AUTOIMMUNE INFLAMMATORY DISEASES: WHEN SELF IS THE ENEMY

By John Clark M.D. NorthernLightsHealthEducation.com

"How long will he have to live in that bubble?" David Vetter, born with a dysfunctional immune system had lived in a sterile plastic "germ-free isolator" world all of his life. The question was; when would science deliver on its quest, through some new technological advance, to find a solution to David's dilemma? If allowed to encounter the environment, the one we live in every day, David would most certainly pick up a pathogen that would end his life. Even NASA got involved! Top engineers put their heads together and crafted a most eloquent space suit for David. But after a few forages out into the real world, David's fears of contamination, microbes and death drove him back to his reclusive spot at Baylor University Medical center. David finally died when an attempt to solve his life-threatening condition with a tissue transplant operation, failed to resolve his immune system deficiency.[1]

If we did not have an immune system, we like David, would die. But where did our immune system come from? "I will praise thee; for I am fearfully and wonderfully made: marvellous are thy works; and that my soul knoweth right well."[2]

THE ADVANTAGE OF AN IMMUNE SYSTEM

The skin is our first line of defense. "Every square inch of human skin consists of 19 million cells, 60 hairs, 90 oil glands, 19 feet of blood vessels, 625 sweat glands, and 19,000 sensory cells that can transmit information at more than 200 miles an hour."[3] What is more, immune cells of the skin secrete antibodies that can stop invaders. And not just from the skin of our bodies, antibodies from the immune system emerge to protect the nose, sinuses, throat, lungs, stomach, and intestines. Without these antibodies from the immune system, we'd all be doomed.

After the skin, our next line of defense centers in our immune system's ability to mount an all-out counterattack to invaders, and I do mean counterattack. These invaders can be identified or unidentified. If the immune system identifies them (has had experience with them before) then it can deal more specifically and carefully with them. If the immune system has never seen them before, then it gets out the big guns and shoots anything that seems out of place. As long as this line of defense only destroys invaders, we are happy. This line of defense is called inflammation. It is especially active to deal with any new injury, antigen, bacteria, or virus.

FRIENDLY FIRE: WHY AUTOIMMUNE INFLAMMATORY DISEASE?

A compromised immune system cannot deal with infections and antigens in its usual healthy way, consequently it resorts to inflammation. Tissue damage often occurs as the body attempts to rid itself of disease.[4] When the only weapon available is a sledgehammer, collateral damage is sure to occur. Autoimmune inflammatory diseases arise under several situations where the immune system is not able to function most efficiently. Inflammation can occur when: the immune system is not in optimal health, the immune system is confused by hostile antigens, the immune system is overstimulated, the immune system's inflammatory process is secretly triggered, or the immune system is overpowered by oxidative stress or other sources of inflammation. A few examples of autoimmune inflammatory diseases that occur under these conditions are rheumatoid arthritis, polymyalgia rheumatica, psoriasis, ankylosing spondylitis, polyarteritis nodosa, scleroderma, inflammatory bowel disease, ulcerative colitis, Crohn's disease, irritable bowel, some cases of type I diabetes, fibromyalgia, multiple sclerosis, systemic lupus erythematosus, allergy, chronic fatigue, and asthma, etc.

WHAT PERTURBS THE IMMUNE SYSTEM

The list of what brings down the immune system so that it resorts to primitive means of defending the body could be very long. We will try to point out some of the ones most common and the most dangerous, rather than giving an exhaustive list.

A compromised immune system cannot deal with infections and antigens in its usual healthy way, consequently it resorts to inflammation.

THE AGING IMMUNE SYSTEM

As we age our immune system tends to lose its acuity making autoimmune inflammatory diseases more likely.[5] Now you may be thinking, "There is nothing I can do about aging!" But, as you will discover, aging can be influenced for better or for worse.

STRESSING THE BODY'S DEFENSES

Stress essentially drives the immune system to suicide. Emotional stress or job "burn out" provokes inflammation, increasing the risk of cardiovascular disease and autoimmune inflammatory disease.[6] Having experienced major stressful life events within the last 2 years increases the risk of developing an autoimmune inflammatory disorder 140%.[7]

ANTIOXIDANTS

A deficiency of antioxidants favors oxidative stress. Oxidative stress kills cells: The immune system then makes anti-bodies to their spilled DNA.[8] Many autoimmune inflammatory diseases are identified by the presence of anti-DNA antibodies.

HEAVY METAL BLUES

Heavy metals increase the body's inflammation, increasing the risk of autoimmune inflammatory diseases. Top heavy metal villains include lead,[9] mercury, beryllium, nickel, chromium, cobalt,[10] cadmium, and vanadium.[11] Mercury increases inflammatory tissue damage by 50%.[12]

THE DRUGGED IMMUNE SYSTEM

Many drugs are known risk factors for these diseases. For example, estrogens: estrogens enhance the release of inflammatory mediators from white cells in the immune system.[13] Oral contraceptive use increases autoimmune inflammatory disease risk by 90%.[14] Hormone replacement therapy increases autoimmune inflammatory disease risk 150%.[15] Pharmaceutical drugs are not the only source of these hormones. Chemicals and animal products are also big sources of hormone and hormone like substances that can cause autoimmune inflammatory disorders.

BETTER LIVING THROUGH CHEMISTRY?

There are many chemicals, especially in some work environments,[16,17] which increase the risk of autoimmune inflammatory disease.[18] For example, hair preparations, especially dyes, increase the risk of an autoimmune inflammatory disease by 90%.[19] Another culprit is Sodium Lauryl Sulphate (SLS), which breaks down the body's barriers to antigen invasion and it also causes inflammation.[20] SLS is the most common major ingredient in shampoo's, toothpaste and other personal care items.

What are you eating? Food preservatives, such as BHA (3-tert-butyl-4-hydroxyanisole),[21] and additives, such as emulsifiants, thickeners, surface-finishing agents and contaminants like plasticizers can trigger inflammation in the body.[22]

Do you eat crackers with soup? The stomach's job is to produce acid for the digestion of food. When alkali substances such as baking soda/powder are ingested, as found in crackers, many biscuits and cakes, the stomach has to work twice as hard to achieve the same level of acidity. Baking soda/powder intake is associated with a 190% increase in risk of stomach cancer, a cancer often the result from increased stomach acidity, irritation, and inflammation.[23]

Toxins and waste products are eliminated through the skin. People avoid jobs that provoke sweat and as a result skin pores become clogged

with waste. Consequently, a greater burden is placed on the liver, bowels and kidneys to dispense of these. This leads to increased inflammation and increased skin, liver, bowel, and kidney disease. Good skin hygiene helps combat inflammatory disease.[24] Good skin hygiene may involve thorough scrubbing, brushing and sweating.

WOULD YOU LIKE THAT FRESH OR ROTTEN?

Can you find a good banana in a dumpster? Aflatoxins, formed in the process of aging or fermenting,[25] are a source of inflammation.[26] Dietary sources of aflatoxins include cheese,[27] wine, vinegar, and any food created by rotting or fermentation. Scientists use weak vinegar solutions to cause inflammatory bowel disease in rats as a model for studying ulcerative colitis and Crohn's disease in humans.[28,29] What is more, chemicals formed when foods are pickled[30] increase oxidative stress, inflammation,[31] autoimmune disease, and cancer.[32,33]

Another source of aflatoxin exposure is the environment. Mold in the environment increases the risk of autoimmune inflammatory disease by 180% for the lungs[34] and 360% for joints.[35] Shade trees and shrubbery close and dense around a house, water-damaged buildings,[36,37,38,39] decaying leaves,[40] compost heaps,[41] sauna baths, wet basements, swamps and lowlands—all are sources of aflatoxins and inflammation. Avoid all decay, both personal and environmental.

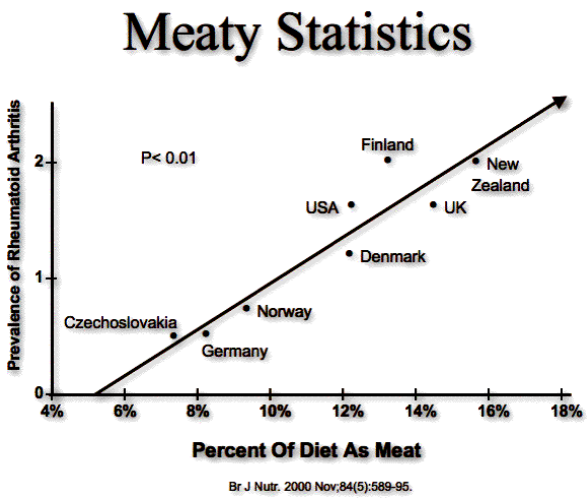
PERFECT HEALTH DEPENDS ON PERFECT CIRCULATION

Inflammation increases when blood flow is congested and slowed.[42,43,44,45] As a result autoimmune inflammatory diseases are more likely with a sedentary lifestyle, tight clothing or cold extremities. On the other hand, when circulation is quickened, inflammation decreases.[46]

In cold weather, wearing short sleeves or short pants exposes the limbs to cooling, chilling the blood back from the extremities to the chest, abdomen and pelvis where inflammation can set in. Additionally, the circadian rhythm (your internal equilibrium clock which controls the balance between inflammation and anti-inflammation) is disrupted,[47] inflammatory mediators are released,[48,49] and the risk of autoimmune inflammatory disease increases.

Studies show that tight clothing has a negative effect on the body. From slowed digestion of food to increased inflammatory mediators, tight clothing is sure to increase one's risk of autoimmune inflammatory disease.[50,51]

Another way in which circulation is unbalanced, and can be the source of inflammation, is through overwork of the brain. Overwork of the brain in the absence of good outdoor physical exercise results in increased inflammation. Inflammatory diseases are significantly more prevalent in those doing mental work compared to those involved in physical labor.[52]



SLEEPING OFF THE DISEASE

Sleep loss is associated with increased inflammation and autoimmune disease.[53,54,55,56] Many of the things that we are discussing affect sleep quality and therefore also affect the risk of disease.

AIR QUALITY CONTROL

Indoor air has far more contaminants than outdoor air. Indoor air contaminants are a source of inflammation. Contaminants include breathable dust, nitrogen dioxide, chemicals such as formaldehyde, aspergillus aflatoxins, and various molds.[57]

DON'T LET THIS ONE GET OUT OF HAND

Some causes of autoimmune inflammatory disease start small and increase with time to something bigger than expected. Improper use of the voice,[58] voice strain,[59] shallow or improper breathing[60,61] can all cause inflammation of the lungs and throat increasing the risk of autoimmune inflammatory diseases.

Repetitive or forceful tasks cause tissue microtraumas leading to inflammation that can spread to the whole body[62] increasing the risk for autoimmune inflammatory disorders.[63,64,65]

CONDIMENTS AND SPICES

Strong dietary condiments and spices can be the source of inflammation leading to autoimmune disease. Mince pies, cakes, preserves, highly seasoned meats with gravies, pickles, excessive salt, grease, pepper, mustard, and ketchup, etc.

Excessive salt intake increases hypertension and renal injury caused in part by oxidative stress and inflammation in the kidneys and blood vessel walls.[66,67]

Red and black pepper significantly increase the stomach's acidity leading to cell destruction, microbleeding, and inflammation.[68] Red pepper increases stomach acid excretion 700%.[69]

STIMULANTS

What about caffeine? Caffeine and its relatives increase the risk of acquiring an autoimmune disease. Once inflammation starts in the body, caffeine can accelerate it by 300%-600%.[70] Chocolate increases the risk by 150%, cola drinks by 120%[71] and coffee 118%.[72]

Does alcohol impair the immune system? Alcohol consumption increases free radical formation and whole-body inflammation.[73] Wine can be especially aggravating, worsening such inflammatory diseases as asthma.[74]

Smoking (even second-hand smoke)[75] causes increased inflammation thereby using up the body's protective antioxidant resources. Toxic fumes and caustic chemicals from burning tobacco increase the risk of acquiring an autoimmune inflammatory disease.[76,77] The risk of acquiring an autoimmune inflammatory disease increases 65% with smoking and 98% with alcohol consumption.[78]

FOOD WOULD YOU LIKE THAT FRESH OR ROTTEN?

Notice that we have been writing about a lot more than just diet. Diet is important, but there is a whole lot more to autoimmune disease than just diet.

SNACK ATTACK!

Fried potatoes, salty snacks, desserts and processed meats are among the top instigators of elevated oxidative stress and whole-body inflammation.[79] Can you name one snack food that is healthy?

WESTERN DIET WOES

A number of studies have identified the western diet, (described variously as including red meat, processed meat, pork/hot dogs, butter, lard, hydrogenated fats, high-saturated fat, high-fat dairy, eggs, french fries, potatoes, regular and diet soft drinks, pizza, refined grains, breads and pastas, coffee and tea, sweets/candy and desserts), as increasing the risk of autoimmune inflammatory diseases by as much as 210%.[80,81,82]

THE KEY IS TO EAT YOUR PROTECTION

Patients suffering from autoimmune inflammatory disease have significantly lower blood antioxidants levels.[83] [84] Studies also show that commercial supplements are of no value in correcting this deficiency.[85] Proper diet is the only solution to poor nutrition and reducing the risk of autoimmune inflammatory disease.[86]

FIBER: START ROUGHING IT

Patients suffering from autoimmune inflammatory disease can also have significantly lower fiber and magnesium intakes. Fibrous foods are usually higher in magnesium. Fiber and magnesium deficiency are associated with a 300%-400% elevation in inflammation.[87]

MINERAL DEPLETION IS A GLOBAL ISSUE

The amount of magnesium in all foods has decreased by 19% in the last 50 years.[88] Low levels of zinc,[89] selenium,[90] and magnesium[91] are associated with increased inflammation. Whole wheat flour has 530% more magnesium, 320% more zinc, and 110% more selenium than white flour.[92] Pumpkin seeds are a rich source of zinc and Brazil nuts are a good source of selenium.

DOUGHNUT DESPAIR

Doughnuts are a huge source of advanced glycation end products! Carbohydrates fried with oil accumulate advanced glycation end products (AGEs), toxins that activate the body's inflammatory mediators.[93,94] AGEs can also be formed in the body if the blood sugar becomes elevated. A slice of 100% whole wheat bread has 536 AGEs units,[95] while a plain-glazed doughnut weighs in at a whopping 425,740 units of AGEs.[96]

HIGH-FRUCTOSE IS HIGH RISK

Fructose (in all its forms, e.g., high fructose corn syrup) activates inflammatory mediators in the liver[97] and blood vessels[98,99] increasing the risk for autoimmune inflammatory disease.

RISK MANAGEMENT

The results of a study that came out of Israel help put things in perspective. Dietary choices that increase autoimmune inflammatory disease risk include sugar (430% increased risk), cholesterol (360%), eggs (350%), saturated fat (animal fat, 310%), soft drinks (300%), and vegetable oil (22%).[100]

FAT AND CHOLESTEROL

Dietary cholesterol is especially harmful.[101] Cholesterol provokes the immune system to increase inflammation.[102] [103] A high-cholesterol diet more than triples the risk of autoimmune inflammatory disease.

High-fat food is at greater risk for lipid oxidation or peroxidation. Cheese is high in fat and is created by decay, thus it is high in oxidized lipids (fats). These oxidized cheese lipids significantly increase the risk of autoimmune inflammatory diseases.[104,105]

Butter significantly increases oxidative stress by stimulating the immune cells to produce inflammation when there is no other reason to be causing inflammation.[106]

High-fat diets increase body inflammation.[107] Of special concern are trans-fats that significantly increase the inflammatory responses of the body. Saturated fat, as found in animal products and tropical oils such as palm oil, have been shown to increase the body's inflammation.[108] Compared to a diet predominating in monounsaturated (vegetable) fat, eating a high-saturated (animal) fat diet increases body inflammation 270%.[109] What's more animals fed a fatty diet develop a high rate of autoantibodies (antibodies against one's own self),[110] a classic finding in autoimmune inflammatory diseases.

Of special concern are oils that have been become oxidized. Oxidized oils pose an immediate and long-term threat to body anti-inflammatory reserves heightening the risk of multiple autoimmune inflammatory diseases.[111,112] Oxidized oils are common to deep fat fryers, fried foods, and packaged foods with a long shelf history.

Cooking food in oil (frying) produces trans-fat,[113] acrylamide[114] and lipid peroxidation.[115,116] These by-products of frying are all stimulators of inflammation leading to increased risk of autoimmune inflammatory disorders.[117] Trans-fat can also be found in hydrogenated and partially hydrogenated vegetable oils, margarines and shortening.

Oxidized cholesterol promotes tissue inflammation and cell death leading to atherosclerosis (inflammatory heart disease) and autoimmune inflammatory disease.[118] Common sources of oxidized cholesterol are spray-dried egg powders (such as found in pancake mixes), Parmesan cheese, butter oil, ice cream, sausages, and beef tallow. Oils and cholesterol are especially apt to oxidation when heated in the presence of air for a longer period for example in deep-frying at fast food restaurants.[119]

SAME FOODS EVERYDAY?

Eating the same foods day after day overwhelms the body's food tolerance mechanisms and can result in food allergy and/or autoimmune inflammatory disease.[120]

ENRICHED!

A diet high in refined carbohydrates negatively affects the balance of free radical generation and antioxidant defense leading to inflammation overload.[121,122,123] A breakfast consisting of a bowl of corn flakes with skimmed milk, a piece of toast and a glass of orange juice converts almost instantly to 16 teaspoons of sugar. Sixteen teaspoons of sugar will increase the body's oxidative stress and inflammation by 240%.[124] A can of soda has 12 teaspoons of sugar.

Patients with autoimmune inflammatory diseases have a high incidence of sensitivity to wheat gluten, as high as 10 times higher than normal individuals.

Refined grain products, (e.g., white bread, white rice, white pasta), tip the body's oxidant/antioxidant balance toward oxidation, increasing inflammation and the risk of autoimmune disease.[125,126]

THE GLUTEN CONNECTION

Patients with autoimmune inflammatory diseases have a high incidence of sensitivity to wheat gluten, as high as 10 times higher than normal individuals.[127]

ANIMAL ANTIGENS

Individuals with autoimmune inflammatory disease show higher than normal sensitivities to animal product antigens; 1200% higher for dairy, 600% for eggs, 460% for pork, and 400% for fish.[127] If you have autoimmune disease or know you should be taking precautions to avoid autoimmune inflammatory disease it might be prudent to stay away from these sources of disease.

MORE ON MILK

The link between dairy and autoimmune inflammatory diseases is multifactorial;[128,129] milk is immunosuppressive,[130] it has many hormones which increase disease risk,[131] milk is the source of many infectious agents (viruses and bacteria) that precipitate autoimmune inflammatory disease,[132] it contains many antigens which initiate the autoimmune process,[133,134,135,136] and milk provokes and aggravates[137] the inflammatory process.[138]

GO BIG RED

Why is red meat red? Heme iron makes red meat red and red cells red. Heme iron increases the body's sensitivity to oxidative stress and inflammation.[139] Consumption of red meat increases the risk of autoimmune inflammatory disease by 130%.[140] Epidemiological studies comparing the amount of meat eaten in countries around the world with how much autoimmune inflammatory disease they have, shows that with increased meat consumption there is increased disease.[141] The message of course is, if you need an autoimmune inflammatory disease, eat more meat.

PROTEIN PORTIONS

Many people these days are worried about whether or not they are eating enough protein in their diet. It is a bit of a mania. In fact, it is actually hard to achieve a low protein diet. Protein, eaten in excess of body needs, increases the risk of autoimmune inflammatory diseases by 190%.[140] (For more information on protein, please refer to our handout and presentation on osteoporosis.)

VARIETY, THE SPICE OF LIFE?

Most people in developed countries like to eat a large variety of food at each meal as though they had to balance their entire life's nutritional requirements at one sitting. Excessive antigenic load, as encountered in a complex meal comprised of multiple diverse foods, can provoke autoimmunity, allergy, and inflammation.[142]

EAT TO LIVE, OR LIVE TO EAT?

Another instigator of the autoimmune inflammatory process is overeating. Overeating provides fuel for a bigger fire then can be healthfully managed. Excessive caloric intake is associated with increased body oxidative stress[143] and increased incidence of autoimmune inflammatory diseases.[82] On the other hand reduced caloric intake decreases autoimmune inflammatory disease risks.[144]

Many people these days are worried about whether or not they are eating enough protein in their diet. It is a bit of a mania. In fact, it is actually hard to achieve a low protein diet.

WEIGHT MANAGEMENT

For the avoidance of these diseases, carrying extra weight is not ideal. Studies show that whole body inflammation increases with increasing body weight.[145,146] Being overweight increases the risk of acquiring an autoimmune inflammatory disorder by 275%.[147]

As a person gains weight, fat tends to gather about the abdomen. While much of this fat is external, a large portion of it is also internal, around the organs. This internal fat is termed organ or visceral fat. Visceral fat is another source of inflammation[148] and oxidized fat. For each 1% increase in visceral fat, the risk for increasing inflammation goes up an additional 140%.[149]

A CASE OF MISTAKEN IDENTITY

Worms—could there be a case of mistaken identity? Trichinellosis, a parasite acquired from eating pork and bear, is associated with increased inflammation.[150,151] Musculoskeletal symptoms include muscle pain, joint pain, muscle weakness, and restriction of joint movements.[152,153]

Trichinellosis is not the only infection implicated in autoimmune inflammatory conditions. Viral and bacterial infections are being implicated more and more in the development of autoimmune inflammatory diseases.[154,155,156,157] Autoantibodies increase with the number of infections a person has suffered in their lifetime.[158] Numerous infections agents, including Salmonella,[159] E. Coli, Streptococcus and Mycobacterium,[160] have been linked to autoimmune inflammatory diseases. The most abundant source of these infectious agents is animal products.[161,162,163,164,165]

NEEDLING THE IMMUNE SYSTEM

There are some risky behaviors that may need to be avoided. One of these, about which more and more scientific evidence is emerging, is vaccination. For example, receiving measles, mumps, and rubella vaccine (MMR) vaccination significantly increases the odds of acquiring chronic inflammatory arthritis.[166] Compared to receiving the common tetanus vaccine: receiving a hepatitis B vaccine increases the odds of acquiring multiple sclerosis by 420%, systemic lupus erythematosus by 810%, and rheumatoid arthritis by 1700%![167]

EXERCISE

It has been said, “If you don’t find time to exercise, you will have to find time to be sick.” When one sits around, it is like a car idling; smoke and fumes build up. For the sedentary individual, inflammation builds up, increasing the risk for autoimmune inflammatory disease.[168]

STRICT SCHEDULE

How regular are you—I mean in your schedule? Studies show that extended and irregular shift work confers an increased risk of contracting an autoimmune inflammatory disease.[169]

RESULT OF PERTURBING THE IMMUNE SYSTEM

Once the immune system becomes off balance it can really fall a long way from normal, resulting in signs and symptoms that culminate in autoimmune inflammatory disease. Besides all the well-recognized autoimmune

inflammatory diseases listed earlier there are other unhappy outcomes to letting the immune system fall into disarray. We will list just a few.

The presence of an autoimmune inflammatory disease is a good sign that the immune system is probably going to have trouble performing its usual function with success. Most autoimmune disease is associated with immune suppression or dysfunction. People with autoimmune inflammatory disorders are 85% more likely to acquire serious life-threatening infections. The most common sites of infection include, joints, skin, soft tissues, and the lungs.[170]

Despite increased medical treatment options, patients with autoimmune inflammatory diseases do not enjoy lengthy lives.[171] Pneumonia, tuberculosis, and liver disease are significantly increased as causes of death in these patients.[172]

Chest pain; should autoimmune inflammatory disease patients be worried? Inflammatory disease not only affects bones, connective tissue, and joints, but blood vessels and heart muscle as well. Patients with autoimmune inflammatory disease are 90% more apt to have congestive heart failure,[173] 95% more likely to die of sudden cardiac arrest, and 220% to have a heart attack.[174]

Many autoimmune inflammatory disease patients suffer from osteoporosis, (thinning of the bones), making them more susceptible to fractures. The inflammatory process involved in autoimmune disease is also a major player in osteoporosis.[175] Many of the medications with which autoimmune inflammatory disease are treated also cause osteoporosis.

Cancer is also often the result of a deficient immune system. Immunity is a function of white blood cells. As a consequence of inflammation and immune compromise, the risk of leukemia (blood cell cancer) increases 150%.[176]

RESTORING AND MAINTAINING THE IMMUNE SYSTEM

Let us change gears now and talk about how to restore a failing immune system and maintain it in a condition to assure the avoidance of further inflammatory disease, its complications, and its pain.

FRESH MORNING AIR

Occupations involving physical work in the open air are protective, while working in artificial, air-conditioned environments increase the risk of contracting an autoimmune inflammatory disease.[177,178] One of the most effective immune boosters is an early morning walk in the fresh air near a body of water as the sun is just coming up.[179]

HAVE YOU SEEN THE SMILING SUN RECENTLY?

Sunlight exposure reduces inflammation in the body.[180,181,182] Sunlight exposure is a major source of vitamin D.[183] Vitamin D deficiency increases the risk of many common cancers, multiple sclerosis, rheumatoid arthritis, hypertension, cardiovascular heart disease, and type I diabetes.[184] It is recommended that at least 25% of your skin be exposed to the sunlight for 20 minutes each day, and longer if you have darker skin.

WHAT ABOUT EXERCISE?

"But I can't exercise, I have pain!" you may be thinking. Exercise tips the inflammatory/anti-inflammatory balance in favor of reduced inflammation and reduced disease risk.[185 186,187,188] With few exceptions, sufferers of autoimmune inflammatory diseases benefit significantly from physical activity, which leads to significant improvements in strength, pain, and fatigue without making the disease worse.[189,190,191 192,193]

As individuals age their immune system declines. Being physically fit helps to slow this decline. The immune system responds positively to moderate exercise, while too much exercise tends to suppress it.[194]

While you are out exercising, as I know you will be, loose clothing is of greater benefit than clothing that restricts movement and blood flow. Tight clothing has been shown to interfere in body temperature variations, blood flow and hormone levels; factors implicated in autoimmune inflammatory disease.[195]

CIRCADIAN RHYTHMS: THE BODIES INTERNAL CLOCK

Our bodies run on clocks. The anti-inflammatory/inflammatory balance cycles on a clock called your circadian rhythm.[196,197] The anti-inflammatory circadian clock malfunctions when meal times are varied, or meals are taken late in the evening,[198,199] sleeping times are varied,[200] insufficient, or shifted to a late bedtime and/or late rise time, a job requires shift work where daily schedules vary on some days, such as on days off or weekends.[201] Regularity in sleeping hours improves overall sleep quality and anti-inflammatory effect. For the autoimmune inflammatory patient, we recommend a strict schedule for sleeping hours with a set nightly bedtime no later than 9:30 p.m., and a set regular rise time between 7.5 and 8 hours later on all weekdays and weekend days.[202] We recommend regular mealtimes every day of the week not varying by more than 5 minutes with no meal later than 5:30 p.m.[197] We recommend regularity in exercising every day of the week including days off and weekends.[203]

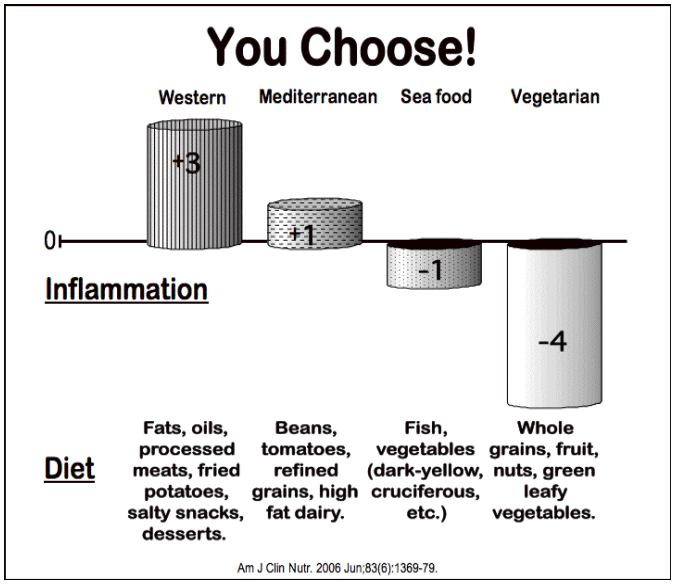
DIETARY CHOICES

A study was performed comparing four diets: (1) fats and processed meats diet (fats, oils, processed meats, fried potatoes, salty snacks, and desserts)—the western diet, (2) beans, tomatoes, and refined grains diet (beans, tomatoes, refined grains, and high-fat dairy products)—a Mediterranean like diet, (3) vegetables and fish diet (fish and dark-yellow, cruciferous, and other vegetables)—sea food diet, and (4) whole grains and fruit diet (whole grains, fruit, nuts, and green leafy vegetables)—vegetarian vegan diet. The western diet raised three markers of inflammation, the Mediterranean diet raised one marker of inflammation, the seafood diet lowered one marker of inflammation and the vegetarian vegan diet lowered four markers of inflammation; showing the superiority of the vegan diet in addressing autoimmune inflammatory diseases.[204]

VEGETARIAN ADVANTAGE

A vegetarian diet has been found to have an anti-inflammatory effect on patients with active autoimmune inflammatory disease.[205,206,207,208] A vegetarian diet stimulates the immune system, improves tolerance to noxious antigens found in less-ideal diets,[209] and is loaded with antioxidant anti-inflammatory vitamins and phytochemicals. Another advantage to the vegetarian diet is its high content of natural antioxidants. Studies show that patients suffering from autoimmune inflammatory diseases eat significantly fewer antioxidant foods.[210] On the other hand studies show that high antioxidant intake decreases the bodies inflammation.[211]

There is a real advantage to eating fruit and vegetables. Fruits and vegetables are high in flavonoids,[212] phytochemicals and antioxidants that have been found to lower the oxidative stress, inflammation and oxidation of lipids (fats) in the body.[213] Fruits and vegetables are high in vitamin A. Deficiency in vitamin A leaves the body unguarded against oxidative stress and autoimmune inflammatory disease.[214,215] Good sources of vitamin A include sweet potatoes,



carrots, kale, spinach, winter squash, cantaloupe, and broccoli.

Whole grains and fiber are also a part of an autoimmune inflammatory disease fighting diet. Diets high in whole grains have been shown to have a protective effect against systemic inflammation reducing the risk of autoimmune inflammatory disease.[216] [217] Fiber, as found in

whole grain products and bran, reduces inflammation in patients with inflammatory disorders.[218]

What about a "low carb" diet for reducing inflammation? To the contrary, low fat, high carbohydrate diets have been shown to significantly reduce whole body inflammation.[219]

What diet provides the maximal amount of antioxidant, anti-inflammatory benefits? Fresh food, that taste of Eden, is most effective. Fresh food is an uncooked vegan diet consisting of berries, fruits, vegetables and roots, nuts, germinated seeds, and sprouts, i.e., rich sources of carotenoids, vitamins C and E, (some call this a "raw food" diet). People on a fresh food diet have been shown to enjoy improvements in symptoms of autoimmune inflammatory disease including pain, joint stiffness, quality of sleep, health quality, cholesterol, and weight management.[220,221]

Another consideration is the health benefits of omega-3 fatty acids. Omega-3 fatty acids are associated with decreased inflammation, improvement in disease symptoms and reduced risk of acquiring autoimmune inflammatory disease.[222,223] Good sources of omega-3 fatty acids are a vegetarian diet, olives, and flax seed. Olives and olive oil, with their high levels of antioxidants, omega-3 fatty acids and phytochemicals, have been found to be helpful in the prevention and treatment of autoimmune inflammatory disorders.[224,225,226,227] The most preferable way to obtain the olive oil is from the eating of whole olives. Results are not immediate but usually felt within 12 weeks.[228]

Are you a python? Do you swallow your food whole?

Another single food we want to mention is lemon juice and citrus. Citrus contains many bioflavonoids, phytochemicals, and antioxidants that have been found to reduce inflammation[229,230,231] and improve symptoms of autoimmune inflammatory disease.[232,233,234]

We mentioned that too much protein has deleterious effects for the would-be autoimmune inflammatory disease survivor. But not all proteins are created equal. Soy protein reduces the risk of autoimmune inflammatory disease by 60% compared to a diet high in animal protein.[235,236]

CHEW YOUR FOOD

Are you drinking juices or slurping smoothies? Are you a python? Do you swallow your food whole? A better method is to chew your food well and savor every bite. The immune tissue in the mouth and throat (tonsils), tests substances coming into the body to let the body know what is food.[237,238,239,240,241] Allergy and autoimmune inflammatory diseases are more likely to flare up when food is not chewed long and well, when the body has not had a chance to recognize the antigens.[242,243]

TEMPERANCE: ABSTINENCE FROM THINGS HARMFUL, MODERATION IN THINGS GOOD

A program designed to benefit patients with autoimmune inflammatory disease will most surely include methods for eliminating the use of such stimulants as tea, coffee, caffeine, tobacco, and alcohol.

Another aspect of temperance is eating moderate amounts of food. When more calories are consumed than are needed, inflammation increases. On the other hand, reducing calorie intake reduces the body's inflammatory responses.[244,245] The goal is to match caloric intake to body energy needs.

Taken a step further, fasting is a quick way to get an energy imbalance under control.[246] Fasting has been found to reduce oxidative stress and inflammation, and improve symptoms of autoimmune inflammatory disease.[247,248]

The outcome of caloric restriction could have another desirable result for some – that being weight loss. Being overweight is associated with increased risk of autoimmune inflammatory disease. Weight loss is associated with a decrease in oxidative damage to lipids (fats) and proteins and decreased inflammation.[249,250,251]

WATER CONSUMPTION: I'LL DRINK TO THAT

Studies show that optimal water intake can lower the risk of autoimmune inflammatory diseases by as much as 60%.[100] On the other

hand, dehydration (being low on water) enhances the inflammatory response of the body to hostile antigens.[252] We recommend 8-12 eight ounce glasses per day.

What water should I drink? We have a saying; "friends don't let friends drink tap water." Contaminated water is also a source of inflammation.[253,254] Water should be obtained which is pure and free from all substances, which can potentially induce inflammation. Depending on your water condition, filtering, distilling or other treatment may be necessary.

Drinking it is not the only beneficial use of water. Many people have discovered the benefits of hydrotherapy, the use of water for treatment of disease and maintenance of health. Acute inflammatory pain can be treated with either superficial heat for reducing guarding (fear and tensing) or with cold for reducing pain.[255] The application of heat or cold to inflamed joints tends to improve pain, joint stiffness, and joint function. The application of cold tends to raise the pain threshold.[256] Contrast (alternating hot and cold) hydrotherapy tends to improve circulation, greatly lowering inflammation.[257,258]

AVOIDING THE OBVIOUS

The avoidance of heavy metal exposure is key to reducing the risk of and/or symptoms of autoimmune inflammatory disease. If a high level of exposure has been experienced in the past, it may be necessary to take steps to eliminate these toxins from the body.

VACCINATION BURNOUT

Repeated over-stimulation of the immune system, as in immunizations, can result in immune fatigue and burnout resulting in increased risk of autoimmune inflammatory disease.

SHOULD I DO A CLEANSE?

When toxin accumulation is the cause of immune dysfunction, toxin elimination may be the only way to get the immune system back in balance. Sweating, something we do not like to do in this day and age, may be just what is needed to expel the aggravating toxins.[259] Skin brushing (exfoliation) can also be a part of this elimination process. Chelation is another effective way to get rid of especially heavy metals.[260] This can often be accomplished with dietary modifications. Sometimes a diet totally devoid for a while of the toxin to be expelled can accomplish the same goal.[261] Some have even found a colon cleanse beneficial.[262] I knew of a patient who was successful at managing their inflammatory arthritis if they did a colon cleanse once a quarter (which made me wonder what they were doing the rest of the time to pollute their colon again).

Another useful modality for the removal of toxins and inflammation is charcoal. Charcoal can be used for inflammation as an oral supplement,[263] and as a topical treatment.[264,265] Studies show that it is effective in reducing the symptoms and signs of autoimmune inflammatory disease.[266]

MIND BODY CONNECTION

Trust in Divine power: Spirituality is associated with less depression and increased feelings of health in patients with autoimmune inflammatory disease.[267,268] Studies reveal that religious intervention such as intercessory prayer increases immune function, improves rheumatoid arthritis, and reduces anxiety.[269] In a study of autoimmune inflammatory patients, six hours of one-on-one intercessory prayer was associated with significant overall improvement in disease that lasted the entire subsequent year of the study's duration.[270]

Given the relationship between stress and autoimmune inflammatory disease, stress reduction should be a priority with autoimmune disease sufferers.[271,272] The dietary changes we advocate have also been found to reduce the psychological symptoms of stress.[273] Improved spiritual health has been shown to be a valuable aid in stress management.[274] Has not God said; "Come unto me, all ye that labor and are heavy laden, and I will give you rest."[275] He is the great burden bearer; trusting in Him alleviates stress.

IN SUMMARY

As you engage in an autoimmune inflammatory disease recovery program you will find it helpful to *eliminate* all:

- Animal products including dairy and eggs.
- Possibly wheat gluten.
- Oxidized oils or cholesterol.
- Refined foods; sugars, starches, grains, and oils.
- Excess dietary calories.
- Foods or drinks created by aging or fermentation.
- Stimulants; coffee, tea, tobacco and alcohol.
- Strong irritating spices.
- Excess body weight.
- Tight clothing and clothing that does not provide adequate and evenly distributed warmth.
- Excessive meals (fasting may be helpful).

As you engage in an autoimmune inflammatory disease recovery program you will find it helpful to:

- Have a regular schedule throughout the day for sleep, meals and exercise.
- Eat a whole plant food diet with plenty of fresh fruits and vegetables, omega-3s and fiber.
- Chew your food thoroughly and swish it around your mouth.
- Make use of pure water: drink plenty, bathe often, and perform hot and cold treatments.
- Make wise application of charcoal as poultices and taken by mouth.

And what dietary program are we really talking about? The original Bible diet! Then God said, "I give you every seed-bearing plant on the face of the whole earth and every tree that has fruit with seed in it. They will be yours for food." "…and you will eat the plants of the field."[276] Should it be any surprise that the Maker of this marvellous immune system, which is designed to protect this marvellous body we have been given, should have the best lifestyle prescription necessary for its upkeep?

For further ideas on how to incorporate what you have just learned into your daily life, see the chapter entitled, "How Can I Apply Healthy Principles in My Daily Life".

---

[1] http://www.pbs.org/wgbh/amex/bubble/gallery/g_05.html
[2] Psalm 139:14, King James Version of the Holy Bible.
[3] http://www.amazingfacts.org/Publications/InsideReport/tabid/123/newsid457/206/Dismembered-Avoiding-an-Out-of-Body-Experience/Default.aspx
[4] Arkwright PD, Abinun M, Cant AJ. Autoimmunity in human primary immunodeficiency diseases. Blood. 2002 Apr 15;99(8):2694-702.
[5] Prelog M. Aging of the immune system: a risk factor for autoimmunity? Autoimmun Rev. 2006 Feb;5(2):136-9.
[6] von Känel R, Bellingrath S, Kudielka BM. Association between burnout and circulating levels of pro- and anti-inflammatory cytokines in schoolteachers. J Psychosom Res. 2008 Jul;65(1):51-9.
[7] Cénac A, Sparfel A, Amiel-Lebigre F, Cleuziou A, Pennec Y, Le Goff P, Mottier D. Effect of stressful life events on clinical development of temporal arteritis and/or polymyalgia rheumatica. Presse Med. 2002 Jun 1;31(19):873-9.
[8] Altindag O, Karakoc M, Kocyigit A, Celik H, Soran N. Increased DNA damage and oxidative stress in patients with rheumatoid arthritis. Clin Biochem. 2007 Feb;40(3-4):167-71.
[9] Valentino M, Rapisarda V, Santarelli L, Bracci M, Scorcelletti M, Di Lorenzo L, Cassano F, Soleo L. Effect of lead on the levels of some immunoregulatory cytokines in occupationally exposed workers. Hum Exp Toxicol. 2007 Jul;26(7):551-6.
[10] Kusaka Y. Occupational diseases caused by exposure to sensitizing metals. Sangyo Igaku. 1993 Mar;35(2):75-87.
[11] Dong W, Simeonova PP, Gallucci R, Matheson J, Flood L, Wang S, Hubbs A, Luster MI. Toxic metals stimulate inflammatory cytokines in hepatocytes through oxidative stress mechanisms. Toxicol Appl Pharmacol. 1998 Aug;151(2):359-66.
[12] Ilbäck NG, Wesslén L, Fohlman J, Friman G. Effects of methyl mercury on cytokines, inflammation and virus clearance in a common infection (coxsackie B3 myocarditis). Toxicol Lett. 1996 Dec;89(1):19-28.
[13] Cushman M, Meilahn EN, Psaty BM, Kuller LH, Dobs AS, Tracy RP. Hormone replacement therapy, inflammation, and hemostasis in elderly women. Arterioscler Thromb Vasc Biol. 1999 Apr;19(4):893-9.
[14] Sanchez-Guerrero J, Karlson EW, Liang MH, Hunter DJ, Speizer FE, Colditz GA. Past use of oral contraceptives and the risk of developing systemic lupus erythematosus. Arthritis Rheum. 1997 May;40(5):804-8.
[15] Sánchez-Guerrero J, Liang MH, Karlson EW, Hunter DJ, Colditz GA. Postmenopausal estrogen therapy and the risk for developing systemic lupus erythematosus. Ann Intern Med. 1995 Mar 15;122(6):430-3.
[16] Röder-Stolinski C, Fischäder G, Oostingh GJ, Feltens R, Kohse F, von Bergen M, Mörbt N, Eder K, Duschl A, Lehmann I. Styrene induces an inflammatory response in human lung epithelial cells via oxidative stress and NF-kappaB activation. Toxicol Appl Pharmacol. Epub 2008 Apr 29.
[17] Sverdrup B, Källberg H, Bengtsson C, Lundberg I, Padyukov L, Alfredsson L, Klareskog L; Epidemiological Investigation of Rheumatoid Arthritis Study Group. Association between occupational exposure to mineral oil and rheumatoid arthritis: results from the Swedish EIRA case-control study. Arthritis Res Ther. 2005;7(6):R1296-303. Epub 2005 Sep 23.
[18] Vojdani A, Ghoneum M, Brautbar N. Immune alteration associated with exposure to toxic chemicals. Toxicol Ind Health. 1992 Sep-Oct;8(5):239-54.
[19] Reckner Olsson A, Skogh T, Wingren G. Comorbidity and lifestyle, reproductive factors, and environmental exposures associated with rheumatoid arthritis. Ann Rheum Dis. 2001 Oct;60(10):934-9.
[20] Fluhr JW, Kelterer D, Fuchs S, Kaatz M, Grieshaber R, Kleesz P, Elsner P. Additive impairment of the barrier function and irritation by biogenic amines and sodium lauryl sulphate: a controlled in vivo tandem irritation study. Skin Pharmacol Physiol. 2005 Mar-Apr;18(2):88-97.
[21] Schilderman PA, ten Vaarwerk FJ, Lutgerink JT, Van der Wurff A, ten Hoor F, Kleinjans JC. Induction of oxidative DNA damage and early lesions in rat gastro-intestinal epithelium in relation to prostaglandin H synthase-mediated metabolism of butylated hydroxyanisole. Food Chem Toxicol. 1995 Feb;33(2):99-109.
[22] Traunmüller F. Etiology of Crohn's disease: Do certain food additives cause intestinal inflammation by molecular mimicry of mycobacterial lipids? Med Hypotheses. 2005;65(5):859-64.
[23] Phukan RK, Narain K, Zomawia E, Hazarika NC, Mahanta J. Dietary habits and stomach cancer in Mizoram, India. J Gastroenterol. 2006 May;41(5):418-24.
[24] Draelos ZD. The effect of a daily facial cleanser for normal to oily skin on the skin barrier of subjects with acne. Cutis. 2006 Jul;78(1 Suppl):34-40.
[25] Hinton DM, Myers MJ, Raybourne RA, Francke-Carroll S, Sotomayor RE, Shaddock J, Warbritton A, Chou MW. Immunotoxicity of aflatoxin B1 in rats: effects on lymphocytes and the inflammatory response in a chronic intermittent dosing study. Toxicol Sci. 2003 Jun;73(2):362-77.
[26] Roy RN, Russell RI. Crohn's disease & aflatoxins. J R Soc Health. 1992 Dec;112(6):277-9.
[27] A case-control study of ulcerative colitis in relation to dietary and other factors in Japan. The Epidemiology Group of the Research Committee of Inflammatory Bowel Disease in Japan. J Gastroenterol. 1995 Nov;30 Suppl 8:9-12.
[28] Lavy A, Naveh Y, Coleman R, Mokady S, Werman MJ. Dietary Dunaliella bardawil, a beta-carotene-rich alga, protects against acetic acid-induced small bowel inflammation in rats. Inflamm Bowel Dis. 2003 Nov;9(6):372-9.
[29] Slaga TJ, Bowden GT, Boutwell RK. Acetic acid, a potent stimulator of mouse epidermal macromolecular synthesis and hyperplasia but with weak tumor-promoting ability. J Natl Cancer Inst. 1975 Oct;55(4):983-7.
[30] Cleary K, McFeeters RF. Effects of oxygen and turmeric on the formation of oxidative aldehydes in fresh-pack dill pickles. J Agric Food Chem. 2006 May 3;54(9):3421-7.

[31] Lynch MP, Faustman C. Effect of aldehyde lipid oxidation products on myoglobin. J Agric Food Chem. 2000 Mar;48(3):600-4.
[32] MacDonald WC, Anderson FH, Hashimoto S. Histological effect of certain pickles on the human gastric mucosa. A preliminary report. Can Med Assoc J. 1967 Jun 10;96(23):1521-5.
[33] Kono S, Hirohata T. A review of gastric cancer and life style. Gan No Rinsho. 1990 Feb;Spec No:257-67.
[34] Kuwahara Y, Kondoh J, Tatara K, Azuma E, Nakajima T, Hashimoto M, Komachi Y. Involvement of urban living environments in atopy and enhanced eosinophil activity: potential risk factors of airway allergic symptoms. Allergy. 2001 Mar;56(3):224-30.
[35] Reckner Olsson A, Skogh T, Wingren G. Comorbidity and lifestyle, reproductive factors, and environmental exposures associated with rheumatoid arthritis. Ann Rheum Dis. 2001 Oct;60(10):934-9.
[36] Gray MR, Thrasher JD, Crago R, Madison RA, Arnold L, Campbell AW, Vojdani A. Mixed mold mycotoxicosis: immunological changes in humans following exposure in water-damaged buildings. Arch Environ Health. 2003 Jul;58(7):410-20.
[37] Jussila J, Komulainen H, Kosma VM, Nevalainen A, Pelkonen J, Hirvonen MR. Spores of Aspergillus versicolor isolated from indoor air of a moisture-damaged building provoke acute inflammation in mouse lungs. Inhal Toxicol. 2002 Dec;14(12):1261-77.
[38] Jussila J, Komulainen H, Huttunen K, Roponen M, Iivanainen E, Torkko P, Kosma VM, Pelkonen J, Hirvonen MR. Mycobacterium terrae isolated from indoor air of a moisture-damaged building induces sustained biphasic inflammatory response in mouse lungs. Environ Health Perspect. 2002 Nov;110(11):1119-25.
[39] Jussila J, Komulainen H, Huttunen K, Roponen M, Hälinen A, Hyvärinen A, Kosma VM, Pelkonen J, Hirvonen MR. Inflammatory responses in mice after intratracheal instillation of spores of Streptomyces californicus isolated from indoor air of a moldy building. Toxicol Appl Pharmacol. 2001 Feb 15;171(1):61-9.
[40] Marsh PB, Millner PD, Kla JM. A guide to the recent literature on aspergillosis as caused by Aspergillus fumigatus, a fungus frequently found in self-heating organic matter. Mycopathologia. 1979 Nov 30;69(1-2):67-81.
[41] Mullins J, Harvey R, Seaton A. Sources and incidence of airborne Aspergillus fumigatus (Fres). Clin Allergy. 1976 May;6(3):209-17.
[42] Signorelli SS, Malaponte MG, Di Pino L, Costa MP, Pennisi G, Mazzarino MC. Venous stasis causes release of interleukin 1beta (IL-1beta), interleukin 6 (IL-6) and tumor necrosis factor alpha (TNFalpha) by monocyte-macrophage. Clin Hemorheol Microcirc. 2000;22(4):311-6.
[43] Tsujii M, Kawano S, Tsuji S, Kobayashi I, Takei Y, Nagano K, Fusamoto H, Kamada T, Ogihara T, Sato N. Colonic mucosal hemodynamics and tissue oxygenation in patients with ulcerative colitis: investigation by organ reflectance spectrophotometry. J Gastroenterol. 1995 Apr;30(2):183-8.Links
[44] Keli E, Bouchoucha M, Devroede G, Carnot F, Ohrant T, Cugnenc PH. Diversion-related experimental colitis in rats. Dis Colon Rectum. 1997 Feb;40(2):222-8.
[45] Scholbach T. From the nutcracker-phenomenon of the left renal vein to the midline congestion syndrome as a cause of migraine, headache, back and abdominal pain and functional disorders of pelvic organs. Med Hypotheses. 2007;68(6):1318-27.
[46] Ma XJ, Yin HJ, Chen KJ. Research progress of correlation between blood-stasis syndrome and inflammation. Zhongguo Zhong Xi Yi Jie He Za Zhi. 2007 Jul;27(7):669-72.
[47] Park SJ, Tokura H. Effects of different types of clothing on circadian rhythms of core temperature and urinary catecholamines. Jpn J Physiol. 1998 Apr;48(2):149-56.
[48] Bøkenes L, Alexandersen TE, Østerud B, Tveita T, Mercer JB. Physiological and haematological responses to cold exposure in the elderly. Int J Circumpolar Health. 2000 Oct;59(3-4):216-21.
[49] Mercer JB, Osterud B, Tveita T. The effect of short-term cold exposure on risk factors for cardiovascular disease. Thromb Res. 1999 Jul 15;95(2):93-104.
[50] Mori Y, Kioka E, Tokura H. Effects of pressure on the skin exerted by clothing on responses of urinary catecholamines and cortisol, heart rate and nocturnal urinary melatonin in humans. Int J Biometeorol. 2002 Dec;47(1):1-5.
[51] Okura K, Midorikawa-Tsurutani T, Tokura H. Effects of skin pressure applied by cuffs on resting salivary secretion. J Physiol Anthropol Appl Human Sci. 2000 Mar;19(2):107-11.
[52] Vikhert AM, Zhdanov VS, Lifshits AM. Arteriosclerosis in men doing physical and mental work. Kardiologiia. 1976 Mar;16(3):119-23.
[53] Irwin MR, Wang M, Ribeiro D, Cho HJ, Olmstead R, Breen EC, Martinez-Maza O, Cole S. Sleep Loss Activates Cellular Inflammatory Signaling. Biol Psychiatry. Epub 2008 Jun 16.
[54] Simpson N, Dinges DF. Sleep and inflammation. Nutr Rev. 2007 Dec;65(12 Pt 2):S244-52.
[55] Irwin MR, Wang M, Campomayor CO, Collado-Hidalgo A, Cole S. Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. Arch Intern Med. 2006 Sep 18;166(16):1756-62.
[56] Palma BD, Suchecki D, Catallani B, Tufik S. Effect of sleep deprivation on the corticosterone secretion in an experimental model of autoimmune disease. Neuroimmunomodulation. 2007;14(2):72-7.
[57] Norbäck D, Wålinder R, Wieslander G, Smedje G, Erwall C, Venge P. Indoor air pollutants in schools: nasal patency and biomarkers in nasal lavage. Allergy. 2000 Feb;55(2):163-70.
[58] J Voice. 1997 Jun;11(2):165-70. The singing/acting mature adult--singing instruction perspective. Westerman Gregg J.
[59] Anikeeva ZI, Pleshkov IV, Bondareva AV. Clinical features of vocal disorders in population of megapolis. Vestn Otorinolaringol. 2007;(1):14-21.
[60] Vassilakopoulos T, Divangahi M, Rallis G, Kishta O, Petrof B, Comtois A, Hussain SN. Differential cytokine gene expression in the diaphragm in response to strenuous resistive breathing. Am J Respir Crit Care Med. 2004 Jul 15;170(2):154-61.
[61] Vassilakopoulos T, Katsaounou P, Karatza MH, Kollintza A, Zakynthinos S, Roussos C. Strenuous resistive breathing induces plasma cytokines: role of antioxidants and monocytes. Am J Respir Crit Care Med. 2002 Dec 15;166(12 Pt 1):1572-8.
[62] Barbe MF, Barr AE. Inflammation and the pathophysiology of work-related musculoskeletal disorders. Brain Behav Immun. 2006 Sep;20(5):423-9.
[63] Kivi P. Rheumatic disorders of the upper limbs associated with repetitive occupational tasks in Finland in 1975-1979. Scand J Rheumatol. 1984;13(2):101-7.
[64] Carp SJ, Barbe MF, Winter KA, Amin M, Barr AE. Inflammatory biomarkers increase with severity of upper-extremity overuse disorders. Clin Sci (Lond). 2007 Mar;112(5):305-14.
[65] Clin Sci (Lond). 2007 Mar;112(5):305-14. Inflammatory biomarkers increase with severity of upper-extremity overuse disorders. Carp SJ, Barbe MF, Winter KA, Amin M, Barr AE.
[66] Lenda DM, Boegehold MA. Effect of a high salt diet on microvascular antioxidant enzymes. J Vasc Res. 2002 Jan-Feb;39(1):41-50.
[67] Chandramohan G, Bai Y, Norris K, Rodriguez-Iturbe B, Vaziri ND. Effects of dietary salt on intrarenal angiotensin system, NAD(P)H oxidase, COX-2, MCP-1 and PAI-1 expressions and NF-kappaB activity in salt-sensitive and -resistant rat kidneys. Am J Nephrol. 2008;28(1):158-67.
[68] Myers BM, Smith JL, Graham DY. Effect of red pepper and black pepper on the stomach. Am J Gastroenterol. 1987 Mar;82(3):211-4.
[69] Vasudevan K, Vembar S, Veeraraghavan K, Haranath PS. Influence of intragastric perfusion of aqueous spice extracts on acid secretion in anesthetized albino rats. Indian J Gastroenterol. 2000 Apr-Jun;19(2):53-6.
[70] Biochem J. 1990 Jul 1;269(1):41-6. Induction of C-reactive protein by cytokines in human hepatoma cell lines is potentiated by caffeine. Ganapathi MK, Mackiewicz A, Samols D, Brabenec A, Kushner I, Schultz D, Hu SI.
[71] Russel MG, Engels LG, Muris JW, Limonard CB, Volovics A, Brummer RJ, Stockbrügger RW. Modern life' in the epidemiology of inflammatory bowel disease: a case-control study with special emphasis on nutritional factors. Eur J Gastroenterol Hepatol. 1998 Mar;10(3):243-9.
[72] Pedersen M, Jacobsen S, Klarlund M, Pedersen BV, Wiik A, Wohlfahrt J, Frisch M. Arthritis Res Ther. 2006;8(4):R133. Environmental risk factors differ between rheumatoid arthritis with and without auto-antibodies against cyclic citrullinated peptides.
[73] Nordmann R. Alcohol and antioxidant systems. Alcohol Alcohol. 1994 Sep;29(5):513-22.
[74] Vally H, de Klerk N, Thompson PJ. Alcoholic drinks: important triggers for asthma. J Allergy Clin Immunol. 2000 Mar;105(3):462-7.
[75] Zhang J, Liu Y, Shi J, Larson DF, Watson RR. Side-stream cigarette smoke induces dose-response in systemic inflammatory cytokine production and oxidative stress. Exp Biol Med (Maywood). 2002 Oct;227(9):823-9.
[76] Oliver JE, Silman AJ. Risk factors for the development of rheumatoid arthritis. Scand J Rheumatol. 2006 May-Jun;35(3):169-74.
[77] Costenbader KH, Feskanich D, Mandl LA, Karlson EW. Smoking intensity, duration, and cessation, and the risk of rheumatoid arthritis in women. Am J Med. 2006 Jun;119(6):503.e1-9.
[78] Pedersen M, Jacobsen S, Klarlund M, Pedersen BV, Wiik A, Wohlfahrt J, Frisch M. Environmental risk factors differ between rheumatoid arthritis with and without auto-antibodies against cyclic citrullinated peptides. Arthritis Res Ther. 2006;8(4):R133.
[79] Nettleton JA, Steffen LM, Mayer-Davis EJ, Jenny NS, Jiang R, Herrington DM, Jacobs DR Jr. Dietary patterns are associated with biochemical markers of inflammation and endothelial activation in the Multi-Ethnic Study of Atherosclerosis (MESA). Am J Clin Nutr. 2006 Jun;83(6):1369-79.
[80] Schulze MB, Hoffmann K, Manson JE, Willett WC, Meigs JB, Weikert C, Heidemann C, Colditz GA, Hu FB. Dietary pattern, inflammation, and incidence of type 2 diabetes in women. Am J Clin Nutr. 2005 Sep;82(3):675-84.
[81] Tola MR, Granieri E, Malagù S, Caniatti L, Casetta I, Govoni V, Paolino E, Cinzia Monetti V, Canducci E, Panatta GB. Dietary habits and multiple sclerosis. A retrospective study in Ferrara, Italy. Acta Neurol (Napoli). 1994 Aug;16(4):189-97.
[82] Ghadirian P, Jain M, Ducic S, Shatenstein B, Morisset R. Nutritional factors in the aetiology of multiple sclerosis: a case-control study in Montreal, Canada. Int J Epidemiol. 1998 Oct;27(5):845-52.
[83] Kacsur C, Mader R, Ben-Amotz A, Levy Y. Plasma anti-oxidants and rheumatoid arthritis. Harefuah. 2002 Feb;141(2):148-50, 223.
[84] Kamanli A, Naziroğlu M, Aydilek N, Hacievliyagil C. Plasma lipid peroxidation and antioxidant levels in patients with rheumatoid arthritis. Cell Biochem Funct. 2004 Jan-Feb;22(1):53-7.
[85] Dunstan JA, Breckler L, Hale J, Lehmann H, Franklin P, Lyons G, Ching SY, Mori TA, Barden A, Prescott SL. Supplementation with vitamins C, E, beta-carotene and selenium has no effect on anti-oxidant status and immune responses in allergic adults: a randomized controlled trial. Clin Exp Allergy. 2007 Feb;37(2):180-7.
[86] Bae SC, Kim SJ, Sung MK. Inadequate antioxidant nutrient intake and altered plasma antioxidant status of rheumatoid arthritis patients. J Am Coll Nutr. 2003 Aug;22(4):311-5.
[87] Bo S, Durazzo M, Guidi S, Carello M, Sacerdote C, Silli B, Rosato R, Cassader M, Gentile L, Pagano G. Dietary magnesium and fiber intakes and inflammatory and metabolic indicators in middle-aged subjects from a population-based cohort. Am J Clin Nutr. 2006 Nov;84(5):1062-9.
[88] Thomas D. The mineral depletion of foods available to us as a nation (1940-2002)--a review of the 6th Edition of McCance and Widdowson. Nutr Health. 2007;19(1-2):21-55.
[89] Frigo A, Tambalo C, Bambara LM, Biasi D, Marrella M, Milanino R, Moretti U, Velo G, De Sandre G. Zinc sulfate in the treatment of psoriatic arthritis. Recenti Prog Med. 1989 Nov;80(11):577-81.
[90] Vunta H, Belda BJ, Arner RJ, Channa Reddy C, Vanden Heuvel JP, Sandeep Prabhu K. Selenium attenuates pro-inflammatory gene expression in macrophages. Mol Nutr Food Res. Epub 2008 May 15.
[91] Almoznino-Sarafian D, Berman S, Mor A, Shteinshnaider M, Gorelik O, Tzur I, Alon I, Modai D, Cohen N. Magnesium and C-reactive protein in heart failure: an anti-inflammatory effect of magnesium administration? Eur J Nutr. 2007 Jun;46(4):230-7. Epub 2007 May 3.
[92] Composition of Foods Raw, Processed, Prepared USDA National Nutrient Database for Standard Reference, Release 18, August 2005. U.S. Department of Agriculture Agricultural Research Service, Beltsville Human Nutrition Research Center, Nutrient Data Laboratory, 10300 Baltimore Avenue, Building 005, Room 107, BARC-West, Beltsville, Maryland 20705.
[93] Uribarri J, Cai W, Sandu O, Peppa M, Goldberg T, Vlassara H. Diet-derived advanced glycation end products are major contributors to the body's AGE pool and induce inflammation in healthy subjects. Ann N Y Acad Sci. 2005 Jun;1043:461-6.
[94] Kislinger T, Tanji N, Wendt T, Qu W, Lu Y, Ferran LJ Jr, Taguchi A, Olson K, Bucciarelli L, Goova M, Hofmann MA, Cataldegirmen G, D'Agati V, Pischetsrieder M, Stern DM, Schmidt AM. Receptor for advanced glycation end products mediates inflammation and enhanced expression of tissue factor in vasculature of diabetic apolipoprotein E-null mice. Arterioscler Thromb Vasc Biol. 2001 Jun;21(6):905-10.
[95] Goldberg T, Cai W, Peppa M, Dardaine V, Baliga BS, Uribarri J, Vlassara H. Advanced glycoxidation end products in commonly consumed foods. J Am Diet Assoc. 2004 Aug;104(8):1287-91.
[96] Wautier JL, Guillausseau PJ. Advanced glycation end products, their receptors and diabetic angiopathy. Diabetes Metab. 2001 Nov;27(5 Pt 1):535-42.
[97] Kelley GL, Allan G, Azhar S. High dietary fructose induces a hepatic stress response resulting in cholesterol and lipid dysregulation. Endocrinology. 2004 Feb;145(2):548-55.

[98] Nyby MD, Abedi K, Smutko V, Eslami P, Tuck ML. Vascular Angiotensin type 1 receptor expression is associated with vascular dysfunction, oxidative stress and inflammation in fructose-fed rats. Hypertens Res. 2007 May;30(5):451-7.
[99] Glushakova O, Kosugi T, Roncal C, Mu W, Heinig M, Cirillo P, Sánchez-Lozada LG, Johnson RJ, Nakagawa T. Fructose Induces the Inflammatory Molecule ICAM-1 in Endothelial Cells. J Am Soc Nephrol. 2008 Sep;19(9):1712-20.
[100] Reif S, Klein I, Lubin F, Farbstein M, Hallak A, Gilat T. Pre-illness dietary factors in inflammatory bowel disease. Gut. 1997 Jun;40(6):754-60.
[101] Yeh YF, Huang SL. Enhancing effect of dietary cholesterol and inhibitory effect of pravastatin on allergic pulmonary inflammation. J Biomed Sci. 2004 Sep-Oct;11(5):599-606.
[102] Li Y, Schwabe RF, DeVries-Seimon T, Yao PM, Gerbod-Giannone MC, Tall AR, Davis RJ, Flavell R, Brenner DA, Tabas I. Free cholesterol-loaded macrophages are an abundant source of tumor necrosis factor-alpha and interleukin-6: model of NF-kappaB- and map kinase-dependent inflammation in advanced atherosclerosis. J Biol Chem. 2005 Jun 10;280(23):21763-72.
[103] Shi Q, Vandeberg JF, Jett C, Rice K, Leland MM, Talley L, Kushwaha RS, Rainwater DL, Vandeberg JL, Wang XL. Arterial endothelial dysfunction in baboons fed a high-cholesterol, high-fat diet. Am J Clin Nutr. 2005 Oct;82(4):751-9.
[104] Shamberger RJ, Shamberger BA, Willis CE. Malonaldehyde content of food. J Nutr. 1977 Aug;107(8):1404-9.
[105] Parke AL, Hughes GR. Rheumatoid arthritis and food: a case study. Br Med J (Clin Res Ed). 1981 Jun 20;282(6281):2027-9.
[106] Fujiyama Y, Hokari R, Miura S, Watanabe C, Komoto S, Oyama T, Kurihara C, Nagata H, Hibi T. Butter feeding enhances TNF-alpha production from macrophages and lymphocyte adherence in murine small intestinal microvessels. J Gastroenterol Hepatol. 2007 Nov;22(11):1838-45.Click here to read Links
[107] Trapali M, Liapi C, Perelas A, Perrea D, Stroubini T, Dontas I, Couvari E, Mavri M, Galanopoulou P. Effect of isocaloric diets and sibutramine on food intake, body mass variation and serum TNF-alpha levels in rats. Pharmacology. 2008;82(1):15-21.
[108] Håversen L, Danielsson KN, Fogelstrand L, Wiklund O. Induction of proinflammatory cytokines by long-chain saturated fatty acids in human macrophages. Atherosclerosis. Epub 2008 May 28.
[109] Perez-Martinez P, Lopez-Miranda J, Blanco-Colio L, Bellido C, Jimenez Y, Moreno JA, Delgado-Lista J, Egido J, Perez-Jimenez F. The chronic intake of a Mediterranean diet enriched in virgin olive oil, decreases nuclear transcription factor kappaB activation in peripheral blood mononuclear cells from healthy men. Atherosclerosis. 2007 Oct;194(2):e141-6
[110] Lin BF, Huang CH, Chiang BL, Jeng SJ. Dietary fat influences Ia antigen expression, cytokines and prostaglandin E2 production of immune cells in autoimmune-prone NZB x NZW F1 mice. Br J Nutr. 1996 May;75(5):711-22.
[111] Lin BF, Lai CC, Lin KW, Chiang BL. Dietary oxidized oil influences the levels of type 2 T-helper cell-related antibody and inflammatory mediators in mice. Br J Nutr. 2000 Dec;84(6):911-7.
[112] Kanner J. Dietary advanced lipid oxidation endproducts are risk factors to human health. Mol Nutr Food Res. 2007 Sep;51(9):1094-101.
[113] Martin CA, Milinsk MC, Visentainer JV, Matsushita M, de-Souza NE. Trans fatty acid-forming processes in foods: a review. An Acad Bras Cienc. 2007 Jun;79(2):343-50.
[114] Naruszewicz M, Daniewski M, Nowicka G, Kozłowska-Wojciechowska M. Trans-unsaturated fatty acids and acrylamide in food as potential atherosclerosis progression factors. Based on own studies. Acta Microbiol Pol. 2003;52 Suppl:75-81.
[115] Viana M, Villacorta L, Bonet B, Indart A, Munteanu A, Sánchez-Vera I, Azzi A, Zingg JM. Effects of aldehydes on CD36 expression. Free Radic Res. 2005 Sep;39(9):973-7.
[116] Sutherland WH, Walker RJ, de Jong SA, van Rij AM, Phillips V, Walker HL. Reduced postprandial serum paraoxonase activity after a meal rich in used cooking fat. Arterioscler Thromb Vasc Biol. 1999 May;19(5):1340-7.
[117] Lopez-Garcia E, Schulze MB, Meigs JB, Manson JE, Rifai N, Stampfer MJ, Willett WC, Hu FB. Consumption of trans fatty acids is related to plasma biomarkers of inflammation and endothelial dysfunction. J Nutr. 2005 Mar;135(3):562-6.
[118] Baranowski A, Adams CW, High OB, Bowyer DB. Connective tissue responses to oxysterols. Atherosclerosis. 1982 Feb;41(2-3):255-66.
[119] Fischer KH, Laskawy G, Grosch W. Quantitative analysis of auto-oxidation products of cholesterol in food of animal origin. Z Lebensm Unters Forsch. 1985 Jul;181(1):14-9.
[120] Ginaldi L, De Martinis M, Monti D, Franceschi C. Chronic antigenic load and apoptosis in immunosenescence. Trends Immunol. 2005 Feb;26(2):79-84.
[121] Dickinson S, Hancock DP, Petocz P, Ceriello A, Brand-Miller J. High-glycemic index carbohydrate increases nuclear factor-kappaB activation in mononuclear cells of young, lean healthy subjects. Am J Clin Nutr. 2008 May;87(5):1188-93.
[122] Mantzoros CS, Li T, Manson JE, Meigs JB, Hu FB. Circulating adiponectin levels are associated with better glycemic control, more favorable lipid profile, and reduced inflammation in women with type 2 diabetes. J Clin Endocrinol Metab. 2005 Aug;90(8):4542-8.
[123] Busserolles J, Rock E, Gueux E, Mazur A, Grolier P, Rayssiguier Y. Short-term consumption of a high-sucrose diet has a pro-oxidant effect in rats. Br J Nutr. 2002 Apr;87(4):337-42.
[124] Mohanty P, Hamouda W, Garg R, Aljada A, Ghanim H, Dandona P. Glucose challenge stimulates reactive oxygen species (ROS) generation by leucocytes. J Clin Endocrinol Metab. 2000 Aug;85(8):2970-3.
[125] Garrett SL, Kennedy LG, Calin A. Patients' perceptions of disease modulation by diet in inflammatory (rheumatoid arthritis/ankylosing spondylitis) and degenerative rthropathies. Br J Rheumatol 1993;32(suppl. 2):43.
[126] Nilsson AC, Ostman EM, Holst JJ, Björck IM. Including indigestible carbohydrates in the evening meal of healthy subjects improves glucose tolerance, lowers inflammatory markers, and increases satiety after a subsequent standardized breakfast. J Nutr. 2008 Apr;138(4):732-9.

[127] Hvatum M, Kanerud L, Hällgren R, Brandtzaeg P. The gut-joint axis: cross reactive food antibodies in rheumatoid arthritis. Gut. 2006 Sep;55(9):1240-7.
[128] Malosse D, Perron H, Sasco A, Seigneurin JM. Correlation between milk and dairy product consumption and multiple sclerosis prevalence: a worldwide study. Neuroepidemiology. 1992;11(4-6):304-12.
[129] Sepcić J, Mesaros E, Materljan E, Sepić-Grahovac D. Nutritional factors and multiple sclerosis in Gorski Kotar, Croatia. Neuroepidemiology. 1993;12(4):234-40.
[130] Stoeck M, Ruegg C, Miescher S, Carrel S, Cox D, Von Fliedner V, Alkan S. Comparison of the immunosuppressive properties of milk growth factor and transforming growth factors beta 1 and beta 2. J Immunol. 1989 Nov 15;143(10):3258-65.
[131] Epstein SS. Unlabeled milk from cows treated with biosynthetic growth hormones: a case of regulatory abdication. Int J Health Serv. 1996;26(1):173-85.
[132] Appelboom T, Durez P. Effect of milk product deprivation on spondyloarthropathy. Ann Rheum Dis. 1994 Jul;53(7):481-2.
[133] Kjeldsen-Kragh J, Hvatum M, Haugen M, Førre O, Scott H. Antibodies against dietary antigens in rheumatoid arthritis patients treated with fasting and a one-year vegetarian diet. Clin Exp Rheumatol. 1995 Mar-Apr;13(2):167-72.
[134] Lerner A, Rossi TM, Park B, Albini B, Lebenthal E. Serum antibodies to cow's milk proteins in pediatric inflammatory bowel disease. Crohn's disease versus ulcerative colitis. Acta Paediatr Scand. 1989 May;78(3):384-9.
[135] Knoflach P, Park BH, Cunningham R, Weiser MM, Albini B. Serum antibodies to cow's milk proteins in ulcerative colitis and Crohn's disease. Gastroenterology. 1987 Feb;92(2):479-85.
[136] Stefferl A, Schubart A, Storch2 M, Amini A, Mather I, Lassmann H, Linington C. Butyrophilin, a milk protein, modulates the encephalitogenic T cell response to myelin oligodendrocyte glycoprotein in experimental autoimmune encephalomyelitis. J Immunol. 2000 Sep 1;165(5):2859-65.
[137] Panush RS, Stroud RM, Webster EM. Food-induced (allergic) arthritis. Inflammatory arthritis exacerbated by milk. Arthritis Rheum. 1986 Feb;29(2):220-6.
[138] Motrich RD, Gottero C, Rezzonico C, Rezzonico C, Riera CM, Rivero V. Cow's milk stimulated lymphocyte proliferation and TNFalpha secretion in hypersensitivity to cow's milk protein. Clin Immunol. 2003 Nov;109(2):203-11.
[139] Balla G, Vercellotti GM, Muller-Eberhard U, Eaton J, Jacob HS. Exposure of endothelial cells to free heme potentiates damage mediated by granulocytes and toxic oxygen species. Lab Invest. 1991 May;64(5):648-55.
[140] Pattison DJ, Symmons DP, Lunt M, Welch A, Luben R, Bingham SA, Khaw KT, Day NE, Silman AJ. Dietary risk factors for the development of inflammatory polyarthritis: evidence for a role of high level of red meat consumption. Arthritis Rheum. 2004 Dec;50(12):3804-12.
[141] Grant WB. The role of meat in the expression of rheumatoid arthritis. Br J Nutr. 2000 Nov;84(5):589-95.
[142] Ferguson AC. Food allergy. Prog Food Nutr Sci. 1984;8(1-2):77-107.
[143] Faine LA, Diniz YS, Almeida JA, Novelli EL, Ribas BO. Toxicity of ad lib. overfeeding: effects on cardiac tissue. Food Chem Toxicol. 2002 May;40(5):663-8.
[144] Bosutti A, Malaponte G, Zanetti M, Castellino P, Heer M, Guarnieri G, Biolo G. Calorie restriction modulates inactivity-induced changes in the inflammatory markers CRP and PTX3. J Clin Endocrinol Metab. 2008 Aug;93(8):3226-9.
[145] Khaodhiar L, Ling PR, Blackburn GL, Bistrian BR. Serum levels of interleukin-6 and C-reactive protein correlate with body mass index across the broad range of obesity. JPEN J Parenter Enteral Nutr. 2004 Nov-Dec;28(6):410-5.
[146] Yudkin JS, Stehouwer CD, Emeis JJ, Coppack SW. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial dysfunction: a potential role for cytokines originating from adipose tissue? Arterioscler Thromb Vasc Biol. 1999 Apr;19(4):972-8.
[147] Symmons DP, Bankhead CR, Harrison BJ, Brennan P, Barrett EM, Scott DG, Silman AJ. Blood transfusion, smoking, and obesity as risk factors for the development of rheumatoid arthritis: results from a primary care-based incident case-control study in Norfolk, England. Arthritis Rheum. 1997 Nov;40(11):1955-61.
[148] Dela Peña A, Leclercq I, Field J, George J, Jones B, Farrell G. NF-kappaB activation, rather than TNF, mediates hepatic inflammation in a murine dietary model of steatohepatitis. Gastroenterology. 2005 Nov;129(5):1663-74.
[149] van der Poorten D, Milner KL, Hui J, Hodge A, Trenell MI, Kench JG, London R, Peduto T, Chisholm DJ, George J. Visceral fat: A key mediator of steatohepatitis in metabolic liver disease. Hepatology. 2008 Aug;48(2):449-57.
[150] Wu Z, Nagano I, Boonmars T, Takahashi Y. Tumor necrosis factor receptor-mediated apoptosis in Trichinella spiralis-infected muscle cells. Parasitology. 2005 Sep;131(Pt 3):373-81.
[151] Chen Z, Suntres Z, Palmer J, Guzman J, Javed A, Xue J, Yu JG, Cooke H, Awad H, Hassanain HH, Cardounel AJ, Christofi FL. Cyclic AMP signaling contributes to neural plasticity and hyperexcitability in AH sensory neurons following intestinal Trichinella spiralis-induced inflammation. Int J Parasitol. 2007 Jun;37(7):743-61.
[152] Akar S, Gurler O, Pozio E, Onen F, Sari I, Gerceker E, Gunes AJ, Akinci B, Birlik M, Akkoc N. Frequency and severity of musculoskeletal symptoms in humans during an outbreak of trichinellosis caused by Trichinella britovi. J Parasitol. 2007 Apr;93(2):341-4.
[153] Ferraccioli GF, Mercadanti M, Salaffi F, Bruschi F, Melissari M, Pozio E. Prospective rheumatological study of muscle and joint symptoms during Trichinella nelsoni infection. Q J Med. 1988 Dec;69(260):973-84.
[154] Zochling J, Bohl-Bühler MH, Baraliakos X, Feldtkeller E, Braun J. Infection and work stress are potential triggers of ankylosing spondylitis. Clin Rheumatol. 2006 Sep;25(5):660-6.
[155] Korzhova TP, Shyrobokov VP, Koliadenko VH, Korniushenko OM, Akhramieieva NV, Stepanenko VI. Coxsackie B viral infection in the etiology and clinical pathogenesis of psoriasis. Lik Sprava. 2001 May-Jun;(3):54-8.
[156] Bartenjev I, Rogl Butina M, Potocnik M. Subclinical microbial infection in patients with chronic plaque psoriasis. Acta Derm Venereol Suppl (Stockh). 2000;(211):17-8.
[157] Ohkusa T, Nomura T, Sato N. The role of bacterial infection in the pathogenesis of inflammatory bowel disease. Intern Med. 2004 Jul;43(7):534-9.
[158] Cainelli F, Betterle C, Vento S. Antinuclear antibodies are common in an infectious environment but do not predict systemic lupus erythematosus. Ann Rheum Dis. 2004 Dec;63(12):1707-8.
[159] Little CL, Richardson JF, Owen RJ, de Pinna E, Threlfall EJ. Campylobacter and Salmonella in raw red meats in the United Kingdom: prevalence, characterization and antimicrobial resistance pattern, 2003-2005. Food Microbiol. 2008 May;25(3):538-43.
[160] Pineton de Chambrun G, Colombel JF, Poulain D, Darfeuille-Michaud A. Pathogenic agents in inflammatory bowel diseases. Curr Opin Gastroenterol. 2008 Jul;24(4):440-7.
[161] Foley SL, Lynne AM, Nayak R. Salmonella challenges: prevalence in swine and poultry and potential pathogenicity of such isolates. J Anim Sci. 2008 Apr;86(14 Suppl):E149-62.
[162] Mataragas M, Skandamis PN, Drosinos EH. Risk profiles of pork and poultry meat and risk ratings of various pathogen/product combinations. Int J Food Microbiol. 2008 Aug 15;126(1-2):1-12.
[163] Douris A, Fedorka-Cray PJ, Jackson CR. Characterization of Salmonella enterica serovar Agona slaughter isolates from the animal arm of the National Antimicrobial Resistance Monitoring System-Enteric Bacteria (NARMS): 1997 through 2003. Microb Drug Resist. 2008 Mar;14(1):55-63.
[164] Khaitsa ML, Kegode RB, Doetkott DK. Occurrence of antimicrobial-resistant salmonella species in raw and ready to eat turkey meat products from retail outlets in the midwestern United States. Foodborne Pathog Dis. 2007 Winter;4(4):517-25.

[165] Aarestrup FM, Hendriksen RS, Lockett J, Gay K, Teates K, McDermott PF, White DG, Hasman H, Sørensen G, Bangtrakulnonth A, Pornreongwong S, Pulsrikarn C, Angulo FJ, Gerner-Smidt P. International spread of multidrug-resistant Salmonella Schwarzengrund in food products. Emerg Infect Dis. 2007 May;13(5):726-31.
[166] Schattner A. Consequence or coincidence? The occurrence, pathogenesis and significance of autoimmune manifestations after viral vaccines. Vaccine. 2005 Jun 10;23(30):3876-86.
[167] Geier DA, Geier MR. A case-control study of serious autoimmune adverse events following hepatitis B immunization. Autoimmunity. 2005 Jun;38(4):295-301.
[168] Fischer CP, Berntsen A, Perstrup LB, Eskildsen P, Pedersen BK. Plasma levels of interleukin-6 and C-reactive protein are associated with physical inactivity independent of obesity. Scand J Med Sci Sports. 2007 Oct;17(5):580-7.
[169] Sonnenberg A. Occupational distribution of inflammatory bowel disease among German employees. Gut. 1990 Sep;31(9):1037-40.
[170] Doran MF, Crowson CS, Pond GR, O'Fallon WM, Gabriel SE. Frequency of infection in patients with rheumatoid arthritis compared with controls: a population-based study. Arthritis Rheum. 2002 Sep;46(9):2287-93.
[171] Gabriel SE, Crowson CS, Kremers HM, Doran MF, Turesson C, O'Fallon WM, Matteson EL. Survival in rheumatoid arthritis: a population-based analysis of trends over 40 years. Arthritis Rheum. 2003 Jan;48(1):54-8.
[172] Hakoda M, Oiwa H, Kasagi F, Masunari N, Yamada M, Suzuki G, Fujiwara S. Mortality of rheumatoid arthritis in Japan: a longitudinal cohort study. Ann Rheum Dis. 2005 Oct;64(10):1451-5.
[173] Nicola PJ, Maradit-Kremers H, Roger VL, Jacobsen SJ, Crowson CS, Ballman KV, Gabriel SE. The risk of congestive heart failure in rheumatoid arthritis: a population-based study over 46 years. Arthritis Rheum. 2005 Feb;52(2):412-20.
[174] Maradit-Kremers H, Crowson CS, Nicola PJ, Ballman KV, Roger VL, Jacobsen SJ, Gabriel SE. Increased unrecognized coronary heart disease and sudden deaths in rheumatoid arthritis: a population-based cohort study. Arthritis Rheum. 2005 Feb;52(2):402-11.
[175] Scheidt-Nave C, Starker A. The prevalence of osteoporosis and associated health care use in women 45 years and older in Germany. Results of the first German Telephone Health Survey 2003. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz. 2005 Dec;48(12):1338-47.
[176] Cibere J, Sibley J, Haga M. Rheumatoid arthritis and the risk of malignancy. Arthritis Rheum. 1997 Sep;40(9):1580-6.
[177] Sonnenberg A. Occupational distribution of inflammatory bowel disease among German employees. Gut. 1990 Sep;31(9):1037-40.Click here to read Click here to read Links
[178] Patberg WR. Beneficial effect of being outdoors in rheumatoid arthritis. J Rheumatol. 2002 Jan;29(1):202-4.
[179] Yamada R, Yanoma S, Akaike M, Tsuburaya A, Sugimasa Y, Takemiya S, Motohashi H, Rino Y, Takanashi Y, Imada T. Water-generated negative air ions activate NK cell and inhibit carcinogenesis in mice. Cancer Lett. 2006; 239(2):190-7.
[180] Dawe RS, Ferguson J. History of psoriasis response to sunlight does not predict outcome of UVB phototherapy. Clin Exp Dermatol. 2004 Jul;29(4):413-4.
[181] Hayes CE, Donald Acheson E. A unifying multiple sclerosis etiology linking virus infection, sunlight, and vitamin D, through viral interleukin-10. Med Hypotheses. 2008;71(1):85-90.
[182] Kreuter A, Hyun J, Skrygan M, Sommer A, Bastian A, Altmeyer P, Gambichler T. Ultraviolet A1-induced downregulation of human beta-defensins and interleukin-6 and interleukin-8 correlates with clinical improvement in localized scleroderma. Br J Dermatol. 2006 Sep;155(3):600-7.
[183] Ponsonby AL, Lucas RM, van der Mei IA. UVR, vitamin D and three autoimmune diseases--multiple sclerosis, type 1 diabetes, rheumatoid arthritis. Photochem Photobiol. 2005 Nov-Dec;81(6):1267-75.
[184] Holick MF. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. Am J Clin Nutr. 2004 Dec;80(6 Suppl):1678S-88S.
[185] Krause Mda S, de Bittencourt PI Jr. Type 1 diabetes: can exercise impair the autoimmune event? The L-arginine/glutamine coupling hypothesis. Cell Biochem Funct. 2008 Jul-Aug;26(4):406-33.
[186] Noskova AS, Margazin VA. Efficacy of intensive therapeutic exercise and interval hypoxic training in rheumatoid arthritis. Vopr Kurortol Fizioter Lech Fiz Kult. 2005 Jul-Aug;(4):17-9.
[187] Hamer M, Steptoe A. Walking, vigorous physical activity, and markers of hemostasis and inflammation in healthy men and women. Scand J Med Sci Sports. Epub 2008 Feb 2.
[188] Hoffman-Goetz L, Spagnuolo PA, Guan J. Repeated exercise in mice alters expression of IL-10 and TNF-alpha in intestinal lymphocytes. Brain Behav Immun. 2008 Feb;22(2):195-9.
[189] Rønningen A, Kjeken I. Effect of an intensive hand exercise programme in patients with rheumatoid arthritis. Scand J Occup Ther. 2008 Apr 7:1-11.
[190] Metsios GS, Stavropoulos-Kalinoglou A, Veldhuijzen van Zanten JJ, Treharne GJ, Panoulas VF, Douglas KM, Koutedakis Y, Kitas GD. Rheumatoid arthritis, cardiovascular disease and physical exercise: a systematic review. Rheumatology (Oxford). 2008 Mar;47(3):239-48.
[191] Lee EO, Kim JI, Davis AH, Kim I. Effects of regular exercise on pain, fatigue, and disability in patients with rheumatoid arthritis. Fam Community Health. 2006 Oct-Dec;29(4):320-7.
[192] de Jong Z, Munneke M, Zwinderman AH, Kroon HM, Ronday KH, Lems WF, Dijkmans BA, Breedveld FC, Vliet Vlieland TP, Hazes JM, Huizinga TW. Long term high intensity exercise and damage of small joints in rheumatoid arthritis. Ann Rheum Dis. 2004 Nov;63(11):1399-405.
[193] Rall LC, Meydani SN, Kehayias JJ, Dawson-Hughes B, Roubenoff R. The effect of progressive resistance training in rheumatoid arthritis. Increased strength without changes in energy balance or body composition. Arthritis Rheum. 1996 Mar;39(3):415-26.
[194] Nieman DC. Exercise immunology: practical applications. Int J Sports Med. 1997 Mar;18 Suppl 1:S91-100.
[195] Lee YA, Hyun KJ, Tokura H. The effects of skin pressure by clothing on circadian rhythms of core temperature and salivary melatonin. Chronobiol Int. 2000 Nov;17(6):783-93.
[196] Cutolo M, Sulli A, Pizzorni C, Secchi ME, Soldano S, Seriolo B, Straub RH, Otsa K, Maestroni GJ. Circadian rhythms: glucocorticoids and arthritis. Ann N Y Acad Sci. 2006 Jun;1069:289-99.
[197] Cutolo M, Masi AT. Circadian rhythms and arthritis. Rheum Dis Clin North Am. 2005 Feb;31(1):115-29, ix-x.
[198] Roky R, Chapotot F, Hakkou F, Benchekroun MT, Buguet A. Sleep during Ramadan intermittent fasting. J Sleep Res. 2001 Dec;10(4):319-27.
[199] Wu MW, Li XM, Xian LJ, Lévi F. Effects of meal timing on tumor progression in mice. Life Sci. 2004 Jul 23;75(10):1181-93.
[200] Carney CE, Edinger JD, Meyer B, Lindman L, Istre T. Daily activities and sleep quality in college students. Chronobiol Int. 2006;23(3):623-37.
[201] Magrini A, Pietroiusti A, Coppeta L, Babbucci A, Barnaba E, Papadia C, Iannaccone U, Boscolo P, Bergamaschi E, Bergamaschi A. Shift work and autoimmune thyroid disorders. Int J Immunopathol Pharmacol. 2006 Oct-Dec;19(4 Suppl):31-6.
[202] Manber R, Bootzin RR, Acebo C, Carskadon MA. The effects of regularizing sleep-wake schedules on daytime sleepiness. Sleep. 1996 Jun;19(5):432-41.
[203] Taylor A, Wright HR, Lack LC. Sleeping-in on the weekend delays circadian phase and increases sleepiness the following week. Sleep Biol Rhythms. 2008; 6:172–179.
[204] Nettleton JA, Steffen LM, Mayer-Davis EJ, Jenny NS, Jiang R, Herrington DM, Jacobs DR Jr. Dietary patterns are associated with biochemical markers of inflammation and endothelial activation in the Multi-Ethnic Study of Atherosclerosis (MESA). Am J Clin Nutr. 2006 Jun;83(6):1369-79.
[205] Kjeldsen-Kragh J. Rheumatoid arthritis treated with vegetarian diets. Am J Clin Nutr. 1999 Sep;70(3 Suppl):594S-600S.
[206] Kjeldsen-Kragh J, Mellbye OJ, Haugen M, Mollnes TE, Hammer HB, Sioud M, Førre O. Changes in laboratory variables in rheumatoid arthritis patients during a trial of fasting and one-year vegetarian diet. Scand J Rheumatol. 1995;24(2):85-93.
[207] Danao-Camara TC, Shintani TT. The dietary treatment of inflammatory arthritis: case reports and review of the literature. Hawaii Med J. 1999 May;58(5):126-31.
[208] McDougall J, Bruce B, Spiller G, Westerdahl J, McDougall M. Effects of a very low-fat, vegan diet in subjects with rheumatoid arthritis. J Altern Complement Med. 2002 Feb;8(1):71-5.Click here to read Links
[209] Hafström I, Ringertz B, Spångberg A, von Zweigbergk L, Brannemark S, Nylander I, Rönnelid J, Laasonen L, Klareskog L. A vegan diet free of gluten improves the signs and symptoms of rheumatoid arthritis: the effects on arthritis correlate with a reduction in antibodies to food antigens. Rheumatology (Oxford). 2001 Oct;40(10):1175-9.
[210] Bae SC, Kim SJ, Sung MK. Inadequate antioxidant nutrient intake and altered plasma antioxidant status of rheumatoid arthritis patients. J Am Coll Nutr. 2003 Aug;22(4):311-5.
[211] Abe S, Tanaka Y, Fujise N, Nakamura T, Masunaga H, Nagasawa T, Yagi M. An antioxidative nutrient-rich enteral diet attenuates lethal activity and oxidative stress induced by lipopolysaccharide in mice. JPEN J Parenter Enteral Nutr. 2007 May-Jun;31(3):181-7.
[212] Kumazawa Y, Kawaguchi K, Takimoto H. Immunomodulating effects of flavonoids on acute and chronic inflammatory responses caused by tumor necrosis factor alpha. Curr Pharm Des. 2006;12(32):4271-9.
[213] Dragsted LO, Krath B, Ravn-Haren G, Vogel UB, Vinggaard AM, Bo Jensen P, Loft S, Rasmussen SE, Sandstrom TB, Pedersen A. Biological effects of fruit and vegetables. Proc Nutr Soc. 2006 Feb;65(1):61-7.
[214] Reifen R, Nur T, Ghebermeskel K, Zaiger G, Urizky R, Pines M. Vitamin A deficiency exacerbates inflammation in a rat model of colitis through activation of nuclear factor-kappaB and collagen formation. J Nutr. 2002 Sep;132(9):2743-7.
[215] Gatica L, Alvarez S, Gomez N, Zago MP, Oteiza P, Oliveros L, Gimenez MS. Vitamin A deficiency induces prooxidant environment and inflammation in rat aorta. Free Radic Res. 2005 Jun;39(6):621-8.
[216] Qi L, Hu FB. Dietary glycemic load, whole grains, and systemic inflammation in diabetes: the epidemiological evidence. Curr Opin Lipidol. 2007 Feb;18(1):3-8.
[217] Qi L, van Dam RM, Liu S, Franz M, Mantzoros C, Hu FB. Whole-grain, bran, and cereal fiber intakes and markers of systemic inflammation in diabetic women. Diabetes Care. 2006 Feb;29(2):207-11.
[218] Jensen MK, Koh-Banerjee P, Franz M, Sampson L, Grønbaek M, Rimm EB. Whole grains, bran, and germ in relation to homocysteine and markers of glycemic control, lipids, and inflammation. Am J Clin Nutr. 2006 Feb;83(2):275-83.
[219] Kasim-Karakas SE, Tsodikov A, Singh U, Jialal I. Responses of inflammatory markers to a low-fat, high-carbohydrate diet: effects of energy intake. Am J Clin Nutr. 2006 Apr;83(4):774-9.
[220] Hänninen , Kaartinen K, Rauma AL, Nenonen M, Törrönen R, Häkkinen AS, Adlercreutz H, Laakso J. Antioxidants in vegan diet and rheumatic disorders. Toxicology. 2000 Nov 30;155(1-3):45-53.
[221] Kaartinen K, Lammi K, Hypen M, Nenonen M, Hanninen O, Rauma AL. Vegan diet alleviates fibromyalgia symptoms. Scand J Rheumatol. 2000;29(5):308-13.
[222] Hagfors L, Nilsson I, Sköldstam L, Johansson G. Fat intake and composition of fatty acids in serum phospholipids in a randomized, controlled, Mediterranean dietary intervention study on patients with rheumatoid arthritis. Nutr Metab (Lond). 2005 Oct 10;2:26.
[223] Lopez-Garcia E, Schulze MB, Manson JE, Meigs JB, Albert CM, Rifai N, Willett WC, Hu FB. Consumption of (n-3) fatty acids is related to plasma biomarkers of inflammation and endothelial activation in women. J Nutr. 2004 Jul;134(7):1806-11.
[224] Linos A, Kaklamanis E, Kontomerkos A, Koumantaki Y, Gazi S, Vaiopoulos G, Tsokos GC, Kaklamanis P. The effect of olive oil and fish consumption on rheumatoid arthritis--a case control study. Scand J Rheumatol. 1991;20(6):419-26.
[225] Wahle KW, Caruso D, Ochoa JJ, Quiles JL. Olive oil and modulation of cell signaling in disease prevention. Lipids. 2004 Dec;39(12):1223-31.
[226] Owen RW, Haubner R, Würtele G, Hull E, Spiegelhalder B, Bartsch H. Olives and olive oil in cancer prevention. Eur J Cancer Prev. 2004 Aug;13(4):319-26.
[227] Aviram M, Eias K. Dietary olive oil reduces low-density lipoprotein uptake by macrophages and decreases the susceptibility of the lipoprotein to undergo lipid peroxidation. Ann Nutr Metab. 1993;37(2):75-84.
[228] Kremer JM. n-3 fatty acid supplements in rheumatoid arthritis. Am J Clin Nutr. 2000 Jan;71(1 Suppl):349S-51S.
[229] Ishiwa J, Sato T, Mimaki Y, Sashida Y, Yano M, Ito A. A citrus flavonoid, nobiletin, suppresses production and gene expression of matrix metalloproteinase 9/gelatinase B in rabbit synovial fibroblasts. J Rheumatol. 2000 Jan;27(1):20-5.
[230] Murakami A, Nakamura Y, Ohto Y, Yano M, Koshiba T, Koshimizu K, Tokuda H, Nishino H, Ohigashi H. Suppressive effects of citrus fruits on free radical generation and nobiletin, an anti-inflammatory polymethoxyflavonoid. Biofactors. 2000;12(1-4):187-92.
[231] Sasaki M, Elrod JW, Jordan P, Itoh M, Joh T, Minagar A, Alexander JS. CYP450 dietary inhibitors attenuate TNF-alpha-stimulated endothelial molecule expression and leukocyte adhesion. Am J Physiol Cell Physiol. 2004 Apr;286(4):C931-9.
[232] Kometani T, Fukuda T, Kakuma T, Kawaguchi K, Tamura W, Kumazawa Y, Nagata K. Effects of alpha-glucosylhesperidin, a bioactive food material, on collagen-induced

arthritis in mice and rheumatoid arthritis in humans. Immunopharmacol Immunotoxicol. 2008;30(1):117-34.
[233] Benavente-Garcia O, Castillo J. Update on uses and properties of citrus flavonoids: new findings in anticancer, cardiovascular, and anti-inflammatory activity. J Agric Food Chem. 2008 Aug 13;56(15):6185-205.
[234] Kawaguchi K, Maruyama H, Kometani T, Kumazawa Y. Suppression of collagen-induced arthritis by oral administration of the citrus flavonoid hesperidin. Planta Med. 2006 Apr;72(5):477-9.
[235] Atkinson MA, Winter WE, Skordis N, Beppu H, Riley WM, Maclaren NK. Dietary protein restriction reduces the frequency and delays the onset of insulin dependent diabetes in BB rats. Autoimmunity. 1988;2(1):11-9.
[236] Mitchell JH, Collins AR. Effects of a soy milk supplement on plasma cholesterol levels and oxidative DNA damage in men--a pilot study. Eur J Nutr. 1999 Jun;38(3):143-8.
[237] Burks AW, Laubach S, Jones SM. Oral tolerance, food allergy, and immunotherapy: implications for future treatment. J Allergy Clin Immunol. 2008 Jun;121(6):1344-50.
[238] Van Hoogstraten IM, Andersen KE, Von Blomberg BM, Boden D, Bruynzeel DP, Burrows D, Camarasa JG, Dooms-Goossens A, Kraal G, Lahti A, et al. Reduced frequency of nickel allergy upon oral nickel contact at an early age. Clin Exp Immunol. 1991 Sep;85(3):441-5.
[239] van Hoogstraten IM, Boden D, von Blomberg ME, Kraal G, Scheper RJ. Persistent immune tolerance to nickel and chromium by oral administration prior to cutaneous sensitization. J Invest Dermatol. 1992 Nov;99(5):608-16.
[240] Huibregtse IL, Snoeck V, de Creus A, Braat H, De Jong EC, Van Deventer SJ, Rottiers P. Induction of ovalbumin-specific tolerance by oral administration of Lactococcus lactis secreting ovalbumin. Gastroenterology. 2007 Aug;133(2):517-28.
[241] Nagatani K, Dohi M, To Y, Tanaka R, Okunishi K, Nakagome K, Sagawa K, Tanno Y, Komagata Y, Yamamoto K. Splenic dendritic cells induced by oral antigen administration are important for the transfer of oral tolerance in an experimental model of asthma. J Immunol. 2006 Feb 1;176(3):1481-9.
[242] Friedman A, al-Sabbagh A, Santos LM, Fishman-Lobell J, Polanski M, Das MP, Khoury SJ, Weiner HL. Oral tolerance: a biologically relevant pathway to generate peripheral tolerance against external and self antigens. Chem Immunol. 1994;58:259-90.
[243] Weiner HL, Mackin GA, Matsui M, Orav EJ, Khoury SJ, Dawson DM, Hafler DA. Double-blind pilot trial of oral tolerization with myelin antigens in multiple sclerosis. Science. 1993 Feb 26;259(5099):1321-4.Click here to read Links
[244] Ugochukwu NH, Figgers CL. Caloric restriction inhibits up-regulation of inflammatory cytokines and TNF-alpha, and activates IL-10 and haptoglobin in the plasma of streptozotocin-induced diabetic rats. J Nutr Biochem. 2007 Feb;18(2):120-6.
[245] Kalani R, Judge S, Carter C, Pahor M, Leeuwenburgh C. Effects of caloric restriction and exercise on age-related, chronic inflammation assessed by C-reactive protein and interleukin-6. J Gerontol A Biol Sci Med Sci. 2006 Mar;61(3):211-7.
[246] Dandona P, Mohanty P, Hamouda W, Ghanim H, Aljada A, Garg R, Kumar V. Inhibitory effect of a two day fast on reactive oxygen species (ROS) generation by leucocytes and plasma ortho-tyrosine and meta-tyrosine concentrations. J Clin Endocrinol Metab. 2001 Jun;86(6):2899-902.
[247] Müller H, de Toledo FW, Resch KL. Fasting followed by vegetarian diet in patients with rheumatoid arthritis: a systematic review. Scand J Rheumatol. 2001;30(1):1-10.
[248] Fujita A, Hashimoto Y, Nakahara K, Tanaka T, Okuda T, Koda M. Effects of a low calorie vegan diet on disease activity and general conditions in patients with rheumatoid arthritis. Rinsho Byori. 1999 Jun;47(6):554-60.
[249] Dandona P, Mohanty P, Ghanim H, Aljada A, Browne R, Hamouda W, Prabhala A, Afzal A, Garg R. The suppressive effect of dietary restriction and weight loss in the obese on the generation of reactive oxygen species by leukocytes, lipid peroxidation, and protein carbonylation. J Clin Endocrinol Metab. 2001 Jan;86(1):355-62.
[250] Dandona P, Weinstock R, Thusu K, Abdel-Rahman E, Aljada A, Wadden T. Tumor necrosis factor-alpha in sera of obese patients: fall with weight loss. J Clin Endocrinol Metab. 1998 Aug;83(8):2907-10.
[251] Jung SH, Park HS, Kim KS, Choi WH, Ahn CW, Kim BT, Kim SM, Lee SY, Ahn SM, Kim YK, Kim HJ, Kim DJ, Lee KW. Effect of weight loss on some serum cytokines in human obesity: increase in IL-10 after weight loss. J Nutr Biochem. 2008 Jun;19(6):371-5.
[252] Morimoto A, Murakami N, Ono T, Watanabe T. Dehydration enhances endotoxin fever by increased production of endogenous pyrogen. Am J Physiol. 1986 Jul;251(1 Pt 2):R41-7.
[253] Pool EJ, van Wyk JH, Leslie AJ. Inflammatory activity as an indicator of water quality: the use of human whole blood cultures. J Immunoassay. 2000 Nov;21(4):387-99.
[254] Pool EJ, Jagals C, van Wyk JH, Jagals P. The use of IL-6 induction as a human biomarker for inflammatory agents in water. Water Sci Technol. 2003;47(3):71-5.
[255] Curković B, Vitulić V, Babić-Naglić D, Durrigl T. The influence of heat and cold on the pain threshold in rheumatoid arthritis. Z Rheumatol. 1993 Sep-Oct;52(5):289-91.
[256] Sluka KA, Christy MR, Peterson WL, Rudd SL, Troy SM. Reduction of pain-related behaviors with either cold or heat treatment in an animal model of acute arthritis. Arch Phys Med Rehabil. 1999 Mar;80(3):313-7.
[257] Rychkova MA, Aĭrapetova NS, Davydova OB, Krivtsova IE, Doronina IuV, Derevnina NA. Contrast baths in the rehabilitation of patients with chronic bronchitis. Vopr Kurortol Fizioter Lech Fiz Kult. 1994 May-Jun;(3):3-6.
[258] Petrofsky J, Lohman E 3rd, Lee S, de la Cuesta Z, Labial L, Iouciulescu R, Moseley B, Korson R, Al Malty A. Effects of contrast baths on skin blood flow on the dorsal and plantar foot in people with type 2 diabetes and age-matched controls. Physiother Theory Pract. 2007 Jul-Aug;23(4):189-97.
[259] Cecchini M, Lopresti V. Drug residues store in the body following cessation of use: Impacts on neuroendocrine balance and behavior - Use of the Hubbard sauna regimen to remove toxins and restore health. Med Hypotheses. 2007;68(4):868-79.
[260] Jiao Y, Wilkinson J 4th, Christine Pietsch E, Buss JL, Wang W, Planalp R, Torti FM, Torti SV. Iron chelation in the biological activity of curcumin. Free Radic Biol Med. 2006 Apr 1;40(7):1152-60.
[261] Barollo M, D'Incà R, Scarpa M, Medici V, Cardin R, Fries W, Angriman I, Sturniolo GC. Effects of iron deprivation or chelation on DNA damage in experimental colitis. Int J Colorectal Dis. 2004 Sep;19(5):461-6.
[262] Horne S. Colon cleansing: a popular, but misunderstood natural therapy. J Herb Pharmacother. 2006;6(2):93-100.
[263] Pederson JA, Matter BJ, Czerwinski AW, Llach F. Relief of idiopathic generalized pruritus in dialysis patients treated with activated oral charcoal. Ann Intern Med. 1980 Sep;93(3):446-8.
[264] Sologub VK, Kaem RI, Pavlova VV, Ustinova TS, Lopatto IuS. Morphological characteristics of the healing of burn wounds covered by an activated-charcoal tissue dressing. Biull Eksp Biol Med. 1989 Mar;107(3):360-3.
[265] Li LG, Chai JK, Guo ZR, Yang HM, Jia XM, Xu MH, Li F, Cao WH, Feng G, Sheng ZY. Application of carbon fiber dressing on burn wounds. Zhonghua Wai Ke Za Zhi. 2006 Aug 1;44(15):1047-9.
[266] Barbas IM, Ermolenko IN, Dozorets DI, Klimova TT, Kozlova IG, Korenko LA, Morozova AA, Skoromets AA, Totolian NA. Enterosorption in the combined treatment of patients with multiple sclerosis. Klin Med (Mosk). 1991 Feb;69(2):88-90.
[267] Bartlett SJ, Piedmont R, Bilderback A, Matsumoto AK, Bathon JM. Spirituality, well-being, and quality of life in people with rheumatoid arthritis. Arthritis Rheum. 2003 Dec 15;49(6):778-83.
[268] Potter ML, Zauszniewski JA. Spirituality, resourcefulness, and arthritis impact on health perception of elders with rheumatoid arthritis. J Holist Nurs. 2000 Dec;18(4):311-31.
[269] Coruh B, Ayele H, Pugh M, Mulligan T. Does religious activity improve health outcomes? A critical review of the recent literature. Explore (NY). 2005 May;1(3):186-91.
[270] Matthews DA, Marlowe SM, MacNutt FS. Effects of intercessory prayer on patients with rheumatoid arthritis. South Med J. 2000 Dec;93(12):1177-86.
[271] Walker JG, Littlejohn GO, McMurray NE, Cutolo M. Stress system response and rheumatoid arthritis: a multilevel approach. Rheumatology (Oxford). 1999 Nov;38(11):1050-7.
[272] Wahle M, Krause A, Pierer M, Hantzschel H, Baerwald CG. Immunopathogenesis of rheumatic diseases in the context of neuroendocrine interactions. Ann N Y Acad Sci. 2002 Jun;966:355-64.
[273] Kjeldsen-Kragh J, Haugen M, Førre O, Laache H, Malt UF. Vegetarian diet for patients with rheumatoid arthritis: can the clinical effects be explained by the psychological characteristics of the patients? Br J Rheumatol. 1994 Jun;33(6):569-75.
[274] Tuck I, Alleyne R, Thinganjana W. Spirituality and stress management in healthy adults. J Holist Nurs. 2006 Dec;24(4):245-53.
[275] Matthew 11:28, King James Version of the Holy Bible.
[276] Genesis 1:29; 3:18 (NIV). Scripture taken from the HOLY BIBLE, NEW INTERNATIONAL VERSION®. Copyright © 1973, 1978, 1984 International Bible Society. Used by permission of Zondervan. All rights reserved. The "NIV" and "New International Version" trademarks are registered in the United States Patent and Trademark Office by International Bible Society. Use of either trademark requires the permission of International Bible Society.