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Could Widespread Selenium Deficiency Have Been A Factor in the Ebola Outbreak?

With the latest Ebola epidemic in Zaire now several months behind us, a new theory suggests that deficiencies of selenium, an essential mineral, may fan this and other viral outbreaks.

In a paper submitted for publication, E. Will Taylor, PhD, and Chandra Sekar Ramanathan, M.Sc., build a compelling case that the Ebola virus contains selenium-dependent genes—and explain how the virus might mutate and spread aggressively when it cannot obtain enough of the mineral. Both researchers are with the Department of Medicinal Chemistry, University of Georgia.

In the spring of 1995, a brief but major outbreak of Ebola hemorrhagic fever erupted in Zaire, killing about 75 percent of the people infected with the virus. It's called "hemorrhagic fever" because blood leaks through the mouth, nose, eyes, and ears. Some eyewitnesses said that victims' organs seemed to "liquefy."

According to Taylor, the Zaire strain of Ebola virus appears to have several genes that produce proteins with an exceptionally high content of selenocysteine, a rare selenium-based amino acid. When selenium levels in infected cells drop, or are low to begin with, the virus reproduces and "escapes" in search of cells with more selenium—spreading the infection throughout the body. In his paper, Taylor outlines the specific sequence of events that might occur on a genetic level.

Compounding the infection, normal immune defenses against the virus would be handicapped if the host—an animal or person—was deficient in selenium. Selenium is a powerful antioxidant and component of another antioxidant, glutathione peroxidase.

"Selenium deficiency has been shown to increase the pathogenicity of several other viruses, particularly coxsackievirus," Taylor said in an interview. "Thus, the finding of selenium-dependent genes in Ebola raises the possibility that selenium deficiency in host populations may actual foster viral replication, possibly triggering outbreaks and perhaps even facilitating the emergence of more virulent viral strains."

He pointed out that selenium-deficient soils and selenium deficiency in humans have been well-documented in Zaire. The nation, formerly the Belgian Congo, is also where the human immunodeficiency virus 1 (HIV-1) emerged—and where the incidence remains particularly high. In addition, Kaposi's sarcoma, which tends to afflict AIDS patients, is widespread among African

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Vitamin E Prevents Exercise Damage to DNA

Although air pollution, exposure to sunlight, and cigarette smoke are signficant sources of damaging free radicals, most free radicals are actually byproducts of the normal process of respiration—breathing. These free radicals can damage deoxyribonucleic acid (DNA) and lead to mutations that cause cancer. In general, the higher the rate of respiration—heavy breathing, so to speak—the greater the number of free radicals produced.

People who exercise, of course, increase their rate of respiration and, consequently, their production of free radicals. But the danger from these free radicals may be particularly acute for people who *over*-exercise—unless they consistently take vitamin E.

Recently, a team of researchers at the University of Ulm, Germany, confirmed that over-exercise damages DNA. In one experiment, they asked eight men ages 29 to 34 to run on a treadmill until exhausted, which took about 18 minutes. When Günter Speit, MD, and his colleagues analyzed the DNA in five subjects' white blood cells, they noted considerable exercise-induced DNA damage.

Next, the researchers gave five of the subjects a multivitamin/multimineral supplement before and after the treadmill exercise. DNA damage was increased, most likely because copper and iron in the supplement generated additional free radicals, according to an article by Speit, and his colleagues in *Mutation Research* (April 1995;346:195-202)

In a third test, five of the subjects received 800 IU of vitamin E two times before the treadmill exercise and once more about a day later. The DNA damage was reduced.

The greatest protective effect of vitamin E, however, occurred when the five subjects took 1,200 IU of vitamin E daily for two weeks before the treadmill exercise. In this test, the vitamin E prevented *all* DNA damage in four of the subjects and reduced DNA damage in the fifth. □

Researchers Decide More Monounsaturated Fat, Fewer Carbohydrates Better for Diabetics

The American Diabetes Association (ADA) has long recommended a high-carbohydrate diet to manage blood sugar levels in noninsulin-dependent diabetes mellitus (NIDDM), known also as Type II or adult-onset diabetes. The general dietary formula has consisted of 55-60 percent carbohydrate, 15-20 percent protein, and less than 30 percent fat (with approximately 10 percent each of monounsaturated, saturated, and monosatured fat).

In a review of recent changes to the diabetic diet, Nancy F. Sheard, ScD, RD, of the University of Vermont, echoed the new recommendation: a diet high in monounsaturated fat, such as the oleic acid found in olive oil, and substantially lower in carbohydrate. Such a diet would provide only 40 percent carbohydrate, 15 percent protein, 25 percent monounsaturated fat, 10 percent saturated fat, and 10 percent polyunsatured fat.

The reason for the change? It turns out that the high-carhohydrate diet often raised raised blood sugar levels, increased insulin levels, and increased the "bad" low-density lipoprotein (LDL) cholesterol and triglyceride.

"Recent studies indicate that a diet high in monounsaturated fat and low in carbohydrate can produce a more desirable plasma glucose, lipid, and insulin profile," Sheard wrote in *Nutrition Reviews* (Jan 1995;53:16-18).

In an attempt at a graceful about face, the ADA last year suggested that doctors and dieticians individualize the diets of diabetics rather than recommend a one-size-fits-all high-carbohydrate diet.

The turnaround is based on a couple of studies showing that high monounsaturated diets lead to a much more desirable blood fat profile, reducing the higher risk of coronary heart disease faced by diabetics.

In one of the studies, 42 diabetics were alternatively placed on the traditional high-carbohydrate diet or on a high-monounsaturated fat diet. The high-carbohydrate diet raised triglyceride levels by 24 percent and LDL levels by 23 percent. In contrast, the high-monounsaturated diet led to a lower-risk lipid profile. (See Garg, A., *JAMA*, 1994;271:1421-8.)

In *Annals of Medicine* (1994;26:469-78), Suvi M. Virtanen, MD, of the University of Helskini, Finland, reviewed the suspected causes of both insulin-dependent (Type I or juvenile-onset) diabetes and NIDDM.

The possible causes of insulin-dependent diabetes include

- the parents' consumption of smoked meat with high levels of nitrosamines, which are chemically similar to a known pancreatic toxin;
- high levels of nitrate in drinking water; and
- high consumption of cow's milk and cereal grains in childhood.

The possible causes of NIDDM include

- obesity, particularly if the fat is in the belly;
- consumption of highly refined, low-fiber carbohydrates; and
- consumption of saturated fats.

"There are reports on associations of reduced insulin sensitivity with high intake of sodium and with reduced intake of magnesium," Virtanen added. □

Researchers Find Benefits in Fish Oil "Loading"

Many marathon runners "load" their bodies with carbohydrates before a big race. Similarly, loading the body with omega-3 fatty acids (fish oils) increases their cardio-vascular benefits.

Elena Tremoli, MD, and her colleagues at the universities of Milan and Palermo, placed 16 healthy volunteers on one of two fish-oil regimens. In one, subjects were given 3 grams of omega-3 fatty acids daily for 12 weeks. In the other, subjects were initially given twice the dose, 6 grams of omega-3 fatty acids for 6 weeks, followed by 3 grams for the remaining 6 weeks. The long-term effects of the treatment were tracked for another 24 weeks.

While both regimens resulted in more favorable blood-lipid profiles, "the combination of a 'loading' treatment with a relatively long-term one results in more pronounced effects on both plasma and platelet variables," wrote Tremoli in the *American Journal of Clinical Nutrition* (March 1995;61:607-13).

After 12 and 18 weeks, platelet aggregation and levels of thromboxane A2 (which promotes clotting) were lower in both groups. The subjects who started out with high doses of fish oils, however, had less platelet aggregation and even lower levels of thromboxane A2. In addition, all of the subjects benefited from lower platelet aggregation for 14 weeks after they stopped taking the fish oil supplements. □

Infants Benefit from Mother's Fish Oil Diet

Pregnant women who consume large amounts of the omega-3 fatty acids give their offspring a head start with extra stores of the nutrient. The the omega-3 fatty acids are essential for the in utero development of the infant's brain and retina, and they prevent premature birth as well.

The latest findings were reported in June 1995 by William E. Connor, MD, at the Second International Congress of the International Society for the Study of Fatty Acids and Lipids, held in Bethesda, Md. Women who ate

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Even After a Bypass, Vitamin E Helps the Heart

It's never too late to benefit from vitamin E supplements—even if you've had coronary artery bypass surgery. That's the finding of researchers at the University of Southern California School of Medicine, who reported the results of a new study in the *Journal of the American Medical Association*.

Howard N. Hodis, MD, and his colleagues came to that conclusion after carefully studying angiograms from 156 patients involved in a larger medical study.

All of the patients, ages 40 to 59, had undergone bypass surgery, and were being carefully monitored for the growth of new fatty deposits on their arteries.

Twenty-nine of the subjects took 100 to 450 IU of vitamin E daily of their own accord, not because the study required it. The other 127 people consumed less than 100 IU of vitamin E, either in the form of supplements or food.

Angiograms—essentially x-rays of the heart—revealed that patients taking 100 IU or more vitamin E daily had much smaller lesions on their arteries than did the men taking less than 100 IU vitamin E, according to Hodis' report in *JAMA* (June 21, 1995;273:1849-54). The benefits are particularly noteworthy because the men taking larger amounts of vitamin E began the study with higher blood levels of cholesterol. Many researchers believe that cholesterol becomes dangerous only when oxidized in the absence of vitamin E. (See related article below.)

So...How Much Vitamin E Helps the Heart?

The 50-year-old controversy surrounding the role of vitamin E in preventing coronary heart disease came to an end of sorts two years ago. That's when Harvard University researchers reported that supplemental doses of the vitamin reduced the risk of heart disease by as much as 54 percent.

Many researchers have focused on *why* vitamin E protects the heart—and how the vitamin interacts with cholesterol. Instead of high levels of cholesterol or the low-density lipoprotein (LDL) form of cholesterol causing heart disease, researchers increasingly believe LDL becomes a problem only when it oxidizes, or turns bad.

In numerous studies, Ishwarlal Jialal, MD, of the University of Texas Southwestern Medical Center, Dallas, has documented that vitamin E (as well as vitamin C) prevents LDL from oxidation. But how much vitamin E is enough?

To determine the minimum effective dose, Jialal gave a group of human volunteers vitamin E in doses of 60, 200, 400, 800, and 1,200 IU per day. After eight weeks, he could find no significant effects from the 60 or 200 IU doses. According to Jialal's report in *Arteriosclerosis, Thrombosis, and Vascular Biology* (Feb 1995;15:190-8), a minimum of 400 IU of vitamin E daily was needed to prevent the oxidation of LDL.

It comes as no surprise, then, that a team of Italian researchers has recommended large doses of vitamin E—in this case, 900 IU per day—in the treatment of elderly patients with heart disease.

Giuseppe Paolisso, MD, and his colleagues at the University of Naples monitored the effects of vitamin E on blood levels of glucose, insulin and triglyceride and the ratio between LDL and high-density lipoprotein (HDL, the "good" cholesterol) among 30 men and women in their 70s.

After four months, they analyzed "fasting" blood samples from the subjects. Blood sugar levels were almost identical between those who did and didn't take the vitamin E. However, blood levels of insulin and triglyceride were significantly lower in the vitamin E group—both signs of reduced heart disease risk. In addition, LDL levels decreased and HDL levels increased, creating a much more favorable ratio between the two.

"We conclude that chronic intake of pharmacological doses of vitamin E might be useful in the therapy of elderly insulin-resistant patients with coronary heart disease," Paolisso wrote in the *American Journal of Clinical Nutrition* (April 1995;61:848-852).

Breast Feeding Boosts Babies' Brains

An analysis of nine-year-old Dutch children has found that those breast fed as infants were neurologically better developed and suffered fewer coordination problems than those fed formula.

C.I. Lanting, MD, of University Hospital Groningen, The Netherlands, compared 135 breast fed and 391 formula-fed children. The children were classified as neurologically normal, slightly abnormal, or abnormal.

The children breast fed for at least three weeks were half as likely to suffer neurological problems, such as motor disorders, than the formula-fed children, according to Lanting's report in *Lancet* (Nov 12, 1994;344:319-322). Breast fed children accounted for 74 percent of those classified as normal and 14 percent of those with neurological problems.

Fish Oils...

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sardines and took fish oil capsules gave birth to infants with omega-3 fatty acid levels 30 times higher than those of women eating an ordinary diet.

Food Allergies Underlie Many Ear Infections

For years, many nutritionally oriented pediatricians, such as Lendon H. Smith, MD, have said that ear infections in children are triggered by allergic-like food sensivities, particularly to cow's milk. In response, conventional doctors stated that the incidence of food allergies was overstated and rare. A major study, published in a leading medical journal, now confirms the likely role of food allergies in ear infections.

Talal M. Nsouli, MD, and his colleagues at Georgetown University, carefully tested 104 children with recurrent middle-ear infections (otitis media) for food allergies. Eighty-one (78 percent) of the children had confirmable food allergies, based on skin prick testing, IgE tests, and food challenges. Nsouli then placed most of the children, ages 1.5 to 9 years, on diets that excluded specific foods for 16 weeks. Meanwhile, he and his associates monitored the childrens' ear infections.

Seventy (86 percent) of the 81 children placed on special diets had a "significant amelioration" of their middle-ear infections. When Nsouli fed suspected allergenic foods—principally milk and wheat—to 70 of the children, 66 (94 percent) developed ear infections, according to his article in *Annals of Allergy* (September 1994;73:215-219).

Nsouli commented that bacterial or viral infections may actually be a consequence of poor drainage and congestion in the middle ear, with allergy being the underlying mechanical cause. Nsouli concluded in *Annals of Allergy* that his study "indicates that food allergy may contribute significantly to a proportion of cases. Thus, the possibility of a food allergy should be considered..."

Mothers might also prevent many ear infections by exclusively breast feeding their infants for at least four months after birth. Burris Duncan, MD, a pediatrician at Steel Memorial Children's Research Center, Tucson, tracked more than 1,000 infants during their first year. Those consuming only breast milk for at least four months had half the incidence of acute ear infections as those who were never breast fed, according to an article by Duncan in *Pediatrics* (May 1993;91:867-872). □

Selenium Deficiency and Ebola...

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subsistence farmers in selenium-deficient regions. In effect, much of the population of Zaire is immune compromised to begin with and highly susceptible to epidemic infections.

Last year, in the *Journal of Medicinal Chemistry* (Aug 19, 1994;37:2637-54), Taylor presented strong theoretical evidence that HIV contained genes that direct the formation of selenium-containing proteins. Because the bodies of healthy people contain only about 10 mg of selenium, the mineral can be quickly consumed by a selenium-hungry virus. (See THE NUTRITION REPORTER, Nov 1994.)

At the time, Taylor suggested that supplemental selenium might prevent HIV from attacking other cells. Evidence from clinical trials shows that selenium supplements do improve the well-being and lifespan of patients with AIDS, and selenium has been shown to inhibit HIV in the test tube (*Antiviral Research*, 1995, 26:A271-86).

Based on Taylor's theoretical genetic analysis, the selenium-dependent proteins in the Ebola virus may require about 10 times as much selenium as those he found in the HIV virus.

Support for Taylor's theory comes from recent studies of how selenium deficiency in animals and people lead to mutations in the coxsackievirus. In *Nature Medicine* (May 1995;1:433-6) Melinda Beck, PhD, of the University of North Carolina (Chapel Hill) and Orville Levander, PhD, of the U.S. Department of Agriculture, described the specific genetic changes that occur when selenium deficiency triggers the coxsackievirus mutation. Once created by selenium deficiency, the mutant virus can infect healthy animals and even people with ample dietary selenium. (See THE NUTRITION REPORTER, July 1995.)

In China, selenium-deficient diets allow the relatively benign coxsackievirus to mutate into a virulent strain that attacks heart muscle, causing a form of heart failure. Zaire might be a parallel situation in which selenium deficiencies lead to mutations in HIV and the Ebola virus.

Editor's note: For a copy of Dr. Will Taylor's paper on Ebola and selenium, write to him at the Department of Medicinal Chemistry, University of Georgia, Athens, GA 30602-2352. □

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