Antibiotics – Society’s Hidden Epidemic

Danielle Johansen
College of DuPage

Follow this and additional works at: http://dc.cod.edu/essai

Recommended Citation
Available at: http://dc.cod.edu/essai/vol13/iss1/19

This Selection is brought to you for free and open access by the College Publications at DigitalCommons@COD. It has been accepted for inclusion in ESSAI by an authorized administrator of DigitalCommons@COD. For more information, please contact koteles@cod.edu.
More often than not, by pursue in their own survival pathogens inflict terrific damage on the hosts they inhabit” (Blaser, 2014, p. 42). Humans themselves can somewhat be described as a type of pathogen. Trying to survive on planet earth and wiping out species as they go, there seems to be little hope. Now, the world population is at risk for the next epidemic, antibiotics. But what sort of an effect can antibiotics have on a person? They make them feel “better” and are nothing more than a small pill in a bottle. Unfortunately, the truth is hidden from the naked eye and scientists are beginning to unravel monumental truths about the current antibiotic epidemic.

It is a fact of life that without microbes, the human race (along with all forms of life) would cease to exist. Microbes are found in and among all forms of life and often share a close symbiotic relationship with their host. However, not all microbes are beneficial and if given the right circumstances, they can inflict serious havoc on the human body. Alexander Flemming's discovery of penicillin was one of the initial discoveries in the prevention of pathogens. Flemming inoculated a plate with the bacterial genus *Staphylococcus* then accidentally left it in the open for weeks. This non-aseptic technique, happening by complete chance, collected a single spore from the bread mold Penicillin which was growing on a loaf of bread in his kitchen. Luckily, before disposing of the plates, Flemming noticed a ring of clearing around the recognizable bread mold. He was able to put two and two together and created one of the first known antibiotics (Blaser, 2014, p. 58). Today, all of the penicillin is a descendent of a single moldy melon that was able to produce mass amounts of units of Penicillin (Blaser, 2014, p. 60). It is remarkable that such a potent medicine was discovered by mistake and it is unfathomable that such a powerful cure was found in a single fruit. Although Flemming's discovery of Penicillin was one of the most monumental discoveries in history, no one ever thought that this miracle drug would lead to the development of today's modern plagues.

One of today's most widely studied human “pathogens” is *Heliobacter pylori*. *H. pylori*, known as a class 1 carcinogen by many, is found in the stomach of humans and has been linked to the development of stomach cancer, increasing the chances by almost double (Blaser, 2014, p. 115). The discovery of this led to a worldwide panic of doctors and patients trying to rid the body of this disease causing microbe. However, this particular bacteria historically was harbored in the guts of some of the earth’s earliest humans and has begun to raise many questions in today's scientific community. Normally, unfavorable life is filtered out through natural selection. This is the stance Blaser (2014) took. He was determined to find out the benefits to this “killer” bacteria that is still till this day feared by so many (p. 128).

In his early research, Blaser (2014) detected a specific virulent strain of *Heliobacter pylori* he called strain cagA. He then noted that individuals positive for the more virulent cagA strain had the highest protection against diseases like GERD, Barrets, and esophageal adenocarcinoma (p. 128) and that as *H. pylori* began to disappear, people started to develop once uncommon diseases. The disappearance of *H. pylori* was linked to increases in asthma, hay fever, celiac’s disease, IBD, autism, GERD, and Crohn’s disease. This inverse relationship between *H. pylori* and the development of disease is due to the effect *H. pylori* has on the immune system (Blaser, 2014, p. 136).

*H. pylori* has a way of indirectly effecting the body’s immune responses. When *H. pylori* is
present, it interacts with the dendritic cells of the stomach. It triggers the dendritic cells to produce more regulatory t-cells (t-reg cells). T-reg cells suppress the immune responses that would normally eliminate \textit{H. pylori}. By doing this, the t-reg cells also suppress other allergic responses such as asthma and even autoimmune diseases. Therefore, the more t-reg cells you have the less your body will respond when it encounters a different foreign substance (Blaser, 2014, p. 139). It is important to understand that these diseases are not merely the result of decreasing \textit{H. pylori}, but, they are due to the use of antibiotics which completely wipe them out. Without \textit{H. pylori} t-reg cells are not produced and the body has an increased sensitivity.

Antibiotics are overly prescribed and the use of them in children is surprisingly high. In a recent study of 1000 children under 2 years old, there was a total of 1,365 antibiotics prescribed with an average of three courses by the age of 2 (Blaser, 2014, p. 71). These numbers are surprisingly high and must be impacting today’s children. One way to really understand the impact of antibiotics on a child is to first look at how farmers have been using them. Antibiotics are often mixed into the feed of various meat animals at doses so low they are considered non-therapeutic. Why would farmers be spending so much money giving their animals antibiotics if they are giving them in such low doses that they wouldn’t even be able to prevent disease? Amazingly, the administration of low dose antibiotics allows the animals to grow much larger and at an increasingly high rate. Does this mean that giving children antibiotics will also fatten them up? This is one of the basis on which Blaser (2014) has based much of his research but is not the sole cause of the obesity epidemic in children (p. 150).

Blaser’s (2014) research began when he noticed that chickens given antibiotics at an earlier age would grow fatter than if the antibiotic was given later in life. He hypothesized that if antibiotics were given to chickens at a young age it would cause them to become fatter. This idea was relayed to humans and he predicted that if a baby was given antibiotics it would predispose the child to become obese. The only issue was that it hadn't been proven, so he began experiments involving the effects of antibiotics on mice (p. 150).

Blaser’s (2014) initial studies involved mice that were given antibiotics at a young age. He termed these studies STAT and found that once the test mice matured they were notably larger than the control mice who weren’t given any antibiotics. But what could cause antibiotics to increase the rate of growth? It was determined that antibiotics cause mice to up regulate the genes that transport fat and that these mice also had higher short-chain fatty acid SFCA levels in their cecum. SFCA digests material and gives the body energy. Therefore, by increasing the amount of SFCA, the mice were able to obtain more energy from their foods and were able to store more fat (Blaser, 2014, p. 155). The discovery that an antibiotic can change your gut microbes to become more efficient at food processing has a lot of potential in global health. Areas that are malnourished or impoverished, such as parts of Africa, could be given antibiotics at an early age to help them throughout life with keeping weight on. The antibiotic epidemic has led to research and discoveries which could be used to treat the world wide epidemic of hunger.

The next significant experiment Blaser (2014) conducted was termed DuraSTAT. In this experiment he wanted to determine whether the length of antibiotic exposure had any effect on the mice. Different sets of mice were given antibiotics for various durations including 4 weeks, 8 weeks, and 28 weeks. Remarkably, all the mice gained the same weight independent of the duration of their antibiotic treatments. Therefore, exposure to antibiotics early in life was enough to have life-long effects (p. 159). The fact that the duration of an antibiotic doesn’t change the growth effect could be potentially a gold mine for farmers who spend a lot of money contaminating all the animal feed with antibiotics. Now, farmers should be able to achieve the same growth effects by only supplying the animals with reduced dosages of antibiotic feed during their first month of life. This could also have a positive impact on society. The decrease in antibiotic use would reduce the possibility for bacterial resistance and would also subject people who eat meat to lower levels of antibiotics.

http://dc.cod.edu/essai/vol13/iss1/19
If low dose antibiotics are enough to elicit their effects of weight gain, what would happen if mice were given a full dose? This is what Blaser (2014) did in his PAT mice experiments. Mice were administered full doses of antibiotics including Tylosin and Amoxicillin. Experimental PAT mice showed an increase in bone area and mineral content but the mice given Tylosin were not able to gain back most of their natural microbiota even later on in life (p. 164). Microbiota are very important at a young age and wiping them out predisposes children to various diseases. There should be a way or system for collecting normal microbiota from small children as a precautionary measure for the future. Much like cord blood harvesting, a fecal sample could be obtained from young children and maintained for the first few years of life. If a child unfortunately has to take an antibiotic within these first few crucial years they will be able to replenish their normal microbiota through a fecal transplant or fecal pills. These types of fecal transplants in recent studies of adults infected with resistant bacteria have shown promising statistics with a 94% cure rate over the 31% cure rate of conventional medicine (Blaser, 2014, p 212). Hopefully, one day children taking antibiotics can be co-treated with their own microbiota and their previously preserved naturally occurring microbe samples will decrease the risk of obesity, asthma, and other modern plague’s that are affected by antibiotic use at a young age.

The use of one’s own microbes as a pro-biotic is very intriguing. According the Blaser (2014), when you get a skin abscess it may initially be exposed to many different microbes. However, almost always, a single microbe will eventually take over and dominate the infection (p. 205). What if that dominate microbe was symbiotically put there purposefully by physicians to protect the patient from infection. In other words, if scientists were able to produce a bacterial GMO that could be introduced into skin infections to keep other harmful bacteria at bay, the patients could reduce their risks of infection. This GMO would either have to be one that responds well to medications or one that can live symbiotically within our bodies. The bacterial GMO must be designed to respond readily to medication so that it can be cleared and removed efficiently once the wound has healed. If Paul Ehrlich found his “magic bullet” in Arsenic (Blaser, 2014, p. 205) then perhaps there exists a chemical that could only kill the particular GMO. However, this sort of treatment must only be used in dire circumstances as to not produce bacterial resistance. An example that may elicit a treatment as radical as this could be severe burn victims. Often, burn victims have a decreased chance of survival due to infection. The GMO bacteria potentially could save their lives by simply preventing the growth of other organisms. GMO bacteria would compete for the nutrients required by other microbes or could possibly even work to produce a “toxin” that prevents the growth other infectious microbes on the damaged tissues.

The idea of helping burn victims brings up so many options when you consider bacteria. Can bacteria be manipulated to grow off dead and decaying flesh? Bacteria are already found in and on dead flesh such as road kill. These bacteria are responsible for the natural decay that occurs after death. In the future, maybe there will be a way to modify a bacteria’s growth requirement so that it is only able to grow off of dead tissue. This bacteria can then be inoculated onto burn victims and will colonize the surface of their burn. Using the dead tissue as “food”, the bacteria will live plentiful “eating away” the dead tissue and producing harmless bi-products until there is no more necrotic tissue left. At this point, the bacteria would be starved and eventually die off leaving the patient free of necrotic tissue and bacteria. So instead of painfully washing and scrubbing the burns the bacteria will be the “magic bullet” and an alternative to modern day antibiotics.

It is extremely hard to watch these preventable diseases manifest themselves in humans. When given the option, many choose the path of antibiotics not because they want to but because they are uneducated. Education is key into the prevention of these future epidemics for both those prescribing and those taking antibiotics. The more antibiotics are used the faster bacterial resistance will develop. It doesn't make sense to be wasting this precious resource [antibiotics] to make our food bigger instead of helping those who are actually sick (Blaser, 2014, p. 82). Instead of spending
a few extra bucks on (for example) more smaller chickens at the super market our society will be spending thousands on medications and health care to soothe its diseased bodies. In the end, “Without microbes, we could not eat or breathe. Without us nearly all microbes would do just fine” (Blaser, 2014, p. 14). Running with the keys, microbes are just waiting for the right scientist to unlock their potential.

References

