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CHLAMYDIA, NOT CHOLESTEROL IS THE CAUSE OF A CORONARY?

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It's beginning to look that way. Some authorities believe that chlamydia is much more of a culprit than cholesterol with regard to causing obstructive atherosclerotic plaque. Many people reading this may have never heard of chlamydia. Those that have, are apt to consider it to be a rather uncommon and harmless pathogen. Yet, the vast majority of us will have been infected by this microorganism at some time in our lives. And, although such incidents are usually mild and short lived, they may have unsuspected and serious long term consequences.

Chlamydia trachomatis infection is the most prevalent sexually transmitted disease in the United States. Over 4 million new cases occur annually in the U.S, but patients usually have very few symptoms. It is more difficult to diagnose in women, where untreated infections can lead to inflammation of the fallopian tubes, ectopic pregnancies, and 50,000 new cases of infertility a year.

Chlamydia trachomatis is hardly a new bug. Its name is derived from the fact that the same organism also causes a contagious conjunctivitis of the eye associated with granuloma formation on the inner lid known as trachoma. Trachoma was recognized thousands of years ago in China, was described in the Ebers Papyrus from ancient Egypt, and is still a major health problem. There are an estimated 500 million cases world wide, including seven million with blindness resulting from scarring. Trachoma is far and away the leading cause of preventable blindness. Indeed, all of the gynecological and ophthalmologic complications resulting from *C. trachomatis* infection could probably be prevented, since antibiotics are extremely effective.

Two other strains, *Chlamydia psittaci* and *Chlamydia pneumoniae* have been identified in recent years. *C. psittaci* infection is acquired through droplet infection from infected birds. It is seen most commonly in employees of pet shops and poultry processing plants, where it can be an occupational hazard. Most birds are usually healthy carriers, but become susceptible to the disease under stressful conditions such as shipping, crowding, starvation, and even egg laying. Person to person transmission is uncommon, but has been seen in outbreaks. Several decades ago, when infected birds from Argentina were shipped to various parts of the world, there were scattered outbreaks of infection associated with death rates up to 40 percent. (Continued on page 2)

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While pet birds remain the greatest hazard, there are also high rates of infection in household cats. Both birds, cats, and possibly other pets may appear healthy, but they can transmit the bacteria to humans, where it can cause a variety of problems that may be difficult to diagnose. In addition to upper respiratory infections, conjunctivitis, glomerulonephritis, and endocarditis due to *C. psittaci* from infected cats and pigeons have also been reported.

But it is *Chlamydia pneumoniae* that has taken center stage. Discovered little more than ten years ago, it has now been found to be a surprisingly common cause of acute respiratory tract infections. Some estimates suggest it may be responsible for up to 10 percent of all the infections we commonly refer to as "flu". Unlike other strains of chlamydia, it spreads readily from person to person by droplet infection, and outbreaks have been reported in schools, nursing homes, and military barracks, with a incubation periods varying from 7-21 days.

Upper respiratory infections can range from having few symptoms, to an influenza like illness with fever, chills, headache, muscle and joint pain, and swelling. The highest incidence of infection is in school children and teenagers. Serologic tests suggest that 40-70 percent of adults have been exposed, but re infection is common, since chlamydia antibodies don't provide much protection.

Bugs Cause Much More Than Infections

It is also believed that *C. pneumoniae* may be responsible for the increasing incidence of asthma that has been observed in recent years. Other microorganisms have been implicated in exacerbating asthma, but studies suggest that acute bronchitis due to *C. pneumoniae* infection may be the culprit in many acute attacks in adults as well as children. Chlamydia IGE antibodies are increased in children with asthma and obstructive airway disease, suggesting not only infection, but also an allergic reaction to the bacteria that can cause further bronchospasm.

The first chronic disease proven to have an unsuspected bacterial cause was peptic ulcer. Although evidence that *Helicobacter pylori* infection might play a role in peptic disease was first proposed two decades ago, it was only officially recognized by the NIH in 1994, when antibiotic therapy was recommended as the treatment of choice for ulcers. *Helicobacter* is now thought to be involved in other gastrointestinal disorders, including esophagitis, certain large and small bowel diseases, and more recently, cancer of the stomach.

Salmonella, shigella, versinia, as well as campylobacter bacteria in foods can not only cause acute gastroenteritis and food poisoning symptoms that last a day or two, but a painful inflammatory arthritis that can persist for months and years. These bacteria and other organisms have also been implicated in Reiter's syndrome, a disorder characterized by inflammation of the eyes, urinary tract and joints. *Ureaplasma urealyticum*, *mycoplasma fermentum*, and *C. trachomatis*, which are sexually transmitted bacteria that produce few symptoms, can also produce this kind of reactive arthritis. And so can *mycoplasma pneumoniae*, which, as its name suggests, causes upper respiratory complaints.

Arthritis can occur in tuberculosis, syphilis, brucellosis, and Lyme disease, or result from gonococcal, streptococcal, staphylococcal, fungal, and viral infections. However these forms of arthritis are quite different, since in such situations, microorganisms attack the joints directly, and can be seen under the microscope or cultured. That is not the case in reactive arthritis, which is more like an allergic response. In some instances, both processes may be involved.

Chlamydia And Atherosclerosis

Other tissues may be affected in a similar fashion. There is growing evidence that chlamydia and other microorganisms may be linked to heart attacks and hypertension because they promote the development of atherosclerotic plaque. How this theory evolved is a fascinating tale of medical detective work and serendipity that could easily be made into a movie.

The story begins about 30 years ago in Taiwan, Thomas Grayston of the University of Washington was testing a vaccine against *C. trachomatis*, the sexually transmitted form of chlamydia that can lead to blindness. He had cultured the eye of a child with failing vision, and grew out an organism that did not resemble any of the known forms of chlamydia. He stored it in the freezer and thought little more about it until 1985, when it showed up again. However, this time it was in blood samples taken from patients in Finland who had been suffering from the "flu". Further studies showed that it could cause a mild pneumonia that was transmitted by droplet infection from coughing and sneezing, so he named it *Chlamydia pneumoniae*. He and his Finnish colleagues began testing other flu victims to see how many of them harbored the organism.

The next chapter in this story takes place in a government laboratory, where technicians found that most of the blood samples in a recent shipment from Finland were testing positive for this specific bacterium. They were therefore concerned that a flu epidemic might be on the horizon, and notified their superiors so that appropriate steps could be taken. To everyone's surprise, it was found that the samples were obtained not from flu victims, but heart attack patients at Helsinki University's Central Hospital. Researchers there had been investigating whether such patients might be more susceptible to various infections, and this procedure had been included as part of the screening process.

To make sure there were no mistakes, the tests were repeated and compared with samples from healthy controls. Of 70 men with clogged arteries, 42 (60%) had antibodies to *C. pneumoniae*, indicating a current or recent infection. Among 41 healthy men of similar age and habits, only 7 (16%) had evidence of these antibodies, a very significant and puzzling difference.

These results were not only difficult to explain, but they also did not jibe with the existing dogma claiming that cholesterol was the cause of coronary heart disease. Grayston was skeptical, and to prove that this was some kind of fluke, began a larger study of heart attack patients and controls in Seattle. As expected, both people with and without clogged arteries had antibodies to *C. pneumoniae*. However, antibody levels were 25 percent higher in those with coronary artery disease. What was equally impressive, was that those with the greatest elevations were twice as likely as those with the least to have significant atherosclerosis. This was strong support for a cause-effect relationship.

There is now additional evidence that this organism can travel from the lungs to the heart and elsewhere, and cause arteries to be clogged with atheromatous plaque. Chlamydia researchers at the Centers For Disease Control and Prevention have shown that the organism can enter the blood stream from the lungs, and it has been identified by DNA testing in obstructive plaque removed from heart attack patients during surgery. Last year, *C. pneumoniae* was actually cultured from plaque in a patient who had required a heart transplant because of severe atherosclerotic disease.

Grayston theorizes that when the organism is inhaled, the immune system marshals its defenses to ward off the intruder. As part of this reaction, macrophages that are supposed to destroy, or literally "eat up" the invaders are sent to the lungs. However, laboratory studies have shown that *C. pneumoniae* is able to survive despite being swallowed up, much like Jonah in the whale. The macrophages carry the bacteria to other parts of the body, and particularly to trouble spots where there is evidence of inflammation or arterial injury due to atherosclerotic deposits. The organisms can then invade arterial walls and continue to reproduce, intensifying the inflammatory response. As a result, other macrophages are attracted to the area, many of which may bring more bacteria that further aggravate the problem. As this vicious cycle is repeated over and over, the arterial lesion keeps expanding and bulging so that the flow of blood is progressively restricted. Eventually, it can slow down to a trickle, thus favoring the development of clot formation that causes complete occlusion.

Are We Being Bugged To Death?

Upper respiratory infections are very common, but generally pose little more than a temporary inconvenience. They often run their course over a few days or a week without any treatment, or respond to antibiotics. There are exceptions like tuberculosis, which can cause long term problems that also affect organs other than the lungs. However, most of the time, recovery from the flu or some upper respiratory infection seems to be fairly complete and uneventful, with few complications or long term effects. This view has now been challenged because of studies showing that arthritis, arteriosclerosis, and possibly cancer may also result from bacterial infections. This is most apt to occur when infections are not recognized or diagnosed and treated correctly. This can readily happen with chlamydia, which often produces minimal symptoms, and only for a day or so.

It is likely that bacteria other than chlamydia may also contribute to heart attacks. Elevated antibodies to *H. pylori* have been reported in at least 20 studies. Individuals with chronic periodontal disease also appear to be at significantly increased risk. A Finnish report showed that even after all conventional risk factors had been accounted for, a significant relationship between dental infections and cardiovascular disease persisted. Research here also confirms that individuals with few or no teeth tend to have much higher rates of heart attacks and strokes. In one study, patients with gum infections due to *Bacterioides gingivalis* had twice as much cardiovascular disease as age matched controls, and one investigator includes a slide warning "Floss Or Die" in his presentations. The National Institutes of Health is apparently impressed, since they are funding a \$1.3 million grant for studying the relationship between gum disease and heart attacks. Patients with chronic sinusitis and bronchitis that are due to other organisms also have a higher incidence of heart attack rates.

Cytomegalovirus (CMV) is a seemingly harmless virus that infects most people if they live long enough. More than two out of three people 60 years old have significant antibody titers. Although it produces few or no clinical symptoms, there is also good evidence that it can cause accelerated atherosclerosis and heart attacks.

Like other members of the herpes virus family, CMV can hide and remain dormant in cells for long periods of time after an infection, producing no signs or symptoms until stress lowers immune defenses. In AIDS patients who have severe deficiencies in immune system function, it has been implicated in variety of illness syndromes, including blindness. In normal individuals, it has been generally viewed as a harmless passenger, but it now seems more likely that it can still provoke a low grade and persistent inflammatory response that stimulates plaque formation.

Patients who have angioplasty or bypass procedures are over five times more likely to have their arteries clog up again if they harbor CMV. In one NIH study of 75 patients undergoing surgery for obstructive coronary atheromatous plaque removal, 49 (75%) had antibodies to the virus. When these patients were examined only six months later, 21 (43%) had evidence of clogging of their arteries due to re growth of the plaque. Of the 26 without antibodies, only 2 (8%) had evidence of significant recurrence. And transplant patients with cytomegalovirus are much more likely to reject their hearts or die within a few years after surgery.

Other herpes viruses may also produce atherosclerosis based on experimental studies conducted by Michael DeBaakey's group at Baylor. One of the sites where they often lie latent is in the smooth muscle lining inner arterial walls. When the virus becomes activated, it manufactures a protein that interferes with the normal mechanisms that keep cell growth in check. As a result, smooth muscle cells proliferate, eventually causing a tumor-like bulge that slows down blood flow. Activation of the virus can result from any arterial injury, such as surgery to remove obstructions, hypertension, cholesterol deposits, or possibly even cigarette smoke. Efforts are now under way to see whether vaccinating experimental animals against CMV will prevent restenosis.

CMV cannot be treated, but chlamydia and bacterial infections can. One study showed that heart attack victims treated with azithromycin for 36 days were much less likely to suffer recurrent problems. In another, roxithromycin significantly reduced angina attacks in unstable patients, and other antibiotics are also effective.

To Treat Or Not To Treat? - Catch 22?

In view of these findings, should all upper respiratory infections be immediately treated aggressively with antibiotics? Most authorities believe that this would do more harm in the long run for several reasons. While there is little doubt that antibiotics can be lifesaving in numerous situations, their indiscriminate use has led to the emergence of a growing number of highly dangerous bacteria in the United States. There are now flesh eating staphylococci that few drugs can touch. Over the past decade, there have been epidemics of a strain of streptococcal pneumonia resistant not only to penicillin, but also all of the newer and more powerful derivatives. It now threatens to be the leading cause of ear and sinus infections, meningitis, and other potentially serious illnesses.

A recent article in the *Journal of the American Medical Association* pointed out that doctors routinely write antibiotic prescriptions for colds, bronchitis, and minor respiratory infections, despite the fact that 90 percent are caused by a virus, and antibiotics are useless. Although most physicians are well aware of this, the authors suggest that they tend to prescribe these drugs because, even though they are unlikely to help, this is what patients expect and want. Some doctors also justify giving antibiotics by claiming they are trying to prevent complications from viral attacks that could lead to bacterial infections. Another problem is that some patients stop taking their medication once they feel better, but before the infection has been eradicated, which leaves only the strongest bugs to survive and spread.

Rural doctors are more likely to prescribe antibiotics because patients travel greater distances to see them. They are apt to have very busy practices because physicians are in short supply, and they don't have the time to explain why antibiotics are really not necessary, and that patients should allow their natural defense mechanisms to get rid of the infection. In an accompanying editorial, the Director of the National Centers for Disease Control and Prevention said that "immediate and aggressive response" is needed to combat the inappropriate prescribing of antibiotics. He feels that all patients who are being prescribed an antibiotic, should consider asking their physician this question, "Do you really think I need this?"

Studies show that individuals from low socioeconomic groups are more resistant to infection and have better immune system function than controls who are more affluent. One explanation for this apparent paradox may be that poorer people don't have as much access to medical care, and when they do, can't afford to pay for office visits or expensive medications. They are less apt to be treated with antibiotics whenever they have a slight fever, productive cough, or other upper respiratory complaints, and infections subside because the body's immune system is stimulated to ward off the invader. As a result, they develop greater resistance to subsequent infections.

Over anxious parents with insurance plans that subsidize office visits and prescription drugs are much more likely to request or even demand antibiotics for their children at the slightest sign of a possible infection. There is not as much stimulation of immune system mechanisms responsible for warding off future attacks in these kids. It's a Catch 22 situation, since although giving antibiotics could increase susceptibility to subsequent infections, it might also significantly reduce the risk for developing a heart attack or stroke due to atherosclerotic plaque in later years.

The incidence of hypertension might also be lowered. In one study, 123 hospitalized patients with severe hypertension were twice as likely to have been previously infected with *C. pneumoniae* than controls. Investigators found that 35 percent of the very high blood pressure group had antibody titers consistent with previous infection. In contrast, this was found in only 17.9 percent of normotensive controls matched for ethnic origin, age, sex, and smoking habits, who had been admitted for various disorders not involving the heart or lungs.

Infection may lead to hypertension by promoting the growth of scar tissue in blood vessels. This could eventually reduce blood flow and increase peripheral resistance, thus leading to persistent hypertension. Excessive scar tissue formation in the eye and fallopian tubes is a characteristic of *C. trachomatis* infection, so it is not surprising that the same response might occur in blood vessels. *C. pneumoniae* has been identified in arterial walls by sophisticated staining techniques, and was recently cultured from carotid artery plaque.

The "Dirty Chicken" Theory And CRP

It has been observed that children reared in poor living conditions tend to become shorter adults, even though their nutritional habits and health histories are no different than those from families with a higher socioeconomic status. This is difficult to explain, although it is likely that their lives may be more stressful, and increased childhood stress has been linked to short stature. On the other hand, as noted previously, kids from families who are better off are much more likely to have received repeated courses of antibiotic therapy while growing up. While inappropriate for the colds for which they were prescribed, they might well have been effective in reducing asymptomatic chronic dental and upper respiratory infections that might cause atherosclerosis years later.

Chickens subjected to stressful, overcrowded, unhygienic conditions also exhibit poor growth despite an adequate diet and nutritional supplements. However, this can be reversed by administering antibiotics. The "dirty chicken" theory proposes that chronic inflammation due to stress or sub clinical infection produces chemicals called cytokines that inhibit growth. Antibiotics reduce the inflammatory process so that normal growth mechanisms can operate. Although chlamydia, helicobacter, and cytomegalovirus infections frequently produce few signs and symptoms of infection, critics of the "dirty chicken" hypothesis have argued that if there were some ongoing inflammatory process that was continually contributing to atherosclerotic plaque, it should be possible to demonstrate this by sensitive laboratory tests. They should be elevated in patients with heart attacks, strokes and other diseases due to advanced atherosclerosis compared to controls. And that is exactly what researchers have now found.

CRP (C reactive protein) has nothing to do with CPR (cardiopulmonary resuscitation), but it has revived the infection/inflammation theory of heart attacks. CRP is not an antibody, but rather a protein compound produced in the liver in response to inflammation and tissue injury that occurs anywhere in the body. It is part of the immune system's response to infection that stimulates the ability of white cells called macrophages to attack, engulf, and inactivate microbial invaders.

Blood levels are also temporarily elevated following a broken bone, severe burn, or any trauma that causes tissue destruction or damage. CRP levels rise during acute heart attacks due to tissue damage, and also provide prognostic significance. Measurements were obtained in 110 patients admitted to a coronary care unit for suspicious chest pain, but without any increase in cardiac enzymes or ECG findings to confirm a heart attack. They were subsequently divided into two groups based on a final diagnosis of either an acute myocardial infarction, or unstable angina. CRP was elevated in 60 percent of the acute myocardial infarction group, compared to only 5 percent of those with unstable angina. Another study measured CRP concentrations in 188 consecutive patients admitted for an acute myocardial infarction. Those with the highest serum CRP values also had the highest mortality rates in the six months following their attack, as might be expected, since this reflected greater inflammation and tissue destruction. Low and high CRP measurements have been found to be helpful in predicting both favorable and unfavorable outcomes in patients with chronic but stable angina.

In the absence of acute injury, increased levels most likely reflect the presence of some chronic inflammatory process. In one study of men at high risk for heart attacks, there were 98 myocardial infarctions and 148 deaths due to coronary heart disease on long term follow-up. Both of these indicators of increased atherosclerosis were significantly correlated with high CRP levels that had been obtained on entry many years before when they were healthy, compared to measurements in 491 controls with no evidence of coronary events.

The best explanation for high CRP would be a chronic inflammatory process due to low grade infection, like chronic bronchitis. This is supported by a study of 20,000 randomly selected men and women, in whom there were 2000 fatal and non fatal coronary events over 13 years. Medical records showed a very clear association between chronic bronchitis and coronary disease, even when all other major risk factors were taken into consideration. These and other reports are consistent with the premise that chronic infection and inflammation contribute to coronary atherosclerosis, and that CRP levels can help predict this.

Risk Factors, Heart Attacks, And Stress

Since elevated C reactive protein is associated with an increase in heart attacks, it is considered a "risk factor", along with premature baldness, prominent ear lobe creases, and some 300 or so other items. The vast majority of these do not cause atherosclerosis, but are merely associated with it. However, interleukin-6 and tumor necrosis factor, which are cytokine components of CRP, can promote the production of inflammatory white cells and macrophages seen in atherosclerotic plaque and the proliferation of smooth muscle cells in the walls of arteries that further restrict blood flow. They also increase levels of fibrinogen, a protein that makes blood platelets clump together to form a clot. People with fibrinogen levels in the top third of the population are twice as likely to develop clogged arteries due to atherosclerosis. These three activities account for almost all the characteristics of atherosclerotic plaque, as well as its obstructive complications. The infection/inflammation hypothesis is supported by the finding of antibodies that block these cytokines in patients with well established coronary atherosclerosis. Learning more about the structure and actions of these antibodies might very well lead to the development of new drugs to retard atherosclerosis.

They may also help to explain the link between certain negative as well as positive risk factors for heart attacks. Having a few drinks of alcohol may help prevent coronaries, because it suppresses production of tumor necrosis factor, and the protective effect of unsaturated fats may also be due to their ability to reduce this and other cytokines. Why a daily baby aspirin helps prevent heart attacks has never been clear, but seems likely be related to its ability to reduce inflammation and clotting tendencies.

Virtually all of the established risk factors are associated with some sort of chronic inflammatory process and are intertwined in other complex ways. Smoking causes low grade inflammation of the lungs, but also periodontal disease and increased fibrinogen levels, and is increased with Type A behavior, all of which can contribute to atherosclerosis and heart attacks by different mechanisms. Unlike chlamydia, there is scant evidence that helicobacter infection is a direct cause of atherosclerosis, but it is associated with low HDL, and high triglyceride and fibrinogen levels which could explain its link with heart attacks.

The acid test of a true risk factor is proving that when it is eradicated, heart attacks don't happen or are remarkably reduced. Type A behavior has more going for it in this respect than cholesterol, hypertension, and smoking combined. Removing hypertension significantly reduces strokes, and risk for lung cancer plummets if you stop smoking. But neither of these interventions alone or combined with cholesterol lowering significantly lowered heart attack rates in one large 8 year multi-center study. Reducing Type A behavior does. In addition, both cigarette consumption and cholesterol levels have been directly linked to the degree of type A behavior in several studies. Type A's have much higher blood pressure surges during stressful challenges than Type B's, and such hyperreactivity has now been linked with increased risk of heart attack.

Stress also reduces immune system resistance to infection, so that normally harmless bacteria and viruses become virulent. The vast majority of people with helicobacter never develop signs or symptoms of peptic disease, and it is likely that the organism starts to attack tissue when stress lowers normal defense mechanisms. Similarly, cytomegalovirus may lie dormant, but like other herpes viruses, becomes activated during stress, and the same may apply to chlamydia. Stress related hormones like noradrenaline can cause direct myocardial damage. And stress has also been shown to increase the production of free radicals that contribute to atherosclerosis.

The development of atherosclerotic plaque is a complex process involving invasion of the artery wall by white cells associated with the inflammatory response to infection and injury, as well as certain microorganisms, proliferation of smooth muscle cells in the inner arterial wall, and the accumulation of oxidized lipids. There is no single cause, and many other influences, such as homocysteine, or antioxidant and B complex vitamins can accelerate or retard this complex process. Infections are an exciting new addition to the cast of characters in this drama. A new study of plaque in the carotid arteries of stroke patients found that almost 75 percent contained bacteria, suggesting a direct involvement that could be treated or prevented. And, there is more.

More Evidence That Heart Attacks Are Caused By Infections, Not Cholesterol

Infections could explain other puzzling aspects of heart attacks. When it started to emerge 50 years ago, coronary heart disease was considered to be due to rich diets and less physical labor. Heart attack deaths rose for the next three decades, but suddenly started to decline in the 60's, long before vigorous treatment of hypertension and high cholesterol, smoking cessation, and increased exercise became popular. Today, although we are fatter and lazier than ever, cardiovascular mortality is less than half of what it was at its peak. This is exactly the pattern one sees with infectious disease epidemics that peak as they spread through susceptible populations, and then taper off as resistance develops. The decline in heart attacks also coincided precisely when broad spectrum antibiotics, especially tetracycline, came into widespread use.

There's lots of other support for the infection/inflammation theory of heart attacks. Their increased incidence in the winter has been attributed to colder weather, but another possibility is increased infections. Heart attack rates increase during and after influenza epidemics not only in the elderly, but also healthy middle aged individuals. Four percent of all people who develop an infection that enters the blood stream suffer an acute myocardial infarction within a month, and up to 10 percent of all strokes are associated with such infections. Abdominal surgery often causes the transient appearance of bacteria in the blood stream, and the incidence of acute myocardial infarction remains high for several weeks post operatively.

However, chronic low grade inflammation due to chlamydia infection appears to be the leading culprit. In one study, researchers obtained blood samples and throat cultures on 61 patients with acute myocardial infarction under the age of 65 on admission to the hospital, and one month later. Tests for *C. pneumoniae* demonstrated chronic infection in 23 patients, acute reinfection in another 12, and about 20 percent reported an upper respiratory infection in the three weeks prior to admission. This data suggests that heart attacks are often triggered by an acute infection superimposed on one that has been chronic, and is supported by Harvard researchers who have been following a large group of doctors taking part in a study of the cardioprotective effect of aspirin. Over an eight year period, there were more than 500 heart attacks and strokes, and initial C reactive protein measurements in this group were compared with an equal number of physicians who remained healthy. The 25 percent with the highest CRP levels had twice as many strokes, and three times more heart attacks than those in the lowest quarter. In other words, their bodies had been fighting something years before they had any symptoms of heart disease. Previous results showed that taking an aspirin reduced heart attacks by 44 percent, but this latest analysis reveals that those with the highest CRP levels had a 55 percent reduction in risk, while aspirin provided little benefit for those with low measurements. Studies are in progress to show that there is also a correlation with *C. pneumoniae*, as has been shown in other research.

Rabbits are notoriously resistant to atherosclerosis, but when researchers recently infected 11 with *C. pneumoniae*, two rabbits developed aortic plaque lesions similar to those seen in humans, and within two weeks! One major pharmaceutical company has already started on a large scale, multi million dollar trial to prove that its antibiotic can prevent heart attacks, and others will likely follow suit, so stay tuned.

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