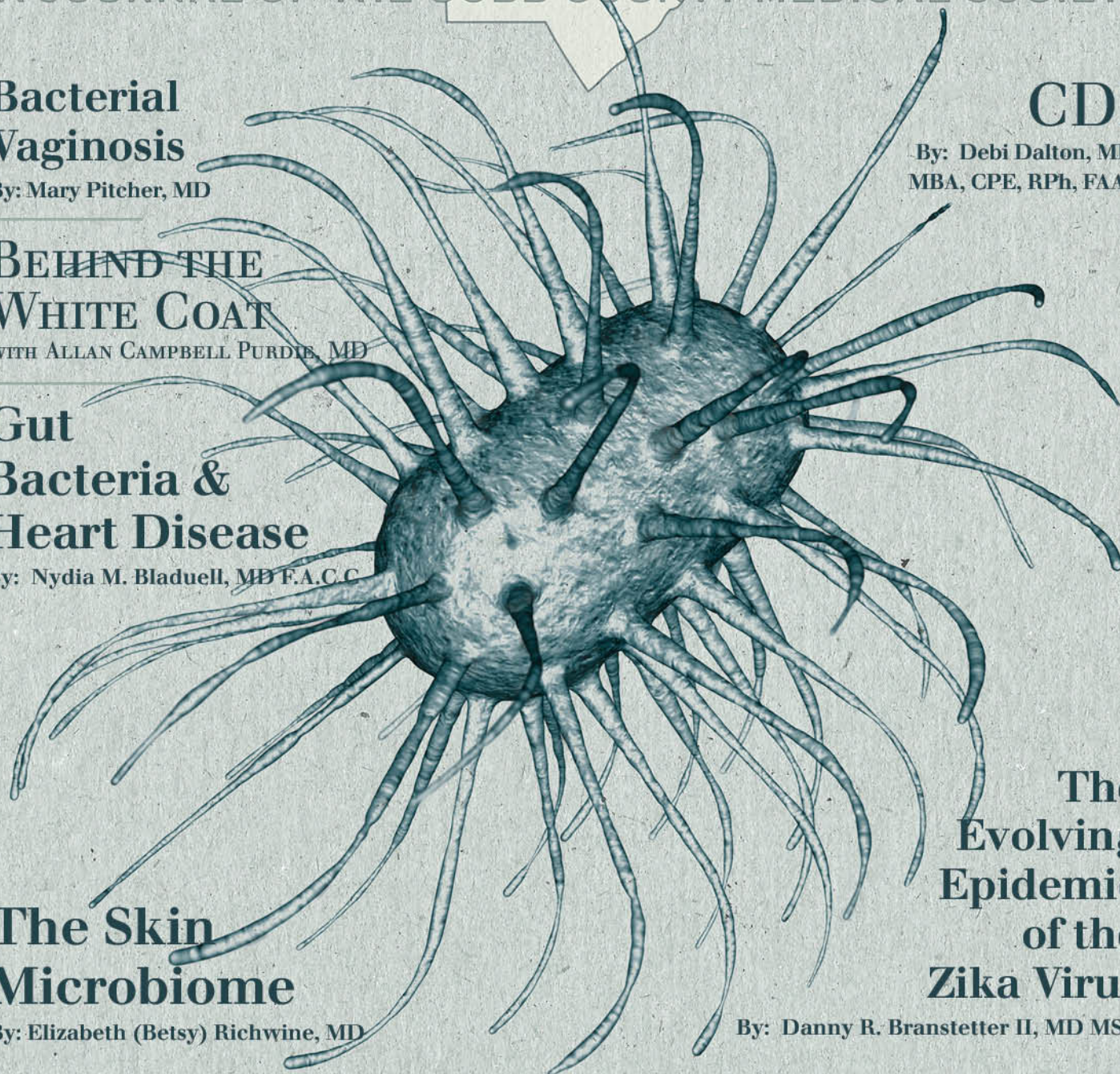
## CDI

By: Debi Dalton, MD,  
MBA, CPE, RPh, FAAP

## The Evolving Epidemic of the Zika Virus

By: Danny R. Branstetter II, MD MSN

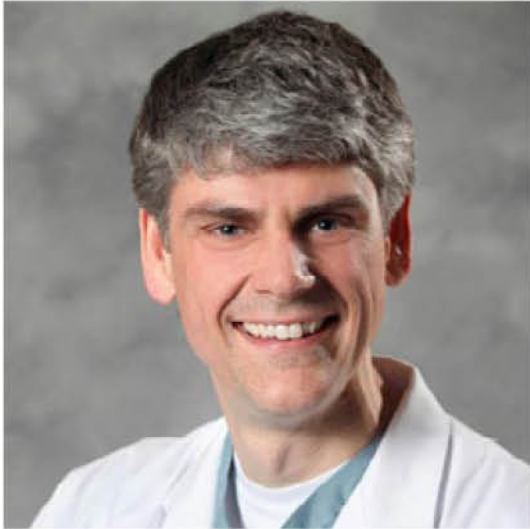
# Bacteria





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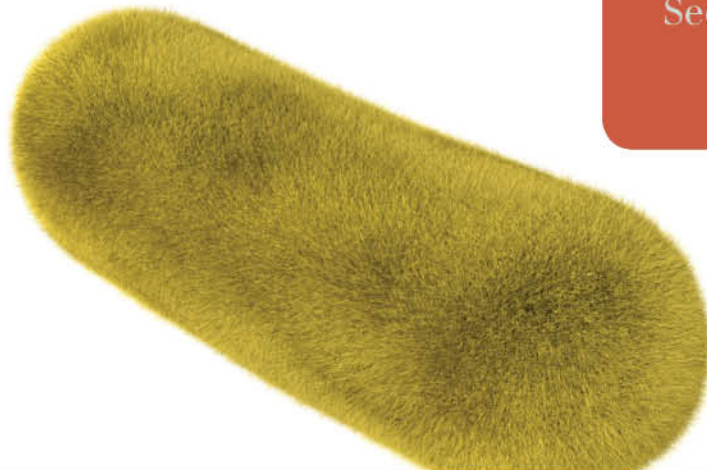
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*"Cancer"*

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**R**eading the articles in this “Bacterias and Viruses” issue of Scripts, has made me think how far we have come in our knowledge and understanding of this field, but at the same time how much more there is to be learned. Medicine can be flashy and we can be blinded when a new procedure or surgical technique is unveiled. But along comes a pandemic like Zika virus, in 2016, to humble and drive us all back to the basics. We covered everything from CDI and BV, to skin and heart, all with a common theme: How all of these diagnoses are tied to those little organisms that we have learned to hate. But maybe we need to learn how to love some of them.

We are entering an era of refining and deepening our knowledge regarding epidemiology of major disease processes and how they are linked to bacterias that live within ourselves. In the future, this understanding will help us to decrease the cost of health care by focusing on primary prevention. The answer lies within ourselves.

I hope you enjoy reading the topics that we covered in this issue, and may they be of help to you as well as to your patients.

NYDIA M. BLADUELL, MD F.A.C.C | *CCMS President* ‘94  
member since

## The Society



**Y**our Medical Society has been busy during the first quarter of 2016, which began with our Annual Dinner with the Legislators. This event is a win-win for both the Society and our legislators. Physicians sat with their respective State Representative or State Senator beside them. We were able to discuss upcoming legislation and how it will affect healthcare and the physician/patient relationship. Then the General Assembly got to work for 40 days. SUCCESS: Truth in Advertising Bill authored by CCMS passed the Legislature and now awaits the Governor’s signature. Thank you to all the Physicians that worked to make this happen.

Our efforts on the other four legislative priorities will be on our agenda for the next session of the General Assembly. However, first there is an election with a candidate to be investigated. Talk to the candidates, invite them for coffee or to visit your office. Tell them about your profession and listen to their thoughts on healthcare. Help us make an informed decision on whom to support.

CCMS is the physician’s (*your*) advocate. We need your input to prioritize our efforts. Contact a Board Member, send an email or attend a meeting. If you are not already a member then join. Support the Cobb County Healthcare Professional PAC.

JOANNE M. THURSTON | *CCMS Executive Director* ‘01  
member since

*Joanne*



# Bacterial Vaginosis

MARY PITCHER, MD  
member since '93

**BV** is the most common vaginal infection in women. Predisposing factors to BV include a lack of Lactobacillus to provide inhibitory control over other vaginal flora, resulting in an overgrowth of virulent bacteria in the vaginal ecosystem. This inappropriate ecosystem can result in many potential complications for the hostess and paves the way for BV to flourish.

Normally the vaginal ecosystem has a number of ways that work together to provide an efficient system to kill unwanted microorganisms. The normal female vaginal flora is comprised of 90-95% Lactobacillus. Lactobacillus produces H<sub>2</sub>O<sub>2</sub> which can inhibit many unwelcome bacteria that do not have the catalase enzyme to detoxify it. The vagina also has serum transudate which contains chloride and peroxidase which work in combination with the H<sub>2</sub>O<sub>2</sub> for even greater inhibition of unwanted bacteria and even viruses.

The enemy of a healthy vagina is the presence of gram negative anaerobes such as strains of Mobiluncus, Prevotella, Ureaplasma and Mycoplasma. These bacteria secrete many substances including sialidase, endotoxin, mucinase and

protease, which increase their ability to invade the upper genital tract and succinate, which inhibits the chemotactic immune response of WBCs. B Vaginalis also invades and secretes a substance which can cleave and destroy protective vaginal secretory IgA.

Women with BV have little to no H<sub>2</sub>O<sub>2</sub> producing Lactobacillus and therefore the opportunistic bacteria are allowed to flourish, providing the perfect environment for BV to multiply and cause symptoms. Symptoms of BV include vaginal discharge and odor, but rarely itching or discomfort. BV metabolizes substrate into amines, which when exposed to alkaline pH (such as with seminal fluid through intercourse) produce the characteristic amine scent. Putrescine and cadaverine are also produced in smaller quantities. This chemical reaction is the basis for the "whiff test" when a sample of vaginal discharge is exposed to 10% KOH. Diagnosis of BV requires 3 of 4 of the following to be present: a positive "whiff test" as outlined above, the presence of clue cells on microscopy, a homogenous skim milk like discharge that uniformly coats the vagina, or a vaginal pH of higher than 4.5.


BV is not sexually transmitted but having BV increases your chance of an STD. Multiple sex partners, douching, and



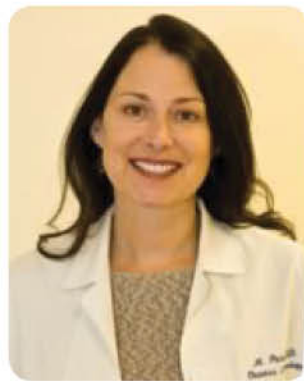
the presence of an IUD increase the chance of BV due to increased potential for disrupting the normal vaginal flora.

Because of the virulent anaerobic bacteria BV travels with, it is associated with a variety of upper genital tract infections and complications. These include: preterm delivery with low birth weight infants and the associated complications, chorioamnionitis, postpartum and post abortion endometritis, PID, and cuff cellulitis after hysterectomy

Recommended primary treatments for BV include Metronidazole orally for one week or the use of vaginal creams, like Metronidazole or Clindamycin. Alternative treatments include Tinidazole orally, Cleocin orally, or Cleocin vaginal ovules. Cleocin ovules can weaken latex and rubber products such as condoms or diaphragms which can have unexpected consequences. BV can often be recurrent and challenging due to the vaginal ecosystem imbalance that predisposed the patient to the infection in the first place. Treatment regimens for recurrent BV normally can last up to six months and often are only temporarily successful. No studies currently support the addition of probiotic or lactobacillus culture as being helpful and treating male partners has not been beneficial.

There are currently no good regimens to restore the vaginal ecosystem back to normal once this upset has occurred and this remains a challenge for both the patient and their physician. 

***“Victory belongs to the most persevering”  
Napoleon Bonaparte***



*Dr. Mary Pitcher is from St. Louis, Missouri. She attended the University of Missouri and completed her residency in OB/GYN at the University of Florida. Dr. Pitcher moved to Marietta and began practice with Dr. Warren Somerlot and Dr. Elizabeth Street.*

*Dr. Pitcher and Dr. Street have added three more physicians to their practice, Drs. Laurie Robbins, Laura Cauthen and Angela Paas. Dr. Pitcher is located at 574 Church Street and has privileges at WellStar Kennestone Hospital.*



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# Gut Bacteria & Heart Disease

Nydia M. Bladuell, MD F.A.C.C.  
member since '94

*Bacteria*

**N**ot a very appealing title, but I think that once finished reading the article you will agree that there is a lot of truth to the saying: “we are what we eat”. Think about it for a moment. In the near future, when you visit your Doctor, your risk factors, like: age, race, hypertension, diabetes, dyslipidemia, diet, obesity, and so forth will have

a new companion: “what kind of gut flora do you carry?”

The researchers at the Cleveland Clinic have identified which bacteria are good and bad for you. It just so happens that the bacteria in patients with more plaque and hardening of the arteries have more genes that cause inflammation. Long term inflammation can lead to different diseases including atherosclerosis.

Bacteria in healthy people have genes that protect them from inflammation by soaking up dangerous chemicals.

Our genes and lifestyles are important in determining our risk of disease, but the genes of the bacteria inside our gut are important as well.




When people think of bacteria they probably think of germs that make them sick. But most bacteria aren't harmful and many are actually helpful. More than a hundred-trillion bacteria live in our guts. They are more numerous than all of a body's cells. Rather than being a nuisance, these bacteria are actually helpful and necessary.

The bacteria that have been identified with increasing atherosclerosis are called *Collinsella*. This bacteria has genes that produce peptidoglycan. The peptidoglycan is found on the outside of the bacteria and causes the immune system to kick into gear. This reaction causes inflammation, which is a risk for developing atherosclerosis. There are other bacteria that protect people from atherosclerosis. These bacteria have more genes for antioxidants, like the ones found in blueberries.

When we eat we are feeding our bacteria. Some of them feed on choline and carnitine, nutrients in red meat, egg yolks, and high-fat dairy products. As they feed they release a chemical called TMA (trimethylamine). The liver turns TMA into TMAO (trimethylamine-n-oxide). TMAO in the blood doubles the risk of heart attacks, strokes, and death. The levels of TMAO in the blood can help predict your risk of heart disease.

## What can we do to help?

Besides moderation when eating meat and other animal products, there is ongoing research with a natural substance called DMB (3,3-dimethyl-1-butanol). In mice DMB has been shown to decrease levels of TMAO, which translates into decreased plaque formation. DMB is found in olive and grape seed oils. Because it is not an antibiotic DMB does not kill "good bacteria," and there is little risk of overusing it or building resistance to it. DMB also has been found in red wines and olive oil, which explains why the Mediterranean diet - which includes both - has been shown to be healthy for your heart.

Until we get the easy fix with a DMB pill to lower TMAO levels without the liver toxicity, use moderation in terms of your animal product caloric intake, enjoy the Mediterranean diet, and think about having a toast to your health with a glass of red wine - if you enjoy wine, that is. 



*Dr. Bladuell is board certified in Cardiovascular Disease and Interventional Cardiology. She joined WellStar Cardiovascular Medicine in April, 2009. She is currently on staff at WellStar Kennestone and Cobb hospitals. Dr. Bladuell is the current President of Cobb County Medical Society.*

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Dr. Allan Purdie

Good Samaritan  
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CLASS OF 1994  
SPECIALTY: FAMILY PRACTICE  
PRACTICE: MEDICAL DIRECTOR OF  
GOOD SAMARITAN CLINIC OF COBB

**Q** WHAT MADE YOU DECIDE TO PRACTICE MEDICINE?

I have been interested in medicine since I was a youth, but I was also interested in ministry. After graduating from Seminary I had the privilege of pastoring a growing church. Yet my interest in medicine didn't wane. Medical Missions was a logical pursuit to combine these two interests. So in my early thirties I started my medical training at Emory University.

**WHAT DO YOU THINK IT TAKES TO BECOME SUCCESSFUL IN YOUR FIELD?**

The concept of "success" is not the same for everyone and it is even not the same for the same person depending on where they are in life's journey. For me, at this stage in my career, I would define success as being able to apply quality medical skills to bring health to the whole person. It is to consider the psychological, social and spiritual well being of my patients. To accomplish this goal requires consistent growing in one's understanding of medicine, people, and a team approach with other others.

**Q** WHAT DOES CCMS STAND FOR AND WHAT DOES IT PROVIDE FOR YOUR COMMUNITY?

CCMS has many of the same goals of The Good Samaritan Clinic of Cobb. Both seek to serve our community by promoting quality health care and considering the welfare of patients. Many of CCMS members have generously given their time and talents to the patients of Good Sam. I am grateful for this wonderful organization and look forward to getting to know the other doctors in our community.

**Q** WHAT DREW YOU TO PRACTICE IN THIS AREA/LOCATION? WHAT BROUGHT YOU TO COBB COUNTY?

I returned to Cobb County after accepting the position at Good Sam. But Cobb County isn't new to either my wife or me. We have both lived in Cobb before. The Smyrna area, in particular, isn't far from my boyhood community. So we're glad to be "home."

***"The concept of "success" is not the same for everyone and it is even not the same for the same person depending on where they are in life's journey."***

**Q** WHAT IS "GOOD SAM" AND HOW ARE YOU INVOLVED WITH THE ORGANIZATION?

Offering quality medical care and sharing the love of Christ to the uninsured and under insured is the two goals of the Good Samaritan Clinic. It was started by concerned citizens of Cobb and is now in its' eleventh year. I started with Good Sam this past January as the Medical Director. We hope Good Samaritan Clinic is more than just a place to receive quality medical and dental care. Our desire it to minister to our patients with whatever resources we have available.

**WHAT DOES THIS ISSUE'S THEME: "BACTERIA" MEAN TO YOU?**

In 1694 Anton Van Leeuwenhoek discovered bacteria through a single lens microscope. Yet it wasn't until 1876 through the work of Robert Koch, that the linkage of bacteria to disease was clearly demonstrated. The understanding of our world, and in particular medical science, has dramatically changed ever since. Bacteria have killed more people than all the wars and natural disasters in the world combined. The invention of antibiotics has saved the lives of millions. Yet our symbiotic relationship with bacteria also enables us to live healthier lives. The horizon offers knew and better understanding of these small creatures that have the potential to harm us and to help us.

**TELL US YOUR FAVORITE LOCAL HANGOUT OR RESTAURANT.**

We are just beginning to explore all the wonderful places in Cobb. However, we do enjoy the flat bread at the Cheesecake Factory.



# The Skin Microbiome: Why It Matters

ELIZABETH (BETSY) RICHWINE, MD  
member since '11

**T**he skin is the largest human organ and serves as a protective barrier from assault by foreign organisms and toxins. The skin also interfaces with the outside environment and thus is colonized by a variety of microorganisms. Much attention has recently been placed on the skin microbiome which can be defined as the total microbial community that lives in association with human skin. A wide array of microorganisms including bacteria, viruses, fungi, and sometimes mites inhabit the cutaneous surface and its appendages (hair follicles, sweat glands, and sebaceous glands).

Molecular techniques have identified a much greater diversity of skin organisms than were previously revealed by culture-based methods in the lab. It is now estimated that over 100 distinct species making up a total of 1 million microorganisms colonize each square centimeter of human skin. Recent research emphasizes the importance of the microbiome in understanding health and disease.

## Factors that contribute to the Skin Microbiome

Development of the skin microbiome is dependent on many factors. For instance, characteristics of a specific site of skin determine the population of bacteria that will colonize the area. Data has revealed that *Staphylococcus* and *Corynebacterium* species prefer areas of high humidity and thus are the most abundant organisms in moist sites (the umbilicus, axilla, inguinal crease, gluteal crease, etc.). These bacteria also process apocrine sweat which results in the odor of sweat in humans. Dry areas like the forearm, buttock, and parts of the hand have the highest diversity of microbes but the lowest absolute numbers.

The variation in the landscape of the skin with all its peaks, invaginations, and appendages allows for colonization by a variety of microorganisms. For instance, the appendage of skin known as the pilosebaceous unit (hair follicle and sebaceous gland) is colonized by microscopic arthropods known as *Demodex* mites. They are part of the normal flora here as they feed on sebum and possibly the epithelial lining of the pilosebaceous unit. They are found in higher



concentration in more sebaceous sites such as the face. Demodex mites have been long been associated with rosacea, facial itching, and chronic blepharitis.

Host factors such as age and sex contribute to the establishment of the microbiome. The skin is sterile in utero, but colonization occurs almost immediately after birth. Anatomical differences in males and females also influence microbial colonization.

An individual's occupation, clothing, and use of skin products may contribute to selecting the inhabitants of the skin. Soaps, cosmetics, and moisturizers alter the skin barrier and thus can potentially influence the skin microbiome. Simple behaviors such as frequency of hand-washing may account for interpersonal differences.

### The Immune System and the Microbiome

The skin microbiome aids in the education of the cutaneous immune system. Skin cells in the epidermis called keratinocytes use receptors to continuously monitor the microbes colonizing the skin. These receptors recognize specific molecular patterns on pathogenic bacteria and fungi. Once receptors are activated the innate immune response is initiated resulting in secretion of chemokines, cytokines, and antimicrobial peptides. These peptides are directly toxic to bacteria, fungi, and some viruses.

In addition to affecting both the skin's adaptive and innate immune response, the skin microbiome is believed to promote some level of immune tolerance. The skin is constantly exposed to numerous microorganisms and is able to distinguish commensal microorganisms from those that are pathogenic. Although the mechanism is not completely clear, the means of differentiation are thought to be due to desensitization of certain receptors by prolonged exposure to commensal inhabitants.

### The Skin Microbiome and Disease


In many skin disorders, the pathogenesis involves a member of the microbial community. Research has shown that some commensal members of the cutaneous microbiome have the potential to become pathogenic under certain conditions.

Sebaceous glands are both relatively anoxic and secrete lipid-rich sebum. They are ideal areas for *Propionibacterium acnes* to reside as this commensal bacterium is a facultative anaerobe that can degrade sebum into free fatty acids. These free fatty acids are beneficial as they make the skin surface more acidic thus preventing invasion by common pathogens like *Staphylococcus aureus* and *Streptococcus pyogenes*. However, *P. acnes* is also associated with acne which is commonly seen in teenage years. During puberty, sebaceous glands mature and increase in their colonization by *P. acnes*. The enzymes secreted by *P. acnes* (lipases, proteases, etc.) damage the lining of the pilosebaceous unit which induces an immune response and starts an inflammatory cascade. Although the development of acne is multifactorial and is influenced by genetics, the immune system, and the environment, *P. acnes* has been implicated in this skin disorder.

Chronic wounds are an example of when commensal bacteria invade and become pathogenic when the skin barrier is breached. The wound is not caused by the bacteria, but their colonization can cause slow wound healing and account for continued inflammation. Additionally, some commensal bacteria are responsible for infection once the skin is breached by non-native objects. *Staphylococcus epidermidis*, a bacterium that is part of normal skin flora, is particularly problematic for patients with in-dwelling medical devices like catheters or heart valves. Virulent strains of this bacterium can form biofilms on such devices. Biofilms are difficult to treat as they protect bacteria from host immune attack and impair diffusion of antibiotics. *S. epidermidis* has become increasingly more resistant to antibiotics like penicillin and methicillin and has been implicated in transfer of antibiotic-resistant genes to the more virulent *S. aureus*.

Seborrheic dermatitis is a pruritic, hyperproliferative skin disorder seen as redness and scale on the scalp, face, behind ears, and chest. Part of its pathogenesis is thought to be due to *Malassezia* species, the most abundant fungal skin commensal. *Malassezia* is seen most commonly in oily areas (such as the ones affected by seborrheic dermatitis) and feeds on human sebum. Lipases secreted by this fungal organism to break down sebum result in free fatty acid metabolites. These metabolites stimulate hyperproliferation, inflammation, and an immune response in the upper layers of skin. Since antifungal topical treatments greatly improve seborrheic dermatitis, this reinforces the hypothesis there is a fungal component to this skin disorder.

### Conclusion

The host-skin microbiota relationship is complex and still being defined. As molecular techniques advance, further identification and characterization of skin microorganisms will occur. Furthermore, greater understanding of the role of the skin microbiome in human skin disorders may lead to new diagnostic and prognostic techniques as well as targeted and unique treatments. 

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*Dr. Richwine is a board certified dermatologist who practices general medical and surgical dermatology. She is a graduate of the Medical College of Georgia and is a partner at Marietta Dermatology and the Skin Cancer Center.*





# CDI: Clostridium Difficile Infection

## What Physicians Need to Know

BY: DEBI DALTON, MD, MBA, CPE, RPH, FAAP  
member since '12

**A**ntibiotics were the miracle drugs of healthcare generations ago and still are to a certain degree. But today they have become medications which can cause mild side effects such as diarrhea, allergic reactions and yeast infections to complications such as anaphylaxis, bloody diarrhea, severe dehydration and even death. The reason these last few complications can occur is because of an emerging superinfection caused by a bacteria called *Clostridium Difficile* or, as commonly known, *C. Diff*. *C. Diff* Infection is most commonly referred to as CDI. *C. Diff* is a bacterium which could reside asymptotically in our intestinal tract. Once it is in our intestinal tract, it can be eradicated with specific antibiotic therapy, but not

always. A patient can remain a carrier for an extended period of time. It afflicts nearly a half a million Americans annually and kills 15,000 each year. This number is bound to grow as it is becoming ever more present in the community, not just in hospitals. Approximately 25% of patients who are successfully treated for their CDI can have a relapse within two weeks of this “recovery”.

Bloody diarrhea is an indication to test for CDI, along with other, more commonly found bacteria such as *Yersinia*, *Campylobacter*, *Salmonella* and *Shigella*. But you must also consider Rotavirus and Norovirus, especially in children. Do not test children for CDI if they are less than a year of age. Greater than three watery or loose stools is also an indication to test. Do not treat empirically. We should know what we are treating and antibiotics are not the answer in most cases. Also beware that some patients are using laxatives and this could mimic CDI with diarrhea, abdominal pain and cramping. CDI patients will be ill appearing, present with a




fever and will have leukocytosis on their CBC.

Do not perform a stool test of cure. Many patients in our community are asymptomatic carriers and will test positive. These patients do NOT require treatment. Hospitalized patients pose a different set of issues. CMS is looking at CDI as part of our Hospital Acquired Infection rates (HAI's). If a patient has been hospitalized for four or more days, starts to have symptoms of CDI, and they test positive, they are considered "Hospital Acquired". Certainly these patients can come from home with CDI and may test positive before those four days. These patients would be considered "Community Acquired" CDI and it would not count against a hospital's infection rates.

Isolation of CDI patients is paramount. In the hospital setting, symptomatic patients who test positive or are suspected CDI and are currently being tested for possible CDI, must be placed in isolation. Gowns and gloves must be worn whenever entering an isolation room, regardless of presumed amount of time spent in the room. Gowns and gloves must be changed immediately if soiled and they must be removed upon exiting from the patient room. Hand hygiene must be performed immediately upon removing the gown and gloves. This entails washing your hands with soap and water to mechanically remove the C. Diff spores. Hand sanitizer will NOT remove the spores but it is an alternative temporarily if a sink is not immediately available.

Antimicrobial Stewardship is also very important with all patients so that we can avoid CDI from occurring. Use narrow spectrum antibiotics, avoid or limit broad spectrum antibiotics if possible, know specifically what you are treating and check sensitivities to ensure targeted therapy. For hospitalized patients, perform a "Time-Out" every 72 hours of antimicrobial therapy to re-evaluate the need to treat with antibiotics, reduce the risk of CDI and help prevent bacterial/antimicrobial resistance. While Proton Pump Inhibitors and Histamine 2 Antagonists have not been shown to be a direct cause of increased CDI, the literature has not completely refuted any contributory basis. Therefore, use these medications when necessary and do not automatically place patients on them unless medically indicated.

On the horizon is a possible new medication which targets the toxins produced by C. Diff. This medication "mops up" the toxins but leaves the bacteria unscathed. This new treatment may be less prone to the bacteria building a resistance to it and will provide physicians with a new option to treat infections such as C. Diff, E. Coli and certain Staph organisms. 



*Dr. Despina "Debi" Dalton is a board certified pediatric emergency physician. She is VP of Medical Affairs at WellStar Douglas Hospital and CCMS past-president.*



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# The Evolving Epidemic of the Zika Virus

BY: DANNY R. BRANSTETTER II, MD, MSN  
member since '15



**C**irca 1947, deep in Entebbe, Uganda a new Flavivirus was discovered while researchers were studying the prototypic Flavivirus, Yellow Fever. Scientist isolated this new mosquito transmitted virus from monkeys in 1952 calling it Zika Virus after the Zika Forest where it was first encountered. Two years later the first human case was identified in Nigeria. Sporadic and isolated outbreaks occurred mainly in Southeast Asia and Africa. Serologic testing revealed as high as forty percent of persons tested in these areas had detectable antibodies. The first outbreak outside Africa occurred in 2007 in Micronesia on Yap Island. Estimates project three quarters of the population to have been infected. Experts at that time identified Zika as an Emerging Pathogen. Zika virus' west to east spread followed previously identified paths of other arboviruses West Nile and Dengue (both flaviviruses) and chikungunya (an alphavirus). 2015 marked a dramatic increase in the number of cases identified in Brazil.


Those affected by Zika virus rarely required medical intervention or hospitalization and it was considered a rather innocuous pathogen. One of the first, if not the first, hospitalization reported was in 2013 from French Polynesia. A young woman experienced Guillain-Barre' syndrome as a complication of Zika Virus infection. Concurrent with the epidemic in Brazil, health officials noted a marked increase in the number of cases of microcephaly in the northeast region. Thus, indicating a possible association with Zika Virus infection. Fetal autopsy report identified Zika Virus in fetal brain tissue by PCR and electron microscopy. Further findings consisted of calcifications in fetal brain and placenta, hydrocephalus and microcephaly.

Flavivirus infection diagnosis requires demonstration of acute and convalescent antibody titers or isolation of the virus from serum by PCR. Flavivirus antibody titer interpretations can be challenging. Cross-reactivity with other flaviviruses and concurrent viral epidemics with overlapping clinical syndromes requires judicious evaluations. Viremia is often short lived limiting the utility of PCR testing. Plaque-reduction neutralizing antibody testing should be performed to compare levels of antibodies to flaviviruses from prior infection or vaccination.

Travelers from endemic areas or those exposed to travelers with symptoms consistent with arboviral infection should be offered appropriate serologic screening. Clinical syndrome of arboviral infection includes fever, rash, conjunctivitis, arthralgia, myalgia, and headache. Geographic area and outbreak data should guide a broad differential based evaluation which could include Dengue, chikungunya, West Nile, malaria and tick borne diseases. Screenings should be coordinated with local public health departments. Attention to possible concurrent illnesses in returning travelers is paramount. Pregnant patient screening should include ultrasound and repeat serologic testing if microcephaly, placenta or fetal brain calcifications are identified. Benefit of amniotic testing remains unknown. Arboviral evaluation needs to be considered in patients with neurologic deficits including encephalitis.

Many questions exist surrounding infection with Zika Vi-

rus including risk associated with pregnancy. Recommendations are updated as knowledge evolves. Currently WHO and CDC recommend limiting travel to areas with ongoing outbreaks. For those residing in or traveling to endemic areas, mosquito avoidance measures including repellents, netting, air-conditioning, long sleeves, and pants are highly recommended. Mosquitos carrying the virus bite throughout the daytime. Exact incubation periods are uncertain but are thought to be approximately 9 days in humans and two weeks in mosquitos. Presence of virus in urine and other body fluids along with sexual transmission have been reported. The extent of infectivity remains unclear. Condom use is effective in preventing sexual transmission. Additional or Enhanced precautions beyond Universal Precautions are not indicated for suspected or confirmed cases.

As increased testing and continued spread of the virus occurs, case occurrences within the United States are an almost certainty. The latest outbreak and guideline recommendations can be found at the CDC website. 



*Dr. Danny Branstetter is a board infectious diseases specialist practicing since 2009. He joined WellStar Medical Group in 2012 at Kennestone.*



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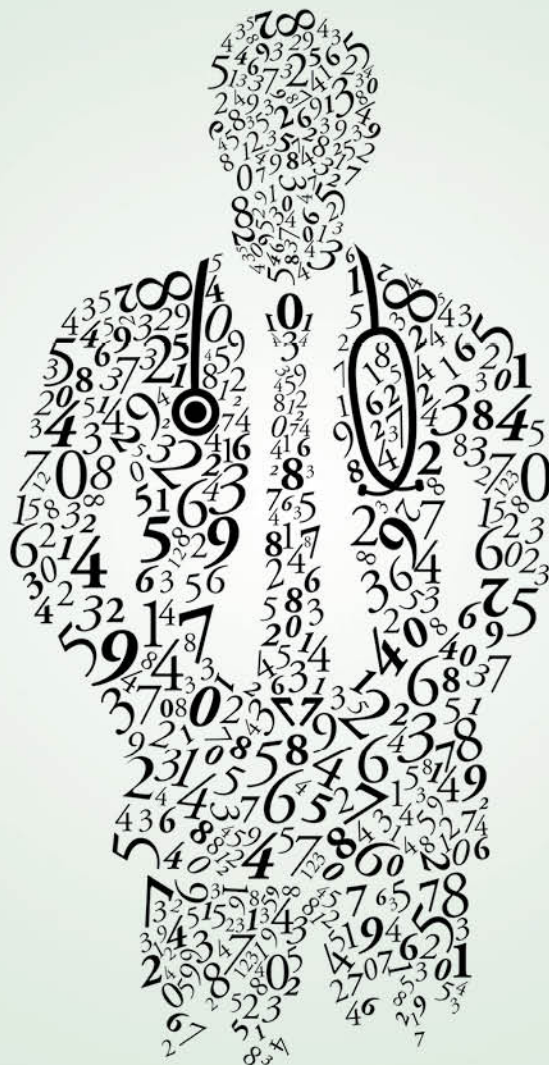
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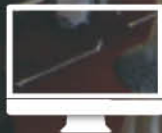
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## Upcoming Events *2016*

May 17, 2016

*Cobb Healthcare Professionals PAC*

*Candidates Reception*

*5:00pm - 7:00pm*

*@ Georgian Club*

May 17, 2016

*General Membership Meeting*

*7:00pm*

*@ Georgian Club*

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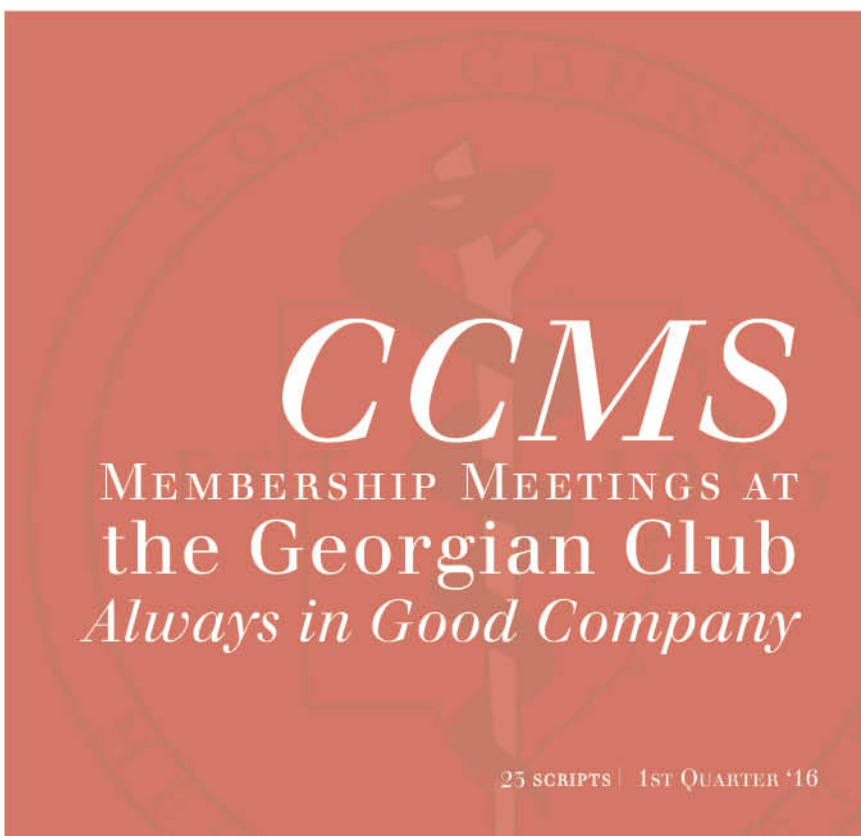
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