# Bio Terror Bible

## EXPOSING THE COMING BIO-TERROR PANDEMIC

**BIOTERRORBIBLE.COM:** In the aftermath of man-made bio-terror generated pandemic, the government and media will be feeding the public any number of different scapegoats allegedly responsible for the pandemic that will likely kill millions.

While some scapegoats (see below) are indeed plausible, it is much more likely that the live pathogens or agents responsible for the pandemic will likely be dispersed via A) chemtrails by government airplanes or drones, B) by the U.S. Postal Service via Tide detergent samples, C) by the government and medical establishment via tainted vaccines, or by D) the portable petri dish commonly known as the Trojan condom.

**Bio-Terror Scapegoats:** Africa, Agriculture (Food & Animals), Airports & Air Travel, Al Qaeda, Bio Labs, Bio-Terrorism Is Easy, Bio-Terrorists (Bio-Hackers), Black Market, Bugs & Insects, Censorship / Lack Thereof, Domestic Terrorists, Exotic Animals (Zoonosis), Government Ineptitude, Mail-Order DNA, Mexico, Missile Shield Failure, Mutation, Natural Disaster, No Clinical Trials (Vaccines), and The Monkeys.

**Title:** Tinkering With The Genes Of Biological Weapons: Genetic Engineering Is Regularly Used To Produce Lethal Bacteria
**Date:** July 13, 2000
**Source:** Sunshine Project

**Abstract:** Investigations by the Sunshine Project show that genetic engineering has been used in the past decade to tinker with the genes of biological weapon agents. Researchers in the USA, UK, Russia, Germany and other countries introduced genes into hazardous bacteria that are likely to enhance the biowarfare possibilities of these microbes. Strains have been designed that can withstand antibiotics, are undetectable by traditional equipment, can overcome vaccines, or that cause unusual symptoms, thereby hampering diagnosis. In general, gene transfer can be used to build more effective biological weapons, it could be used to broaden the military biological warfare spectrum, making it more difficult to fight and control bioweapons.

"*Military research seems to be out of control*", says Jan van Aken, genetic engineering expert of the Sunshine Project. "*Many research projects have a clear offensive potential. To just stick the label 'defense' on it is not enough. We urgently have to draw clear lines and prohibit genetic engineering with biological weapon agents.*"

At the same time, it is very unclear that efforts to strengthen the Biological Weapons Convention will succeed in the round of negotiations currently underway in Geneva. In light of the increasing biowarfare threat, the international community decided in 1994 to negotiate a Protocol to strengthen the Biological and Toxin Weapons Convention (BTWC). (1)

Considering that the biowarfare threat is dramatically increasing due to the speedy development of genetic engineering, a Bioweapons Convention that it not updated to reflect new technological realitites will not create global security. "*In light of recent advancements in genetic engineering, updating and reinforcement of international law that outlaws bioweapons is urgently needed.*" says Edward Hammond of the Sunshine Project's Seattle office. A strong Protocol will be a first step, that enhances tranparency,

making it more difficult for countries to conceal a bioweapons program, for example, in the guise of pharmaceutical research.

**Genetic Engineering: A New Class Of Biological Weapons**
It sounds like science fiction, but it is a deadly reality: lethal microbes, with no cure, invisible to detection systems, and able to overcome vaccines. In 'defensive' programs, researchers in the USA, UK, Russia and Germany have genetically engineered biological weapons agents, building new deadly strains. And this is probably only the tip of the iceberg.

Genetic engineering can be used to broaden the classical bioweapons arsenal. Through genetic engineering, bacteria can not only be made resistant to antibiotics or vaccines, they can also be made even more toxic, harder to detect, or more stable in the environment. By using genetic methods that are standard procedures in thousands of labs worldwide, bioweapons can be made more virulent, easier to handle, and harder to fight. In short, more effective.

Military experts are perfectly aware of the danger of genetically engineered bioweapons, as their traditional defense measures - e.g.detection methods or vaccines - are easily sidestepped by the artificial microbes. The speedy development of genetic engineering is one driving force to strenghten the Bioweapons Convention and establish a verification system.

**Example 1: Bacteria Causing Unusual Symptoms**
Researchers from Obolensk near Moscow inserted a gene into *Francisella tularensis*, the causative agent of tularemia and a well known biological weapon agent. The gene made the bacteria produce beta-endorphin, an endogenous human drug, which caused changes in the behaviour of mice when infected with the transgenic bacteria. (2) According to the published results, the endorphin gene was not introduced into a fully virulent strain, but only into a vaccine strain.

If inserted into virulent *F. tularensis*, the victims would not show the usual symptoms of tularemia, but instead unusual symptoms that would obscure the diagnosis and delay therapy. Development of symptom-altered BW-agents has been identified as one possible application of genetic engineering for BW purposes by the US Department of Defense. (3)

**Example 2: Transferring A Lethal Factor To Harmless Human Gut Bacteria**
Genetic engineering could make previously harmless bacteria lethal biological weapons by introducing deadly genes from a highly pathogenic organism. This was done by US researchers as early as 1986. They isolated the gene for the lethal factor of Bacillus anthracis, the causative agent of anthrax, and introduced into Escherichia coli, a normally harmless gut bacteria. The US team reported that the lethal factor protein was active in E. coli and displayed the same deadly effects as it did when in its native B. anthracis. (4)

**Example 3: Antibiotic Resistant Anthrax And Tularemia**
Antibiotic resistance is often used as a marker gene in genetic engineering experiments. However, the very same genes could render biological weapons more dangerous by making agents less treatable. Any experiment with biological weapons agents using antibiotic resistance genes has a strong offensive potential, even if in the contect of âdefensiveÔ research. Despite this obvious problem, there is a long list of questionable experiments:

German military researchers at the *Santitaetsakademie der Bundeswehr* in Munich, the main BW research facility of the German army, cultured genetically engineered *Francisella tularensis* subsp. *holarctica* bacteria (5), a close relative of the causative agent of tularaemia. An antibiotic resistance marker gene (tetracyclin) was been inserted into these bacteria.

Recently, researchers from Porton Down in the UK used genes conferring resistance to antibiotics for genetic studies in fully virulent strains of anthrax. (6) In the late 1980s, a researcher at the University of

Massaschussetts in Amherst also introduced antibiotic resistance genes into anthrax, making it less treatable with antibiotics. (7)

There are even more cases: Researchers from the Institut Pasteur in Paris (8) and from a Russian laboratory in Obolensk (near Moscow) (9) introduced antibiotic resistance genes into anthrax bacteria.

All these studies are allegedly "basic research", where antibiotic resistance is used as a marker gene. But it is obvious that the very same genetically engineered bacteria can be used to design more effective bioweapons compared to the natural anthrax strains.

**Example 4: Invisible Anthrax**
In December 1997, the same Russian research group from Obolensk published a paper in a British scientific journal on another effort to genetically engineer anthrax. (10) By putting new genes into fully pathogenic strains of anthrax, the scientists altered anthraxÔs immunopathogenic properties, making existing anthrax vaccines ineffective against the new genetically-engineered types.

In most cases, detection of bioweapons relies on molecular recognition of the microbe using antibodies similar to the human immune system. Altering the immunogenicity not only overcomes vaccinations; but also the detection systems.

Western military experts were alarmed by this work. The chief of the bacteriology division at the US Army Medical Research Institute of Infectious Diseases (USAMRIID) in Fort Detrick, Md, Col. Arthur Friedlander, commented: "*This is the first indication we're aware of in which genes are being put into a fully virulent strain. They genetically engineered a strain that's resistant to their own vaccine, and one has to question why that was done*". (11)

The Russian researchers also constructed a new vaccine against the new strain. This is of particular importance, as it could enable an army to use such a bioweapon by vaccinating their soldiers against a specific strain, while the enemy remains vulnerable. The case is an example of the frightening potential of genetic engineering applied to biological weapons research (Sunshine Project, 2000).

**Title:** Losing The Race With Bugs: Bacteria Beats New Drugs
**Date:** April 25, 2002
**Source:** UCLA

**Abstract:** Cheetahs eat gazelles. The fastest cheetahs catch more gazelles and breed more; and over generations, cheetahs get faster. But gazelles evolve, too. Faster gazelles live longer and breed more; over generations, they get faster, too.

The same evolutionary dynamics apply to humans and bacteria. We develop antibiotics that kill bacteria. They evolve resistance. We develop better drugs. They evolve resistance to the new drugs.

Cheetahs and gazelles evolve at the same pace. From about 1945 to the early 1980s, humans developed new drugs faster than bacteria evolved. But bacteria now are changing faster than our drugs.

The bugs are winning the race. The more antibiotics we use, the quicker they evolve resistant strains.

A common bacterium called pneumococcus, which causes ear and sinus infections as well as more serious illness, first showed resistance to penicillin in the 1960s. Into the early 1990s, only 5% of cases were resistant, according to the Centers for Disease Control and Prevention. By the end of the 1990s, penicillin couldn't touch nearly 40% of cases in some parts of the U.S.

Tuberculosis will kill more this year than last because a drug-resistant strain has evolved. "Strains of five bacterial species capable of causing life-threatening illnesses already evade every antibiotic in the clinician's armamentarium," says Stuart Levy, a Tufts University microbiologist.

The science is clear. The medical establishment is alarmed. The bioterrorism threat intensifies concern. The issue is: what to do?

Think of antibiotic effectiveness as a natural resource, like fish, that we're depleting rapidly, suggests economist Ramanan Laxminarayan of Resources for the Future, a think tank in Washington, D.C. "Everyone harvests this resource, caring only about himself and ignoring the potential harm to others," he says.

Each commercial fisherman profits by catching more fish, no matter how depleted the ocean stocks. Each parent will press a pediatrician for a drug if there's any chance it will cure a child. Yet if every parent and pediatrician does the same, they will speed the evolution of drug-resistant microbes. And what drug company will enlist its marketers to prod doctors to prescribe its antibiotics less?

Until now, the main remedy has been preaching, the equivalent of pleas to commuters to carpool. Government, doctors' groups and insurers are trying to persuade patients and doctors to avoid antibiotics where they won't work, in treating viral infections, for instance.

In northern California, Kaiser Permanente, the big HMO, has reduced antibiotic use by 30% during the past two years by showing doctors how their prescription patterns differ from peers and using posters to educate patients. The CDC, among other things, offers doctors "viral prescription pads" with treatment tips so patients whose ailments can't be helped by antibiotics don't go away empty-handed. It sees signs that this public-relations campaign is succeeding.

Such education is essential, but it won't suffice. So in quiet conversations, scientists and economists are beginning to think about stronger medicine.

One option is discouraging unnecessary drug use by charging consumers more for the most-overused antibiotics or for newer, heavily promoted drugs that ought to be held in reserve. Increasing drug prices -- even if only for people whose insurance policies cover most of the cost -- sounds jarring. But Mr. Laxminarayan draws the parallel to the campaign against smoking, which, he notes, "was accomplished through both cigarette-tax increases and information campaigns" after public pressure overwhelmed opposition from smokers and tobacco companies.

This approach assumes that resistance is simply caused by overuse. It isn't. Higher prices or an antibiotic tax won't solve the problem of incomplete treatment -- not finishing a prescribed dose or, in poor places, not having enough medicine to kill bacteria -- which also gives the bugs an edge.

The bugs also get an edge when doctors all tend to use the same drugs. Despite the famously decentralized U.S. health-care system, the five most commonly used antibiotics account for 80% of all antibiotic prescriptions.

To save money, insurers, hospitals and HMOs often limit the menu of drugs available, reasonably seeking to use the most cost-effective medicine. But using different drugs for the same ailment in different people or at different times, much as farmers rotate crops, may be prudent. This requires more coordination than is possible in the decentralized U.S. system, although some hospitals, prodded by the CDC, are moving in this direction.

Another solution would be to pull ahead of the microbes. A new pneumococcus vaccine will help. But we also need new potent families of antibiotics. We haven't found one in decades, and big pharmaceuticals firms are devoting R&D money to more-lucrative drugs that treat chronic conditions such as cancer or impotence.

So there is talk, and not just from drug companies, of new ways to stimulate research into new antibiotics. One possibility is tinkering with patent rules to make them broader, both to lure research money and to give drug companies more incentive to market drugs with an eye to the evolutionary dangers.

Devising the right remedies and selling them won't be easy. It never is when near-term interests, whether those of patients or of drug companies, diverge from the long-term interests of humankind (UCLA, 2002).

**Title:** A Weapon Weakened: Antibiotics
**Date:** February 24, 2003
**Source:** LA Times

**Abstract:** Since hitting the market in 1987, Cipro has been the penicillin of its time, good for knocking out a wide variety of infections. But an increasing percentage of bacteria have grown resistant to this powerful antibiotic, narrowing treatment options and reminding us that microbes find ways to overcome every assault.

Researchers writing in the Feb. 18 issue of the *Journal of the American Medical Assn.* found that in hospital intensive care units, fewer bacteria responsible for respiratory and urinary tract infections are responding to Cipro. An analysis of bacteria samples from hospitals in 43 states plus the District of Columbia found that the percentage of bacteria like *Pseudomonas* and *E. coli* that are susceptible to Cipro fell from 89% in 1990-93 to 76% in 2000.

"The biggest fear is we are losing the battle, that nature can stay ahead of us with mutations," said Dr. Keith Beck, an infectious disease specialist at Harbor-UCLA Medical Center in Torrance.

For now, the arsenal isn't empty. Doctors treating vulnerable hospitalized patients can attack infections with so-called gram-negative bacteria like *Pseudomonas* using existing antibiotics, such as some penicillins and cephalosporins, and aminoglycosides like gentamicin and amikacin. Often, they'll use a combination of these drugs. Instead of overusing fluoroquinolones like Cipro and Levaquin for pneumonia, physicians can still rely on macrolides like erythromycin and clarithromycin (Biaxin) and cephalosporins, which do not create as much gram-negative resistance problems. Although all bacteria have inner-cell membranes, gram-negative bacteria are tough targets because they have an outer membrane that keeps some antibiotics from entering the cell; gram-positive bacteria have a single membrane.

But there's a misperception among consumers that there will always be a new antibiotic around the corner.

"It's not true anymore," said Dr. Stuart B. Levy, director of the Center for Adaptation Genetics and Drug Resistance at Tufts University in Boston. The antibiotic pipeline has slowed in recent years, even as the time it takes for a new drug to lose its effectiveness grows ever-shorter.

For gram-positive bugs, such as streptococcus and staphylococcus, there are powerful new drugs like Zyvox and Synercid and one still in trials called Daptomycin. But for the gram-negatives, there are fewer options. Some promising approaches are coming from small biotechnology companies. Levy started his own to develop new forms of tetracycline that get around the resistance problem. He's also working on molecules that interfere with a bacterium's ability to cause infection.

Although bacteria become resistant through mutations or by picking up resistant genes from other bugs, some of the problem is preventable.

About 75% of all antibiotics prescribed in the United States are given for upper respiratory illnesses: colds, sore throats, bronchitis, sinus and ear infections. Yet, at least half of those prescriptions aren't needed because the infections are caused by viruses, not bacteria. Every time patients take them unnecessarily or improperly -- for example, by not finishing a full course -- the strongest bugs survive the antibiotic hit and flourish.

Levy, founder of the international Alliance for the Prudent Use of Antibiotics, says consumers have come to think antibiotics kill everything: "They believe they're cure-alls; they believe they deserve to have them." And they have ready access through compliant doctors and online pharmacies. He cited the example of Americans stockpiling Cipro after it was prescribed to those potentially exposed to anthrax.

To curb inappropriate use, some hospitals have had success restricting antibiotic prescriptions.

Levy warns that by overusing antibiotics, "we are sowing the seeds of our own destruction. You can't imagine these fabulous drugs are creating in their wake the biggest problem we've ever faced" (LA Times, 2003).

**Title:** CDC To Mix Avian, Human Flu Viruses In Pandemic Study
**Date:** January 24, 2004
**Soure:** CIDRAP

**Abstract:** One of the worst fears of infectious disease experts is that the H5N1 avian influenza virus now circulating in parts of Asia will combine with a human-adapted flu virus to create a deadly new flu virus that could spread around the world.

That could happen, scientists predict, if someone who is already infected with an ordinary flu virus contracts the avian virus at the same time. The avian virus has already caused at least 48 confirmed human illness cases in Asia, of which 35 have been fatal. The virus has shown little ability to spread from person to person, but the fear is that a hybrid could combine the killing power of the avian virus with the transmissibility of human flu viruses.

Now, rather than waiting to see if nature spawns such a hybrid, US scientists are planning to try to breed one themselves—in the name of preparedness.

The Centers for Disease Control and Prevention (CDC) will soon launch experiments designed to combine the H5N1 virus and human flu viruses and then see how the resulting hybrids affect animals. The goal is to assess the chances that such a "reassortant" virus will emerge and how dangerous it might be.

CDC officials confirmed the plans for the research as described recently in media reports, particularly in a Canadian Press (CP) story.

**Two ways to make hybrids**
The plans call for trying two methods to create hybrid viruses, CDC spokesman David Daigle told CIDRAP News via e-mail. One is to infect cells in a laboratory tissue culture with H5N1 and human flu viruses at the same time and then watch to see if they mix. For the human virus, investigators will use A (H3N2), the strain that has caused most human flu cases in recent years, according to the CP report.

The other method is reverse genetics—assembling a new virus with sets of genes from the H5N1 and H3N2 viruses. Reverse genetics has already been used to create H5N1 candidate vaccines in several laboratories, according to Daigle. The National Institutes of Health (NIH) said recently it would soon launch a clinical trial of one of those vaccines.

Of the two methods, the co-infection approach was described as slower and more laborious, though closer to what happens in nature.

Any viable viruses that emerge from these processes will be seeded into animals that are considered good models for testing how flu viruses behave in humans, according to Daigle. The aim will be to observe whether the animals get sick and whether infected animals can infect others.

The World Health Organization (WHO) has been "pleading" for laboratories to do this research, because it could provide some evidence to back up the agency's warnings about the risk of a flu pandemic, according to the CP report.

Klaus Stohr, head of the WHO's global influenza program, was quoted as saying that if none of the hybrids caused disease, the agency might be inclined to dial down its level of concern. But if the experiments produce highly transmissible and pathogenic viruses, the agency will be more worried, he said.

**Safety precautions**
Because of the obvious risks in creating viruses with the potential to spark a pandemic, the work will be done in a biosafety level 3 (BSL-3) laboratory at the CDC in Atlanta, Daigle told CIDRAP News.

"We recognize that there is concern by some over this type of work. This concern may be heightened by reports of recent lab exposures in other lab facilities," he said. "But CDC has an incredible record in lab safety and is taking very strict precautions."

Daigle said the US Department of Agriculture requires that highly pathogenic avian influenza (HPAI) viruses be treated as "Select Agents" and that research on them must be done in BSL-3 labs with "enhancements." These include "special provisions to protect both laboratory workers and the environment."

BSL-3 is the second highest level of laboratory biosecurity. It is used for work with pathogens that may cause serious or potentially lethal disease if inhaled, such as tuberculosis or St. Louis encephalitis, according to the CDC.

CDC experiments with HPAI viruses have to pass reviews by the agency's Institutional Biosafety Committee and Animal Care and Use Committee, Daigle said. The facilities involved are inspected by the USDA and the CDC's Office of Safety and Health, and staff members who work with Select Agents require special clearance.

**It's been done before**
The upcoming experiments will not break entirely new ground for the CDC, the CP story revealed. The agency already has made hybrid viruses with H5N1 samples isolated from patients in Hong Kong in 1997, when the virus first caused human disease.

The results of that research have not yet been published, and the CDC has said little about them. In the CP report, Dr. Nancy Cox, head of the CDC's influenza branch, commented only, "Some gene combinations could be produced and others could not."

Daigle added little to that. He said, "The reassortment work with the 1997 isolate was intermittently interrupted with SARS [severe acute respiratory syndrome] and then the 2004 H5N1 outbreak. We are currently concentrating our efforts on understanding the pathogenicity of the 2004 strains (non-reassortants) in mammalian models."

He said the CDC hopes to prepare a report on that research "in the near future" (CIDRAP, 2004).

**Title:** Super-Bacteria Eat Antibiotics For Breakfast
**Date:** April 3, 2004
**Source:** Discovery

**Abstract:** Antibiotics are meant to kill bacteria, so it might be disheartening to learn that some bacteria can literally eat antibiotics for breakfast. In fact, some species can thrive quite happily on nothing *but* antibiotics, even at high concentrations.

The rise of drug-resistant bacteria poses a significant threat to public health and many dangerous bugs seem to be developing resistance at an alarming rate. The headline-grabbing MRSA may be getting

piggybacks from livestock to humans, while several strains of tuberculosis are virtually untreatable by standard drugs.

But a startling new study reveals just how widespread antibiotic resistance really is. Gautam Dantas from Harvard Medical School managed to culture antibiotic-eating bacteria from every one of 11 soil samples, taken from farmland and urban areas across the US. All eleven were positively loaded with a diverse group of bacteria that were extremely resistant to a wide range of antibiotics at high concentrations.

**Soil Super--Bugs**

In their natural environment, these soil bacteria are frequently exposed to a massive array of antibiotics from plants and other microbes, and have evolved ways of detecting and evading them. These resistant strains act as a living reservoir of innovative genetic means of resisting antibiotics, known as the 'antibiotic resistome'.

Dantas searched for resistant bacteria by culturing colonies that could grow in solutions where antibiotics were their only source of carbon. He tested 18 different antibiotics that are used to kill a variety of different bacterial species. Some of these were natural, others man-made; some were old, others new. But every single one managed to support at least one strain of bacteria. Six of them, including commonly used drugs like penicillin, vancomycin, ciprofloxacin and carbenicillin, even managed to feed bacteria from all 11 soils.

The degree of resistance in the soil bacteria was nothing short of extraordinary. Dantas cultured a representative set of 75 resistant strains and found that on average, they resisted 17 of the 18 antibiotics at low concentrations of 20 milligrams per litre (full bars in image below). But even at higher concentrations of 1 gram per litre (filled bars in image below), each strain managed to stand firm against an average of 14 out of 18 drugs.

When Dantas studied some of these strains more closely, he found that they nullified the drugs using similar techniques to the drug-resistant versions of disease-causing bacteria. Some shunted the antibiotics out of their cells with molecular pumps, others used enzymes to cut up the drugs, and yet others reprogrammed their own genetic code to deprive antibiotics of their targets.

**Reservoir of Resistance**

The real danger is that the soil-living species could provide new defences that more dangerous ones can draw on to shrug off our best drugs. Bacteria are capable of passing genetic material between one another as easily as two humans might swap business cards, making it trivial for the soil super-bugs to pass their crucial genes on to more dangerous species. To see how easily this could happen, have a look at this earlier post about how the food poisoning bug *Salmonella* has passed a resistance gene on to the Black Death bacterium.

In principle, bacteria should be more able to successfully take up resistance genes from other closely related species. It's worrying then that Dantas's antibiotic-eaters belonged to such diverse groups. By establishing a family tree of the different strains, he found that they were members of at least 11 different bacterial groups, although over half of them came from just two orders – the *Burkholderiales* and the *Pseudomonadales*. These include a wide variety of species that are known to infect hospital patients with weakened immune systems.

They are known for their large genome sizes (well, large for bacteria anyway) and some groups have suggested that these sizeable genomes allow them to metabolise a wide range of chemicals, antibiotics included. This unusual diet will come as no surprise to many a microbiologist. Bacteria can colonise some of the most extreme environments on the planet and can survive on the most unlikely to food sources,

from crude oil to toxic waste. Now, it seems that they can also survive solely on chemicals that are meant to kill them (Discovery, 2008).

**Title:** Bird Flu Virus Has Mutated Into Form That's Deadly To Humans
**Date:** March 6, 2008
**Source:** Natural News

**Abstract:** The avian flu has undergone a critical mutation making it easier for the virus to infect humans, according to a study conducted by researchers at the University of Wisconsin at Madison and published in the journal *PLoS Pathogens.*

"We have identified a specific change that could make bird flu grow in the upper respiratory tract of humans," lead researcher Yoshihiro Kawaoka said.

The H5N1 strain of influenza, also known as "bird flu," has decimated wild and domestic bird populations across the world since it emerged between 1999 and 2002. This highly virulent variety of the flu has been identified as a public health concern because in the past, varieties of influenza have mutated and crossed the species barrier to humans.

Since 2003, 329 humans have been confirmed infected with H5N1, with 201 fatalities. The vast majority of these worked closely with infected birds, such as in the poultry industry.

One of the primary things that keeps bird flu from infecting humans is that the virus has evolved to reproduce most effectively in the bodies of birds, which have an average body temperature of 106 degrees Fahrenheit. Humans, in contrast, have an average body temperature of 98.6 degrees, with temperatures in the nose and throat even lower (91.4 degrees). This vast temperature difference makes it very difficult for the bird flu virus to survive and grow in the human body.

In the current study, researchers found that a strain of H5N1 has developed a mutation that allows it to thrive in these lower temperatures.

"The viruses that are circulating in Africa and Europe are the ones closest to becoming a human virus," Kawaoka said. But he pointed out that one mutation is not sufficient to turn H5N1 into a major threat to humans.

"Clearly there are more mutations that are needed. We don't know how many mutations are needed for them to become pandemic strains."

"We are rolling the dice with modern poultry farming practices," warned consumer health advocate Mike Adams, author of the book How to Beat the Bird Flu. "By raising chickens in enclosed spaces, treating them with antibiotics, and denying them access to fresh air, clean water and natural sunlight, we are creating optimal conditions for the breeding of highly infectious diseases that can quickly mutate into human pandemics," Adams said. "Given current poultry farming practices, it is only a matter of time before a highly virulent strain crosses the species barrier" (Natural News, 2008).

**Title:** Drugs That Work Against Each Other Could Fight Resistant Bacteria
**Date:** December 13, 2008
**Source:** Discovery

**Abstract:** When normal bacteria are exposed to a drug, those that become resistant gain a huge and obvious advantage. Bacteria are notoriously quick to seize upon such evolutionary advantages and resistant strains rapidly outgrow the normal ones. Drug-resistant bacteria pose an enormous potential threat to public health and their numbers are increasing. MRSA for example, has become a bit of a media darling in Britain's scare-mongering tabloids. More worryingly, researchers have recently discovered a strain of tuberculosis resistant to all the drugs used to treat the disease.

New antibiotics are difficult to develop and bacteria are quick to evolve, so there is a very real danger of losing the medical arms race against these 'super-bugs'. Even combinations of drugs won't do the trick, as resistant strains would still flourish at the expense of non-resistant ones. Antibiotic combos could even speed up the rise of super-bugs by providing a larger incentive for evolving resistance.

Clearly, fighting the rapidly evolving nature of bacteria is a dead end. So Remy Chait, Allison Craney and Roy Kishoni from Harvard Medical School used a different strategy – they changed the battle-ground so that non-resistant bacteria have the advantage. And they have done so using the seemingly daft strategy of using combinations of drugs that work poorly together, and even those that block each other's effects.

The trio looked at two strains of the common bacteria Escherichia coli – one that was normal, and another that was resistant to doxycycline. Doxycycline is widely used to fight off a variety of bacterial invaders, but resistant E.coli use a specialised molecular pump to remove the drug. It can withstand 100 times more doxycycline than its normal counterparts.

First, the team hit the two strains with doxycycline and erythromycin, a combination of drugs that work particularly well together and enhance each other's effects. The resistant strain was certainly more vulnerable to this double-whammy, but as expected, it always outperformed the normal bugs. With that advantage and enough time, it would inevitably evolve resistance to both drugs.

But Chait managed to remove this evolutionary impetus by combining doxycycline with a third drug, ciprofloxacin, a combination that would normally be useless. Doxycycline actually blocks the effects of ciprofloxacin, and the two drugs together are weaker than either alone. Predictably, the resistant bug did what it had evolved to do – it pumped out doxycycline. But in doing so, it also unwittingly removed the block on ciprofloxacin, restoring this second drug to its full killing power.

The normal strain encountered no such problem. By leaving the drugs alone, it never faced the full effects of either, and out-competed their more heavily-pummelled resistant cousins.

Chait cautions that it's too early to transfer his findings across to hospital beds. The experiment used non-lethal antibiotic concentrations in a very controlled environment. But they have certainly pointed other researchers down a new and interesting path.

Combinations of drugs that block each other have previously been dismissed by doctors because they would require higher doses. But Chait's study suggests that they could be the key to controlling bacterial drug resistance. We clearly can't stop bacteria from evolving, but we can certainly steer the course of that evolution in our favour (Discovery, 2008).

**Title:** New Flu Strain Is A Genetic Mix
**Date:** April 24, 2009
**Source:** Reuters

**Abstract:** A deadly swine flu never seen before has broken out in Mexico, killing at least 16 people and raising fears of a possible pandemic. World Health Organization officials said the flu has killed about 60 Mexicans.

**Here are some facts about the virus and flu viruses in general:**

**1.** The World Health Organization has confirmed at least some of the cases are a never-before-seen strain of influenza A virus, carrying the designation H1N1.

**2.** Although it's called swine flu, this new strain is not infecting pigs and has never been seen in pigs. The threat is person to person transmission.

**3.** It is genetically different from the fully human H1N1 seasonal influenza virus that has been circulating globally for the past few years. The new flu virus contains DNA typical to avian, swine and human viruses, including elements from European and Asian swine viruses.

**5.** The World Health Organization is concerned but says it is too soon to change the threat level warning for a pandemic-- a global epidemic of a new and dangerous flu.

**6.** When a new strain of flu starts infecting people, and when it acquires the ability to pass from person to person, it can spark a pandemic. The last pandemic was in 1968 and killed about a million people.

**7.** Seven people in the United States have been diagnosed with the new strain. All have recovered, but the U.S. Centers for Disease Control and Prevention expects more cases.

**8.** Flu viruses mutate constantly, which is why the flu vaccine is changed every year, and they can swap DNA in a process called reassortment. Most animals can get flu, but viruses rarely pass from one species to another.

**9.** From December 2005 through February 2009, 12 cases of human infection with swine influenza were confirmed. All but one person had contact with pigs. There was no evidence of human-to-human transmission in those cases.

**10.** Symptoms of swine flu in people are similar to those of seasonal influenza -- sudden onset of fever, coughing, muscle aches and extreme tiredness. Swine flu appears to cause more diarrhea and vomiting than normal flu.

**11.** Seasonal flu kills between 250,000 and 500,000 people globally in an average year.

**12. In 1976 a new strain of swine flu started infecting people and worried U.S. health officials started widespread vaccination. More than 40 million people were vaccinated. But several cases of Guillain-Barre syndrome, a severe and sometime fatal condition that can be linked to some vaccines**, caused the U.S. government to stop the program. The incident led to widespread distrust of vaccines in general (Reuters, 2009).

**Title:** Swine Flu Smoking Gun? CDC Was Combining Flu Viruses In 2004
**Date:** April 29, 2009
**Source:** Natural News

**Abstract:** Last week, when what is now called a "swine flu" was first reported to be infecting and killing some people in Mexico, health officials noted it was a strain of flu never before seen. In fact, it is technically incorrect to call this simply a "swine" flu. Analyses showed it's a mixture of swine, human and avian viruses, according to the Centers for Disease Control (CDC). Moreover, it is genetically different from the fully human H1N1 seasonal influenza virus that has been circulating globally for the past few years. ***Bottom line: the new flu virus contains DNA from avian, swine viruses (including elements from European and Asian viruses) and human viruses.***

So did this curious mixture just develop naturally, out of the blue? Is it the result of inhumane farming practices, as the Humane Society of the United States (http://www.hsus.org/) has suggested, that exposes immune-compromised pigs to all sorts of animal and human feces?

Well, maybe. But let's go back and look at the facts to see if any other scenario could be possible.

First of all, there's the troublesome detail that the virus has elements that come from multiple continents. Then there's the fact that true swine flu is only rarely transmissible to humans -- this flu is spreading human-to-human, most likely because it contains DNA from human flu.

Could someone have deliberately mixed these viruses together? Is that possible? Absolutely.

Was this virus mixing being done artificially in the lab, or had it already been done? Yes.

Who was blending potentially swine, human and/or avian viruses in labs? Were those horrible generic boogie men known to Americans far and wide as "terrorists" doing it? There's no proof of bioterrorism at work here yet. However, there is evidence the United States government has been working on concocting new flu virus blends.

So could the hysteria-provoking, new swine flu have escaped from a lab? Or was it deliberately released as some kind of test? When these kinds of questions are asked, the knee-jerk reaction of the mainstream media (MSM) is to giggle and talk about "conspiracy theories" and to joke about wearing tinfoil hats.

But here's the potential smoking gun, the facts that suggest a potential source of the pandemic could be CDC labs. And at the very least, this possibility deserves thoughtful examination and research.

The University of Minnesota Center for Infectious Disease Research and Policy (CIDRAP) is hardly a place most Americans have heard about and, apparently, the Center's web site has news the MSM isn't familiar with, either. But information they published years ago has now taken on an urgent importance. ***CIDRAP, along with the Canadian newspaper Canadian Press (CP), revealed back in 2004 that the CDC was launching experiments designed to mix the H5N1 (avian) virus and human flu viruses. The goal was to find out how likely it was such a "reassortant" virus would emerge and just how dangerous it might be.*** Of course, it's logical to wonder if they also worked with the addition of a swine flu virus, too.

Here's some background from the five-year-old report by the University of Minnesota research center: "One of the worst fears of infectious disease experts is that the H5N1 avian influenza virus now circulating in parts of Asia will combine with a human-adapted flu virus to create a deadly new flu virus that could spread around the world. That could happen, scientists predict, if someone who is already infected with an ordinary flu virus contracts the avian virus at the same time. The avian virus has already caused at least 48 confirmed human illness cases in Asia, of which 35 have been fatal. The virus has shown little ability to spread from person to person, but the fear is that a hybrid could combine the killing power of the avian virus with the transmissibility of human flu viruses. ***Now, rather than waiting to see if nature spawns such a hybrid, US scientists are planning to try to breed one themselves -- in the name of preparedness."***

And CDC officials actually confirmed the government had plans for the research. The CIDRAP News folks did a great job covering this important issue, which was apparently mostly ignored by the MSM back in 2004, and CIDRAP News wrote to the CDC for information. This e-mail produced an answer from CDC spokesman David Daigle who admitted the CDC was working on the project in two ways. "One is to infect cells in a laboratory tissue culture with H5N1 and human flu viruses at the same time and then watch to see if they mix. For the human virus, investigators will use A (H3N2), the strain that has caused most human flu cases in recent years," the CIDRAP story stated. This co-infection approach was described as slow and labor-intensive. However, it was a way to produce a new virus that appeared to be closer to what develops in nature.

There was another, faster way CDC scientists could create the mix, too. Called reverse genetics, it involves piecing together a new virus with genes from the H5N1 and H3N2 viruses. Reverse genetics had already been used successfully to create H5N1 candidate vaccines in several laboratories, the CDC's Daigle wrote. "Any viable viruses that emerge from these processes will be seeded into animals that are considered good models for testing how flu viruses behave in humans... The aim will be to observe whether the animals get sick and whether infected animals can infect others," he revealed in his e-mail.

What's more, the CP reported the CDC had already made hybrid viruses with H5N1 samples isolated from patients in Hong Kong in 1997, when there was the first outbreak of that virus, dubbed the "Hong

Kong flu". It is not clear if the results of that research were ever published. Back in 2004, Dr. Nancy Cox, then head of the CDC's influenza branch, would tell the CP only: "Some gene combinations could be produced and others could not."

The CP's report noted that the World Health Organization (WHO) had been "pleading" for laboratories to do this blending-of-viruses research. The reason? If successful, these flu mixes would back up WHO's warnings about the possibility of a flu pandemic. In fact, Klaus Stohr, head of the WHO's global flu program at the time, told the CP that if the experiments were successful in producing highly transmissible and pathogenic viruses, the agency would be even more worried -- but if labs couldn't create these mixed flu viruses, then the agency might have to ratchet down its level of concern.

The 2004 CIDRAP News report addressed the obvious risks of manufacturing viruses in labs that, if released, could potentially spark a pandemic. However, the CDC's Daigle assured the Minnesota research group the virus melding would be done in a biosafety level 3 (BSL-3) laboratory. "We recognize that there is concern by some over this type of work. This concern may be heightened by reports of recent lab exposures in other lab facilities," he told CIDRAP. "But CDC has an incredible record in lab safety and is taking very strict precautions."

Five years later, we must ask more questions. Were those safety measures enough? Was the CDC creating or testing any of these virus mixes in or near Mexico? What other potentially deadly virus combinations has the US government created? Don't US citizens, as taxpayers who funded these experiments, have a right to know? And for all the residents of planet earth faced with a potentially deadly global epidemic, isn't it time for the truth? (Natural News, 2009).

**Title:** Norway Says Found H1N1 Mutation In Flu Victims
**Date:** November 20, 2009
**Source:** Reuters

**Abstract:** Norwegian health authorities said on Friday they have discovered a potentially significant mutation in the H1N1 influenza strain that could be responsible for causing the severest symptoms among those infected.

"The mutation could be affecting the virus' ability to go deeper into the respiratory system, thus causing more serious illness," the Norwegian Institute of Public Health said in a statement.

There was no reason to believe the mutation had any implication for the effectiveness of flu vaccines or antiviral drugs made by groups such as Roche (ROG.VX), GlaxoSmithKline (GSK.L), Novartis (NOVN.VX) and AstraZeneca (AZN.L), the authorities said.

The World Health Organisation said that the mutation did not appear to be widespread in Norway and the virus in its mutated form remained sensitive to antivirals and pandemic vaccines.

A similar mutation had been detected in H1N1 viruses circulating in several other countries, including China and the United States, in severe as well as in some mild cases, it said.

"Although further investigation is under way, no evidence currently suggests that these mutations are leading to an unusual increase in the number of H1N1 infections or a greater number of severe or fatal cases," the WHO said in a statement.

H1N1, a mixture of swine, bird and human viruses, has killed at least 6,770 people globally, according to its latest update.

In Norway the mutation was found in the bodies of two people killed by the virus and of one person made seriously ill. The two infected by the mutated virus who died were among the first fatalities from the H1N1 pandemic in Norway, the institute said.

It was unclear whether the mutated virus was transmitted among humans, the health authorities said.

"Based on what we know so far, it doesn't seem like the mutated virus is circulating in the population, but rather that spontaneous changes have happened in the three patients," director Geir Stene Larsen at the public health institute said in the statement.

Norway has seen relatively more fatalities in the flu pandemic compared to the size of the population versus other European countries, with 23 confirmed deaths.

Public health authorities have said this could be due to the country being hit early in the pandemic's northern hemisphere winter wave, before a mass vaccination programme got underway.

"Nevertheless, it is important to study if there's still something about the Norwegian fatalities that separate us from other countries, and that make us learn something that strengthens our treatment of the seriously ill," director Bjorn-Inge Larsen at the Norwegian Directorate of Health said.

Dr. Anne Schuchat of the U.S. Centers for Disease Control and Prevention said, "This mutation has been seen sporadically."

She said it is sometimes seen in patients who have mild influenza symptoms.

"I think it is just too soon to say what this might mean long term," Schuchat told reporters in a telephone briefing. (Reporting by Richard Solem; Additional reporting by Stephanie Nebehay in Geneva and Maggie Fox in Washington; Editing by Matthew Jones and Louise Ireland) (Reuters, 2009).

**Title:** Fighting Bacteria With Bacteria – Common Nose Germ Provides New Weapon Against Superbugs
**Date:** May 19, 2010
**Source:** Discovery

**Abstract:** Our bodies are under siege, constantly fighting back assaults from disease-causing bacteria. But we are also home to many harmless bacterial species that are share our bodies to no ill effects. Now, it seems that these 'commensals' could be our hidden allies against their harmful cousins. In one such ally, a group of scientists has just discovered a potential new weapon against *Staphylococcus aureus*.

*S.aureus* is incredibly common, colonising the noses of a third of people in the USA, UK, Japan and other countries. Often, these colonies do nothing untoward, but if a full-blown infection sets in, the result can include life-threatening diseases like pneumonia, meningitis, toxic shock syndrome, endocarditis and sepsis. With the rise of MRSA and other staph strains that shrug off our most common antibiotics, the threat posed by this common nose bug has never been greater.

But *S.aureus* doesn't have our noses to itself. It has to jostle for space with a close relative called *Staphylococcus epidermidis*. It's the most common commensal in our noses and, indeed, the most common contaminating bacterium in laboratory equipment. *S.epidermidis* is harmless, except in people whose immune systems have been compromised. But more interestingly, it has the ability to stunt the growth of its more infamous cousin. Now, Tadayuki Iwase from Jikei University has isolated the protein it uses to do so.

Iwase swapped the noses of 88 volunteers and found that virtually all of them were colonised by *S.epidermidis*. However, *S.aureus* had only set up shop in just under a third. On the whole, the two

bacteria seem to be able to co-exist in harmony, but Iwase found that some strains of *S.epidermidis* are anathemas to *S.aureus.*

Specifically, they caused problems for *S.aureus's* ability to set up biofilms, the bacterial equivalent of cities. Thousands of bacteria swarm within these communities, embedded in a slimy matrix of DNA, proteins and sugars. Within biofilms, bacteria are harder to kill, making them an important public health challenge. But according to Iwase, some strains of *S.epidermidis* not only prevent *S.aureus* from creating biofilms, they also destroy existing ones. People who were colonised by these defensive strains were around 70% less likely to be colonised by *S.aureus*.

To work out the weapon that was keeping the rival bacteria are bay, Iwase let cultures of *S.epidermidis* cut a swath through *S.aureus* biofilms and analysed their secretions when the destruction had reached its peak. He managed to isolate a single protein called Esp or '*S.epidermidis* serine protease' in full. The protein was absent from strains that couldn't wipe out *S.aureus* biofilms and present in strains that could. If Iwase gave the latter bacteria them a chemical that negates the Esp protein, or if he removed the esp gene from them entirely, they lost their competitive edge against *S.aureus*.

Esp even works in tandem with our own defensive proteins, including one called hBD2 (human beta-defensin 2) that's secreted by our skin cells. Alone, hBD2 can kill bacteria but it's a bit of a wimp about it, while Esp (for obvious reasons) has no bacteria-killing ability of its own. But together, their powers are far greater, and they effectively kill *S.aureus*, even when it was under the protection of biofilms. (The idea that the two proteins have co-evolved with one another is an intriguing question for another time.)

As a final test, Iwase introduced the competitive strains of *S.epidermidis* into the noses of volunteers who were already colonised by *S.aureus*. Sure enough, these transplanted bacteria eliminated their evolutionary cousins. Even a purified dose of Esp alone did the trick.

These experiments are very exciting. Humans are fighting a pitched (possibly losing) battle against staph and MRSA in particular, and our antibiotic arsenal is falling short. What better source of new weapons than other bacteria that have been fighting the same fight for millennia? Obviously, there's a lot of work to do to turn Esp into a viable treatment, but this study is a promising first step.

Even better, it seems that, for some unclear reason, *S.aureus* can't evolve resistance to Esp. With its biofilms under attack, you would expect *S.aureus* to quickly adapt, but after a year of culturing the two species together, Iwase couldn't find any evidence that of resistance (Discovery, 2010).

**Title:** Charitable Bacteria Protect Vulnerable Sisters From Antibiotics
**Date:** September 1, 2010
**Source:** Discovery

**Abstract:** Humans are capable of great charity, taking hits to their bank accounts and bodies to benefit their peers. But such acts of altruism aren't limited to us; they can be found in the simple colonies of bacteria too.

Bacteria are famed for their ability to adapt to our toughest antibiotics. But resistance doesn't spring up evenly across an entire colony. A new study suggests that a small cadre of hero bacteria are responsible for saving their peers. By shouldering the burden of resistance at a personal cost, these charitable cells ensure that the entire colony survives.

Henry Lee from the Howard Hughes Medical Centre assaulted a vat of *Escherichia coli* with increasingly strong waves of the drug norfloxacin, always using just enough to seriously impede their growth without killing them outright. As expected, the group became more resistant over time. By the end of the experiment, they were shrugging off doses of antibiotics that would have previously killed them.

But Lee found that not all the bacteria were equal. Most still remained vulnerable to the drug, and the group's overall defences were bolstered by a small group of highly resistant individuals. The leaders of the resistance ~~had all developed a mutation in a gene called~~ all had particularly high levels of a protein called tryptophanase. Tryptophanase breaks down the amino acid tryptophan and produces indole, a chemical that acts like a call to arms. It rallies the colony into action.

When bacteria detect indole, they start mass-producing molecular pumps that evict any drugs that have breached their walls. With these molecules, the beleaguered bacteria can pump out norfloxacin faster than it can kill them.

Indole also tells bacteria to start toughening up. In response, the cells tune down certain genes that norfloxacin would normally use to kill them and tune up genes that protect their insides from damage. By producing indole, the most resistant bacteria were prompting changes in their weaker neighbours that greatly increased the amount of norfloxacin they could withstand.

When Lee peered into the genes of the most resistant cells, he found that their own resistance was the result of several personal adaptations that averted death by norfloxacin. They had altered genes that would normally be targeted by the drug, removing its targets. They had switched on genes that protect them from chemical damage or that mass-produce produce drug-pumps. None of these mutations affect the production of indole; they just gave the mightiest cells the chance they needed to produce this rallying chemical.

When Lee challenged his bacteria with another drug called gentamicin, he found exactly the same thing – a resistant elite promoting the survival of the group by releasing waves of indole. This seems to be a general tactic, rather than a drug-specific one.

Producing indole isn't easy; it takes energy to manufacture. Why should a small number of bacteria shoulder this burden to protect other members of the colony? Lee thinks that relationships are the answer. Having multiplied from common ancestors, the bacteria in the group are all related to one another and carry virtually the same genes. In this light, making a small sacrifice for the sake of genetically identical others is a good move (Discovery, 2010).

**Title:** Tough Bacteria Use Domesticated Viruses To Resist Antibiotics
**Date:** January 5, 2011
**Source:** Discovery

**Abstract:** Even bacteria get sick. Tiny though they are, bacteria can be infected by even tinier viruses known as phages. Like tiny hypodermic needles, phages inject their genetic material into their bacterial hosts, turning them into factories for making more phages. The host usually dies in the aftermath. But some bacteria have turned these enemies into their allies. By adding the viruses' DNA into their own genomes, they have become superbugs, able to tolerate harsh environments and shrug off antibiotics.

Once phages have injected their genes into a bacterium, they can make copies of themselves in two ways. The first is a brutish approach. The genes commandeer the host, using it to manufacture new viruses that eventually burst out of the cell – this is the lytic cycle. Alternatively, the phage DNA can infiltrate the bacterium's genome, becoming part of it. When the bacterium divides in two, it copies the phage's genes along well as its own. This is the lysogenic cycle, an altogether stealthier approach to making more phages.

Within the bacterial genome, the viral DNA is called a prophage. After being copied many times over in these new surroundings, it can pop out again to create a new phage. The prophage is little more than a genetic parasite. But sometimes, a prophage gets trapped by a crippling mutation. Unable to pop out, it becomes a genetic fossil, forever stuck within its host and destined only to preserve a trace of a past infection.

These captives are called cryptic prophages and they can make up a fifth of a bacterium's DNA. Their existence is puzzling. Bacteria are known for having small, streamlined genomes, yet in they have foreign and potentially harmful viral DNA loitering among their genes. Why?

To find out, Xiaoxue Wang from Texas A&M University found all nine cryptic prophages from the common bacterium *Escherichia coli* and, with care and precision, snipped them all out. And to his surprise, the bacteria were the worse for it.

The prophages weren't essential by any means. Without them, the bacteria survived quite reasonably, although they grew more slowly than normal strains. But they proved to be wimps when challenged with difficult conditions. They became up to 400 times more sensitive to antibiotics. They succumbed more readily to extremely salty or acidic conditions. And they were almost completely unable to form biofilms – fortified 'cities' where the microbes gather under the shelter of substances that they themselves secrete.

In many of these cases, Wang could weaken the bacteria by removing a single prophage, which suggests that many of the genes are active parts of the host. The cryptic prophages are no longer selfish parasites, nor are they truly passive fossils. Rather, they have been domesticated to serve their host.

There are other examples of phages bestowing important powers upon the bacteria they infect. *E.coli* is typically harmless but if it gets infected with the right phage, it can turn into a monster that causes dysentery. The phage inserts two genes into the bacterium's genome, which allow it to produce poisons called Shiga toxins. Phages carry the CTX toxin that the bacterium *Vibrio cholerae* needs to cause cholera. Phages allow the bacteria that causes anthrax to find shelter in the guts of earthworms. Phages even allow bacteria to come to the aid of aphids. But in these cases, the phage genes need to pop out of their host. In the case of the cryptic prophages, even though the viral genes stay put, the bacterium still reaps the benefits.

Bacteria are great survivors, able to adapt to a wide variety of conditions, from oil-soaked oceans to arsenic-rich lakes to antibiotic-treated humans. Wang's study suggest that phages could provide bacteria with new ways of coping with these environments, maybe even acting as vehicles for transporting genes from one species to another. He writes, "In effect, the cell uses the tools it obtained from its former enemy, phage, to cope with new environments."

Now that we know about these alliances, we could use them to our advantage. Wang suggests that we could find new ways of preventing bacteria from resisting our antibiotics by blocking the proteins produced by their domesticated viruses (Discovery, 2011).

**Title:** Fighting Evolution With Evolution – Using Viruses To Target Drug-Resistant Bacteria
**Date:** May 31, 2011
**Source:** Discovery

**Abstract:** We are losing the war against infectious bacteria. They are becoming increasingly resistant to our antibiotics, and we have few new drugs in the pipeline. Worse still, bacteria can transfer genes between each other with great ease, so if one of them evolves to resist an antibiotic, its neighbours can pick up the same ability. But Matti Jalasvuori from the University of Jyvaskyla doesn't see this microscopic arms-dealing as a problem. He sees it as a target.

Usually, antibiotic-resistance genes are found on rings of DNA called plasmids, which sit outside a bacterium's main genome. Bacteria can donate these plasmids to one another, via their version of sex. The plasmids are portable adaptations – by trading them, bacteria can rapidly respond to new threats. But they aren't without their downsides. Plasmids can sometimes attract viruses.

Bacteriophages (or “phages” for short) are viruses that infect and kill bacteria, and some of them specialise on those that carry plasmids. These bacteria may be able to resist antibiotics, but against the phages, their resistance is futile.

Scientists have known about these plasmid-hunting phages for over four decades, but Jalasvuori has only now shown that they could prove useful to us. He found that the phages can dramatically reduce the level of antibiotic resistance in colonies of bacteria, by selectively assassinating the plasmid-carriers.

Jalasvuori worked with two common gut bacteria – *Escherichia coli* and *Salmonella enterica* – both of which carried plasmids with antibiotic-resistance genes. In the absence of phages, all of the bacteria resisted antibiotics. When Jalasvuori added a phage called PRD1, that proportion fell to just 5% within 10 days.

The bacteria adapted to the phage assault by jettisoning their plasmids, and with them, their antibiotic-resistance genes. These survivors were now resistant to *phages,* but the vast majority of them could once again be killed by antibiotics.

The method isn’t perfect. A small proportion of the bacteria resisted both phages *and* antibiotics. However, Jalasvuori found that they also formed smaller colonies and had lost the ability to swap genes between one another. Their invincibility came at a substantial cost – compared to normal cells, they were hobbled eunuchs.

Targeting plasmids is a clever strategy that uses the rapid evolution of bacteria against them. Rather than coming up with new weapons in an ever-escalating arms race, Jalasvuori made it too costly for bacteria to keep their defences. It’s like tackling gun crime by penalising gun ownership rather than developing better bullet-proof vests.

However, Jalasvuori is refreshingly cautious about his work. He says, “There are a number of important caveats to these promising preliminary results.” For a start, his bacteria evolved under the threat of phages, but not antibiotics. If they had been exposed to both, there would almost certainly have been more double-resistant strains, which could have ultimately found ways of getting over their weaknesses.

On top of that, not all plasmids are the same; some could potentially hide from threatening phages, and go on to harbour resistance genes. Finally, as Jalasvuori writes, “As with all test-tube studies, the relevance to natural environments is unclear.”

It’s debatable whether this would ever lead to a practical way of dealing with drug-resistant microbes, but it’s certainly a lead. And with a problem as worrying as antibiotic resistance, every lead is an interesting one (Discovery, 2011).

**Title:** House Mice Picked Up Poison Resistance Gene By Having Sex With Related Species
**Date:** July 21, 2011
**Source:** Discovery

**Abstract:** Since 1948, people have been poisoning unwanted rats and mice with warfarin, a chemical that causes lethal internal bleeding. It’s still used, but to a lesser extent, for rodents have become increasingly resistant to warfarin ever since the 1960s. This is a common theme – humans create a fatal chemical – a pesticide or an antibiotic – and our targets evolve resistance. But this story has a twist. Ying Song from Rice University, Houston, has found that some house mice picked up the gene for warfarin resistance from a different species.

Warfarin works by acting against vitamin K. This vitamin activates a number of genes that create clots in blood, but it itself has to be activated by a protein called VKORC1. Warfarin stops VKORC1 from doing its job, thereby suppressing vitamin K. The clotting process fails, and bleeds continue to bleed.

Rodents can evolve to shrug off warfarin by tweaking their vkorc1 gene, which encodes the protein of the same name. In European house mice, scientists have found at least 10 different genetic changes (mutations) in vkorc1 that change how susceptible they are to warfarin. But only six of these changes were the house mouse's own innovations. The other four came from a close relative – the Algerian mouse, which is found throughout northern Africa, Spain, Portugal, and southern France.

The two species separated from each other between 1.5 and 3 million years ago. They rarely meet, but when they do, they can breed with one another. The two species have identifiably different versions of vkorc1. But Song found that virtually all Spanish house mice carry a copy of vkorc1 that partially or totally matches the Algerian mouse version. Even in Germany, where the two species don't mingle, a third of house mice carried copies of vkorc1 that descended from Algerian peers.

What does the Algerian version of the gene do? Song found out after getting a tip from a pest control officer who she works with. He told her that he was having trouble getting rid of house mice in a German bakery, even after trying a powerful second-generation rodenticide called bromadiolone, or "super-warfarin".

The officer sent over some of these resistant mice and when Song looked at their genes, she found a surprise. Both copies of their vkorc1 genes were perfect matches for the version carried by Algerian mice, but the rest of their genes showed them to be house mice. This tiny out-of-place gene made all the difference – it made the house mice nigh-invulnerable to warfarin and its chemical relatives. Super-warfarin kills around 85% of normal house mice, but it only worked against 9% of the German ones with the Algerian gene.

By the time humans developed warfarin, Algerian mice already had a head-start in resisting it. These rodents live in open, scrubby habitats and they feed mostly on seeds. They don't get a lot of food that's rich in vitamin K, such as leafy green vegetables and Song thinks that their vkorc1 genes have adapted to help them cope with this vitamin deficiency – indeed, it's one of the fastest-evolving genes in its entire genome.

It just so happens that the same adaptations also allow the mice to resist pesticides like warfarin that target vitamin K. It's probably no coincidence that other rodents which specialise on grains – such as the golden hamster and Egyptian spiny mouse – also tend to tolerate warfarin-based chemicals.

The Algerian mice transferred their resistance to house mice by breeding with them, somewhere between 5 and 32 years ago. Hybrids between the two species would normally suffer from physical problems that limit their survival in the wild, and around half of them are sterile.

But these mice were buoyed by their warfarin-resistant copies of vkorc1. At a time when humans were using warfarin and related poisons, these hybrid mice had suddenly gained a valuable defence, one powerful enough to compensate for their other disadvantages. They survived and mated with other house mice, spreading the resistance gene to their own pups.

In this way, the mice are rather reminiscent of bacteria. Individual bacteria can develop genetic tweaks that render them invulnerable to antibiotics, but they can also pick up such mutations from one another. They do so via their equivalent of sex – a process called conjugation where genetic material passes across physical bridges, established across two bacteria. The house mice have done something similar, picking up a warfarin-resistant version of vkorc1 by having sex with Algerian mice.

Humans were probably responsible for these lucky liaisons. The two species used to live in completely different parts of the world. They would never have met, had humans not brought house mice with them as they expanded into Western Europe. Once the two species showed up in the same place, they started mating. Later, humans were again responsible for giving the hybrids an edge over their pure-bred house mouse relatives. Our attempts to kill them merely unveiled a strength that had been hiding for centuries (Discovery, 2011).

**Title:** Bacteria: Resisting Antibiotics Since At Least 30,000 BC
**Date:** August 31, 2011
**Source:** Discovery

**Abstract:** The rise of drug-resistant bacteria is one of the most important threats facing modern medicine. One by one, our arsenal of antibiotics is coming up short against microbes that can pump them out, slip under their notice, deactivate them, or even eat them. But these tricks aren't new. Bacteria have been defeating antibiotics for millennia, long before Alexander Fleming noticed a piece of mould killing off bacteria in a Petri dish. And the best proof of that longstanding struggle has just emerged from the ice-fields of Alaska.

In 30,000-year-old samples of frozen soil, Vanessa D'Costa and Christine King from McMaster University have found a wide variety of antibiotic-resistant genes. They would have allowed ancient bacteria to shrug off many modern drugs such as tetracyclines, beta-lactams and vancomycin.

Vancomycin resistance is especially interesting. This drug has traditionally been used as weapon of last resort, a drug to use when all others have failed. When vancomycin-resistant bacteria first emerged in 1987, it was a surprising blow. Since then, resistant versions of more common bacteria, such as staph (VRSA) have reared their heads.

These superbugs neutralise vancomycin using a trio of genes known collectively as vanHAX. Together, they alter the protein that's attacked by the drug, rendering it useless. D'Costa and King found that their ancient sequences include the entire vanHAX cluster. They even resurrected these ancient genes, created proteins from them, and showed that they have the same shape, and do the same thing, as their modern counterparts.

D'Costa and King write that their results disprove the idea that antibiotic resistance is a modern phenomenon. Instead, it's been part of bacterial life long before the modern use of antibiotics. But I'm really not sure how many people would still hold to that view. First, many antibiotics come from natural sources. Penicillin, the first to be synthesised, famously comes from Fleming's surreptitious mould. These natural antibiotics evolved to keep bacteria at bay between 40 million and 2 billion years ago, so it's extremely likely that bacteria have been resisting them for just as long.

Second, we know that the environment is teeming with resistance genes. In her own earlier study, D'Costa found that soil bacteria are a massive reservoir for resistance genes – a "resistome " – which infectious bacteria could draw upon. Meanwhile, Gautam Dantas found that our soils are so full of resistant bacteria that random sampling produced strains that not only resist antibiotics, but actually eat them. He also found that the bacteria in our guts are another reservoir of resistance.

Regardless, D'Costa and King's point stands: they have certainly found the oldest known examples of resistance genes. There have been similar claims in the past, but all of them controversial. Bacteria are so omnipresent that any team claiming to have found ancient samples must bend over backwards to prove that these aren't modern contaminants. And none of the previous groups did this well enough, which means that their claims have not been replicated.

To show that their samples are authentically ancient, D'Costa and King pulled out all the stops. They did all of their lab work in special clean rooms. They showed that their samples included DNA from other

animals that lived at the right time, such as mammoths, but nothing from species that are common today, like elk, moose or spruce. They even sprayed their drilling equipment, and the surface of their unearthed ice cores, with glow-in-the-dark bacteria. This way, they could immediately tell if anything from the outside world had leached into the interior parts of the cores – the parts where they drew their samples from. Nothing had.

So what does this mean for the problem of antibiotic resistance today? Is this an old problem that is being blown out of proportion? Can we let the wanton use of antibiotics in modern healthcare and agriculture off the hook? Hardly. These conditions still create intense evolutionary pressures that favour the rise of resistant bacteria. The fact that resistant genes are widespread and ancient does not change that. It simply means that in times of need, beleaguered bacteria have a vast and longstanding range of defences to draw from. For every new sword that we fashion, there is a millennia-old shield lying around, just waiting to be brandished again (Disocvery, 2011).

**Title:** FAO Warnings Follow Rise In Replikins Count For Both H5N1 And Swine Flu (H1N1)
**Date:** August 31, 2011
**Source:** Replikins

**Abstract:** The possible combination of influenza strains H1N1 (high infectivity) and H5N1 (high lethality) is a matter of global concern (1, 2). Bioradar UK Ltd announced today (3) first, that the Replikin Counts of the two virus strains have risen simultaneously, not seen previously. Additionally, the rise is to their highest levels in 50 years (H1N1, 16.7; H5N1, 23.3), and that clinical outbreaks of each strain are now occurring. These simultaneous conditions may increase the risk that the two virus strains might come into contact with each other more frequently, facilitating transfer of genomic material to form a hybrid (Replikins, 2011).

**Title:** Five Easy Mutations To Make Bird Flu A Lethal Pandemic
**Date:** September 16, 2011
**Soure:** New Scientist

**Abstract:** H5N1 bird flu can kill humans, but has not gone pandemic because it cannot spread easily among us. That might change: five mutations in just two genes have allowed the virus to spread between mammals in the lab. What's more, the virus is just as lethal despite the mutations.

"The virus is transmitted as efficiently as seasonal flu," says Ron Fouchier of the Erasmus Medical Centre in Rotterdam, the Netherlands, who reported the work at a scientific meeting on flu last week in Malta.

"This shows clearly that H5 can change in a way that allows transmission and still cause severe disease in humans. It's scary," says Peter Doherty, a 1996 Nobel prizewinner for work in viral immunology.

H5N1 evolved in poultry in east Asia and has spread across Eurasia since 2004. In that time 565 people are known to have caught it; 331 died. No strain that spreads readily among mammals has emerged in that time, despite millions of infected birds, and infections in people, cats and pigs. Efforts to create such a virus in the lab have failed, and some virologists think H5N1 simply cannot do it.

The work by Fouchier's team suggests otherwise. They first gave H5N1 three mutations known to adapt bird flu to mammals. This version of the virus killed ferrets, which react to flu viruses in a similar way to humans. The virus did not transmit between them, though.

Then the researchers gave the virus from the sick ferrets to more ferrets - a standard technique for making pathogens adapt to an animal. They repeated this 10 times, using stringent containment. The tenth round of ferrets shed an H5N1 strain that spread to ferrets in separate cages - and killed them.

The process yielded viruses with many new mutations, but two were in all of them. Those plus the three added deliberately "suggest that as few as five are required to make the virus airborne", says Fouchier. He will now test H5N1 made with only those five.

All the mutations have been seen separately in H5N1 from birds. "If they occur separately, they can occur together," says Fouchier. Malik Peiris of the University of Hong Kong, a flu virologist, says this means H5N1 transmissible between humans can evolve in birds, where it is circulating already, without needing to spend time in mammals such as pigs.

Peter Palese, a flu specialist at Mount Sinai Medical Center in New York City who has expressed doubts that H5N1 can adapt to mammals, is not convinced.

"Ferrets are not humans," he says. "H5N1 has been around for a long time" and failed to mutate into a form that can jump between people.

"That it has not adapted doesn't mean it cannot," replies Jeffery Taubenberger of the US National Institutes of Health in Bethesda, Maryland, who studies how a bird flu became the deadly pandemic of 1918 (New Scientist, 2011).

**Title:** Making Viruses The Natural Way
**Date:** December 2, 2011
**Source:** Discovery

**Abstract:** When it comes to viruses, we humans like to pretend we know much more than we really do. It's understandable. The influenza virus, for example, has only ten genes. It is just a shell that delivers genes and proteins into a host cell, where it hacks the biochemistry to manufacture more viruses. It seems like such an easy biological problem to solve.

Yet the flu and other viruses hide a complexity which virologists have only partly uncovered. The idea that someone could intentionally design a super-lethal virus from scatch–as plausible as it may seem–is, for now, a delusion.

If you've been following the news this past week, you may think I've just been proven wrong. Reports have surfaced about two teams of scientists producing flu viruses that could potentially kill millions if they escaped from the labs. The scientists have the viruses locked up tight for now, and government officials are debating whether they can publish their results. (New Scientist and Science have excellent reports.)

So is this evidence that scientists have become viral Frankensteins, who can engineer pathogens at will? Hardly.

The new research is part of a long-running struggle to understand how new flu strains arise. It's clear that all flu viruses that infect humans ultimately evolved from viruses that infect birds. From time to time, people can pick up these viruses, which infect their airway. Depending on the strain, bird flu may be harmless or lethal to humans. But for the most part, it can't get from one human to another. It's too well adapted for life in birds.

On rare occasion, a bird flu does manage to adapt to humans. It may experience natural selection, it may pick up some genes from human flu viruses, or both. Scientists are still trying to figure out what it takes for a flu virus to make this transition. It's an important question, not just as a matter of fundamental biology but as a matter of global health. When new bird flus jump to humans, we lack immune defenses against them, and they can thus cause worldwide pandemics.

Flu experts have had their eye on one strain of bird flu in particular for a while now: H5N1. It's proven extraordinarily lethal, and yet, since it first came to light in 1997, it hasn't managed to make the big leap and start spreading from person to person. If you get H5N1, you're in big trouble. But not many people get it. Yet.

Does this mean that H5N1 just doesn't have what it takes to become the next great pandemic? Or does it mean the virus simply hasn't evolved the right recipe yet?

Scientists have tried to answer this question by tinkering with the virus. Instead of trying to make a virus that spreads among people, they infected ferrets, which turn out to have much the same experience with the flu as we humans do. In April, CDC scientists published the latest of these studies. They focused their attention on a protein called hemagglutinin, which flu viruses use to get into host cells. Based on earlier experiments, the CDC scientists reasoned that the right tweak to the structure of hemagglutinin in H5N1 could switch it from binding strongly to bird cells to mammal cells.

But their rational tweaks failed. They concluded that there was a lot more to becoming a human flu that we don't yet understand.

The studies that have now hit the news have succeeded where other experiments have failed. The difference is that instead of trying rational tweaks, the scientists sat back and let evolution do the tweaking.

According to the news reports, the scientists used a tried-and-true method known as serial passage. You infect an animal. It gets sick. You wait for the virus to replicate inside its animal host–as new mutants arise and natural selection favors some mutants over others–and then take some viruses from the sick animal and infect a healthy one. You repeat this, moving the virus from host to host.

Interesting things can happen when you let viruses evolve under these conditions. Natural selection can produce viruses with many new mutations, which together let them reproduce faster in the lab than their ancestors. And those viruses, in some cases, can be a lot more dangerous than their ancestors.

Back in 2007, for example, a virologist named Kanta Subbarao and her colleagues transformed the SARS virus this way. SARS evolved from a bat virus, crossing over into humans in 2003. It killed over 900 people before it mysteriously disappeared. Subbarao wanted to find a way to study SARS in lab animals, such as mice. Mice normally don't get sick from human SARS viruses, though, even though the virus can replicate at a low rate inside them. Even when mice are genetically engineered so that they can't develop an immune system, SARS can't harm them.

So Subbarao and her colleagues that instead of changing the mice, they'd change the virus. They inoculated mice with the SARS virus, gave it a chance to replicate inside them, and then isolated the new viruses to infect new mice.

Over the course of just 15 passages, it changed from a harmless virus into a fatal one. One sniff of SARS was now enough to kill a mouse.

As Martin Enserink reports in *Science*, the new experiments on bird flu were similarly effective. They turned H5N1 into a ferret flu in just 10 generations. By the time the scientists were done, they no longer had to ferry the flu from one ferret to the next. A healthy ferret just had to be placed near a sick one; the virus could travel through the air. When they examined the new strain, they discovered five mutations in two genes. All five mutations have been found in natural H5N1 viruses–just not all in one virus.

A mammal-ready flu virus was beyond human reason, in other words, but it was fairly easy for evolution to find, given the right condtions. That suggests that H5N1 may not have far to evolve to make us its host.

Of course, a serial passage experiment is not identical to the flu's natural world, where it circulates among millions of birds and sometimes encounters people. But it's disturbingly close.

And if it's so easy for mutations to turn H5N1 into a human flu, the experimental viruses have a lot to tell us about what we may be facing in the future. There's no point in condemning the scientists for tampering with nature. They were watching nature do what it does disturbingly well (Discovery, 2011).

**Title:** The Polio Genome
**Date:** 2012
**Source:** NMAH

**Abstract:**

It's now possible to go from data printed on a piece of paper or stored in a compute and, without the organism itself, re-construct a life form.

John LaMontagne, National Institute of Allergey and Infectious Diseases, 2002

A genome is the genetic material of an organism. In 1981, two different research groups, Vincent Racaniello and David Baltimore at Massachusetts Institute of Technology and Eckard Wimmer's team at State University of New York, Stony Brook, published the poliovirus genome. They used an enzyme to switch the single strands of viral ribonucleic acid—RNA—to double strands of deoxyribonucleic acid—DNA—and then determined the sequence of adenine, thymine, guanine, and cytosine encoding the five molecules that are the substance of the virus's existence.

Poliovirus lacks the ability to correct its mutations, so its genome evolves at one to two nucleotide substitutions per week. It is always changing.

In 2002, investigators at the State University of New York in Stony Brook used the published genetic sequence to synthesize a DNA version of poliovirus. Then they used an enzyme to convert the DNA to RNA and grew the virus in a cell-free extract. Animal tests showed that the synthesized poliovirus caused paralysis.

"I did not use any machine for sequencing the poliovirus genome. It was all done by hand—my hands! I used what was known as the 'Maxam-Gilbert' method, in which four different chemical reactions are carried out on the DNA. The products are then fractionated on thin polyacrylamide gels, which were poured manually, run, and then carefully removed from the plates, dried, and exposed to X-ray film. The sequencing 'ladders' were then read by myself on a light box and entered manually into a computer. But we didn't have individual computers back then, so I used a terminal hooked up to an MIT central computer."
—*Vincent Racaniello, 1981* (NMAH, 2012).

**Title:** Bird Flu Mutation Study Stopped In Fear Of Deadly Global Outbreak
**Date:** January 21, 2012
**Source:** Russia Today

**Abstract:** Under pressure to put their research on hold due to fear of a biological disaster, an international team of scientists have voluntarily suspended their study on an advanced, incredibly deadly mutation of the H5N1 bird flu.

In an effort to better understand the deadly bird flu virus, Ron Fouchier of Erasmus Medical College in the Netherlands, Adolfo Garcia-Sastre of Mount Sinai School of Medicine in New York and Yoshihiro Kawaoka of the University of Wisconsin, Madison have been slaving over their study of the avian influenza. In conducting their own research, the team of scientists was able to mutate the original H5N1 virus into a much more lethal form to see how the outbreak could increase in intensity if not controlled outside of the lab. As word came around late last year that their research had returned a variation able to

induce an international outbreak, however, the scientific community urged them to abandon their study in fear that the mutated strain would escape the lab and cause a deadly, worldwide outbreak.

With the fear failing to subside weeks later, the team of scientists has temporarily halted their research.

In its natural form, the bird flu virus has led to nearly 600 known cases and 340 deaths since it was discovered in 2003. That year there were only four outbreaks, all in East Asia, although in the years since an outbreak has claimed lives as far west as Egypt. The scientists were studying what damage a mutated strain of the virus could bring, but the US National Science Advisory Board for Biosecurity cautioned them to refrain from publishing the results of their finding, fearful that it would influence budding bioterrorists to use the study to create their own strain and launch an epidemic.

Despite the Board's urging, others in the science community were skeptical. *"In the end, is the likelihood of misuse outweighed by the danger of beginning a Big Brother society?"* Professor Wendy Barclay of Imperial College London asked the Daily Mail last month.

The researchers say in a letter published in the journals Nature and Science on Friday that they will take a two-month break from their efforts. Since news of their study caught wind, the US government, the World Health Organization and other international bodies have been evaluating a way to go about publishing the findings in periodicals eventually, taking into account their research but avoiding the publishing of a how-go guide for biological warfare.

*"We realize that organizations and governments around the world need time to find the best solutions for opportunities and challenges that stem from the work,"* the scientists write.

*"We hope that by having a calm and reasoned discussion of the facts, scientists and biosecurity experts can reach a better understanding and find ways to enable the research to go forward while minimizing risks,"* adds Kawaoka (Russia Today, 2012).

**Title:** Big Pharma Creates Resistant "White Plague" Through Mass Drugging
**Date:** March 21, 2012
**Source:** Natural Society

**Abstract:** Thanks to widespread and unnecessary usage of antibiotics throughout the modern world, a heavily drug-resistant form of tuberculosis is now striking fear into the hearts of scientists and doctors alike. Affecting both poor and rich, those affected with the disease are put into quarantine and injected with a large number of super drugs. If the disease were to spread and develop, tuberculosis experts are worried that medical professionals would be helpless to stop it — at least when it comes to more of big pharma's drugs. Natural solutions do exist, and they don't involve the very drugs that *spawned* the 'white plague' in the first place.

India is receiving the bulk of the blame for spurring on the drug-resistant killer, as the country is known for its massive overuse of antibiotics. In fact, India has the most cases of multi-drug resistant tuberculosis in the world, with more than 100,000 cases of the disease. While multi-drug resistant tuberculosis is still quite deadly, it is the 'extensively drug-resistant' and '*totally* drug-resistant' tuberculosis that worries many health organizations and officials.

**'Totally a Man Made Disease'**
Make no mistake that this is not a 'natural' evolution of disease, but a result of excessive drug use made possible by big pharma and mainstream health officials. Even members of the World Health Organization's 'Stop TB Partnership' are outraged over the man-made disease progression, with member Lucica Ditiu stating that the drug-resistant TB "is a totally man-made disease". Dr. Zarir Udwadia, also a TB specialist from India, had similar statements, explaining that that resistant strains were "an accident waiting to happen."

Dr. Udwadia published a report in the journal Clinical Infectious Diseases last year documenting four cases of totally drug-resistant tuberculosis. Currently, he has about twelve cases of the resistant disease with no treatment options left, and three have already died. Each medicine the doctor used to combat the mutated bacteria failed, with the bacteria immune to 12 drugs total. Dr. Udwadia explains that to even get to the point of developing such a drug resistant strain, it requires severe misuse of antibiotic drugs:

"To get to this stage, you have to have amplified resistance over years, with loads of misuse of (antibiotic) drugs. And no other country throws around second-line drugs as freely as India has been doing."

**Real Solutions**

It is clear that the resistant strain is a real threat to public health, with many experts concerned about a potential pandemic. Unfortunately these very same individuals who blow the whistle over the new resistant 'white plague' being a man-made disease are turning to even more pharmaceuticals to 'treat' the condition. This is a serious web of drug use, with drugs creating problems that require even *more* drug usage. There's simply no room for a cure within this drug paradigm, because even if they make a drug powerful enough to wipe out the resistant tuberculosis bacteria, it comes with an onslaught of symptoms that 'require' more drugs.

In one case of treatment, for example, Anna Watterson was given so many drug injections in an attempt to treat the resistant disease that she was heavily bruised, constantly nauseous, and *unable to go out into the sun*.

Instead of subjecting yourself to this 'drug web', you can utilize natural solutions that will also serve to enhance other biological aspects of your life as well. Vitamin D3, for example, can not only boost your overall immunity and resistance to tuberculosis, but it can also help fight the disease once you've been infected. Scientists have even found that vitamin D intake can significantly reduce tuberculosis associated mortality on a global scale. But what if you're infected with the totally resistant mega bacteria?

Garlic has been found to outpace drugs in the treatment of resistant tuberculosis, putting pharmaceuticals to shame and of course boosting your overall health in the process. This has been proven by more than one piece of peer-viewed research, with scientists finding garlic to be one of many natural solutions that should be considered by all medical professionals. Amazingly, there are 43 other natural substances documented as powerful solutions to tuberculosis, virtually all of which most doctors ignore. In the abstract of the study from the University of Health Sciences in Pakistan, scientists state:

"Alternate medicine practices with plant extracts including garlic should be considered to decrease the burden of drug resistance and cost in the management of diseases. " Big pharma's drugs spawned this new plague, so why take them to fight it? Empower your health naturally through nutrient-dense foods, supplements, and pure water. In particular, stock up on vitamin D and turmeric — they will be highly beneficial in the event of a pandemic or disease outbreak (Natural Society, 2012).