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DIARRHEA DIGEST

DIARRHEA DIGEST is an irregular publication of TECHLAB®, Inc. dedicated to the etiology, diagnosis, and therapy of diarrheal diseases and related aspects of intestinal ecology

Fall 2011

E. coli 0104:H4 --- the outbreak strain in Germany

From May to June 2011 there was an outbreak of 3,602 cases of diarrhea caused by Shiga-toxin-producing *Escherichia coli* (STEC). The outbreak originated from northern Germany and resulted in 908 cases of hemolytic uremic syndrome (HUS) and 47 deaths (1,2). The source of the outbreak was contaminated raw fenugreek sprouts. According to the Robert Koch Institut the peak of the outbreak was May 22, 2011. The outbreak strain affected primarily women, resulting in severe neurological complications (3,4). Properties of 0104:H4 outbreak strain include:

- Incubation of 8 days
- HUS developed 5 days after symptoms, compared to 7 days for 0157(1)
- Resistant to ampicillin, cefotaxime, ceftazidime, streptomycin, sulfamethoxazole, trimethoprim, cotrimoxazole, tetracycline, nalidixic acid
- Sensitive to imipenem, kanamycin, gentamicin, chloramphenicol, and ciprofloxacin (4).
- Ferments sorbitol

The Germany outbreak strain was identified as serotype O104:H4 and contained virulence features that were seen in enteroaggregative *E. coli* (EAEC) and enterohemorraghic *E. coli* (EHEC). EAEC are known to cause persistent diarrhea in infants and children in developing countries (5) and EHEC is well known as a food borne pathogen causing diarrhea after contaminated food is ingested. The current outbreak strain was be missed by standard procedures that include Sorbitol MacConkey (SMAC) plates (4).

The pAA plasmid carried by the outbreak strain encodes (i) aggregative adherence fimbriae (AAF/I), (ii) Aat complex (enteroaggregative ABC transporter that transports dispersin onto the bacterial cell surface), (iii) the dispersin protein (to control electrostatic attraction between the AAF and bacterial surface), and (iv) AggR (regulates AAF) that are all characteristics of EAEC (2, 6). The *E. coli* O104:H4 contains a toxin-encoding phage that is similar to 933W phage found in EHEC (2) but with one nucleotide change in each of the subunits (Stx2A and Stx2B) (4). The Germany outbreak strain contains another virulence factor referred to SPATEs (Serine Protease Autotransporter Toxins). The O104:H4 strain has a combination of SPATEs that include SepA, SigA, and PIC. PIC promotes colonization of the gut by clearing the mucin from epithelial cells. SigA causes rounding of enterocytes by clearing the cytoskeletal protein spectrin which maintains the plasma membrane and cytoskeletal structure of cells. SepA function is not known. EAEC usually do not code for more than two SPATEs unlike the outbreak strain. Strain O104:H4 also had virulence factors that included long polar fimbriae (lpf) and *iha* homologue adhesion that are associated with EHEC and colonization of the gut. The O104:H4 outbreak strain contained plasmid pESBL that encodes for extended spectrum beta lactase CTX-M-15 that is a recent addition and not in the 2001 strain, but the antibiotic susceptibility remained the same between both strains (2).

The *DIARRHEA DIGEST* is now green. Just like previous paper issues, the green version will be an irregular publication and it will be available on our website. The green version may not be as easy to take to the bathroom, but by saving trees, the green version will help make sure that you don't run out of toilet paper.

A serotype O104:H4 was previously identified in 2001 in Germany (01-09591; HUSECO41) and 2002 in Central Africa (55989). The O104:H4 strain from Germany is referred to as LB226692. The 2011 strain was similar to EAEC strain O104:H4 from Central Africa (55989) (7) and is not the first EAEC to acquire the Stx2 phage (e.g., O111:H2) (2,8).

In 2011, Mellmann proposed two evolutionary models to describe the origin of the 2011 outbreak strain that included (i) the common ancestor model suggesting a O104 progenitor or (ii) the linear ancestry model that suggest that all EHEC O104:H4 originated from the prototypic EAEC 55989. They believe the common ancestor model is probably the most likely situation in this case. The 55989 strain was more than likely derived from a progenitor STEC O104:H4. The 55989 strain has an *stx* integration site at *wrbA* and carries *iha* (9,10). The 55989 strain was formed by six insertion events, whereas LB226692 was formed by three insertion events and 01-09591 was formed by one insertion event. The 2001 isolate kept the AAF/III fimbriae that was also in 55989 and obtained the type IV pilus and TEM-1. The German 2011 O104:H4 strain obtained plasmids containing AAF/I fimbriae, TEM-1 and CTX-M-15 beta lactamase and lost the AAF/III fimbriae (9). Overall, with the additions of CTX-M-15, AAF/I and the combinations of virulence factors from EAEC and EHEC, this O104:H4 strain is highly virulent.

Davina Campbell

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Caught Brown-Handed --- Heard on the local radio station --- A man slipped over to his neighbor's yard and attempted to steal some cow manure to put on the mushrooms he was growing. The man stated that he needed the manure because mushrooms do much better with cow manure and besides, the stuff was just laying there.

No Vacancy…The Concept of Competitive Exclusion

If you have ever circled a parking lot numerous times in search of a parking spot, you have a general idea of how microbes prevent other bacteria from colonizing your intestinal tract.

The gut is a densely populated ecosystem where bacterial counts range from 10^4 cells per gram in the stomach to 10^{11} per gram in the colon. This biome also boasts a diversity of over 2000 known species of bacteria with many residents still unidentified [1]. Some of the most well known inhabitants include *Bacteroides*, *Lactobacillus*, *Clostridium, Fusobacterium*, *Bifidobacterium*, *Eubacterium*, *Peptococcus*, *Peptostreptococcus*, *Escherichia* and *Veillonella* [2]. We rely on these organisms to synthesize volatile short chain fatty acids, vitamins and other metabolic precursors. More importantly, these inhabitants provide protection from foreign or pathogenic microbes that may be passing through as well as stimulate host immunity. The most basic way bacteria achieve this is known as competitive exclusion. Competitive exclusion is defined as the elimination of another organism by reducing its chances of survival by nutrient availability, attachment sites and reproduction [1].

The innate microbiota becomes established in the early stages of the host's life and maintains a foothold in the gut by binding to the intestinal epithelial [3]. In some instances the native bacteria defend their "home" by producing antimicrobial protein compounds that act as bacteriocins or colicins to reduce the fitness of invading organisms [1]. *Lactobacillus* and *Bifidobacterium* are known to produce short chain (volatile) fatty acids capable of inhibiting the growth of pathogens such as *Salmonella* by creating a more acidic environment [2,4]. Bacteria in the gut must also be able to reproduce rapidly due to the sloughing off of intestinal epithelial cells and passage of waste through the tract. In the event of diarrhea, the host flushes out the intestinal tract as a way to flush unwanted toxins, pathogens or food stuffs out of the body. Therefore large numbers determine which members remain to colonize and which members become displaced. This concept also holds true if the host undergoes antibiotic therapy.

Aside from diarrheal episodes, the diet of the host plays a major role in the diversity of the microbiota. Fructo-oligosaccharides, manno-oligosaccharides, inulin and dietary fibers that cannot be used directly by the host's digestive machinery are utilized by the bacteria in the large intestine [1]. This in turn, boosts the population of the specific genera that are able to benefit from the energy source. Substrates which allow such boosting of existing beneficial bacteria are known as prebiotics. Weanling pigs fed manno-oligosaccharide supplemented feed for a week prior to, during an infection with *Salmonella typhimurium* DT 104, and two weeks after infection had less severe diarrhea, fever and shorter illness duration than pigs only given the commercial feed diet [5]. This is partially due to the significant increases of *Bacteroidetes* and *Lactobacillus* within the intestinal population outcompeting *Salmonella* for colonization sites [5]. Additionally, pigs on the prebiotic supplement gained weight more effectively than sick pigs on the commercial diet [5]. Unfortunately, the benefits from prebiotics only continue as long as the host continues to regularly ingest them [1]. As with any ecological system, the population of the gut can only be sustained if the nutrition source is available. An alternative strategy is to ingest the beneficial bacteria in the form of probiotics.

Probiotics interact with other microbes to alter adhesion of pathogens via secretions of antimicrobial substances or competing for carbohydrate-binding specificities [6]. Additionally, probiotics interact with the host by adhering to the intestinal mucosa stimulating immune responses to increase mucin production, a barrier of the intestinal epithelial [2]. Bacteria often considered probiotic include species of *Lactobacillus*, *Bifidobacteria* and *Propionibacteria*. In order

for probiotics to be effective, they must pass through several stressors including stomach acid, aerobic conditions, pancreatic enzymes and bile salts [3]. The organism should be non-pathogenic and preferably should be isolated from the same species as the intended host [2]. Patients on extensive antibiotic therapies may be given probiotics to reduce the incidence of antibiotic associated diarrhea [7]. In an effort to keep beneficial bacterial populations high, children undergoing antibiotic therapies have been administered *Lactobacillus reuteri* as a preventative for antibiotic associated diarrhea [7]. Probiotics also show promise for individuals with irritable bowel syndrome, inflammatory bowel disease, diarrhea and obesity [7, 8]. As with prebiotics, the level of beneficial bacteria can be altered if/when probiotic regimens end [4].

Synbiotics utilize probiotics with prebiotic compounds. Combining the probiotic organism with its desired substrate enhances the chances of survival through the gut [8]. The most common genera selected for synbiotics are *Lactobacillus* and *Bifidobacterium* which are then partnered with an oligosaccharide such as lactulose, galacto-oligosaccharides and fructo-oligosaccharides [1, 9]. This dietary strategy could assist in the prevention of diabetes, obesity, non-alcoholic liver disease, inflammatory bowel disease and certain cancers [8, 10]. In a study where healthy individuals ingested a synbiotic snack composed of fructo-oligosaccharides and *Lactobacillus helveticus* and *Bifidobacterium longum* for one month, the overall gut profiles of the individuals did not change but the amounts of volatile (short chain) fatty acids, ketones, carbon disulfide and methyl acetate produced significantly increased, indicative of beneficial gut metabolic activity [8]. Another study examined the benefits of lactic acid bacteria and four fiber compounds (inulin, pectin, betaglucan and resistant starch) as part of postoperative care for individuals that had undergone pylorus-preserving pancreatoduodenectomy [10]. This surgical procedure involves re-sectioning the duodenum, gall bladder, bile duct, pancreas and distal pylorus and runs the severe risk of bacterial infection. In addition to the mandatory antibiotic therapy, one group received the synbiotic while the other group received only the four fibers for 8 days after surgery. Results from the study indicate the synbiotic group had a reduced hospital stay and significantly less days of antibiotic therapy were needed (2±5 days versus 10 ±14 days). Additionally, only 5 out of 40 patients that were on the synbiotic acquired a bacterial infection versus 16 out of 40 in the fibers only group. [10]

The synergy of host-microbe interactions allow our immune system to ward off disease and promote digestive welfare. By harnessing the ecologic mechanisms of these microscopic sentinels, pathogens are left to "circle the block".

Heather Totty

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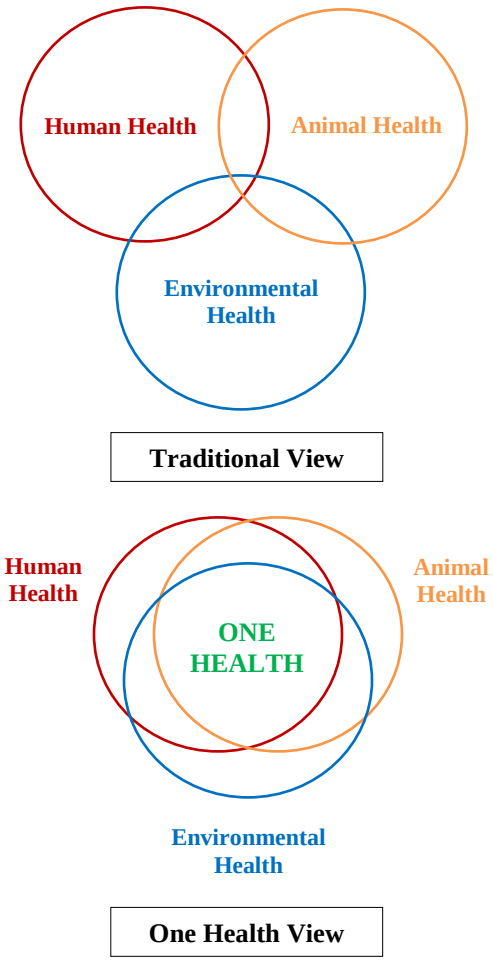
One Health

In 1807, Dr. Benjamin Rush addressed the University of Pennsylvania's Medical School with a lecture entitled *On the Duty and Advantages of Studying the Diseases of Domestic Animals and the Remedies Proper to Remove Them.* The speech emphasized security of the human food supply, evolution of emerging human diseases in animals, influences of animal husbandry on the human ecosystem, and co-development of disease treatments in humans and animals. To an audience intently focused on human medicine, the topics seemed out of proper context. But interestingly, this was an early example of a now rapidly developing "One Health" philosophy of public health.

Today, numerous international and national organizations support the One Health initiative. Medical schools, veterinary schools, and programs in environmental science have added curricula that address overlapping interests and fully encourage interdisciplinary collaborations. Patterns of training and experimentation have evolved from a traditional public health intervention strategy to a One Health strategy that tightly binds the disciplines (see figure). Goals of the One Health model are far reaching and involve governments, health professionals and individual citizens. Current efforts include:

- ❖ Disease surveillance using a pathogen-focused view instead of species-focused view; effectively recording zoonoses.
- ❖ Development of diagnostics, therapeutics and vaccines with cross-species integrity and impact.
- ❖ Enhanced communication between disciplines at conferences, in journals, and through implementation of allied health networks.
- ❖ Education of government officials and the public through timely and accurate interactions with media groups.
- ❖ Renewed emphasis on comparative medicine and establishment of environmental studies within veterinary and medical programs.

Public Health Intervention Strategies



Adapted from Lamielle, G. *One Health Newsletter*. Spring 2011.

More information regarding One Health and the organizations involved can be found at the following sites:

http://www.onehealthinitiative.com/index.php

http://www.onehealthcommission.org/

http://cdc.gov/onehealth/

http://www.avma.org/onehealth/

http://www.vetmed.ucdavis.edu/onehealth/

J. Herbein

The Stick Test --- it's not rocket science but it's more complicated than it appears!

We have been hearing about using the stick test to determine which specimens should be tested for *C. difficile* and its toxins so we actually ran a small in-house study to investigate the stick test in a little more detail. In our study, we included solid specimens, semi-solid, and liquid specimens, and we used commercial medical grade 6 inch wooden applicator sticks --- we were not able to find out which type of wood, although we doubt it's mahogany or balsam. Specimens were from hospitalized patients and their consistency (the specimen, not the patient) was determined based on the Bristol Stool Chart, which we consider to be very informative and descriptive. In fact, we think the stool chart would go well as a wall decoration in some college dorm rooms.

All three groups of specimens exhibited similar prevalence rates for *C. difficile* and its toxins, whether we used antibody-based tests for antigen and toxin or PCR for the toxin B gene (*tcdB*). The high number of positive solid specimens didn't surprise us too much --- we knew that many asymptomatic patients picked up *C. difficile* while in the hospital.

When we used the stick test on these specimens, the results with the solid and liquid specimens were what you would predict --- solid specimens stand the stick up so you don't test them. With liquid specimens, the stick falls over (translated "diarrhea"), so you test them. Like we said, it's not rocket science. But the semi-solid group posed a challenge. Roughly half of the specimens stood the stick up and half the specimens let it fall over. This raises a big question mark in our minds, because in our experience, many of the specimens submitted for *C. difficile* testing fall into the semi-solid group. In fact, we probably see more in this category than in the "liquid" category. This means that many patients who have loose stools --- at least we consider semi-solid specimens to be loose, and most patients with semi-solid specimens would agree that their bowel movements had changed --- would not be tested. Are we "over-interpreting" our stick results? Do you have any suggestions or comments --- other than take the stick and shove it?

Weird Wallabies

What's the difference between a cow and a Tammar Wallaby? Gas of course! While the Tammar Wallaby consumes a diet very similar to ruminants - a variety of grasses - these neat marsupials emit a fifth of the methane of most ruminants. Ruminants harbor methanogens in their gut which produce methane via enteric fermentation. Researchers from Australia and the United States identified the WG-1 bacterial species (*Succinivibrionaceae*), which accounts for this reduction in methane by producing succinate instead.

You may think this is only a fun fact; however the Tammar Wallaby could help reduce methane emissions around the world! According to the Environmental Protection Agency (EPA), ruminants produce 80 million metric tons of methane each year. This accounts for 28% of the methane emissions from human-related activities worldwide! In 2009 enteric fermentation was the second largest contributor to human-related sources of methane emissions in the United States after natural gas systems. If researchers could figure out how to adjust the microbiota of ruminants to include more WG-1 than methanogens this could make a huge dent in methane's major contribution to global warming.

What else does the Tammar Wallaby bring to the table with WG-1? Succinate, produced as a byproduct in WG-1 metabolism, is a major component of the citric acid cycle (a.k.a the TCA or Krebs cycle) – a metabolic pathway that allows organisms to gain energy from carbohydrates, fat, and proteins. Ruminants using typical enteric fermentation lose between 2-12% of the energy contained in the foods they consume. This means that when the Tammar Wallaby is digesting its own food, it is actually more efficient because the byproducts of fermentation from WG-1 are used again to create more energy for the wallaby. WG-1 provides high hopes for saving our planet while still giving the Tammar Wallaby a little more energy and reason to keep on hopping!

Rebecca Easley

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From ICAAC 2011

Keep an eye on the new *C. difficile* antibiotic Fidaxomicin (also called Dificid). Dr. Stuart Johnson (Loyola University, Maywood, IL) reported that this macrolide antibiotic looks promising. Fidaxomicin is active against Gram-positive bacteria, inhibits protein synthesis, and is poorly absorbed by the gut, allowing high intestinal concentrations to be achieved. The clinical cure rate for Fidaxomicin was comparable to Vancomycin, and importantly, showed a significantly lower recurrence rate for non-027 infected patients.

Dogs do it --- why don't we??

If you go to a restaurant and see a piece of steak with a nutritional value of 63% protein, 25% carbohydrate, 3% lipid, and 9% minerals, you'd think that it sounds pretty healthy, right? Okay, but what if we went one step further and said that it contains 100% human excrement --- ok, 100% human poop. Then your reaction probably would be a little different. In fact, your reaction would probably be a lot different and you'd comment that this is nothing but a crock of ….! And you would be right. But the story goes even farther.

Here is a summary of the story as it appeared on news networks. In Japan, the Tokyo Sewage Service covers more than 13 million people, and they approached the Okayama Laboratory with a problem --- too much sewer mud (another term for human excrement). So what could they do about it? Well, research scientists are supposed to be creative, and in this case, they certainly came up with something novel. They really cooked up a good one here, so to speak. Human excrement is chocked full of protein as evidenced by the 63% protein levels in the nutritional value. Much of the protein is provided by all the bacteria that are present --- both good and bad bacteria. So to make the stuff safe to eat, the excrement was cooked to kill all the bacteria. The proteins were then extracted --- this is an important psychological step. They don't simply take the excrement, shape it into a hamburger, and cook it on the grill. Then soy protein is added to enhance the flavor. Through some type of magic, the stuff is processed into a textured "meat" and red food color is added to make it look like beef. If you watch the YouTube video on the process, the material looks a little like textured tofu with some pretty rotten-looking colors.

The news items went on to say that the material has been taste-tested and yes, the tasters confirmed that the "poop meat" tastes

kind of like beef --- not like chicken. There were a couple of key points made by the researchers. First, the market price for this had not been established. (Personally speaking, we think we would have to be paid, and paid well, to eat it). Second, when this product hits the market, be sure that you always order it well-done --- as if it would be prepared any other way! Were there any reasonable scientific reasons for doing this? The "scientists" argued that it provided another food source --- recycled food that is --- and said it would cut down on greenhouse gas emissions. However, they did allow for the fact that most people would have trouble getting past the yuck factor and actually pay $$ for a crapburger.

The story went out on different news agencies and got some quick responses on the message board:

- "Eat s___ and die!
- Why does my steak have corn in it?
- And we thought hotdogs were bad!
- Eat more chikin.
- What's next --- diet pee?
- Another reason to be a vegetarian.

So back to our title --- dogs eat their feces, why don't we? Well, we seriously doubt that people are ready to go this far. We may use feces to improve our complexions (at least this was done years and years ago), burn the stuff for fuel (remember the photos of cow patties splattered on a wall to be dried), cover our gardens with it (very commonly done), and transplant it into sick people to make their intestines healthy (this works great in patients with recurrent *C. difficile* disease who don't respond well to vancomycin) --- but we're not yet ready to make it into hamburgers and eat it. Is this a good hoax? If not, then like the fitness folks tell us, we could very well be what we eat!

Prove to that special person you care by buying them a luxury toilet!

- Uses 25% less water than your standard flush toilets so you save money for going to the movies
- Saves wear and tear on your back because the toilet has a motion detector that automatically opens the lid (hopefully the motion detector works quickly enough)
- The toilet can tell when you are standing, which automatically tells it to use a low-water flush
- Has a retractable self-cleaning bidet wand that is remote-controlled --- it aims the water, adjusts the water pressure and temperature, and switches on an air dryer when it's time --- no comments are needed for this point
- There is a heated seat so that you can adjust the seat to your own personal sit-down temperature
- There are ground level vents to blow warm air at your feet if you are barefooted
- The bowl automatically cleans itself and has a charcoal filter to eliminate odors
- It will get you in the mood (we wondered about this one) by providing ambient lighting through side panels --- but no coffee, bran muffins, or book is provided
- There is a touchscreen wireless remote for your MP3 player or radio, with sound coming out of 15-watt speakers strategically placed behind the toilet --- we're concerned about this one. It's bad enough dealing with speakers blasting from cars, much less from the bathroom.
- Your price is only $6,000 --- and it says "I care about you in a way no other gift can say it"!

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