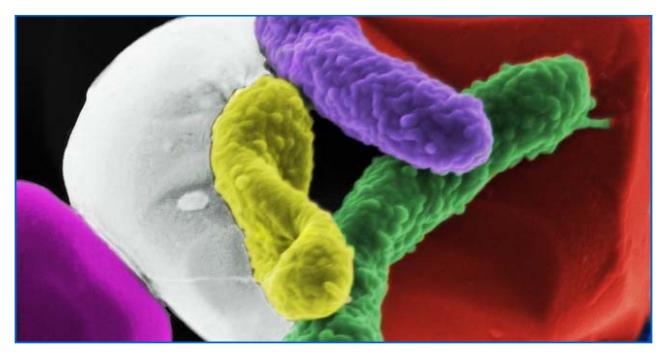


Antibiotics: Killing Off Beneficial Bacteria ... for Good?

By Maryn McKenna Magust 26, 2011 | 12:30 pm | Categories: Science Blogs, Superbug



It's an accepted concept by now that taking antibiotics in order to quell an infection disrupts the personal microbiome, the population of microorganisms that we all carry around in our guts, and which vastly outnumbers the cells that make up our bodies. That recognition supports our <u>understanding of *Clostridium difficile* disease</u> — killing the beneficial bacteria allows *C. diff* room to surge and produce an overload of toxins — as well as the intense interest in <u>establishing a research</u> program that could demonstrate experimentally whether the vast industry producing probiotic products is doing what it purports to do.

But implicit in that concept is the expectation that, after a while — after a course of antibiotics ends — the gut flora repopulate and their natural balance returns.

What if that expectation were wrong?

In a provocative editorial <u>published this week in Nature</u>, Martin Blaser of New York University's Langone Medical Center argues that antibiotics' impact on gut bacteria is permanent — and so serious in its long-term consequences that medicine should consider whether to restrict antibiotic prescribing to pregnant women and young children.

Early evidence from my lab and others hints that, sometimes, our friendly flora never fully recover. These long-term changes to the beneficial bacteria within people's bodies may even increase our susceptibility to infections and disease. Overuse of antibiotics could be fuelling the dramatic increase in conditions such as obesity, type 1 diabetes, inflammatory bowel disease, allergies and asthma, which have more than doubled in many populations.

Among the findings he cites in support: The population-level observation that the incidence of infection with *H. pylori*, the bacterial cause of gastric ulcers, has declined over decades just as the incidence of esophageal cancer has risen. In addition, he offers his own research group's observation that children who don't acquire *H. pylori* are at greater risk of developing allergy and asthma, and their findings that eradicating *H. pylori* affects the production of the two hormones, ghrelin and leptin, that play a role in weight gain.

Are antibiotics to blame for the decline in *H.pylori*? Blaser points out that the organism is vulnerable to the same antibiotics that are prescribed to children for ear infections and colds — and that children routinely receive up to 20 courses of antibiotics before they reach adulthood. In addition, he says, one-third to one-half of women in the industrialized world receive antibiotics during pregnancy. Couple that with the increasingly large percentage of children born by Caesarean section — who by skipping their trip through the birth canal miss their first exposure to friendly bacteria — and the result, he says, is that "each generation… could be beginning life with a smaller endowment of ancient microbes than the last."

Finally, he points to evidence that antibiotic use permanently changes the composition of the gut microbiome, <u>altering the balance</u> of <u>bacterial species</u> and <u>maintaining resistant bacteria</u> in the gut.

The function and influence of the microbiome — in the gut, on the skin and everywhere in the body — is a huge research issue right now, with the founding by the National Institutes of Health of the <u>Human</u> <u>Microbiome Project</u>, not to mention continuing debates over the accuracy of the "<u>hygiene hypothesis</u>" and speculation that altering gut flora could influence everything from <u>obesity</u> to <u>depression</u>. This proposal dovetails with those inquiries — and also (you knew I had to get there eventually) with ongoing concern about antibiotic over-use encouraging the emergence of resistant organisms.

It's understood that antibiotics are already over-prescribed <u>in adults and children</u>; reining in overprescribing is one of the most difficult tasks in controlling the spread of superbugs. This new hypothesis, Blaser says, ought to put more force behind the push to reduce antibiotic overuse, especially in early life:

We urgently need to investigate this possibility. And, even before we understand the full scope, there is action we should take.

Cite: Blaser MJ. <u>Stop the killing of beneficial bacteria</u>. Nature 476, 393–394 (25 August 2011). doi:10.1038/476393a

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