Myth provides problem-solving opportunity

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A commonly believed myth can provide a provocative starting point for a new topic in the classroom. Students are interested because the topic sounds familiar and relevant. You can have students use scientific methodology to discredit the myth and learn to use these processes to evaluate other myths and product claims.

It isn't easy, however, to dispel an often repeated myth. For example, gastric and duodenal ulcers, or Peptic Ulcer Disease (PUD), have been attributed to diet, stress, socioeconomic condition, and genetics—but only recently to infection. A decade after the discovery of the bacterial cause of PUD, only 5% of PUD patients in the United States received antibiotic therapy.² One article¹ even attributed ulcers to psychological problems in the mothers of ulcer patients! Other studies showed increased PUD in populations who have recently left rural areas and moved to (presumably stressful) cities. The studies do not account for the possibility that bacteria can be easily transmitted via food or water in the more crowded urban area.

The following questions provide a framework for having students use clinical data to draw conclusions about PUD, and offers an opportunity for class discussion, group activity, and quantitative analysis in a single 50-minute period. Start by asking question 1 and, after a few minutes of discussion, move on to question 2, and so on.

1. Which of the following occupations do you expect to have the highest incidence of ulcers?

Most stressful jobs	Least stressful jobs
Inner city high school teacher	Forester
Police officer	Telephone line worker
Miner	Toolmaker
Air traffic controller	Repairperson
Medical intern	Natural scientist ³

After students have given you their answers, you share your insights. There is no difference in the incidence of ulcers among the occupations listed. Although stress, diet, and cigarette smoking may irritate an ulcer, they do not cause ulcers.

In the 1940s, spiral-shaped bacteria were observed in removed stomachs but these findings were not considered significant because it was believed that bacteria could not live in the stomach. You can ask students to propose a reason (high acidity) for this. In 1975, these bacteria were again observed. But this time they were seen in living stomachs using fiber optic technology. Moreover, the bacteria were seen in 80% of patients with gastric ulcers. Of course, the bacteria could be growing after an ulcer is established and not be the cause of the ulcer. The bacteria are *Helicobacter pylori*.

2. Epidemiologists, the "disease detectives," often use relative risk to look for a correlation between exposure and disease. In 1984, two Australian physicians, Barry Marshall and Robin Warren, collected the following data on patients experiencing stomach pain. Calculate the relative risk for PUD associated with *H. pylori*.

After showing students how to construct a 2 X 2 table (below) they can work in small groups to complete the calculations. The answers are shown in color.

	Have ulcers	Do not have ulcers	Attack rate
H. pylori present	(a) 27	(b) 2	$\frac{a}{a+b} = (e) \ 0.93$
<i>H. pylori</i> absent	(c) 4	(d) 29	$\frac{c}{c+d} = (f) \ 0.12$
Relative risk = $\frac{e}{f}$	= 7.8		

A follow-up study compared the outcome of patients treated with a standard acid suppressor with patients treated with antibiotics that kill *H. pylori*. All patients were healed 8 weeks after treatment.

	Relapsed after 3 months	Healthy after 3 months	Relapse rate		
Acid suppressor	(a) 16	(b) 5	(e) 0.76		
Antibiotics	(c) 1	(d) 16	(f) 0.06		
Relative risk = $\frac{e}{f}$ = 12.7					

Students should determine that the presence of *H. pylori* increases the risk of ulcers 7.8 times. The relative risk of relapse with acid suppressant therapy increases 12.7 times compared to antibiotic therapy. This provides strong evidence that *H. pylori* is the cause of PUD.

3. What information would you need to prove that *Helicobacter pylori* causes peptic ulcers?

Here, you can work through Koch's postulates with your students. Barry Marshall did inoculate himself with *H. pylori* and caused gastritis, a precursor to PUD. Marshall treated his gastritis before developing ulcers.

continued on page 5

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Myth Provides Problem-Solving Opportunity continued from page 4

4. Were Koch's postulates fulfilled?

Marshall says they were not. (If your textbook doesn't have a figure illustrating Koch's postulates, you could use the one at <<u>http://www.smccd.net/accounts/case/koch></u>)

- 5. What treatment would you recommend for PUD?
 - a) An antacid b) An acid suppressor
 - c) A bland diet d) An antibiotic

6. Which group do you expect to have the highest incidence of PUD?⁴

- a) U.S. population before 1950
- b) U.S. population after 1950

The incidence of PUD has decreased since 1950. This should reinforce that stress (of urban living) does not cause ulcers. It is also an opportunity to discuss the roles of sanitation and water treatment in preventing disease transmission. The appearance of ulcers in families can be explained by sharing fomites and contact with body fluids in a household as opposed to genetics or mom's mental health.

7. *H. pylori* has been implicated as the cause of stomach cancer. Which group do you expect to have the highest incidence of stomach cancer?

- a) Adults in developed countries
- b) Adults in developing countries

Stomach cancer is one of the most common malignancies in the world, although it is relatively uncommon in the United States. Over 80% of the population of developing countries and only 30–50% of the population of developed countries have *H. pylori* by early adulthood. Consequently, stomach cancer rates are very high and continuing to rise in developing countries.

8. *H. pylori* produce the enzyme urease that hydrolyzes urea to produce ammonia. Of what value is ammonia to *H. pylori*?

The bacteria are able to neutralize the acid in their microenvironment, which allows them to grow where other bacteria cannot grow.

References

- 1 Journal of Chronic Diseases 20:435-456, 1967.
- 2 Archives of Internal Medicine 157:1489-1494, July 14, 1997.
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- 4 "U.S. Population before 1950, U.S. Population after 1950," Archives of Internal Medicine 157:1489-1494, July 14, 1997.
- 5 ASM News 61:21-26, 1995.

Editor's Note: Christine L. Case is co-author of *Microbiology: An Introduction, Seventh Edition* (Benjamin Cummings, 2001).

What freshmen don't know

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About five years ago I returned from a detour into administration to teaching. I was so enthralled with the idea of returning to teaching, that I assigned myself an introductory biology course for new freshmen. While I have not regretted my decision, I have wrestled with a question faced by many faculty: why do so many freshmen do so poorly in introductory science courses?

During the first semester with my freshmen, I was rolling along until the first lecture examination. Disastrous! Shameful! Just plain ugly! Over 50% of this brilliant group of students scored less than 65% on an easy, straightforward test. What went wrong? Who messed up, teacher or students or both? I decided to get some answers.

First, I asked for an early anonymous written evaluation from each student. According to my class, I was THE MAN. After reading notes full of superlatives, I realized that my freshmen were judging the quality of the class and the instruction by my enthusiasm and humor. They were unable to identify problems with the learning process. They too were frustrated by their performances on an examination they judged to be "fair," "on material they expected," and "really fairly easy in hindsight." I resolved to dig deeper into this mystery.

I got my first glimpse of the problem while I was attempting to answer a student's question after class by referring to her lecture notes. I couldn't find the section of her notes that covered the topic in question. I knew that she had attended every lecture and wrote frenetically as I spoke. But where in her notes was the stuff I wanted to show her? I hurriedly grabbed notebooks from every other student still in the lecture hall and frantically turned pages. The difficulty became obvious. During my lecture, students were accurately capturing less than 60% of the information and actually hearing and processing much less than that. Then they dutifully put the notebooks away until the next class, never revisiting those notes until the week of the test.

The root of the problem was apparent to me: my freshmen came to me with a collection of facts learned in high school, solid SAT scores that predicted brilliant academic careers, but no tools or strategies for handling college-level work. They needed a plan, an organized set of guidelines and strategies to help them survive until they evolved into mature, sophisticated learners.

How to generate this? I assembled a group of "volunteers" in my laboratory late one afternoon. Together *continued on page 6*

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