

The Newsletter of THE AMERICAN INSTITUTE OF STRESS

Number 3

1993

DEBUNKING THE DIET-CHOLESTEROL-HEART ATTACK MYTH:

"Ignore the experts and eat what you like"

M. Oliver The London Times, 1992

The certitude that cholesterol and diet are major contributors to heart attacks is so prevalent and pursued with such passion, that any contrary conviction is currently considered heretical. However, not only is there no proof to defend this dogma, but the preponderance of evidence is clearly contradictory. Analyzing how this myth materialized and matured is fascinating, especially with respect to its culmination in the concurrent Quixotic quest for "risk factors". The history of medicine is replete with analogous dietary doctrines that subsequently fell into disrepute. The reasons why a society tends to select a particular diet is most apt to depend on what is readily available to them, although this is often modified by certain taboos. Some primitive tribes forbade eating tortoise for fear it would slow down the hunters, while others abstained from eating rabbit, deer or jackal meat, lest it cause their warriors to

be timid. In old Madagascar, soldiers were prohibited from eating hedgehog to avoid any tendency to curl up like as these animals do when confronted with a stressful situation. Since oxen were believed to be "weak in the knees", this part of their anatomy also had to be avoided. From a historical perspective, the current preaching seems somewhat like putting old wine in new bottles, but that has not stopped it from turning sour.

The Birth Of The Dietary Fat - Cholesterol Taboo

Our cholesterol taboo probably began around 90 years ago, when the Russian physician, Nikolai Anichkov, demonstrated that he could produce atherosclerotic lesions in rabbits by feeding them a high cholesterol diet. This attracted relatively little interest until the Korean War, when the Army dispatched a team of pathologists to perform thorough autopsies

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The Newsletter of
**THE AMERICAN INSTITUTE OF
 STRESS**

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studies on soldiers killed in battle. Their main objective was to study the ballistic characteristics of fatal wounds. Since the average age of the victims was 22, it came as quite a shock to discover that more than three out of four showed evidence of significant coronary artery atherosclerosis. In 10 per cent, this was sufficiently severe to almost completely obstruct at least one major coronary vessel. However, none of these individuals had any cardiac complaints, and it would probably be more than twenty or thirty years before it was likely that they might actually suffer a heart attack.

Because it became apparent that significant atherosclerosis was already present in these otherwise healthy young men, it seemed prudent to attempt to prevent the process long before the onset of clinical symptoms. It was postulated that the basic problem was because the American diet was too high in fat, which resulted in an elevation of blood cholesterol that caused atherosclerotic deposits, much as Anichkov had reported in his rabbits. Further support came from surveys reporting that Greek and Japanese populations who consumed much less animal fat than Americans, had heart attack rates almost 90 per cent lower than ours. One staunch advocate of the fatty diet - heart attack

hypothesis, who flaunted the statistics obtained from seven countries, concluded:

There is a remarkable relationship between the death rate from degenerative heart disease and the proportion of fat calories in the national diet. A regular progression exists from Japan through Italy, Sweden, England and Wales, Canada and Australia to the United States. No other variable in the mode of life besides the fat calories in the diet is known which shows.....such a constant relationship to the mortality rate from coronary or degenerative heart disease.

Most vegetarians I ever see looked enough like their food to be classed as cannibals.

Finley Peter Dunne

The Framingham Study And Risk Factors

The Framingham study was able to demonstrate a direct correlation between elevation in blood cholesterol and an increase in the incidence of heart attacks. This was interpreted as confirming that elevated serum cholesterol caused heart attacks, and was therefore a risk factor that required eradication. However, association never proves causation, and you can't use a statistic to prove another statistic. There are close to 300 conditions or "risk factors" that have been associated with heart attacks, and new ones like increased serum iron and male vertex baldness pop up every few weeks. However, like elevated serum cholesterol, these are simply statistical associations. It does not mean that they have anything to do with causing heart attacks, and in view of the confusion, it would be more appropriate to refer to these as "risk markers". Getting a hair transplant or flattening out an earlobe crease will not prevent a coronary. Nevertheless, despite the fact that the Framingham study showed no relationship between a high fat diet and increased serum cholesterol, or any link between a high fat diet and heart attacks, America was subjected to an unparalleled media blitz and crusade designed to wage war on cholesterol.

Free Radicals And Antioxidants

Cholesterol is an essential building block for hormones and other important compounds, and a vital constituent of cell membranes. It is a large, inert molecule, and it has always been difficult to understand how it could cause the pathology seen in atherosclerotic plaque. Neither cholesterol, or even unmodified low density lipoprotein, are capable of producing the chronic inflammatory changes and foam cells that characterize this lesion. The real culprit appears to be oxidized LDL resulting from free radical activity or the intake of trans-fatty acids from products such as margarine and partially hydrogenated oils. The oxidation of LDL is prevented by substances known as antioxidants which scavenge the body searching for free radicals, and inactivate them. As demonstrated at a session I recently chaired at the Fifth International Montreux Congress on Stress, this may explain the cardioprotective effects of vitamins C, E and beta carotene, as well as peanuts, olive oil, other monounsaturated fats, yogurt, and red wine, all of which have powerful antioxidant properties. Lowering serum cholesterol per se does not reduce atherosclerosis. Reduction of elevated cholesterol in experimental animal studies using the leading drug lovastatin failed to prevent the development of atherosclerotic plaque. However, when probucol was given in dosages sufficient to achieve the same degree of cholesterol lowering, atherosclerosis was significantly diminished. The reason for the difference between these two drugs is that probucol has antioxidant properties whereas lovastatin does not.

Stress And Cholesterol

As in previous years, another Congress session was devoted to demonstrating that stress has a much more powerful influence on serum lipids and cholesterol than dietary fat intake. Stress related hormones elevate serum lipids by breaking down fat stores in the body. Stress also contributes to accelerated atherosclerosis because it promotes free

radical production. Free radicals are responsible for other manifestations of the aging process, such as wrinkled skin, grey hair, cataracts, and cancer, which helps to explain the close links between stress and premature aging. The well established cardiotoxic effects of increased stress related catecholamines and corticoids were discussed, and other relationships between stress and heart attacks, and sudden death were also reviewed.

High Fat Diets Do Not Cause Heart Attacks

If we reexamine more carefully the evidence that has contributed to the current confusion concerning cholesterol and coronary atherosclerosis, and the reputed role of high fat diets, then a different picture is painted. It was not pure cholesterol that Anichkov fed his rabbits, but rather oxidized cholesterol, which, as we have seen, is quite different. The marked atherosclerotic lesions described in Korean War casualties quite likely resulted from their diet which included powdered eggs and other rations rich in cholesterol-oxidation products. Young Americans killed in civilian accidents do not show similar lesions at autopsy, and the microscopic appearance of any atherosclerotic deposits found is quite different, since marked inflammatory changes are minimal.

The high fat diet hypothesis has been refuted by numerous observations. Coronary heart disease is relatively uncommon in the African Massai or the Eskimos, both of whom have high fat diets. At the height of the U.S. epidemic, heart attack rates were lowest in those midwest farming areas where fat consumption was the highest. In some surveys, increased dairy consumption has been associated with a decreased incidence of coronary heart disease. Several years ago, The New England Journal of Medicine reported a well documented case of an 86 year old man with no history of heart disease and a normal electrocardiogram, who had consumed

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two dozen eggs every day for the previous 15 years. As reported at this Congress, eating fresh eggs and dairy products may lower coronary risk because of the natural antioxidants contained in these foods, and increased consumption of peanuts, yogurt, and the high fat "Mediterranean" and "French Paradox" diets, protect against heart attacks because of their antioxidant effects.

When the report demonstrating a correlation between national fat consumption and coronary disease mortality was reexamined, epidemiologists wondered why only seven countries had been selected. When the statistics available from 22 countries were included, that theory completely disintegrated. Fat consumption in Israel and Mexico was similar, but cardiovascular deaths were eight times higher in Israel. U.S. fat consumption was only slightly higher than Norway's, but our cardiovascular death rate per capita was more than three times greater. Although Framingham heart attack rates did show a correlation with increased cholesterol levels, a similar relationship was found for cigarette smoking and hypertension. This was also misinterpreted as indicating a causal connection, rather than merely some statistical association. If that were true, then eliminating or significantly reducing any or certainly all of these so called "risk factors" should help to prolong life. However, none of the large scale intervention studies that achieved these objectives demonstrated any such benefits. After seven years of the 115 million dollar MRFIT study, cholesterol, blood pressure and smoking were satisfactorily lowered in the intervention group. Despite this, they had just as many heart attacks as untreated controls, and cardiovascular deaths were actually higher in hypertensives treated with drugs.

Part of the secret of success in life is to eat what you like and let the food fight it out inside.

Mark Twain

The Commercial Cholesterol Crusade

There is growing evidence that we have been the victims not only of poor science, but deliberate

deception due to the systematic suppression of information that would reveal damaging defects in the design of various drug studies, or lack of support for their preconceived suppositions. The objective of The National Cholesterol Education Program was "To reduce the prevalence of elevated blood cholesterol in the United States and thereby contribute to reducing coronary heart disease morbidity and mortality". The government sponsored crusade was supported by panels of hand picked cardiologists and scientists, who recommended following a strict low fat diet for three months. If this did not achieve results, then an even more severe regimen was imposed for a similar period, and if this was not successful, patients would have to take powerful cholesterol lowering drugs forever. However, there was no evidence that reducing dietary fat as opposed to total calories would lower blood cholesterol levels, and more importantly, absolutely no proof that lowering cholesterol could save lives. Indeed, all the cholesterol lowering drug trials have shown just the opposite.

The cholesterol campaign was launched in 1985 with authoritative promises of progressive health rewards as the serum cholesterol fell every few milligrams. However, the investigators were well aware that laboratory measurements of cholesterol throughout the country were extremely inaccurate and could lead to unnecessary lifetime drug therapy for several hundred thousand individuals. When 117 blood specimens of employees from one company were sent to one respected commercial laboratory, 74 per cent of them fell into the treatment group, although independent testing of the same samples found that only 25 per cent should have been in this arbitrary category. Even a recent survey of the 5000 laboratories approved by the American College of Pathologists found that one out of five had an error rate of nine per cent. This means that an actual cholesterol of 220 might be recorded anywhere from 187 to 267. Three out of four of the major commercial clinical laboratories are not even in the ACP program, and one can only imagine what the error rate is for poorly regulated

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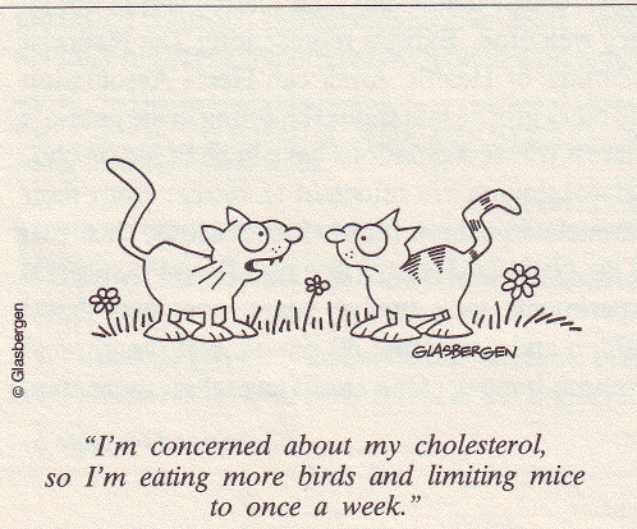
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forty years and continues on in its futile attempt to prove a flawed premise.

The Cholesterol Cartel And Educating The Public

It is thus difficult to find sponsorship for programs that run contrary to current diet-cholesterol dogma, or to explain the important contribution of stress to coronary heart disease. Such educational efforts provide little financial incentives. We are grateful for the support of the Biotonus Clinic which has consistently made it possible to attract an impressive array of authorities from all over the world to this and previous Congresses. Their state of the art presentations and cutting edge discussions are conducted in an atmosphere of academic freedom that encourages critical objective analysis not motivated by commercial concerns. While it is not possible to review all the material presented in an article such as this, our conclusions were perhaps best summed up by Dr. George Mann, one of the participants and a respected pioneer in the field, who early on identified the cardiotoxic effects of trans-fatty acids such as those found in margarine and partially hydrogenated oils.

"Saturated fat and cholesterol in the diet are not the cause of coronary heart disease. That myth is the greatest scientific deception of this century, perhaps of any century."



Can Odors Make You Smarter and Thinner?

Aromatherapy has been increasing in popularity, particularly with respect to the use of certain scents to reduce stress and/or induce a state of deep relaxation. Recent reports now suggest that certain smells can also be used to promote weight loss or improve memory. According to the Smell and Taste Treatment and Research Foundation, regularly inhaling the scent of Fritos when you are hungry can help you lose weight. The chemical 2-acetylpyridine smells like Frito corn chips, and in one study, a group of men and women who inhaled it whenever they felt hungry lost over 4 pounds in a 4 week period, compared to a loss of just over a pound in controls inhaling a placebo odor.

In another study, students were taught to memorize 12 pleasant and 12 unpleasant nouns on 2 separate occasions. During one of the sessions, they were also exposed to either jasmine, incense, or Ralph Lauren perfume. Students who inhaled any of the scents during the learning process were able to recall both sets of words with much greater ease and accuracy, compared to aroma free sessions. No particular scent showed any significant superiority. It was suggested that the presence of the aroma became associated with the learning process and somehow was able to improve retrieval capabilities.

Brain, Mind and Common Sense, 11/92
USA Today, 3/12/93

Nothing awakens a reminiscence like an odour.

Victor Hugo

The Stressful Health Effects of Unemployment

Several studies in the United States over the past few decades have demonstrated a close association between economic recessions and increased

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rates of overall illness. Decreased access to medical care may be a factor, but a report from the United Kingdom also emphasizes the key role unemployment plays in causing family disruption and mental health problems due to psychosocial stress. Unemployment represents the leading cause of poverty in the United Kingdom and its effects on health can be surprising. One national survey revealed that 1 out of 10 affected children under the age of 5 misses a daily meal, and the figure is twice as high for their jobless parents who can no longer afford to buy food. Another study found that unemployed men were 11 times more likely to attempt suicide than working controls.

Non-workers of both sexes were also approximately 30% more likely than others to report chronic illness, and in addition to suicide, particularly high levels of lung cancer, accidents and heart disease were associated with long term unemployment. As the Employment Minister noted in the House of Commons, "In plain English, it kills and maims. It kills mainly by way of cancer, suicides, accidents and violence. For a middle aged man to lose his job doubles his chance of dying in the next ten years". When the government indicated they were planning to lay off an additional 30,000 coal miners, the Editor of the British Medical Journal predicted that many of them would die prematurely, especially from "suicide, cancer, accidents and violence....Exactly how unemployment kills is unclear, but it is through a combination of poverty, stress, adoption of unhealthy behaviors and a devastating affect on mental health."

Some suggest that that it may be difficult to tell which comes first, since people in poor health might be more likely to lose their jobs. However, a 20 year UK study found that the unusually high death rate of unemployed men could not be explained by the state of their health before they lost their jobs. Emotional disturbances seem to be the most common manifestations of the stress of unemployment, and "evidence that unemployment causes a deterioration in mental health is extremely strong".

Further support for this comes from the finding that mental health improved significantly when unemployed individuals were returned to the workforce.

Lancet, 1/23/93

*Oh! poverty is a weary thing, 'tis full of grief and pain;
It keepeth down the soul of man, as with an iron chain.*

Mary Howitt

Can Noise Stress Create Protective Earplugs?

Exposure to 95-decibel sound, similar to that experienced while riding in a noisy subway, can cause slight temporary hearing loss after only a few hours. However, exposure to 105-decibel jet engine sound for 8 or more hours will produce significant and permanent damage. Since chinchillas ears are anatomically similar to humans, they were exposed to 95-decibel sound for six hours a day for 10 days to study the nature of noise induced hearing loss. Researchers expected to find that hearing progressively worsened with each repeated exposure. They were therefore surprised to discover that just the opposite occurred, since temporary hearing loss became less severe after each exposure. After a week of quiet, the same chinchillas were then subjected to 48 straight hours of 105-decibel sound, with equally surprising findings. The resultant reduction in hearing acuity was only about 10 decibels, a deficit that would not be appreciated in normal daily activities. However, a control group of chinchillas who had not experienced the previous 95-decibel exposure lost an average of 30 decibels, enough to make it difficult to hear soft conversation.

These findings are consistent with the *Stage of Resistance* of Selye's *General Adaptation Syndrome*, during which the body's resistance to a stressor increases. It has been suggested that the resistance to noise stress damage demonstrated in this study results from sound sensitive cells in the inner ear that produce protective proteins to reduce further damage.

In Health, July/August 1991

Book Reviews • Meetings and Items of Interest

Book Review

Mental Health in The Workplace: A Practical Psychiatric Guide, Jeffrey Kahn, Editor, Van Nostrand Reinhold New York, 1993. No. of pages: 462. Price \$59.95

This volume offers a comprehensive guide to dealing with the identification and treatment of mental health problems encountered in the workplace. The 18 chapters are divided into three parts, and are primarily authored by physicians with a wide range of expertise in various relevant areas. Part 1, entitled Problems In Adaptation, contains a broad overview of the problems associated with the recognition, assessment, and management of workplace mental health disorders. Other chapters address the role of health care providers and delivery systems with respect to such important issues as cost containment, and ethical considerations such as preserving confidentiality. It also includes a historical review of the development of the field of occupational psychiatry, by Alan McLean, one of the pioneers in this field. Part 2, Common Organizational Problems, deals with disability determinations, workers' compensation concerns, executive stress, emotional crises in the workplace, and problems related to organizational changes. The discussion of the effects of impending or actual dismissal unfortunately neglected to mention its significant contribution to cardiovascular and immune system disorders. The final section, Employee Problems, addresses anxiety and other stress related disorders such as depression and burnout, the complex area of substance abuse. Chapters are also devoted to a discussion of psychotic, psychosomatic and somatoform disorders commonly encountered, and these are often illustrated by useful case reports.

One drawback or deficiency, was the lack of any

discussion dealing with the design, delivery, and cost effectiveness of stress management programs for the workplace. This is currently a top priority for Employee Assistance Programs and is a leading objective in the Public Health Service's strategic report on improving the health of the nation in the coming decade. As in most multiauthored works, the style is somewhat uneven. However, although geared to health care professionals, the text should be readily comprehensible to lay readers. Helpful charts and tables are of high quality, and timely references are provided for those who wish to obtain more detailed information on specific subjects.

Meetings and Items of Interest

June 6-9 Shadyside Hospital - 1993 Meeting of the Academy of Behavioral Medicine Research: Behavior, Health and Aging, Nemacolin Woodlands, Farmington, PA (412) 623-2393

June 10-11 Corporate Leadership & Innovation in Maternal & Child Health, The New York Palace, New York City (800) 872-0094 to register

June 11-13 Harvard Medical School, Massachusetts General Hospital, Department of Psychiatry - Psychiatric Disorders Associated with Female Reproductive Function, Copley Plaza Hotel, Boston (617) 432-1525

June 20-23 International Conference - Healing: Beyond Suffering or Death, Montreal Convention Centre, Montreal, CANADA (514) 848-1133

June 23-26 Become A Stress Trainer, 5th Annual Conference, Donner Lake, CA. Contact Dr. Edmond C. Hallberg, 470 West Highland Ave., Sierra Madre, CA 91024 (818) 355-1325

ISSN # 1047-2517

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**THE AMERICAN INSTITUTE OF
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