Name _____ Date _____
Teacher _____

MRSA texts
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1. Connie's Story: A Nurse's Personal Experience with MRSA (video text)

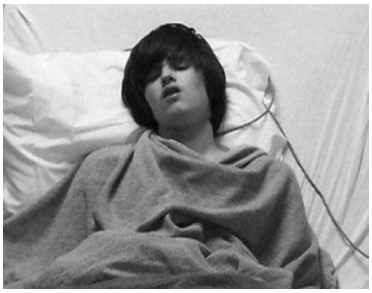
The voices of patients are often missing from discussions of the impact of medical errors and adverse events. Ms. Constance Lehfeldt is a former nurse who developed a methicillin-resistant *Staphylococcus aureus* (MRSA) infection, which ultimately led to a devastating series of complications. Although the exact source of her MRSA infection remains unclear, it manifested itself after her surgery at a hospital in the Peace Health system and left her with mild speech problems and blindness in one eye. Connie's Story was produced for the Agency for Healthcare Research and Quality by editors from the University of California, San Francisco.

Source: http://webmm.ahrq.gov/perspective.aspx?perspectiveID=58

2. Kansas City Teen Gets MRSA From Attempted Lip Piercing, Almost Dies

Kansas City Teen Gets MRSA From Attempted Lip Piercing, Almost Dies

Published May 12, 2008 / FoxNews.com



A Kansas City-area teenager who tried to pierce his lip with a needle from a first-aid kit ended up with a staph infection that almost killed him.

Zeke Wheeler of Blue Springs is recovering at Children's Mercy Hospital after several surgeries on his knees and hips to remove the drug-resistant infection called Methicillin-resistant Staphylococcus aureus infection. Now the 15-year-old high school freshman faces heart surgery, more hospitalization and a long course of antibiotics.

The boy's father — John Wheeler — said Wednesday that the boy was at home ill with flu and bronchitis on April 8 and tried to pierce his lower lip. A week later the boy felt feverish and went to an emergency room, where he was diagnosed with a viral infection.

Not until he was at Children's Mercy was he found to have MRSA. Dr. Robyn Livingston, director of Infection Control at Children's Mercy Hospital, told KCTV-5, "If MRSA gets into the blood stream, you're talking about infection on the heart, pneumonia, into the bone that may require surgical intervention." Every part of Wheeler's body is now affected, Livingston added. He's had six blood transfusions, and three knee and two hip surgeries.

Dr. Joseph Rahimian, an infectious disease specialist at St. Vincent's Hospital in New York City, said he treats about 15 cases of MRSA each week. "It's a bacteria we're seeing more and more frequently in communities and hospitals," Rahimian said. "It could be because of the increased use of antibiotics." Rahimian said he thinks Wheeler will recover, although the teenager might have some chronic consequences. "I guess there is a lesson in this," Rahimian said. "If you are getting a piercing, it should be done with someone who knows what they are doing, and it should be done with a clean, sterilized needle with someone who knows where the major vessels are to avoid injecting into an artery or vein."

— The Associated Press contributed to this report

Source: http://www.foxnews.com/story/0,2933,354696,00.html#ixzz1m0Zzjt9b

3. Superbug' MRSA Worries Doctors, Athletes

'Superbug' MRSA Worries Doctors, Athletes
Jan. 13, 2005 --

Ricky Lannetti was once the picture of health -- a big, strong college football player.

In the fall of 2003, he had led his team to a big victory, catching more passes than anyone and securing a spot in the national semifinals. But sometime after that game he caught something else.

"They didn't know what they had. They were as confused as I was," his mother, Teresa, told ABC News. "They had five different antibiotics in him, but they finally said, 'We can't handle it.'" On Dec. 6, 2003, one week after his last game, Lannetti died.

There's still a lot of mystery surrounding how Lannetti, 21, got sick in the first place and why his illness progressed so quickly. But one thing is clear: He had an infection caused by a bacteria generally found on the skin or in the nose, called MRSA, or methycillin resistant *staphylococcus aureus*.

MRSA is the kind of germ doctors have worried about for years: some call it a "superbug," a germ the usual antibiotics won't kill.

Worse, it can cause trouble quickly. What starts as a skin infection, can become a deadly pneumonia or blood or bone infection in a matter of days if not treated correctly.

Delicate Choices

Up until recently, doctors hadn't seen MRSA in healthy young people outside the hospital, said Dr. Robert Daum of University of Chicago Hospitals. "MRSA is a denizen of the hospital," he said. "It lives here."

But now, 65 percent of the staphylococcus infections coming into his emergency room in otherwise healthy kids are MRSA, he said. To him, that rate of growth is alarmingly fast -- a cause for concern.

MRSA is resistant to anywhere from 15 to 30 different antibiotics. That means when it's detected, a doctor has only a very small number of compounds at hand that are able to kill it.

Daum said he has seen some patients with MRSA that are worse off for having seen a doctor that could not recognize it. The patients were treated with regular antibiotics -- and that gave the germ more time to do damage in the body.

"We've seen a lot of kids that come in here that needed intensive care and in fact have died that have started off by being out in the community, where they get an old treatment and then come in here having failed it," he said.

Evolving Quickly

Most MRSA infections begin with a cut or a bruise, which is why some of the worst outbreaks have happened to football teams.

"I think you'd be hard-pressed right now to find a college athletic department that has not seen it in some shape or form with some of their athletes," said Ron Courson, the athletic trainer for the University of Georgia football team. Eight players on his team had MRSA infections this season.

A communal locker room, with many people in one area, can help bacteria spread, he said. "You may have athletes sharing equipment such as passing a towel from one person to the next person on the sideline."

Even the NFL has had its share of problems: players such as Kenyatta Walker of the Tampa Bay Buccanneers and Junior Seau and Charles Rodgers of the Miami Dolphins reportedly have been hospitalized with serious MRSA infections.

Daum's biggest concern is that as MRSA continues to evolve, it will become resistant to even more antibiotics.

"Bacteria are unlike us humans. We have a generation time of about 25 years. They have a generation time of 20 minutes," he said. "They can adapt pretty fast."

Daum said he is seeing a strain in the Midwest that is so severe, it has caused deaths even when the right antibiotic is used.

Source: http://abcnews.go.com/Health/Primetime/story?id=410908&page=1&singlePage=true

4. How long do microbes like bacteria and viruses live on surfaces in the home at normal room temperatures?

How long do microbes like bacteria and viruses live on surfaces in the home at normal room temperatures?

Posted 08.22.2002 at 11:43 AM 2 Comments

Art Dekenipp
Alvin, Texas

The answer is probably not what you want to hear: Microbes can live on household surfaces for hundreds of years. The good news, however, is that most don't. Some well-known viruses, like HIV, live only a few seconds.

Microbes, of course, are everywhere. Each square centimeter of skin alone harbors about 100,000 bacteria. And a single sneeze can spray droplets infested with bacteria and viruses as far as 3 feet. The microbial life span depends on many factors, says Philip Tierno, director of microbiology and diagnostic immunology at the New York University School of Medicine. Because viruses must invade cells of a living host to reproduce, their life spans outside are generally shorter than that of bacteria, which reproduce on their own. Although viruses can survive outside a host on household surfaces, their ability to duplicate themselves is compromised-shortening the virus's life span.

Humidity also makes a difference; no bacteria or virus can live on dry surfaces with a humidity of less than 10 percent. Any sort of nutrients-food particles, skin cells, blood, mucus-helps microbes thrive, which is why your kitchen sponge is a breeding ground.

Bacteria called mesophiles, such as the tuberculosis-causing Mycobacterium tuberculosis, survive best at room temperature and are likely to thrive longer than cold-loving psychrophiles or heat-loving thermophiles. According to Tierno, at room temperature and normal humidity, Escherichia coli (E. coli), a bacteria found in ground beef that causes food poisoning, can live for a few hours to a day. The calicivirus, the culprit of the stomach flu, lives for days or weeks, while HIV dies nearly instantly upon exposure to sunlight. Other microbes form exoskeleton-like spores as a defense mechanism, like the bacteria *Staphylococcus aureus*, which is responsible for toxic shock syndrome, food poisoning, and wound infections. In this way, they can withstand temperature and humidity extremes. Tierno says this bacterial spore can survive for weeks on dry clothing using sloughed skin cells for food. The Bacillus anthracis, the anthrax bacteria, can also form spores and survive tens to hundreds of years.

Worried that your home is a hospitable habitat? Tierno says simple hand washing can greatly reduce your risk of picking up germs. Using a disinfectant on high-traffic surfaces-doorknobs, kitchen counters, and sinks-also helps eliminate unwanted household guests.

Edited by Bob Sillery
Research by Reed Albergotti and Emily Bergeron

Source: http://www.popsci.com/scitech/article/2002-08/how-long-do-microbes-bacteria-and-viruses-live-surfaces-home-normal-room-tem

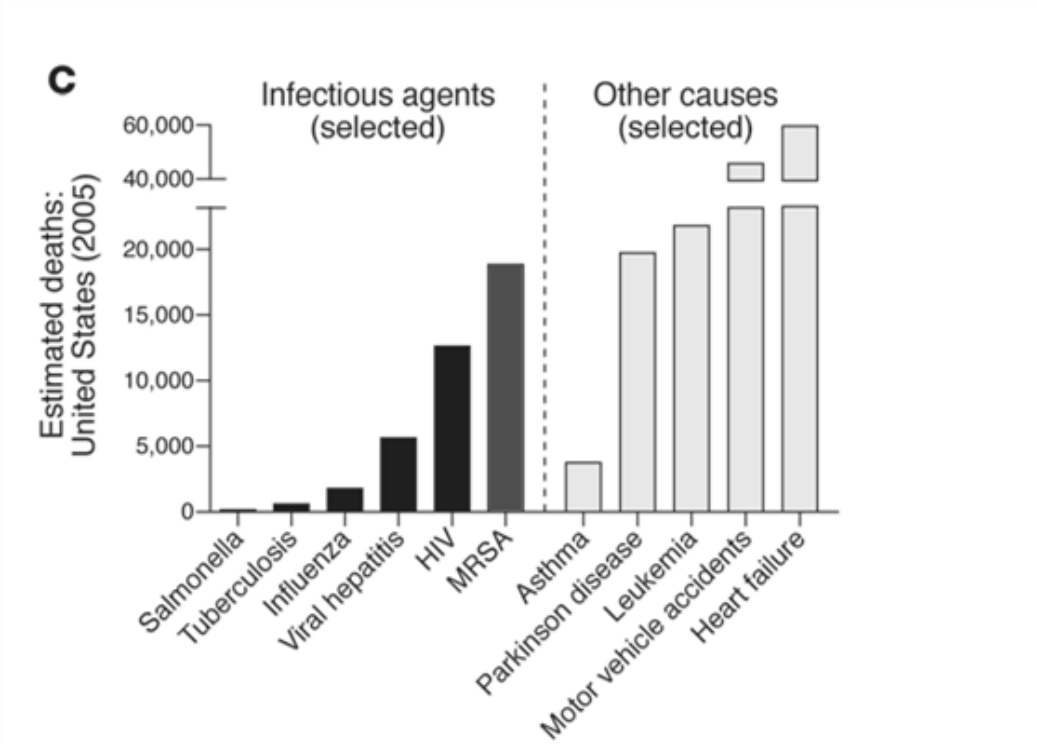
5. Antibiotic Resistance

Antibiotic / Antimicrobial Resistance

Antibiotics and similar drugs, together called antimicrobial agents, have been used for the last 70 years to treat patients who have infectious diseases. Since the 1940s, these drugs have greatly reduced illness and death from infectious diseases. Antibiotic use has been beneficial and, when prescribed and taken correctly, their value in patient care is enormous. However, these drugs have been used so widely and for so long that the infectious organisms the antibiotics are designed to kill have adapted to them, making the drugs less effective. People infected with antimicrobial-resistant organisms are more likely to have longer, more expensive hospital stays, and may be more likely to die as a result of the infection.

Source: http://www.cdc.gov/drugresistance/index.html

6. Comparison of Estimated Death in U.S. in 2005



(**C**) Comparison of estimated deaths in the United States in 2005 due to individual infectious agents or other causes. Data are CDC estimates from National Vital Statistics Reports (12) and Klevens et al. (11). Deaths associated with MRSA infection are based on the estimated number of in-hospital deaths rather than attributable mortality, whereas data for all other causes of mortality are based on US Standard Certificate of Death. Note also that mortality due to MSSA is not included, and thus estimated mortality associated with all *S. aureus* infections is not shown.

Source: http://www.jci.org/articles/view/38226, Frank R. DeLeo, Henry F. Chambers, *J. Clin. Invest.* 2009; **119**(9):2464

7. MRSA History

Late 1880s
Scottish surgeon Alexander Ogston identifies a bacterium, *Staphylococcus aureus*.

1928
British scientist Alexander Fleming discovers the first antibiotic, penicillin.

1941
Penicillin becomes available in the United States and England. The first penicillin-resistant Staphylococcus aureus is reported a short time later.

Late 1940s
One-quarter of *Staphylococcus aureus* bacteria in hospitals are penicillin-resistant.

1958
Vancomycin, still considered an antibiotic of last resort, is introduced.

1959
The antibiotic methicillin is introduced.

1961
Doctors find the first cases of methicillin-resistant *Staphylococcus aureus* (MRSA).

1960-1967
Infrequent hospital outbreaks of MRSA in Western Europe and Australia

1968
First hospital outbreak of MRSA in the United States at the Boston City Hospital, Massachusetts

1968–mid 1990s
Percent of *Staphylococcus aureus* infections in hospitalized patients that were caused by MRSA increased slowly but steadily.

1982
Large outbreak of MRSA infections among intravenous drug users in Detroit, Michigan

Late 1980s–1990s
Outbreaks of MRSA noted in Australia among Aboriginal populations with no exposure to hospitals.

1998–2008: The CA-MRSA Epidemic Decade
While rates of HA-MRSA (Hospital Acquired) infection remained stable, rates of CA-MRSA (Community Acquired) increased.

Mid-1990s
Scattered reports of CA-MRSA infections in children in the United States.

1999
First reports of healthy, young children dying of severe MRSA infections

2002
Doctors find in the United States a strain of Staphylococcus aureus highly resistant to vancomycin.

2005
CA-MRSA risk factors identified to date include: athletes, military recruits, incarcerated people, emergency room patients, urban children, HIV patients, men who have sex with men, indigenous populations.

Today
Over 95% of *Staphylococcus aureus* worldwide is penicillin-resistant and 60% is methicillin-resistant.

Sources: http://mrsa-research-center.bsd.uchicago.edu/timeline.html
http://articles.latimes.com/2006/feb/26/science/sci-staph26/3

8. Contagion Movie Trailer (video text)

Source: *Contagion*, Directed by Steven Soderbergh, written by Scott Z Burns. Warner Bros Entertainment Inc, 2011. On general release in the USA and on release in the UK later in October, 2011. Watch a trailer at http://contagionmovie.warnerbros.com

9. Superbug, Super-fast Evolution

April 2008

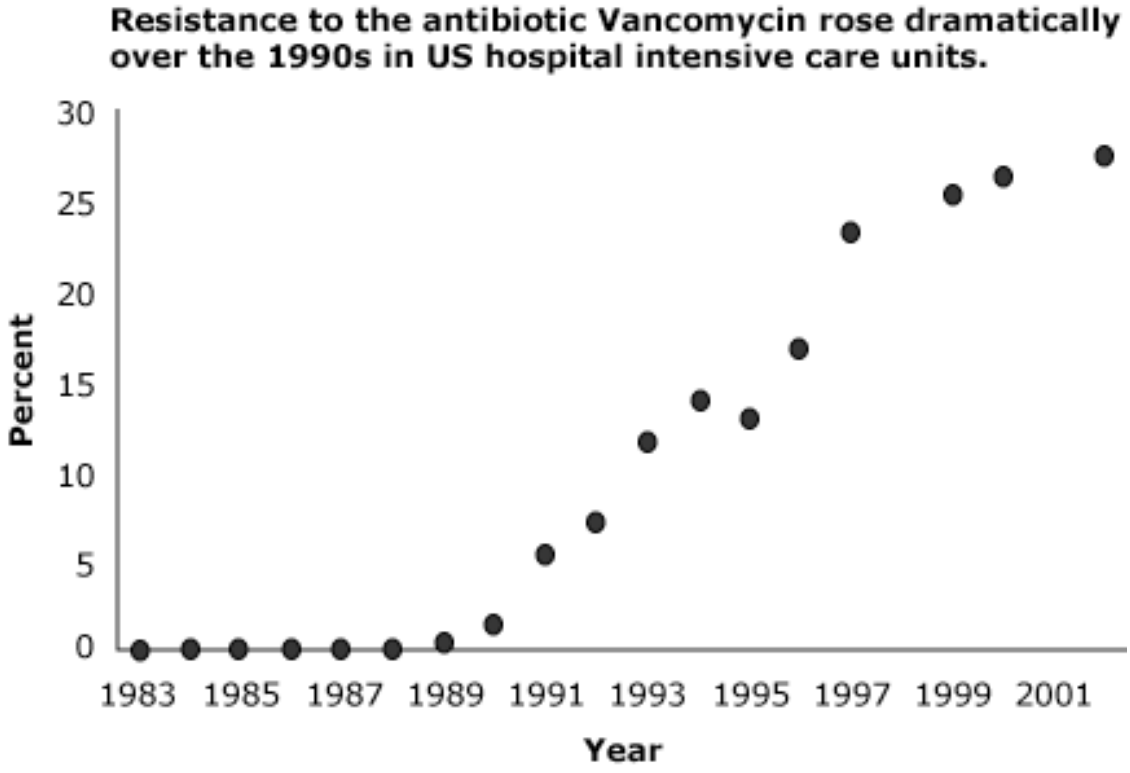
Fascination with tiny microbes bearing long, difficult-to-pronounce names is often reserved for biology classrooms — unless of course the bug in question threatens human health. MRSA (methicillin-resistant *Staphylococcus aureus*) now contributes to more US deaths than does HIV, and as its threat level has risen, so has the attention lavished on it by the media. At this point, almost any move the bug makes is likely to show up in your local paper. Last month saw reporting on studies of hospital screening for MRSA (which came up with conflicting results), stories on MRSA outbreaks (involving both real and false alarms), and media flurries over the finding that humans and their pets can share the infection with one another. Why is this bug so frightening? The answer is an evolutionary one.

Where's the evolution?

MRSA is resistant not only to the antibiotic methicillin, but also to whole other suites of our drugs, making it very difficult to treat and, occasionally, deadly. Modern strains of MRSA did not, however, show up out of the blue. In the early 1940s, when penicillin was first used to treat bacterial infections, penicillin-resistant strains of *S. aureus* were unknown — but by the 1950s, they were common in hospitals. Methicillin was introduced in 1961 to treat these resistant strains, and within one year, doctors had encountered methicillin-resistant *S. aureus*. Today, we have strains of MRSA that simultaneously resist a laundry list of different antibiotics, including vancomycin — often considered our last line of antibacterial defense.

How did *S. aureus* morph from a minor skin infection to a terror? When the media report on MRSA and other drug resistant pathogens, they often say that such pathogens have recently "emerged" — that they've "developed" resistance or "learned" to evade our drugs. In fact, it's more accurate to say that these bugs have *evolved* resistance. It's particularly ironic that newspapers might shy away from describing bacterial evolution as such because, when it comes to evolution, bacteria have most of the rest of us beat.

10. Resistance to Vancomycin

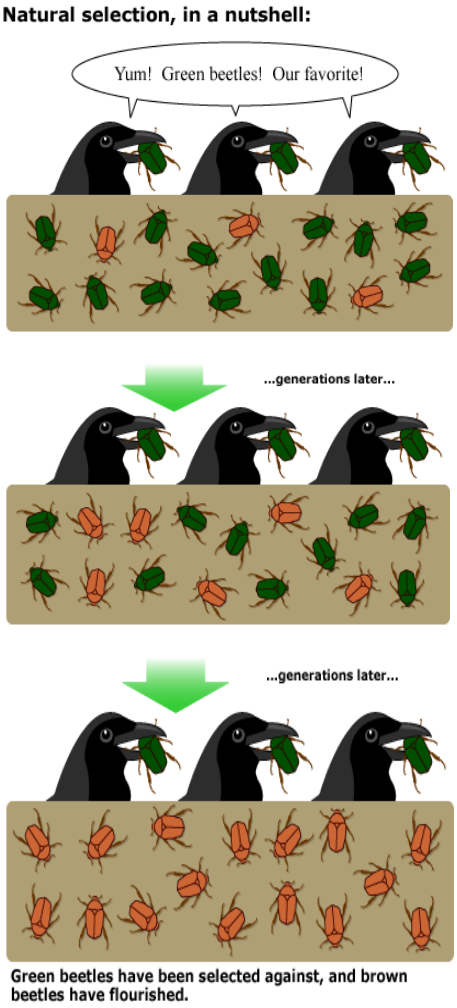


Excerpted from "Battling bacterial evolution: The work of Carl Bergstrom. " Copyright 2011 by The University of California Museum of Paleontology, Berkeley, and the Regents of the University of California

11. Battling Bacterial Evolution

Battling Bacterial Evolution: The Work of Carl Bergstrom

Dr. Carl Bergstrom manages evolution. From his laboratory at the University of Washington, Carl figures out how to control the evolutionary future of microbe populations, nudging them towards particular destinies and away from others. His laboratory does not look like a traditional biology lab; rather than test tubes or microscopes or Petri dishes, the rooms are full of computers, whiteboards, books, and coffee machines.



Hooked on natural selection

Carl had always been interested in biology but got hooked on evolution after encountering Darwin's basic idea of natural selection. The concept is simple, but incredibly powerful…

Natural selection is simply the logical result of four features of living systems:

- **variation** - individuals in a population vary from one another
- **inheritance** - parents pass on their traits to their offspring genetically
- **selection** - some variants reproduce more than others
- **time** - successful variations accumulate over many generations

Natural Selection and Antibiotic Resistance

Natural selection can operate in any population, but Carl focuses much of his work on bacterial populations that impact public health… Carl's work tackles the very real problem of the evolution of antibiotic resistance by bacterial populations in hospitals.

Antibiotics, such as penicillin, are drugs that kill or prevent the growth of bacteria. When antibiotics were first discovered, they seemed to represent a miracle cure for human diseases like pneumonia, typhoid, bubonic plague, and gonorrhea. However, almost immediately after the introduction of antibiotics, bacteria began to up the stakes — resistant strains of bacteria soon evolved that could grow even in the presence of a particular antibiotic, rendering our drugs ineffective in battling these resistant infections.

How exactly does antibiotic resistance evolve? How have such small and simple organisms managed to repeatedly outpace our drugs? The process is quite simply evolution by natural selection.

Bacteria are great evolvers for many reasons. For example, their short generation times and large population sizes boost the rate at which they can evolve.

12. Modification by Natural Selection

Modification by Natural Selection

Darwin proposed that the environment may affect individual organisms in a population in different ways because individuals in a species are not identical. Some organisms have traits that make them better able to cope with their environment. Organisms that have a greater number of these favorable traits tend to leave more offspring than organisms with fewer beneficial traits. Darwin called the different degrees of successful reproduction among organisms in a population natural selection.

If a trait both increases the reproductive success of an organism *and* is inherited, then that trait will tend to be passed on to many offspring. A population of organisms **adapt** to their environment as their proportion of genes for favorable traits increases. The resulting change in the genetic makeup of a population is evolution. In an evolving population, a single organism's genetic contribution to the next generation is termed **fitness.** Thus, an individual with high fitness is well adapted to its environment and reproduces more successfully than an individual with low fitness.

Bear in mind that natural selection is not an active process. Organisms do not purposefully acquire traits that they need, although it may seem that this is true. The environment "selects" the traits that will increase in a population. The kinds of traits that are favorable depend on the demands of the environment. An organism may be able to run fast, or it may be strong or have coloring that acts as camouflage from predators. Traits that are favorable for some organisms in some environments are not necessarily favorable for all organisms or all environments. For example, the large body size of large mammals such as the elephant would not be beneficial to a species of flying birds if size prevented flight. A favorable trait is said to give the organism that has it an **adaptive advantage.**

Selection conditions change as the demands of the environment change. For example, a significant change in climate or available food can cause rapid evolutionary change as populations adapt to the change. If the environmental change is too extreme, however, populations cannot adapt quickly enough and they become extinct.

Excerpted from MODERN BIOLOGY page 287

13. Growth and Reproduction

Growth and Reproduction

Bacterial cells grow by a process called binary fission: One cell doubles in size and splits in half to produce two identical daughter cells. These daughter cells can then double in size again to produce four sibling cells and these to produce eight, and so on. The time it takes for a bacterial cell to grow and divide in two is called the doubling time. When nutrients are plentiful, the doubling time of some bacterial species can be as short as twenty minutes. However, most bacterial species show a doubling time between one and four hours. A single bacterial cell with a one-hour doubling time will produce over 1 million offspring within twenty hours. If left unchecked, a single E. coli bacterium replicating once every twenty minutes could replicate to equal the mass of Earth in twenty-four hours. The enormous increase in cell numbers that accompanies this exponential growth provides these simple unicellular organisms with an incredible growth advantage over other unicellular or multicellular organisms.

Source: http://www.biologyreference.com/Ar-Bi/Bacterial-Cell.html#ixzz1RG7ByBLw

14. Wash your hands

Wash your hands

Human skin — even in the most healthy of us — is teeming with bacteria. Most of those bacteria only cause disease under special circumstances. But everyone also carries potentially dangerous germs from time to time, such as staph, strep, and the intestinal bacteria that cause food poisoning and diarrhea. Sad to say, health care personnel — including your doctors and nurses — are particularly likely to carry the most troublesome bacteria, especially on their hands. And although viruses don't set up shop on the skin the way bacteria do, the viruses that cause diarrhea and respiratory infections — from the sniffles to the flu — can hang around on the hands long enough to spread from person to person.

If your skin is covered with so many bacteria, why don't they make you sick more often? Although the skin is a hospitable resting place for bacteria, it is also a tough barrier that prevents hostile bugs from reaching the body's vulnerable internal tissues. Ironically, perhaps, some of the traditional methods of removing bacteria from the skin can disrupt the skin's own defenses. Scrubbing can produce tiny abrasions that allow bacteria to sneak into your tissues. Detergents and even plain water can remove the skin's oils, which have important antibacterial properties.

Good handwashing, then, involves two potentially conflicting goals, removing microbes while still keeping your skin healthy.

Preached but not practiced

Handwashing is good advice — but do Americans follow it?

Often, we don't. When investigators surveyed public restrooms around the country, they found that only 83% of people washed up after using the toilet. Do posted reminders to "Please Wash Your Hands" help? When researchers tested this simple strategy, they found that handwashing improved in women but not in men.

The gender gap applies to hospitals, too. In one study, female physicians washed their hands after 88% of patient contacts, but male doctors washed after just 54%.

Does it work?

Yes. Just 30 seconds of simple handwashing with soap and water reduces the bacterial count on health care workers' hands by 58%. And there is an even better way: Alcohol-based handrubs reduce counts by 83%.

What's best?

Soap and water is the time-honored technique, and it does work. In fact, it's still the best way to remove visible soilage and particulate material. But as the public has become concerned about the risk of infection, soaps with antibacterial additives have gradually taken over 45% of the market. It's understandable, but it's not helpful; antibacterial soap is no better than ordinary soap, and the additives actually increase the risk of allergic reactions and other side effects.

Plain soap will do the job — and so will plain water. Tap water is excellent, and cool or lukewarm temperatures serve as well as hot water. If soap and water are not available, antibacterial wipes can help. Although they are not as effective, they will reduce bacterial counts. Washing with soap and water is the best way to remove dirt, but waterless, alcohol-based handrubs are even better at killing germs. Handrubbing is faster and more convenient than handwashing, and it's also easier on the skin. Hospitals are switching to handrubs because they kill more bacteria and viruses and they are used more regularly.

When and how

How should you wash? Wet your hands with water, then apply the soap to your palms. Rub your hands together briskly for at least 15 seconds before rinsing.

Wash your hands before each trip to the dining room and after each trip to the bathroom. Wash after handling diapers and animals. Wash before and after you handle food. Wash after you take out the trash, work in the yard, clean the house, repair the car, or do other messy chores. Wash before and after sex. Wash after you come in contact with anyone who is sick. If you follow reasonable guidelines you'll be washing often, but you won't become obsessive or compulsive. Be careful, not fearful.

August 2006 Update

Source: http://www.health.harvard.edu/fhg/updates/update0806d.shtml

15. The success of evolutionary engineering

Responding to the pervasive reach of evolution in medicine, scientists have developed an impressive series of innovative methods to slow the pace of evolution. The following examples show that successful methods often slow evolution for clearly evolutionary reasons and that these approaches may be generalizable to other systems.

Drug overkill and HIV triple drug therapy. The evolutionary biology hidden in this strategy is simple: a strong, multi-drug overdose leaves no viruses able to reproduce, and so there is no genetically based variation in fitness among the infecting viruses in this overwhelming drug environment. Without fitness variation, there is no evolutionary fuel, and evolution halts.

Table 3. The success of evolutionary engineering: mechanisms that reduce evolution can and do work on all three parts of the evolutionary engine.

MECHANISMS THAT WORK TO SLOW EVOLUTION

Method of slowing evolution	**Example**
Reduce variation in a fitness-related trait	
Drug overkill with multiple drugs	Triple-drug therapy for AIDS Pesticide pyramiding
Ensure full dosage	Direct observation therapy of tuberculosis
Reduce appearance of resistance mutations	Engineer RT gene of HIV-1
Reduce pest population size	Integrated pest management of resistant mutants Nondrug sanitary practices
Reduce directional selection	
Vary selection over time	Herbicide rotation Vary choice of antibiotics, pesticides or antiretrovirals
Use nonchemical means of control	Integrated pest management
Limit exposure of pests to selection	Withhold powerful drugs, e.g., restricted vancomycin use
Avoid broad-spectrum antibiotics	Test for drug or pesticide susceptibility before treatment of infections or fields
Reduce heritability of a fitness-related trait	
Dilute resistance alleles	Refuge planting

Direct observational therapy. Tuberculosis (TB) infects 1/3 of the world's population and is difficult to treat because it requires 6 months of medication to cure. Partial treatment has resulted in the evolution of multi-drug resistance. Direct observational therapy has been used to improve patient compliance during the whole treatment regimen, reducing evolution of resistance by ensuring a drug dose long enough and severe enough to completely eradicate the infection from each person.

Withholding the most powerful drugs. The antibiotic vancomycin has been called the "drug of last resort," because it is used only when other, less powerful antibiotics fail. Withholding the most powerful drugs lengthens their effective life span because overall selection pressure exerted by these drugs is reduced, slowing the pace of evolution.

Screening for resistance. Screening infections for sensitivity to particular antibiotics before treatment allows for a narrow=range antibiotic to be used instead of a broad-spectrum antibiotic. Reduced use of a broad-spectrum antibiotic slows evolution of resistance as in the mechanisms above. Similarly, farmers are advised to check their fields after pesticide treatment and hten change the chemical used in the next spraying if many resistant individuals are discovered. Screening for pest susceptibility reduces use of chemicals for which resistance has begun to evolve.

Adapted from www.sciencemag.org SCIENCE VOL 293 7 SEPTEMBER 2001