Northeast Branch Newsletter

Number 138 Winter 2014-2015

2014 Programs in Review

Climate Change and Infectious Diseases: Knowns and Unknowns

(L-R) NEB-ASM Council Members: Alfred DeMaria,Jr. MD (President); Irene George (Secretary); Speaker David N. Fisman, MD; NEAACC Board Members: Dr. George Parsons, Dr. Mark Kellogg (Secretary), Dr. Rabie Al-Turkmani (House of Delegates Representative), Dr. Mahdi Garelnabi (Program Chair)

Global climate change has implications for all aspects of the environment, society and human activity, including health, epidemiology, and clinical manifestations of disease. Clinical laboratory professionals should be aware of these implications for their clinical practice, and as informed citizens engaged in public discourse.

Dr. David Fisman, infectious disease physician, epidemiologist and Professor of Epidemiology at the University of Toronto's Dalla Lana School of Public Health spoke on *Climate Change and Infectious Diseases: Knowns and Unknowns*, at the fourth dinner-meeting jointly sponsored by the Northeast Branch of the ASM and the Northeast Section of the American Association for Clinical Chemistry. This was held on March 13, 2014 at the Hilton Garden Inn in Waltham, MA. Dr. Fisman is also a practicing

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Keeping Out of Touch: The Role of High Touch Surfaces in Infection Transmission in Home and Community Settings and the Implications for Cleaning and Disinfection

The second program of the year, which was co-sponsored by the Northeast Branch and the American Society for Clinical Laboratory Science of Central New England, was held on April 23, 2014 at Rachel's Lakeside in Dartmouth, MA. Elizabeth Scott, PhD, Co-Director of the Simmons Center for Hygiene and Health in Home and Community and Associate Professor at Simmons College in Boston, MA, spoke on *Keeping Out of Touch: The Role of High Touch Surfaces in Infection Transmission in Home and Community Settings and the Implications for Cleaning and Disinfection*.
Dr. Scott has a long-standing interest in the field of microbial environmental hygiene. She has experience in industrial-based research projects evaluating bacterial content in the domestic environment and the effectiveness of disinfectants in the home. In addition, she has university-based research on the effectiveness of surface disinfectants under "in-use" conditions.

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NEB Council Meetings

Council Meetings this year will continue to be held at the William A. Hinton State Laboratory Institute in Jamaica Plain. Members and all interested microbiologists and scientists are welcome to attend. Please notify Irene George, Secretary at (508) 785-0126 in advance.

Membership Notes

Dues reminders for 2015 have been sent to our membership via e-mail. Members who did not provide an e-mail address were contacted by postal service. Membership forms may be found on the NEB website or you may join the both the ASM and the Northeast Branch online through the ASM eStore. Please make the necessary corrections to your demographics and return dues to the Treasurer. Emeritus members need to reply if they wish to remain on the mailing list. Changes only may be e-mailed to: NEBranch-ASM@comcast.net. Please check mailing labels on postal correspondence as they reflect existing information.

Although membership in the national branch automatically makes you a member of the local branch in some organizations, this is NOT the case in the ASM. *To be both a National Member and a NEB member, you have to join each individually.* The Northeast Branch currently has 202 members.

Council Election Results

Congratulations to the following NEB members whose terms as Branch Officers began July 2014. Nancy S. Miller, President; Patricia Kludt, Treasurer; Paulette Howarth, National Councilor; Frank Scarano, Alternate National Councilor and Beverley Orr, Local Councilor. We are looking forward to working with everyone in planning a busy year!

Student Chapters

The NEB is associated with three active student chapters. The Boston-Area Student Chapter, the University of New Hampshire Chapter in Durham, NH, and the Maine Society of Microbiology, Orono, ME. We look forward to collaborating with them again in the coming year.

FUTURE PROGRAMS

LOCAL PROGRAMS: Local Meeting announcements and registration materials are posted on our website: http://www.northeastbranchasm.org or through the ASM website: http://www.asm.org

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March 19, 2015
Dinner Meeting:
Supporting Genomics in the Practice of Medicine

Speaker: Heidi Rehm, Director of the Laboratory for Molecular Medicine at Partners Healthcare Personalized Medicine. Sponsored jointly by the Northeast Branch and the Northeast Section of the American Association for Clinical Chemistry.
Location: Forefront Center for Meetings & Conferences, 404 Wyman St.,Waltham, MA.
Contact: Carol L. Finn 508-584-5173

April 29, 2015
New England Laboratory Director's Meeting, Publick House, Sturbridge, MA

October 20-21, 2015
50th Annual Region I Meeting
Sponsored by the Northeast, Connecticut Valley, Eastern New York, and New York City Branches of the ASM.
Location:The Lantana, 43 Scanlon Drive, Randolph, MA
Symposia – Posters – Exhibitors
A 50th Anniversary Toast

NATIONAL MEETINGS:

May 2-31, 2015. ASM Conference for Undergraduate Educators, Austin, TX
http://www.asmcue.org/

May 30-June 2, 2015. 116th ASM General Meeting, New Orleans, LA.
http://gm.asm.org/

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Infectious Diseases clinician at the Toronto Western Hospital. He trained in clinical infectious diseases at Beth Israel Deaconess Medical Center in Boston and from 1999 to 2001 was an AHRQ Fellow in Health Policy at Harvard Centre for Risk Analysis. He has previously held faculty positions at McMaster, Princeton, and Drexel Universities. His interests are in analytical and mathematical epidemiology of infectious diseases, and in health economic evaluation of communicable disease control programs.

Dr. Fisman first spoke about the trends and projections in climate change. He showed the "inevitable" slide of greenhouse gases, carbon dioxide, methane and nitrous oxide (IPCC, 4th Assessment Report, 2007). He spoke of climate change as a process where a physical component of our planet, which we experience as weather, seems to be shifting in very dramatic ways. Upstream from that, as best as we can understand, seems to be a changing atmospheric concentration of different gases. We speak of them as greenhouse gases because just as glass in a greenhouse traps heat, these gases seem to trap temperature at the level of the earth's surface. Carbon dioxide gets a lot of play but methane and nitrous oxide are also greenhouse gases. As the world has industrialized, starting in about the 18th century, the atmospheric concentrations of carbon - as evidenced by ice core measurements - have exploded and have reached unprecedented levels. Where does all that carbon come from? Looking at graphs showing relative carbon emissions per capita by country, it is evident that the wealthier countries are emitting the lion's share of carbon that is driving the changes in climate. South of the equator there is much less carbon emission per capita. (Sources: worldmapper.com and gapminder.com). Dr. Fisman then showed a slide of Canada's contribution to the global carbon footprint. When viewed using Google Earth, a gigantic scar on the earth known as the Tar Sands Project is seen in Northern Alberta, which has the world's 2nd largest petroleum reserves. Extracting that petroleum, some of which is sent to the US, accounts for 6% of Canada's GDP. The heavy crude oil (bitumen) however is mixed with sand, clay and water, and

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it takes about a barrel of oil (one barrel's worth of energy) to separate one barrel of bitumen/mud**)** mixture. Therefore this is a major source of air pollutants in Alberta and greenhouse gas emissions in Canada.

The world's mean temperature is actually rising at a fairly marked rate; this year globally is the fourth warmest year on record. When using models, projections and progressions, what you see when looking back to the mid-nineteenth century is an accelerated warming (IPCC, 4th Assessment Report, 2007). Dr. Fisman mentioned that models however, are always idealized and simplified representations of very complex systems in reality**,** and can be both useful and sometimes inaccurate. But when we try to model what is happening to global ocean and land temperatures, such models seem to simulate and explain pretty well what is happening.

A National Oceanic and Atmospheric Administration (NOAA) report (June 2009) synthesizes a number of plausible climate projections for the United States (US). It projects the number of days per year that the temperature will be above 100°F in a particular area of the country under six different emissions scenarios**,** and projects plausible futures based on how much carbon tends to be released into the atmosphere, etc.

A low emissions scenario predicts very warm-hot temperatures seven years from now. Death Valley will obviously still be warm, but we have the central valley of California, southern Texas and a large swath of the Midwest becoming very hot.

A high emissions scenario for 2080-2099, would be a nightmare, as it projects that large portions of the US, such as the Midwest, may be like Death Valley, CA. When talking about mitigating the effects of climate change, we potentially talk about trying to engineer our way around situations like this, where large chunks of the country will look like Death Valley.

From these IPCC reports we see that global mean temperatures and global average sea levels have been rising continuously in the last hundred years; while northern hemisphere snow and ice cover has been decreasing. This is already a reality as seen by events such as the Muir Glacier in Alaska, which has been retreating since 1941 and is being replaced by a salt-water lake as seen in 2010.

We face some hard choices as a result. Short term economic and business interests many times supersede future global problems. A graph of climate change and disaster frequency due to floods, earthquakes, thunderstorms, etc. from 1950-2009 shows an increasing frequency in natural catastrophes, which were projected by the IPCC in 2007 as increasing in frequency. There was $1 billion economic loss and 50 fatalities during this time period. It is impossible however, to say that all of these, or to pinpoint which of these**,** occurred as a result of global warming, except that there is a net change in frequency and strength in events over time. There were seven significant natural catastrophes in the US in 2009 alone, the highest number to date.

A model using a sea level rise of five to seven feet, examined how many people are involved in floods annually. Temperature anomalies were plotted against the population at risk and cost and annual investment required; a 2°C temperature shift in 100 years was projected. The model shows a massive risk for individuals living in coastal areas, but also shows that there is a net cost savings if you invest heavily in infrastructure such as enhanced coastal protection.

What is in store for us in the future? Projections in climate change in North America include**:** increased temperature, increased rainfall, increased drought and wildfires (extended periods of drought followed by heavy rain all at once due to decreased carrying capacity of water by dry air), and increased frequency of extreme weather events.
(Intergovernmental Panel of Climate Change [IPCC] 4th Assessment 2007)

Moving away from generic climate change Dr. Fisman spoke of what such changes have to with infectious diseases. He spoke of ecosystems, which are complex biological systems and include both living and non-living components; we are part of ecosystems, as are pathogens and vectors. A physical attack on ecosystems by changes in temperature, water availability, ocean pH (via CO2), or and frequency of extreme events such as fire and floods, stresses living components of the

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environment. In order to survive, they have to adapt or change in some manner.

We already see the health effects of climate change. Direct consequences have been heat related mortality; injuries due to hurricanes, tornadoes and fires with significant loss of life; and displacement of populations to coastal flooding and desertification. We can also see indirect consequences of climate change**,** such as changes in the incidence and distribution of infectious diseases, either because of the pathogen, or displaced populations that may be living in refugee camps which are an excellent breeding ground for disease.

In diseases with environmental reservoirs, climate change can affect food and water. Lack of water can result in the use of wastewater for irrigation, etc. Extreme weather events can also results in "inoculation" of pathogens into humans, such as melioidosis resulting from monsoon-like weather in Australia.

Many communicable diseases, especially respiratory pathogens, are very seasonal, which implies that there is some unknown environmental driver. The seasonality of influenza, our "flu season" in reality is due to due to environmental change. Such diseases can be influenced by disturbances in the seasonal patterns of transmission (environmental changing) and mass movement and crowding of populations via social disruption.

It is also recently believed that other diseases such as nosocomial pathogens, endemic mycoses and some other diseases may be influenced by climate change. Dr. Fisman showed a model predicting a relationship between latitude and the odds of Gram-negative bacteremia in hospitalized patients with bacteremia in 22 cities. Data were consistent with prior reports of elevated risk of Gram-negative bacteremia with warmer temperatures nearer the equator - the further away from the equator, the more Gram-positive bacteremia. This has important implications for climate change and is still under study.

Climate change can also impact vector-borne diseases by changing ecosystems and the ranges of amplifying hosts and insect vectors. A model showing a prolonged transmission cycle of West Nile virus projects that an earlier onset of spring temperatures can result in earlier egg-laying and larval development due to higher temperatures, and a large increase in West Nile infection in humans. Infections might start in June or July instead of August. Thus West Nile and EEE are greater threats with warming temperatures than when it is cooler. Climate may also have something to do with the spread of lyme disease. Ticks basically need blood meals repeatedly to assure they molt and go on to the next life stage in one or two seasons. With shorter summers and longer winters such as occurred in Canada fifteen-twenty years ago for example, *Ixodes scapularis* tick populations could not complete a life cycle in one year and could not establish themselves locally. This is not the case now; ticks are now well established and all life stages can be found. Canada now has its own endemic population.

Speaker David N. Fisman, MD

What are the implications as far as human epidemiology is concerned? A group from Michigan, Ethiopia and Columbia looked at malaria dynamics in areas where malaria is endemic in valleys and uncommon on surrounding mountains. They looked at mean altitude (cases/time) in mountainous regions of Columbia and Ethiopia and how that tracked with temperature. Malaria dynamics show increased biting rates and increased larval development at higher temperatures. There is reason to think the effects of warmer temperatures on vector dynamics and increased rainfall could facilitate the movement of malaria up hillsides. In some countries this is significant because a lot of economic and productive

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activity occurs at higher elevations because of the populations living there, such as in Bogota. Results showed that the median altitude of cases seems to increase over time in both countries; plotting mean temperature/median altitude shows a near linear relationship, the warmer it is, the higher up the cases are going. This is new work in the field although this observation and projection has been around for about 10 years (AS Siraj et al, Science 2014).

Dr. Fisman returned to the topic of vector-borne diseases, focusing on lyme disease in North America. One of his colleagues in Montreal is interested in ticks and in 2006 tried to link up ITCC climate-warming model projections of lyme disease, i.e., when can we expect to see lyme in Canada over time? A graph of model-derived temperature limits for *Ixodes scaluparis* used a baseline up to year 2000; at this time lyme was probably endemic in Nova Scotia and Southern Ontario. Their projection was that by 2020 there may be a bit of lyme in western Ontario and a some movement into the prairie provinces of Saskatchewan and Manitoba, then and coming into Alberta, which is very cold, in the 2080's.

The reality is that this is occurring much faster than projected. One example was reported by ProMed in July, 2008, where the first case of Canadian human granulocytic anaplasmosis was reported in Alberta. The patient, an older man and avid dog walker, had not traveled outside the city of Calgary in many years. He was thought to have been bitten by a tick during his walks through local wooded recreational areas.

Dr. Fisman and colleagues constructed a lyme disease risk map, looking at overall incidence over time of lyme in the US from 1993 to 2007, and the disease is gradually climbing. They then looked at changes in lyme disease risk (density over time), state by state, in the US. They found that the further north you go the change tends to be up. The further south you go, the delta tends to be flat or to decrease.

Dr. Fisman then went on to speak of mosquitos and climate change. Another study found that in different states there do seem to be different temperature and precipitation effects on West Nile with heavy rains. It is unclear why the heavy rains affect case occurrence, while temperature does drive normal larval maturation and biting rates up. In trying to predict future disease trends weather risks are sometimes not readily identifiable.

El Niño data was used to get some sense of the big picture in the US and Canada as to what climate change means to communicable disease events. El Niño's are periodic irregular thermal inversions in the Pacific Ocean associated with extreme weather events, heavy precipitation and elevated temperature. These occur naturally and may provide insight into climate change but because they occur naturally and are relatively rare, other indices may need to be evaluated. A distributed lag model of vector-borne disease risk showed two peaks in risk, consistent with the biology of most vector-borne diseases. This suggests that different effects may be operating over different time periods; we may have short-term effects of warming and rainfall and longer effects of warming and rainfall. The overall relative risk estimates of hospitalization for vector-borne diseases can be generated from this. The implication is that we can expect an increase in vector-borne diseases.

Relatively less work has been done on water and foodborne diseases and their potential to change under the climate change scenarios, than on vector-borne diseases. Viral, bacterial and protozoan pathogens such as *Salmonella* and *Shigella* and toxin-producing *E. coli*, continue to be important causes of morbidity in North America. Many pathogens causing gastroenteritis have a marked seasonality. In the summertime we have *Campylobacter* and protozoans, in the winter there is always norovirus, rotavirus etc. This also applies to pneumonic pathogens such as *Legionella*, for which there may also be environmental drivers, as it has a marked summertime seasonality in many regions. There is good empirical data to suggest that large waterborne disease outbreaks are often preceded by usually large rainfall events. Looking at cholera cases in Haiti and the difficulty there dealing with sewage, if you have a large rainfall that brings sewage into drinking water, it is not surprising that infectious diseases increase. However, while this may be expected in countries such as Haiti, Dr. Fisman mentioned a disaster in Ontario about 15 years ago, where mandated testing of water by provincial public health laboratories was

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eliminated, and the largest outbreak of toxigenic *E. coli* in their history occurred, including a number of fatalities. The disaster was actually preceded by a fifty-year rainfall event, in this is a large cattle-farming area, with wells used for drinking water. Thus extreme rainfall carried cattle feces from the field into the wells, and there was no public health oversight of water quality. Hurricane Katrina showed us that strong winds can whip water and waterborne pathogens around. There was much unusual *Vibrio* disease in New Orleans afterwards (MMWR, Sept, 23, 2005). Extreme weather events such as tornadoes, hurricanes and cyclones, which are projected to increase, can generate aerosols and environmentally abundant pathogens can be forcibly inoculated into human skin and soft tissues. An increase in the severe of respiratory infections *(B. pseudomallei*) after monsoon rains in Northern Australia was observed. *Clostridium* infections, atypical mycobacterial infectious of skin an soft tissues, and multi-drug-resistant gram negative rod infectious were seen as a results of the 2004 South Asian tsunami.

Ecologists seem to think that environmentally abundant pathogens can cause outbreaks only when the reproductive number exceeds a certain threshold. When temperatures increase (looking at cholera and ocean temperatures) there will be a longer time period during the year when a pathogen can cause disease. In five Philadelphia counties there was a massive surge in legionellosis in the early 2000's, which was not expected. This was similar to cholera data from Haiti, where upstream surges in relative humidity seem to be strongly associated with downstream risk of legionellosis.

A slightly different approach was used to study *Campylobacter* (Philadelphia, 1993-2007), which is a summertime seasonal pathogen. The predictors for disease here were increasing temperature and humidity, and interestingly, decreasing river temperature at 0-4 week lags. *Campylobacter* apparently survives better in cooler surface waters. This lends credence to another model, where we have an environmental sink of *Campylobacter* which is moved around by vectors like flies. If you look at generation times for houseflies they increase very markedly with ambient air temperature. All these interactions and relationships are probably much more complicated, and when you see an environmental signal like summertime seasonality of campylobacteriosis, you may be seeing abundant environmental contamination with the organism.

Dr. Fisman then spoke on mitigation and vulnerability, which is very important as projections have the earth warming by two or so degrees in the next hundred years. Populations particularly vulnerable to climate change include the usual suspects in terms of public health. Heat stress affects the elderly, pregnant women, those with chronic medical conditions and infants and children; air pollution affects children, athletes, and those with pre-existing heart or lung disease. Those vulnerable in extreme weather events will be the poor, pregnant women, people with chronic medical conditions, mobility and cognitive restraints; in food and water-borne illnesses the immunosuppressed, elderly and infants are vulnerable. Vector-borne diseases affect children, pregnant women and outdoor workers. Basically, all people who already bear the brunt of any population level health stress will continue to bear the brunt of population level heath stresses but more so under climate change, i.e. those who already are at increased risk relative to the population as a whole presumably under the climate change scenario will bear much of the brunt of further inducements.

If we look at something like poverty and climate change, the question of dengue in Chicago is a controversial one. One reason to think that the impact of dengue in any parts of Chicago may be less than is suggested comes from a CDC study done in 2007, looking at twin cities of Matamoros, Mexico and Brownsville TX. Mosquitos in both cities are likely to be infected with dengue; however dengue seroprevalence is markedly higher on the Mexican side of the border than on the American side. An ecological analysis done region by region showed that elevated seroprevalence was associated with a low income of less than $100/month. Important variables related to poverty included availability of air conditioning, window screens, street drainage, storage of water in roof tanks that may be a good habitat for larvae, etc. Therefore there

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is reason to believe that poverty at a city, regional, or even individual level can dramatically modify the risk people will experience when it comes to climate change; we already see this with a disease like dengue.

Another example of vulnerability is the summer London heat wave of 2003 for which heat stress and age were plotted. While there was an overall increased mortality in people ages 15-74, there was nearly twice the mortality in those over age 75, and an even higher mortality in this age group in late summer.

Much of climate change evolves around the concept of One Health which promotes the idea that the health of people, animals and the environment are inextricably linked; we have rapid environmental change and it intersects with communicable diseases. Zoonotic diseases are emerging due to an increase in human and wildlife interaction due to increased farming/deforestation. We know that many emerging infectious diseases are either vector-borne or zoonotic and it is estimated that these constitute 70% of the emerging and reemerging infections. Many of these have important environmental drivers. We see yellow fever with urban sprawl into areas that were previously rain forests, animal trade and animal consumption is linked to SARS and nipah virus. etc. There are many challenges with habitat loss and environmental and climate change. We can expect other parts of our ecosystems to be vulnerable to stresses that will influence the animal and vector components of the environment.

Dr. Fisman spoke of interdisciplinary and transdisciplinary research, and said that there needs to be interaction, cooperation and collaboration in the natural sciences, engineering, and all other disciplines in order to have a fully dimensional understanding of infectious disease and to understand the implications of climate change and the challenges we will be facing. We need to break the interdisciplinary boundaries, employing those with skills that are not discipline specific. Some of the challenges we are facing recently, especially in climate change**,** are unprecedented challenges outside the **"**tool box**"**, and not something that can be studied in graduate school or elsewhere.

We do have complex systems approaches to understanding how disease, environment, human-animal interaction might affect the contours of infectious disease epidemics. Lloyd-Smith et al. in Science in 2009 did a literature search and looked at the number of published models for fifty-one selected zoonotic threats; only six had >20, five had 11-20 and forty had 1-10 modeling studies published. They also compared temporal profiles of total research effort and modeling effort for three recently emerging zoonoses, SARS-CoV, *Borrelia burgdorferi* and West Nile virus. For each of the three zoonoses the number of modeling studies was 1/10th of the total number of research papers. It is quite obvious that there is a tremendous amount of work to be done in understanding environmental and climatic drivers.

In conclusion, Dr. Fishman reiterated that global climate change probably is real and probably has major implications for human health. Regarding mitigation, impacts on ecosystems will change the distribution and burden of vector-borne infectious diseases, and the impact is likely to be borne primarily by already-vulnerable individuals and groups. Changes in epidemiology may already be underway. He lastly added that surveillance is so important and so often forgotten about; good public health cannot be done without good infectious or chronic disease surveillance; it would be like flying a large plane without instruments. For example, lyme became a notifiable disease in Canada only in 2009 therefore lyme disease trends are unknown. There are other threats in very vulnerable populations where life expectancy is twenty years or so shorter than for other Canadians. They experience health threats associated with climate change such as echinococcosis and blastomycosis, which are not reportable in the US or Canada. Therefore you have diseases that are concentrated in very vulnerable populations that we are not watching.

Keeping Out of Touch **(Cont. from pg 1)**

Speaker Elizabeth Scott, PhD

Dr. Scott has also studied the survival and transfer of potential pathogens on inanimate surfaces and provided insight into the chain of events that can result in errors of hygiene and the consequences of such errors. It is clear that while foodborne disease is one of the major preventable hygiene problems, there are many other related hygiene issues in the home, the community, and in the food and hospitality industry. Dr. Scott has developed an approach to hygiene practice based upon a risk analysis of an environment and its occupants. The aim is to recognize the level of risk and to produce an appropriate and flexible hygiene policy. The strongest driving force for Dr. Scott's work has been the desire to offer practical information on matters of hygiene to non-scientific audiences who need to put the information into practice.

Infectious disease in the human population most likely became an issue about 10,000 years ago when humans started to move from a hunter-gatherer lifestyle to an agrarian lifestyle, and they started living together in communities. Perhaps, most importantly, they started living together closely with their animals.

Notion of cleanliness began to evolve in London in the early 18th century. Dr. Scott showed a cartoon called "The Morning Gossip", that depicted three housemaids outdoors in the street near the places where they worked, carrying their brooms and mops. What is not depicted in this image is that a strict notion of cleanliness began to evolve at this time because of the appalling conditions of the streets in the cities in the early 18th century. The streets of London were described as "stinking masses of human sewage and animal manure". So people were sent outside to clean the immediate environment outside the homes of wealthy people. In fact, the age of sanitary reformers and the history of advice on hygiene, cleaning and infection control, dates back to at least the mid-nineteenth century. The sanitary reformers were the people who began to address the problems of overcrowded slums, poorly organized sanitation, the filthy streets, and the abominable water supplies. These people were actually the precursors of the public health movement that started in London and quickly moved to the United States.

Edwin Chadwick in 1842 wrote one of the early important reports, *Report on Sanitary conditions of the Laboring Populations of Great Britain* and painted a dismal picture of the vile, overcrowded conditions of England's working poor. The"Miasma" theory of disease also existed, where disease was thought to be caused by bad smells and bad air caused disease. That really led to an emphasis on ventilation, pure air, and odor-free drains. The nurse Florence Nightingale wanted to bring some science to infection control and nursing, and picked up on these themes when she wrote *Notes on Nursing*: in 1859. She listed five essentials for a healthy home: pure air, pure water, efficient drainage, cleanliness and light. This is remarkable in that all this occurred before these people understood the germ theory of disease.

Growing cities in America were over-whelmed with sanitary problems in the 19th century and none more so then New York City. There are many descriptions of filthy streets, disease, overcrowding, and epidemics of yellow fever, cholera, and typhoid. The leading citizens of New York got together in 1864 and formed the New York Council of Hygiene and Public Health. This group reported on the unsanitary conditions in New York City and within a year the Metropolitan Board of Health was formed and was given powers to enforce sanitary measures. This resulted in great improvements and reductions in outbreaks of disease. Again this was all before the germ theory of disease was widely accepted. It was the late 19th century before the medical community started to accept the germ theory and even later when it was accepted by the non-scientific community.

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In the latter half of the 20th century there was a focus on infection control in health care settings; here was the wonderful age of antibiotics. The Surgeon General at that time, William Stuart, made his famous announcement that "Infectious Disease is beaten!", but it never was and only increased. At the same time there was a focus on cleanliness and environmental hygiene in hospitals, but the accepted regime at the time was that the environment played a minor role in the spread of healthcare-associated infections. At the same time there were societal changes occurring that had an impact on infections. It was the era when large numbers of women went to work and had careers. People were leaving rural areas to work in the cities and increased global travel made foreign countries and their diseases easily accessible. The globalization of the food supply brought foreign foods and disease to the local dinner table (you could get traveler's diarrhea at home now just by going to the local grocery store!). In addition, groups of susceptible populations were now brought together in daycare and eldercare settings.

Today there about thirty million deaths annually attributed to infectious disease, with a large increase seen during the AIDS epidemic. We now see a reversal of the role contaminated surfaces play in hospitals; they are now thought to be responsible for endemic/epidemic transmission of pathogens such as *C. difficile*, VRE, MRSA, norovirus and others. Environmental decontamination is thought to play an important role in their control.

Dr. Scott's focus has always been on environments outside of clinical settings, i.e. on home and community settings. She was initially involved in a study that looked at bacteria in homes. Subsequently she continued to work in this area because in the United States, Western Europe and Japan we see a growing immune-compromised population, and we have emerging pathogens such as MRSA that are active in community settings, and there is antibiotic resistance. There are increasing groups of susceptible individuals and home-care nursing; more people are being nursed at home than in hospitals today. Patients are being discharged shortly after surgery, women are being sent home immediately after giving birth and soon afterwards the child may go to daycare. This leads to a whole new set of problems as pathogens can be passed around quickly. Dr. Scott believes there is declining support for hygiene practices in this country. There was (is) also the belief that you won't get sick at home, and arguments that we are "too clean", we use toxic chemicals too liberally at home, and is it right to kill microbes?

Microbial ecology of the indoor environment is a growing scientific field and the question here is what exactly is the ecology, is there a normal flora? We know that wherever there are humans, surfaces are going to be contaminated by organisms we shed. We really don't know the answer. Most of the data we have today has been collected by "snapshots", by sampling an environment and taking samples back to the laboratory. But does the ecology of an environment really change over time according to temperature, humidity, food you eat, etc. It is hard to compare studies done to date because people use numerous different sampling techniques. We know quite a bit about surfaces in the clinical environment, but do we expect nonclinical environment surfaces to be the similar or different? It is also difficult for the general public to accept the fact that there are "good germs" in addition to the "bad germs" they are accustomed to hearing about. In the indoor environment we actually find many representatives of human flora, opportunistic pathogens and some pathogens.

Dr. Scott commented that we know more about the ecology of planes that about our indoor living environment. One study showed a wide diversity of bacterial contamination on frequently touched surfaces of planes and included organisms known to be opportunistic pathogens. Fifty-eight genera were identified using DNA sequencing and BLAST analysis to identify sequences in Genbank. The highest diversity was found on lavatory surfaces including door handles, toilet handles and sink faucets (McManus & Kelly 2005, J. Appl Micro). Species most frequently found belonged to five genera commonly associated with humans*: Streptoccus, Staphylococcus, Coryne-bacterium, Propionobacterium* and *Kocuria*. No one has done such a comprehensive genomic study on our indoor living environments!

Keeping Out of Touch (Cont. from pg 10)

Surfaces in our homes and environments are very complex; risk of infection is generally specific to the type of surface and setting. There are various types of flooring, for example, wood vs carpeting, an (office) floor vs (daycare) floor where opportunistic pathogens may be present. The activities occurring on the floor also need to be taken into account. A floor at home is not too much of a risk unless you have pets running around and perhaps shedding *Salmonella*, and small children crawling around on the floor. Organisms do not fly off surfaces onto humans on their own. The type of surface, nature of contamination of the surface, and most importantly, the extent to which we touch those surfaces determines risk. Floors are therefore not too great a risk, more contamination occurs on higher surfaces.

In order for a surface to pose a risk, you need a pathogen (bacteria, virus, fungus or parasite) and source of pathogenic organisms, which can be people who are infected, carriers, animals in the home, raw foods brought into the home, and temporary reservoirs for the pathogens such as surfaces and equipment we have at home. The means or mode of transmission can be direct or indirect. A vulnerable population is also necessary, such as the very young, the elderly, immunocompromised persons and pregnant women.

Community-based infections are those circulated in the home and other related settings, such as daycare, schools and offices. These include gastrointestinal illness, respiratory infections, skin and wound infections. For these three types of illness there is a chain of transmission that can involve and include inanimate surfaces.

Infectious gastrointestinal diseases are common, but underreported; up to 50% of transmission occurs person to person (in the United Kingdom). There are about 48 million cases of foodborne illness/year in the USA alone, and over 40% of reported outbreaks occur at home. Organisms primarily involved include norovirus, rotavirus and *Campylobacter*. Surfaces such as cutting boards can become contaminated, and illness spread due to poor food and kitchen hygiene.

We also have acute respiratory infections

University of MA Dartmouth
Student Attendees

caused by cold viruses (80% by rhinovirus) and influenza epidemics. Adults have 1.5-3 respiratory infections/year and children under 5 years have 3.5-5 infections/year. Recent influenza epidemics in the USA included 36,000 deaths and 114,000 hospitalizations. These illnesses lead to loss of productivity and present an economic burden. It was learned only in the past few years that hands and surfaces play an important role in the spread and transmission of influenza.

Skin infections, such as community-acquired-MRSA (methicillin-resistant *S. aureus*), which are a major concern in home and community settings, are also common but very under-reported. Staphylococci are documented to be able to survive well on environmental surfaces and can be transferred directly by skin to skin contact and by indirect contact from surfaces. A 2004-05 population study in the San Francisco area showed that 85% of infections occurred outside of healthcare.

The transmission equation shows that hands and high touch surfaces are the two components of infection transmission. Surfaces are a temporary reservoir for pathogens that are contaminated, for example, by humans, pets, and raw foods, and these surfaces in turn can infect other humans, pets, and foods. Pathogen transfer from a surface to hands is highly variable and is species and strain specific; transfer rates appear to be highest from non-porous surfaces.

Contrary to common belief (are we still in the age of miasma theory?), the risk of infection is relatively low from wet reservoir sites such as a bathroom for enteric pathogens such as *E. coli*,

Keeping Out of Touch (Cont. from pg 11)

Salmonella, Shigella or norovirus, unless there is an outbreak or enteric infection. Other reservoirs or sites of pathogen dissemination can be wet mops, sponges, rags, etc., used in cleaning, where the risk of cross-contamination to other surfaces is constant. These items can spread *E.coli* and other coliforms. *Salmonella, Listeria,* MRSA, and fungi.

The risk of infection by hand and food contact surfaces is variable but constant and increases when there are vulnerable people in a particular setting or in an infection outbreak. This applies to organisms such as *E. coli* and coliforms at home; enterics, cytomegalovirus and rotavirus in daycare; MRSA at home and in gyms; vancomycin-resistant enterococci (VRE) in long-term care facilities, and rhinovirus and influenza in offices (who cleans keyboards?). Dr. Scott cited studies that showed *Campylobacter* and *Salmonella* in kitchens on hands, cutting boards and rags (from handling raw chicken). Poliovirus was found on 13% of hand contact surfaces following vaccination of infants. A rhinovirus study in 2007-08 placed 40 volunteers with colds into hotel rooms in which hand contact surfaces had been decontaminated. After one night, rhinovirus was found on 35% of hand contact surfaces sampled in the hotel rooms (door handles, light switches, phones) which are never cleaned by hotel cleaning crews. Another study showed that when hand contact surfaces in hotel rooms were deliberately contaminated with rhinovirus, 60% of volunteers staying those hotel rooms overnight became infected.

Floors, carpets, athletic mats and soft furnishings can also transfer pathogens. *C. difficile* spores, VRE, Group A streptococci are seen in long term care facilities; MRSA is also seen here and in homes and athletic facilities, enterics and rotavirus in daycare, norovirus in hotels and cruise ships etc.

Dr. Scott described several studies showing that study pathogen survival on inanimate surfaces varies. Most gram positive bacteria survive for months on dry surfaces; community acquired-MRSA survives weeks to months on vinyl and plastic fomites; many gram negative bacteria survive for months; GI tract viruses (as rotavirus), survive up to 2 months; and most respiratory viruses survive only for a few days.

Dr. Scott, in a Simmons Publication reported sampling 35 "healthy" homes, selected at random. Thirty-two hand contact surfaces were sampled for a baseline study and with few exceptions, nearly all hand contact surfaces were positive for *S. aureus*; MRSA was isolated from 13 sites in 9 homes. None of these surfaces were considered to be bacterially "dirty" and looked clean. Additional information was requested from these households in order to put these finding in context, such as place of work, infants in daycare, any pets, use of a gym, visits to eldercare facilities, antibiotic treatment, etc. Rigorous statistical analysis of this data showed that homes that had a cat in them were 8 times more likely to have MRSA on home surfaces; she also mentioned that 5% of healthy cats are reported to carry MRSA.

An additional study of household surfaces was conducted in 9 homes; 495 samples were taken (Scott et al, AJIC, 2009). MRSA was found on dish towels, pet food dishes, infant high chairs, bathroom tub and sinks, doors etc. 70% of the surfaces within a 1.5 radius of a hospital were contaminated; that question now is whether community environmental surfaces near hospitals are reservoirs for gram-negative nosocomial pathogens (Rose et al, AJIC, 2014).

Dr. Scott showed a table taken from her 2013 publication listing common touch surfaces associated with the potential transmission of bacterial and viral pathogens in home and community settings. Outbreaks in homes and the community are usually not investigated she said. Organisms isolated in the home setting were *Salmonella typhimurium* from the refrigerator and vacuum cleaner; *Salmonella enteriditis* from toilet surfaces and *Salmonella species* from worktops, sinks and towels; *Shigella sonnei* from toys and toilet seats, sporadic *E. coli* 0157 from kitchen work surfaces, *S. aureus* from clothing, household linens and communal laundry; community-associated MRSA from door knobs, sofas, computer surfaces and joysticks, door handles and computer surfaces; and influenza A virus from handles, faucets, computers and TV remote controllers, switches and telephone receivers. Norovirus was found in long-term care facilities on the toilet seat, dining room table, and elevator button; on cruise ships contaminated communal

Keeping Out of Touch **(Cont. from pg 12)**

bathroom surfaces; in hotels, carpets, toilet surfaces and other frequently touched surfaces had the pathogen. Rotavirus was found in daycare on the water play table, telephone receiver and moist surfaces. Coffee cup handles were found to be contaminated with rhinovirus in controlled community settings and influenza A virus was found on towels and medical carts in a nursing home. [E.scott, American Journal of Infection Control 41(2013) 1087-1092].

Dr. Scott concluded the talk by saying that indoor environments are quite complex and large populations are at risk in the home. One study involved culturing nursing student scrubs when they finished their shift. Approximately 6% of the scrubs were contaminated with *S. aureus.* After laundering, about half were still contaminated. Dr. Scott emphasized that we must focus our studies more on high contact surfaces; pathogens survive on and are transmitted to and from these. To date implications are that the chain of transmission, i.e. hands and high touch surfaces, need to be interrupted to prevent disease. More evidence-based studies are need in this area.

Food for Thought: Cheese Rinds as Model Microbial Ecosystems

The third program of the year sponsored by the Northeast Branch was held on June 16, 2014 at the Hilton Garden Inn in Waltham, MA. Benjamin E. Wolfe, Ph.D. spoke on *Food for Thought: Cheese Rinds as Model Microbial Ecosystems.* Dr. Wolfe is a microbiologist at Harvard University and uses microbial communities of food to address fundamental questions in microbial ecology and evolution. He received his B.Sc. from Cornell University and his Ph.D. from Harvard University. He is currently a post-doctoral fellow with Rachel Dutton at Harvard's FAS Center for Systems Biology and will hold the position of Assistant Professor of Microbiology at Tufts University Department of Biology in September 2014. Benjamin teaches microbiology courses at the Harvard Summer School and Boston University's Gastronomy Program. He also writes about microbes for various publications including *Lucky Peach* magazine, and writes an online series about the biology of food for *Boston* magazine. Dr. Wolfe believes that food is a powerful tool for teaching the general public about science and is especially passionate about using food as a medium to improve microbial literacy.

*(*L-R) NEB President-Elect Nancy Miller, MD ; Gregory Reppucci, Education Chair; Speaker Benjamin Wolfe, PhD; Pat Kludt, Treasurer,and Alfred DeMaria, President.

Dr. Wolfe talked about the goal of the last four years of research to understand how microbes work. Most of what we know was learned from single species microbial models but we are trying to apply this to the multi-species world in which we live. Interactions are extremely complicated however and evolution may differ from that seen in the laboratory environment. We need to study model multi-species microbial ecosystems and Dr. Wolfe is exploring how interactions in microbial communities on cheese rinds evolve over time.

Why study cheese rinds? Cheese is aged traditionally and multi-species biofilms (rinds) occur on cheese surfaces which are comprised of molds, yeast and bacteria. Culture-based studies suggest rind communities are low in diversity; bacteria and fungi live together thus providing a simple model system. Cheese is easily accessible in stores and the microbes can be cultured using petri dishes. Since cheese is likewise a defined substrate the natural habitat of these organisms is easily reproduced. Many cheesemakers produce cheese with rinds thus we

Food for Thought **(Continued. from pg 13)**

have a diversity of cheese rind communities available to study. Questions Dr. Wolfe hopes to answer by laboratory studies are what the patterns of microbial diversity are in nature in these cheeses, how microbial communities are formed, what factors drive community assembly, and are what the molecular mechanisms underlying community assembly.

Dr. Wolfe showed a diagram describing how cheese rinds form. Rinds, the outside shell of cheese are usually edible, and are produced from fermented milk, which consists of solid curds and liquid; the curds are then drained. Depending of the type of cheese, the curds can be heated, salted (using brine or dry salt), and eventually pressed into wheels of various shapes and sizes. These can be aged from weeks to years in a damp, cool place.

Microbes in the rinds can come from a variety of sources. They are present in the raw milk, domestic bacterial "starter" cultures which are purchased are frequently used, and cheese houses have their own microbes in the air, etc. There are also viable microbial cells in salt (ocean microbes), and in caves (cave microbes), where cheese is sometimes stored.

Cheese rinds are formed by the microbes present. Starter cultures and other bacteria continue to grow and metabolize the interior of the cheese, while the exterior is colonized by bacteria, fungi and molds that form a multispecies biofilm (the "rind"). These various bacteria, fungi and molds give cheese their specific characteristics. For example a mold can be inoculated into milk which forms a white rind (bloom) as seen on Camembert cheese. Washed rind cheeses are regularly washed with brine while aging resulting in a washed "stinky" rind that has a yellow/orange biofilm on it such as Limburger. Holes in Swiss cheese result from lactic acid fermentation by a bacterium that produces CO_2 and propionic acid, which gives the Swiss cheese one of its characteristic flavors. The organism grows under anaerobic conditions and CO_2 trapped inside the cheese produces the bubbles/holes seen in Swiss cheese. Or you can do little to the wheels of cheeses and allow ambient microbes to colonize and produce their own rind. Managing microbial rinds involves an incredible amount of work, which can be reflected in the price of a particular cheese. For example, artisan cheese is produced at Jasper Hill Farm in Greensboro, Vermont. Each wheel of cheese is touched by people, some are washed twice a week, holes have to be poked into blue cheese weekly to allow the molds to do their work, and they even have a robot washing heavy wheels of cheese. The function of a rind is to protect and preserve the cheese as well as make it aesthetically pleasing. These surface microbes release enzymes that also age and flavor the cheese. A particular cheese will look similar and have similar organisms if the right environment is created, regardless of whether the cheese is made is California, Vermont or France; geography is not significant.

Dr. Wolfe noted that little is known about what is on rinds and then spoke of research which is being conducted on cheese rinds. Italy & Europe characterized a few rinds using culture-dependent techniques. Neither metagenomic studies nor a widespread geographic sampling has been done to date, and no North American samples were ever studied. Laboratory studies conducted at the Harvard Laboratory are trying to answer what the patterns of microbial diversity are in nature, how microbial communities form, what factors drive communist assembly, and what are the molecular mechanisms underlying community assembly.

A large scale study of microbial cheese rind diversity involved rinds from 137 different cheeses collected from the US and 10 different countries in Europe. There were 362 wheels of cheese and included 24 samples of bloomy rind, 52 samples of washed rind, and 61 samples of natural rind. To measure microbial diversity, DNA was released from the cells, purified, amplicons were produced, and microbial profiles and relative abundance of microbes in a sample were observed (PCR based amplicon sequencing). A metagenomics approach can also be used where the DNA of the entire rind community is sequenced. Distribution of fungi and bacteria genera across 137 cheeses showed similarities across different cheeses. There was much variation between cheeses but rind communities were relatively simple in composition. There were about 7 bacterial genera per cheese and 3 fungal genera per cheese.

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The microbes in each rind can be grown and their growth requirements analyzed. Among microbes found were staphylococcus (coagulase negative, not known to be pathogenic and sometimes inoculated using starter cultures), corynebacteria (no human pathogens found), *Pseudomonas fragi* (and others associated with spoilage only), *Vibrio casei* (isolated by the French and has no virulence factors) and *Aspergillus*, which lives on plants was found in low numbers and may be isolated from rinds if herbs were added and not sterilized. *Fusarium* was also found on washed rind cheeses. Overall, there were no pathogenic concerns.

Bloom cheese rinds are enriched with molds and *Proteobacteria* that love to grow on crustaceans (which are made of chitin). Salt (sea salt) is a great environment for *Pseudo-alteromonas*, *Vibrio* and *Halomonas*. Natural rind cheese is enriched with molds and *Actinobacteria;* these microbes love dry environments and the cheese dries out. Other organisms seen on these rinds are staphylococci and others found on human skin, yeast, and various filamentous fungi. Washed rind cheeses appear to be "hybrids" and have a mixture of both bloomy and natural rind microbes.

Two types of bacteria new to food microbial ecosystems and cheese were identified in this study, *Nocardiopsis*, and *Yaniella*, which is found only on Swiss alpine style cheese. Cheese makers were unaware of growing these. It was found that rind type and composition best explain patterns of rind community diversity and moisture correlates with rind composition. Geography is not significant in regard to microbes present. Cheeses may look similar regardless of whether they are made in England or New England; some even have similar microbes. Whether it be made in California, Vermont or France, if the right environment is created, similar organisms will be found at the genus level. Metagenomics revealed novel contributions of marine bacteria such as *Pseudoalteromonas* genomes and enzymes. This particular organism uses gene to attach to crabs as well as to cheese.

Dr. Wolfe's research tries to answer how communities assemble on rinds, how microbes get there and how many get there. They can be introduced by starter cultures found in milk, however, species interaction must exist. In an *in vitro* reconstruction of cheese rind communities in the laboratory, cheese rinds were swabbed and inoculated into 96-well tissue culture plates containing cheese curd. This was done using a cheese from VT. At 63 days full rind development was seen and patterns of microbial succession were highly reproducible.

Plots were made of data vs time and bacteria vs fungi. A shift from a predominance of each colonizing bacterial species to a predominance of molds was seen. This reproducible pattern was seen in each batch of cheese therefore we can recreate succession of microbial laboratory. There is usually a change from yeast to mold and staphylococci to actinomycetes.

Laboratory experiments also confirmed inter-kingdom (bacterial-fungal) positive and negative interactions, such as the production of molecules that inhibit or stimulate bacterial growth. For example, a fungus can produce an enzyme that breaks down proteins and provides amino acids for the bacteria to grow. Research currently being done is focused on these mechanisms . "Fungal superhighways" were also observed on rinds and the question is whether bacteria use fungal hyphae to move around. Fungal networks therefore could facilitate bacterial dispersal within the rind. The molecular basis of microbial interactions, which involves much complex chemistry, is also being studied in an attempt to determine how microbes communicate and interact with each other.

The study of the microbial ecosystems in cheese rinds has proven valuable to artisan cheesemakers and the industry which is using these microbes to make cheeses. Large companies are buying up starter cultures, and may sometimes discontinue some, therefore limiting the diversity of cheese flavors. Most microbes are French and most American cheesemakers desire American microbes, and are therefore looking for microbes in the United States that can be used as starter cultures, such as the endemic strain of *Geotrichum*. They are finding that cheeses made using native cultures instead of industrial ones are better. This would also give American cheesemakers more diversity in the American cheese market. Some artisan cheese makers are investing in on-site laboratories in order to make more varieties

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than currently exist.

Dr. Wolfe believes that food is a powerful tool for teaching the general public about science and is especially passionate about using food as a medium to improve microbial literacy. Results of laboratory studies discussed will be published in Cell in July 2014.

Whither Malaria - From Control to Eradication

Malaria was the subject of the fourth program of the year sponsored by the Northeast Branch on September 18, 2014, and was held at the Hilton Garden Inn in Waltham, MA. Dr. Regina Rabinovich, M.D, Ph.D. is a global health executive with over 25 years of experience in the research, public health, and philanthropic sectors, with focus on strategy, analytics, global health product development, and the introduction and scale-up of tools and strategies resulting in impact on endemic populations. Currently, she is the 2012-2013 ExxonMobil Malaria Scholar in Residence at Harvard University. Prior to joining Harvard, she served as Director of the Infectious Diseases Unit at the Bill & Melinda Gates Foundation (from 2003-2012), overseeing the development and implementation of strategies for the prevention, treatment, and control of diseases of particular relevance to global health, including malaria, pneumonia, diarrhea, and neglected infectious diseases.

Malaria is a global disease which places 40% of the world's population at risk, results in about 220 million cases of illness, and half a million deaths. In the past decade, there has been enormous scale-up of the malaria toolbox: vector control with insecticide-treated bed nets and indoor residual spraying, and diagnosis/treatment with combination therapy. Dr. Rabinovich spoke on how the malaria community is doing at reaching 2015 delivery and impact goals and what else will be required to achieve long term goals; she also covered product development and field implementation. The potential for impact of a vaccine, a robust drug pipeline, and novel vector control tools was explored. Novel strategies targeting elimination include enhanced surveillance and intervention on the major infectious reservoir – humans. Major threats to progress include drug and insecticide resistance, weakening financial commitments, and cross-border and cross-sectoral challenges. Scientific and political solutions were discussed. It is clear that if pressure on the parasite is not maintained, there will be resurgences in areas that are achieving control. She asked whether we are poised for a public health crisis or can we define what will be needed to optimize transition into an exit strategy for malaria?

Dr. Rabinovich first defined malaria as a parasite, then an infection, and an infectious disease; however it is definitely not a single disease but as variety of diseases as defined by Lowell Coggeshall in 1952. Lewis Hackett in 1937 wrote "Everything about malaria is so moulded and altered by local conditions that it becomes a thousand different diseases and epidemiological puzzles. Like chess, it is played with a few pieces, but is capable of an infinite variety of situations.'' Dr. Rabinovich reminded us of this throughout the talk. In this respect, how do we simplify the intervention package and what we want to achieve she asked? Are we flexible enough to deal with the multitude of variations we will find?

Dr. Rabinovich showed a diagram of the *Plasmodium* life cycle from a vaccine perspective, showing antibodies and T-cells. A mosquito feeding on a person introduces sporozoites which go to the liver where they remain for a few weeks, then infect and lyse red blood cells; disease symptoms appear by day 9-11. She pointed out that the most common vaccine targets sporozoites, which are present in the bloodstream at that point for only 5 minutes, as the infection is cleared quickly by the liver. She also pointed out that this cycle can continue for 10 years, and that in a moderately endemic area, 1% of mosquitos will be infected.

Variation is the key element of how one has to think about this disease. There are multiple ecologies worldwide (forests, near infected bodies of water, urban malaria, etc.), with at least five *Plasmodium* species infecting humans, *P. falciparum, P. vivax, P. malariae, P. ovale*, and *P. knowlesi*. Each species is composed of multiple strains and simultaneous infection with

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different strains of malaria can occur. *P. falciparum* is most likely to kill and we are learning more about *P. vivax* regarding its ability to cause both chronic illness and death. Several mosquito vectors can infect humans and human clinical disease can vary from asymptomatic to coma. Immune responses vary, which is challenging for vaccines. There is also vector feeding heterogeneity, requiring different interventions. Therefore malaria is a "complex and heterogeneous biological phenomenon". This is important to remember because we need to simplify it.

Malaria transmission existed everywhere worldwide in 1945 except in several countries such as Mongolia, Scandanavian countries, Greenland, and New Zealand. Even swampy Washington, DC at that time had polio, malaria, and yellow fever! This was not so long ago, then changes occurred! A Malaria Eradication postage stamp was issued in the United States in 1962 with the elimination of malaria transmission here in 1951 during the Global Eradication Program. From then until now we have had about 2000 cases/year, with some local transmission because we have a vector in the south, but we do have a good health and public health infrastructure to take care of this. But what happened everywhere else? A 1999 chart showed over three million deaths from malaria in Africa in 1900, about 2.5 million in the Americas, and about 300,000 in Asia. DDT started being used for control in the 1930's and there was a precipitous drop in cases until the late 1970's. The WHO Malaria Eradication Campaign was launched globally in 1955 following the success of the Malaria Elimination Program in the US; it was concluded to be a failure and was stopped in 1974-75; surprisingly, it never attempted to attack the location where most of the malaria exists. The Campaign worked in the US, in several Latin and European countries, but was never attempted in Africa, where 80% of the deaths occur, because it was too difficult to do in that country for numerous reasons and could not be done with the available means. And this was driven by drug resistance. *M. falciparum* had become chloroquine, sulfadoxine-pyrimethamine, mefloquine and DDT resistant.

(L-R) Speaker Regina Rabinovich, MD, MPH, NEB Past-President Jeffrey Klinger and NEB President, Nancy Miller, MD

What changed over the past decade that led to underlying problems we faced with malaria in the year 2000? There were dramatic funding increases. The Global Fund and malaria initiatives were created, and we are now probably burning about 2-3 billion annually, including research and development funds. Malaria control was done successfully in colonial times with public health measures, water, DDT, spraying, etc. but that infrastructure and the entire system fell apart. No one believed in that day and age that scaling up programs in Africa could make a difference. There was also an emphasis on disease eradication; smallpox, rinderpest, guinea worm, and polio were targeted instead.

We likewise faced demographic changes: economies in countries that we thought were really poor were changing. Nigeria, for example, where most of the deaths from malaria occur, would have financial advantages from trade. How does one handle projections for the future for such countries in poverty where economic development is occurring?

Malaria transmission worldwide in 2008 was quite different from that in 1945. The US, Canada, most of Europe and Australia were now clear of malaria. The countries that had planned for elimination of malaria are now malaria free; how did that happen? Melinda Gates in 2007 stated that any goal short of eradication of malaria is accepting it and that is just unacceptable. It's rich countries saying they don't need to eradicate malaria worldwide as long as they have eliminated it in their own countries.

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The Gates Foundation looked at what it takes to sustain the malaria eradication program at 80% implementation of the anti-malaria package, and 5-6 billion/year would be needed just to control malaria in all countries. The malaria team said it was not possible to spend this much- it was one-third of the overseas development funding for everything The exit strategy would be to eradicate it, and then we didn't have the right diagnostics, vaccines, drugs, or tools to eradicate the disease.

Dr. Rabinovich then differentiated between eradication and elimination. Elimination is a soft language applied to a public health threat and is usually used to refer to a geographic location, a defined area. WHO defined malaria eradication in 1963: "Malaria eradication is to extirpate the roots of the infection-the parasites…so that the parasites will find none to transmit." Looking at the research & development pipeline, how can we make eradication possible?

The global diversity and behavior of the *Anopheles* vector greatly influences and determines the type of intervention needed. This is important when you are making a genetically modified mosquito; each species needs to be changed. There is a preponderance of one species over another in different places and there are also behavioral differences in the numerous species worldwide; endophilic species prefer to rest indoors, while exophilic species prefer to rest outdoors, though this can differ regionally based on local vector ecotype. Indoor walls of houses need to be sprayed every 4-6 months with a long-lasting insecticide for indoor types. Emptying a home of all furniture, spraying, waiting for the spray to dry, then replacing furniture is a tedious process and works in many cases but depends on the density of the population. Control of outdoor mosquitos is more difficult.

While we have really done well with malaria control, we currently need to be worried about Africa said Dr. Rabinovich. Drug resistance has previously arisen along the Thai-Cambodian borders and now there is resistance to artemisinin. From here, resistance can jump to India and eastern Africa, and there is a huge campaign underway to eliminate malaria in this region as a way to eliminate the parasite that carries resistance.

Another cause for concern in African vectors over the past decade has been pyrethroid resistance and vector behavioral changes. *Anopheles* mosquitos have become resistant to insecticides in 64 countries and counting. Indoor biting mosquitos can change to outdoor biting, and biting preferences can change from nocturnal to diurnal. New vector interventions for this have included insecticide-treated bed nets, special repellents and larvicides. The Integrated Vector Control Consortium in on track with a number of such interventions and research into the problem. A number of these are already in use and work well.

One thing that was done after the 2007 malaria decline was not published until January 2011. Proposed key responses to malaria elimination included discussing eradication as a goal instead of control. Discussions included single exposure drugs, single dose drugs for mass administration, things that interrupted transmission, surveillance as an intervention, vaccines, new diagnostics, predictive modeling, etc. Much work needed to be done, interventions needed to be combined and the system had to be improved. But by the 2011 publication date, these ideas were already two years old.

The 2013 WHO World Malaria Report of incidence and mortality showed that by 2012, about 30 countries had scaled up malaria control. However, we still have those countries such as Nigeria that are responsible for 60% of the deaths and 50% of the cases. The difference is that we started off with 240 million cases and about 1 million deaths; today we have about 210 million cases and 660,000 deaths. Deaths have been cut by about half, to the best of our knowledge, but we still have the number of cases because this was not a transmission stopping/blocking initiative, it was a disease-control initiative.

Dr. Rabinovich also spoke of the varied health systems that exist in Africa, therefore malaria is controlled and managed by different people, even in a single country. There are public (Zambia) and private sector (Nigeria) systems; for example each state in Nigeria has different health systems and the infrastructure for malaria management is enormous. The

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private sectors are interesting in that they can be for profit, premium for profit or non-governmental. Public sector systems can vary from health extension workers (Ethiopia) to primary health centers (Madagascar).

Current core primary malaria control interventions include Insecticide-treated bed nets, rapid diagnostics with finger-prick, drugs that are now readily available as a combination (artemisinin combination therapy, ACT), vs previously used single drugs (primaquine), and indoor residual spraying.

Fifty countries will attempt to reduce malaria case incidence by 75% by 2015 but these account for only 3% of total estimated cases; about 50% of the cases come from African countries, and there is insufficient data to access trends there. This should have us worried about what we know is going on and much energy will be put into surveillance to learn what we do not know.

Morality rate estimates between 2000 and 2012 showed a 42% decline globally and 48% decline in children under 5 years of age because of the way interventions were distributed. Tests currently used to detect malaria parasites are microscopy and Rapid Diagnostic Tests (RDTs). Interventions were focused on symptomatic people where parasitemia can be picked up readily by a trained microscopist. However, diagnosis of symptomatic persons seeking care is the tip of the iceberg only, while infected asymptomatic people not seeking care are the majority of people in malaria endemic area. These people have lower levels parasitemia that will be missed by the available tests as the levels are below the limit of detection of microscopy or current RDTs. Therefore mosquitos biting these people will most likely not transmit the disease but they do constitute a huge reservoir for transmission.

Data were generated in 2009 comparing the prevalence of *M. falciparum* infection in asymptomatic people as determined by polymerase chain reaction (PCR) vs microscopy. At the lowest prevalence 10-35% of the infections were missed, at a higher prevalence 50-79% were missed.

Dr. Rabinovich described two other important points to consider in malaria control. If the disease is interrupted but not eliminated, a high resurgence will occur in about two years, as has occurred in numerous countries where programs have scaled up then relaxed or ended. Resurgence occurred in 36 of 49 (73%) countries participating in the 1955 Global Malaria Eradication Program that failed to eliminate malaria. The disease had been reduced to 18 cases in Sri Lanka; it no longer became a priority and now they have 160,000 cases. The other point is that if malaria is eliminated, it remains eliminated. The disease cannot reestablish itself and this situation can actually be sustained if you have a good infrastructure and intervention package to identify and treat cases. This is seen in the 70 countries that eliminated malaria between 1945 and 2010.

Another cause for concern in African vectors over the past decade has been pyrethroid resistance and vector behavioral changes. Indoor biting mosquitos can change to outdoor biting, and biting preferences can change from nocturnal to diurnal. New vector interventions for this have included combination bed nets, special repellents and larvicides. The Integrated Vector Control Consortium in on track with a number of such interventions and research into the problem. A number of these are already in use and work well.

A strong global malaria vaccine pipeline exists and a vaccine is feasible. People living in endemic regions have become clinically immune from severe disease, rradiated sporozoites have been found to protect human volunteers from malaria challenge, and passive transfer of antibody protects human volunteers. Researchers are looking for places to intervene and are targeting all life stages of the parasite. One new strategy is to interrupt transmission from the midgut of the mosquito. There are several vaccine candidates in Phase 2b trials but the only one in Phase 3 trials in Africa is RTS,S, which demonstrated an efficacy of 50%. Vaccines have to be very safe to be used for eradication. Drugs as artemisinin can also be used but the drug must be used in a very different way to block transmission. All these strategies are collaborations between academia and industry. New medicines being developed for eradication include single dose drugs, but

Whither Malaria (Continued. from pg 21)

how these will be used is under examination in population trials using a number of strategies; for example, should we test and treat, or treat and don't test, etc.

We currently don't have new drugs or vaccines but these no doubt will be introduced in the next 3-5 years. New strategies have always been a problem as there will need to be changes made in the countries involved. The funding of programs involves multilaterals, foundations, research and academia, the private sector, donor countries, NGOs, and the countries themselves, which put in more funding annually. Therefore malaria control cannot be a single effort; there has to be cooperation and coordination. How can this be done especially in the post 2015 agenda asked Dr. Rabinovich. What is changing?

The World Bank has become focused on the eradication of extreme poverty, and regarding United Nations (UN) Millenium Development Goals, a high level UN panel commented that we have been doing this (malaria) for fifteen years, what did it achieve? Malaria is competing against a broader agenda of chronic diseases, such diabetes, hypertension, etc., and does health remain an agenda? Does it involve malaria? There is also the malaria eradication strategy focusing on the human reservoir and strategic use of drugs in large scale programs led by Alan Magill at the Gates Foundation who joined the Global Health Program in 2012.

Numbers currently show that progress has been made in fighting malaria. Much is known about the hardest geographic, population and strategic areas. There are only two alternatives left failure or success. The question is whether we should be approaching eradication, and the alternative is failure. Dr. Rabinovich also spoke of managing our knowledge of malaria. We have much data and experience with the disease, but there is much more to collect, and much to learn in the field that needs to be used to fill in major gaps. There is also managing uncertainty.

Regarding the approach to elimination, we know malaria thrives in the most distant disenfranchised at risk populations. If we don't start with Nigeria we will be dealing with Nigeria in the very end, and we need early successes. How do we organize ourselves? How do we bring the leaders to the table to account for the success of their organizations, and how do we achieve these goals? She quoted Don Hopkins/Carter Center who said "There is no point at which an eradication campaign gets easier." This will go on for a long time said Dr. Rabinovich.

Malaria is down to 500-600,000 deaths/year which is good, but we need to do more by way of controlling the disease. There is a projected shortage of about 20 million dollars for 2014-2015 funding for malaria while 2500 million dollars is needed just to sustain the elimination program. Dr. Rabinovich concluded with the words of Nelson Mandela, which were " It always seems impossible…until it is done." Malaria eradication may not happen in our lifetime she said, but it is something we cannot fail to think about.

Malaria: Additional Cause for Concern-from the Boston Globe

(excerpts from an article by Jeffrey Gettleman, New York Times)

Zambia - Mosquito nets are widely considered a magic bullet against malaria – one of the cheapest and most effective ways to stop the disease that kills at least half a million Africans each year. But mosquito nets not being used as global health experts have intended, they are being used as fishing tools. Across Africa, mosquito–net fishing is a growing problem, an unintended consequence of one of the biggest and most celebrated public health campaigns in recent years. The nets have saved millions of lives, but scientists worry about the collateral damage: Africa's fish.

Part of the concern is the scale. Mosquito nets are now a billion-dollar industry, with hundreds of millions of insecticide-treated nets distributed in recent years, and many more on the way.

Net holes are smaller than mosquitoes and the mesh traps much more life than traditional fishing nets do. Scientists say that could imperil stressed fish populations, a critical food source for millions of the world's poorest people.

Scientists are hardly the only ones alarmed. Fistfights are breaking out on the beaches of Madagascar between fishermen who fear that

Malaria **(Continued from pg 22)**

the nets will ruin their livelihoods and those who say they will starve without them. Congolese officials have snatched and burned the nets and in August, Uganda's president, threatened to jail anyone fishing with a mosquito net.

Many of these insecticide– treated nets and dragged through the same lakes and rivers people drink from, raising concerns about toxins. One of the most common insecticides used by the mosquito net industry is permethrin, which the US Environmental Protection Agency says is "likely to be carcinogenic to humans" when consumed orally. The EPA also says permethrin is "highly toxic" to fish.

The leading mosquito net manufacturers say their products are not dangerous. Still, many nets are explicitly labeled: "Do not wash in a lake or river." Some labels go even further, warning people to pour any water used in washing a net into a hole in the ground, "away from home, animals and wells".

The Secret Lives of Parasites: A Cultural Perspective

(L-R) Speaker Rosemary Drisdelle and NEB Councilor/Program Chair Carol L. Finn

The final Northeast Branch program of the year was presented on October 27, 2014. Rosemary Drisdelle, a freelance writer and medical technologist certified by the Canadian Society for Medical Laboratory Science, spoke on *The Secret Lives of Parasites: A Cultural Perspective.* After fifteen years in the microbiology laboratory at the Queen Elizabeth Health Sciences Centre in Halifax, Nova Scotia, she focused on the fascinating world of parasites and subsequently obtained her Advanced Registered Technologist certification in parasitology in 1995. Continuing her studies, she graduated from Mount Saint Vincent University in 2005 with a BA in sociology. This eclectic educational mix gives her a unique perspective on the relationships between parasites and people in both the global and medical areas. She continues to teach both clinical parasitology and forensic parasitology to medical residents and forensics students. Her book *Parasites: Tales of Humanity's Most Unwelcome Guests* was published by the University of California Press in 2010.

Rosemary introduced us to parasites by giving examples of their ubiquitous prevalence among humans. She showed photos of *Demodex* mites and informed us that they live in the hair follicles and sebaceous glands, mainly on our face. A recent study published in North Carolina showed that 100% of people over the age of 18 had *Demodex* mites. "That pretty much levels the playing field, doesn't it?" she said. The mites in a skin section look like cigars with four pairs of legs and a long tail, and fit nicely inside a hair follicle where they eat dead skin and secretions. They come out and walk on your scalp while you're asleep! Most of us are unaware of this; however, since we don't see them we are not disturbed by it.

She then showed a photo of *Toxoplasma gondii and explained that t*he Centers for Disease Control currently estimates that 1 in 5 or 6 Americans are infected with it. *Toxoplasma* is more common in people of poor economic social groups. Therefore, she said, look around at your neighbors; it would be safe to say that someone in the audience is carrying the organism.

The definition of a parasite is something that lives on or in a host, getting all it needs from the host. We are living in a time when people are in one of two camps she said. Some think that in developed countries there are no parasites or that they are insignificant; others think everyone is full of parasites and that they are dangerous. Both think eradication is necessary. Rosemary however, thinks all organisms have some value; parasites balance the population and ecosystem; some need food others are food; even parasites have parasites.

Secret Lives of Parasites (Cont. from pg 23)

Rosemary then described the life cycle of a hookworm. Hookworms enter a human host from the ground, are carried into the lungs by the blood stream, are coughed up, and swallowed, then suck blood and lay their eggs in the small intestine. The eggs are then released with feces into the environment, and the cycle repeats. The hookworm however, lives in a limited geographical area; the eggs need warmth. Socioeconomically, it is a disease of poverty and is found where toilets are not available or are not used. The parasite does not pass from person to person. Symptoms include wasting, stunted growth etc. Having a hookworm infection and having the disease are two separate items; having a few parasites only may even be beneficial. She spoke of the "Old Friends Hypothesis" which is that organisms such as parasites and bacteria evolved with humans; reduced exposure to them could

North Shore Community College Students and Speaker Rosemary Drisdelle (C-L) and Prof. Gregory Reppucci C-L)

possibly be involved in immune system disruption. If the immune system evolved to kill invading organisms; immune responses need to be down-regulated so that we would not attack ourselves. Some diseases such as inflammatory bowel disease, multiple sclerosis, allergies, Crohn's disease, diabetes, depression and perhaps Alzheimer's may benefit from having the parasites present. Hookworms may be legally sold in England, they may also be shipped to Canada, and are sold over the internet. Rosemary witnessed such a sale; the hookworms were put on a bandage, placed on the buyer's skin and allowed to penetrate

The hookworm is also a historian. If the human migratory route to the Americas is mapped, hookworm would be found in South America and could not have come here through the frigid Behring Strait. This tells us that the Behring Strait was not the only way humans came to North America; they also came from the south. In addition, during the Civil War, the level of hookworm infestation was higher in the slave population than among poor southern whites. It is thought that hookworm arrived in the Americas with African slaves. However, the slaves had less disease than the whites, who were more susceptible (was this a factor in helping the North win the Civil war?).

Rosemary returned to *Toxoplasma*, a parasite of cats, its only definitive host. She commented that this is an organism to watch and study. *Toxoplasma* needs to be eaten by cats. Normally rats and mice fear cats, but when they are infected with *Toxoplasma*, the rodents lose their fear and are easily caught and eaten by the felines. Thus the intermediate host (rodents) and definitive host (felines) are brought together by the parasite. One-third of the world's population is thought to be infected with *Toxoplasma*. Rosemary described its life cycle which concludes with cats passing billions of oocysts into the soil. *Toxoplasma* can also infect warm-blooded hosts such as humans, who are dead-end hosts. While healthy people may only have mild flu-like symptoms, the disease can be serious in immunocompromised people and pregnant females, in which there may be abortion, impaired fetal brain development, etc. While *Toxoplasma gondii* infection is not considered to be serious, what does it actually do to humans? Suicide and depression have been linked to high levels of *Toxoplasma* antibodies in the blood. Countries with high rates of *Toxoplasma* infection have higher suicide rates but the link to *Toxoplasma* is yet unclear. Other behavioral changes observed in infected vs non-infected people are slower reaction times, a shorter attention span, females becoming more outgoing and persistent, males taking more risks, being more jealous and suspicious, and both genders fearing uncertainty. Therefore *Toxoplasma* may affect human behavior as it

Secret Lives of Parasites **(Cont. from pg 24)**

does in rodents but many cultural factors may also play a role.

Guinea pig domestication, particularly in areas where the "Kissing Bug" is found, is thought to have also resulted in the domestication of the triotomid that that carries *Trypanosoma cruzi*, the cause of Chagas Disease. The triatomid was originally thought to have been a parasite of wild guinea pigs, but was brought indoors when guinea pigs were domesticated. It quickly adapted to a human food source, and now lives in human homes. One result of this is that blood and organ donors must be screened for the disease as the parasite has been shown to be transmitted in this way.

Rosemary showed a photo of a community in Thailand, where people, pigs and chickens reside in the same home. Pigs are allowed to run free to eat anything they can find such as food scraps and even human waste that is usually deposited on the ground, as there may be only one toilet per village.

In Mexico, a similar situation exists with the pork tapeworm *Taenia solium*, and she presented a diagram of its life cycle. Pigs ingest human feces or other material contaminated with worm eggs or proglottids, and humans in turn are directly infected by eating contaminated, raw or undercooked meat. In humans, larvae can migrate anywhere in the body, including the brain, and depending on their location in the brain, this can result in epilepsy, seizures and neurocystercosis. In endemic areas about 25% of people are infected. These infections are due to poor hygiene and lack of fuel for cooking. Thus our cultural behaviors have made parasites successful.

Rosemary illustrated how our view of parasites is very different from what the perception of other people can be by showing a photo of a village on a river in Cambodia. She traveled on that river, sitting on the roof of a boat; the river was the center of the community for the local people. Closer to a large lake nearby there were floating villages; everyone lived above the water, including animals that were kept on rafts. These people used river water for food preparation, cooking, washing dishes and clothing, and bathing. At a floating restaurant, the toilet was a hole in the floor over the water. It could be assumed that all people on the river must have parasites due to the highly contaminated water, but here intestinal worms would be a way of life. The people look healthy and may have good immune systems even though they do have a parasitic disease. One of the parasites, the Chinese liver fluke, for example, lays eggs in fresh water which are eaten by snails; a larval stage infects freshwater fish, which are in turn eaten (undercooked) by humans. Adult flukes, present in the liver and bile ducts, produce eggs which are excreted into the water in feces via privies built over the water to serve as food for the fish; and so the cycle continues. It has only been about 50-60 years since we have been able to prevent ourselves from getting worm infections; and we still get pinworm today. However, it would be a daunting task to bring sanitation to this area. Therefore it is important to remember that not everyone is repulsed by parasites; to many people they are just part of the fauna.

Some parasites show an evolutionary adaptation to the environment and can control us as well as other hosts. There are parasites in crabs for example, that can turn a male crab into a female and causes it to care for the parasite's young, as well as a parasite that will force an ant to climb to the top of a blade of grass until sheep come along and eat it. The horsehair worm found in freshwater or marine environments parasitizes grasshoppers. The infection causes the grasshopper to seek water and drown itself so the worm can get back to its watery environment to complete its life cycle. Another similar adaptation of a parasite to its environment is seen with the Guinea worm *Dracunculus medinensis* in humans. Female worms in the abdominal cavity migrate to subcutaneous tissues, and form uncomfortable blisters that itch and burn on the extremities. When the blister is immersed in cool water (pond, river, water bucket) to relieve the discomfort, the worm expels larvae into the water, which are ingested by water fleas, and humans in turn drink the contaminated water, repeating the cycle. There are no drugs to treat Guinea worm disease and it has caused much debilitation and misery. The Guinea Worm Eradication Program, supported by the Carter Center and other organizations, is involved in bringing about cultural changes such as

Secret Lives of Parasites **(Cont. from pg 25)**

educating people to filter water and not to drink from unsafe sources, not to soothe lesions in a pond but to use a bucket. These cultural changes have successfully decreased the disease worldwide to only 148 cases, and soon this may be the second disease, next to smallpox, to be eradicated.

Thus our various cultural behaviors have made parasites successful and they affect our daily lives more than we can imagine, in both good and bad ways, and they in turn adapt to our changes; and this may always be the case. Humans have spread pinworm, hookworm and *Toxoplasma* worldwide. However, as she previously stated, Rosemary believes that all organisms have some value, and play a role in our lives, including parasites.

Science Fair Winners

Annual support was again provided by the NEB to the five Massachusetts regional fairs (Worcester Regional Science and Engineering Fair, Rensselaer-BCC Science Fair, Somerville Science Fair, South Shore Regional Science Fair, Boston Public Schools Science Fair), the Massachusetts State Science Fair, and the Vermont State Science Fair.

Following are some of this year's winners of the NEB awards and their projects. Congratulations again to the students for their outstanding work. . Names of the winners at the Region 4 Somerville Science Fair, the Massachusetts State Science Fair and the VT State Science and Mathematics Fair were not available at this time.

Region 2: Worcester Regional Science and Engineering Fair: *Early Diagnosis of Herpes viridae Viral Infections by MIP's.* Xiayue Wang, Mass Academy of Math and Science, Worcester, MA.

Region 3: Bristol Community College-Rensselaer Polytechnic Institute Regional Science Fair: *DNA Damage.* Jillian J. Braga, Age 17, 11th Grade, New England Christian Academy, Berkley, MA.

Region 5: The South Shore Regional Science Fair had two winners: Sophia AiDong, Age 15, Grade 10, Plymouth North High School with *UV Light, the Key to Bacterial Dominance* and Phya Han, Age 17, Grade 11, from North Quincy High School with *Digestion of Different Proteins in Stomach Acid.*

Region 6: Boston Regional Science Fair: "*Novel Engineered Oral Vaccine Against HIV/AIDS.* Jonathan Zou, Boston Latin School, Boston, MA.

Thank you again to NEB members who served as judges.

New England Microbiology Laboratory Directors Meetings

The New England Microbiology Laboratory Directors group has been meeting at the Publick House in Sturbridge twice a year for the past thirty years in order to share information and their experiences in the laboratory. The informal half-day agenda consists of presentations by attendees. The meetings are attended by physicians, laboratory directors, epidemiologists and laboratorians from New England. Please contact Alfred.DeMaria@state.ma.us if you would like to receive meeting information. Meetings are supported in part by the NEB.

66th ASCLS:CNE Annual Convention

The 66th American Society for Clinical Laboratory Science Annual Convention was held at the Rhode Island Convention Center in Providence, RI on April 29-May 1, 2014. It was jointly sponsored with the American Association for Clinical Chemistry (AACC), Board of Rhode Island Schools of Allied Health (BRISAH), Bay State Chapter Clinical Laboratory Managers Association (CLMA), Northeast Branch of the American Society for Microbiology (NEB-ASM), Rhode Island Cytology Association (RICA), and Rhode Island Society for Histology.

Boston Bacterial Meeting

The NEB was again one of the sponsors of the annual Boston Bacterial Meeting (20th BBM 2013) which was held at the Harvard University Science Center on June 12-13, 2014. The meeting attracts Boston-area researchers who are studying the biology of microorganisms in either academic or industrial settings. The NEB Booth was again manned by students from the Boston Area Student Chapter of the American Society for Microbiology.

Packaging and Shipping Division 6.2 Hazardous Materials

This intermediate-level, one-day program was held in December and was designed for laboratorians who package, ship, and transport Division 6.2 hazardous materials such as patient specimens and cultures, A comprehensive overview of regulations applicable to packaging and shipping laboratory specimens was provided. Lectures, demonstrations, and group exercises were used to provide instruction on complying with international, federal, and local transportation regulations. Faculty were from the Hinton State Laboratory Institute and included Tanya Swanson, BS, MT, Packaging and Shipping Division 6.2 Materials Coordinator and Supervisor, Bioterrorism Response Laboratory and Cynthia Condon, BS, M(ASCP), RN Laboratory Coordinator Bioterrorism Response Laboratory.

Hospital Response to Chemical Emergencies

The program was designed to help health care personnel, hospital emergency department professionals and clinical laboratory staff to better respond to chemical emergencies by providing information on the public health response to chemical emergencies and how to properly collect, package and ship appropriate clinical specimens for chemical analysis. The roles of the MDPH HSLI, Massachusetts and Rhode Island Regional Poison Center, Control Center,Centers for Disease Control and Prevention (CDC), and other state and federal agencies during a chemical emergency was also discussed.

Four programs were held at Falmouth Hospital, Cape Cod Hospital, snd DelValle Institute. The program was sponsored at no charge by the Massachusetts Department of Public Health, (MDPH), Poison Control Center of MA and RI, and the Northeast Branch-ASM. Faculty included Jennifer Jenner, Ph.D., Coordinator, MDPH Chemical Threat Response Laboratory and Nicole Gethin, M.S., Assistant Coordinator, MDPH Chemical Threat Response Laboratory.

Agents of Bioterrorism: Sentinel Laboratory Training

This program is designed to provide timely information to help clinical laboratorians understand their role in the Laboratory Response Network as they rule-out organisms and serve as sentinels for persons who may fall ill due to a bioterrorist event. It provided an overview of the clinical laboratory's role in the presumptive identification of primary agents of bioterrorism. Laboratory demonstrations and hands-on learning exercises outlined the microbiology of these agents so that participants can recognize their culture, staining and biochemical characteristics. The safety implications of handling suspected organisms in clinical isolates and suspected toxins was also discussed.

All faculty were from the Hinton State Laboratory Institute, MDPH and included Cheryl Gauthier, MT(ASCP), Director, Bioterrorism Response Laboratory, Sandra Smole, Ph.D., Director, Division of Molecular Diagnostics & Virology, Scott Hennigan, Supervisor, Molecular Diagnostics Laboratory, Tanya Swanson, BS, MT, Supervisor, Bioterrorism Response Laboratory, and Cynthia Condon, BS, M(ASCP), LRN Coordinator.

Northeast Branch of the American Society for Microbiology

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January 1, 2015 - December 31, 2015

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Are you interested in any of the following Branch activities?

__ Working on Committees
__ Running for Office

MEMBERSHIP OPTIONS

__ Individual ($ 15.00 annually)
__ Individual ($40.00 / 3 years)
__ Student ($ 10.00 annually)
__ Emeritus (No charge)
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Renewals postmarked after September 1, 2014 will be effective January 1, 2015. Please make checks payable to: NORTHEAST BRANCH -ASM and send with this form to:

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