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Short Communication

Nature vs. Nurture – Will the Species Survive?

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During the last half-century, human beings have developed new processes to both produce and preserve food for immediate and long term consumption. Such abilities would presumable result in increased survival of the species by reducing starvation and provide for times of need. The exact opposite has occurred; however, with recent increases in obesity and weight related disease (eg. heart disease, diabetes mellitus, hypertension, certain forms of cancer, et cetera) leading to an increase in the incidence of these diseases and related deaths. Arguments over who is to be blamed for these increased health problems has lead to various discussions implicating the food industry, the government, the pharmaceutical industry, and others, with efforts to even blame our ancestors by convincing ourselves that it is our genetic nature guised in the politically acceptable term, the "thrifty gene" hypothesis which is to blame. In fact for many people, any explanation except for individual accountability is an acceptable cause for the problem, but failure to correct the cause of the problem does not solve the problem, it merely masks it [1-4].

Having been involved in research [1-26] for almost four decades involving heart disease and the dietary factors which produce the inflammatory changes within our bodies which lead to this immunologic disease, it is clear that we need to consider this problem from a more fundamental perspective, namely a discussion of nature (that which we are genetically born with) versus nurture (those behaviors we exhibit in perpetuation of the species). The changes in our production, storage, utilization and consumption of food is clearly a change in behavior and such changes in behavior occur much faster than genetic mutation can occur. As we are seeing, the wrong behavior in any situation can be threatening to the survival of the species. Survival of the fittest proposes that only those species with the genetic predisposition for survival, which also exhibit the behavioral traits for survival, will endure changes presented to them. These changes may occur from sources outside the control of the species (eg. volcanic eruptions, temperature changes, meteorite impaction, et cetera), or they may be precipitated by the species itself. Like all species, we Homo Sapien Sapiens, have the ability to affect our environment for better or worse. In our earlier development, decisions to leave areas of protection (trees, caves, et cetera) and venture out into open areas where we were exposed, produced a situation where we were more the hunted than hunter. These efforts were successful, not because of a sudden genetic shift in the species (there is no data to support such a theory), but rather, a shift in our behavior (the ability to make and use tools/weapons) and our working together to out think our more physically agile and aggressive opponents. Those human ancestors who did not adopt such behavioral changes, but ventured out onto the plains without the necessary behavior to ensure their safety, undoubtedly provided lunch for other species which was the more fit for their environment. Hence, survival of the more adept species prevailed [5-8].

In the February 7, 2003 issue of Science, several authors discussed the need to further understand the neurohumoral pathways involved in the regulation of eating - hunger versus satiety. While such efforts are of scientific value, our focus as a society appears to be more related to the pharmaceutical development of drugs to manipulate these pathways than the understanding that

behavioral change is needed. In other words, the focus has been to influence nature and not nurture. Such efforts fail to recognize the multiple lessons from the past that we should have learned regarding our efforts to manipulate nature, ignoring the impact of nurture, which can lead to serious consequences [9,10]. The issue here has not been a change in the genome of the human species, but behavioral changes which threaten our survival. For example, the genetic structure of Japanese immigrants who have migrated to the United States in the last 50 years, has not been associated with a genetic shift in the population, yet changes in the consumption of high fat, high calorie foods has lead to an increase in heart disease, diabetes mellitus, obesity, elevated lipid (cholesterol, triglycerides) levels, and certain types of cancers, when compared with their Japanese ancestors of only two generations earlier. Similar problems have occurred among the children of China and Spain in a single generation; reflecting changes in (behavioral) eating patterns and not genetic shifts. Other groups frequently discussed, to support the need for manipulation of the neurohumoral pathways include the PIMA Indians, who have demonstrated a significant increase in obesity and diabetes during the last several decades resulting from changes in dietary and lifestyle habits, which when coupled with their leptin resistance, accelerated their inherent risk of disease; however, their leptin resistance existed long before their dietary changes and did not produce a health problem until changes in dietary patterns occurred following high calorie, high saturated fatty foods provided by the US government. Hence, the presence of a potential genetic problem was not realized until the behavioral changes occurred to make the problem a reality [11-18].

Efforts to manipulate the chemical processes of our bodies may result in more devastating problems than we currently have or are presently capable of understanding. For example, numerous anorexic medications have been used in an effort to result in weight loss. These medications have resulted in ocular problems [27], pulmonary problems [28, 29], hepatic [30] problems, and valvular problems [31-35]. Leptin itself was discussed throughout the Science articles as a regulator of hunger and like so many other substances found in the human body; leptin has more than one effect. It is now known, that leptin is involved with immunologic response and altering leptin levels whether by medication or dietary efforts, may improve heart disease by reducing the inflammatory component of the disease [19,20]. The utilization of medications may however affect more than just this component of leptin while the dietary changes which reduce caloric and saturated fat content have been shown to lower weight, which will not only reduce leptin levels, but the other inflammatory components of heart and other diseases as well. Our studies [2-14, 18, 20-21, 23-26] have looked at both the behavioral component of changing eating habits as well as the effect of medications. These studies have clearly shown that the successful approach to weight loss must include several key components. The problem is a behavior of excess consumption of calories and fat, not an excess of food availability. Individuals must make decisions several times a day regarding food consumption and the type of food they will consume. They do so based upon the information they hear and read. Clever advertisements and marketing by people hoping to profit by influencing human behavior has lead to more misinformation than information. These strategies have been used previously by suppliers of tobacco and alcohol products and clearly focus on influencing behavior and not genes [21-25].

Our research [2-14, 18, 20-21, 23-26] has shown that people clearly can loose weight and keep it off, by changing their eating behaviors. To do this one has to first accept that this is a behavior problem, which can and must be influenced through changes in behavior alone, instead of resigning to a naïve assumption that our genes are driving us to over consume food. An interesting twist on this approach has been the popularization of diets, which tell people they can consume as much food as they want and still lose weight. These extreme diets may suggest that one component of the diet alone is at fault, negating almost a century of scientific work establishing the need for balance with reduction in total caloric and saturated fat intake [26,27]. In fact, the reason for the success of such extreme diets is the reduction in total caloric intake, which results in weight loss. The second law of thermodynamics still applies! The consequence of extreme diets, however, is the potential adverse effects seen when heart disease, kidney disease, liver disease, bone disease and other health problems occur. As our research has shown, changes in eating behaviors with a balance in protein, carbohydrates and fat is not dependent upon the gene pool of people changing their eating patterns. Many eating patterns are learned (home, school, work, et cetera) and can be influenced through these same groups [29-32].

In the mid 1900s, we as a species, for better or worse, learned how to increase our production of our food, as well as our preservation of food by hydrogenation. We have not focused nearly enough on the quality of this food and its impact on our health. In 1999, as a member of the University of Northern Iowa advisory board, I was asked what I thought the greatest healthcare problem would be for the United States in the next century. My response, then as now, was simple and concise. Unless we change our current eating behavior we will become a bimodal society. My explanation is as follows. Those members of our society who have taken better care of themselves have not only benefited from this health of earlier years, but can currently take advantage of our ability to keep people alive longer. While we can extend the quantity of life, this does not mean we are improving the quality of life. Our school age children (those from Kindergarten through 12th grade) are showing ever-increasing problems resulting from increased weight and sedentary lifestyles. These problems affect ¼ to 1/3 of these children and include heart disease with elevated cholesterol levels, high blood pressure, diabetes mellitus and problems with weight (both overweigh and obesity). This increased incidence of health problems could very easily result in our children dying in their 20's-50's instead of their 70's-80's. As a consequence, the average life span will decrease. They will live long enough to conceive. This will, in the short span, lead to a stable population of children, and a stable population of older adults with a drop in numbers of people between 30-60 years of age. This bimodal population will lead to a healthcare crisis for two major reasons. First, the increased cost of caring for increasing numbers of people with heart disease, diabetes mellitus, hypertension, and certain types of cancer, and

secondly, because the adults who will be responsible for paying the taxes (30-60 year olds) to pay for healthcare will be fewer in number. This reduction in individuals financially supporting the healthcare system will result in a financial burden that cannot be maintained and the system will collapse, resulting in problems with delivery of healthcare and further problems. Perhaps this is the crisis, which will be necessary for us to change our behavior, analogous to watching our ancestors being eating on the Serengeti [33].

However, unlike natural disasters, which we cannot directly influence, we have produced this one and the human species need not eat itself into extinction. The same efforts being employed to develop drugs to manipulate our genetic response to eating could be put to better use to produce foods which have higher nutritional values, with less saturated fat and more complex carbohydrates. These foods are produced by many of the same companies who make the medications we take as a society. Similarly, our efforts could be directed towards improving our dietary and exercise patterns, beginning with our children and incorporating all age ranges. This would be considerably less costly to our society than the scenario outlined above. Like our eating behaviors, the decision to change the foods we make available for consumption, is also a conscious (behavior) decision, which in the end may determine the survival of the species. The only question is have we evolved enough to survive or just enough to cause our own extinction [34-36].

References

1. Ornish D, Brown SE, Scherwitz LW, Billings JH, Armstrong (1990) Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. Lancet 336: 129-133.
2. Fleming RM, Rater D, Ketcham K (1993) Reducing cholesterol and triglycerides in the elderly patient by diet alone. The council on arteriosclerosis for the 66th Scientific Sessions of the AHA, pp. 127.
3. Fleming RM, Rater D (1993) Dietary changes without medications can equally reduce cholesterol in both the young and older patient. The council on arteriosclerosis for the 66th Scientific Sessions of the AHA, pp. 128.
4. Fleming RM, Rater D, Ketcham K (1993) Studying the effect of medications on cholesterol and triglycerides in subjects not receiving dietary counseling. The council on arteriosclerosis for the 66th Scientific Sessions of the AHA, pp. 128.
5. Fleming RM, Ketcham K (1993) Dietary reinforcement is an integral component of cholesterol reduction. The council on arteriosclerosis for the 66th Scientific Sessions of the AHA, pp. 128.
6. Fleming RM, Fleming DM, Gaede R (1994) Hyperlipidemic elderly patients: Comparing diet and drug therapy. The council on arteriosclerosis for the 67th Scientific Sessions of the AHA, pp. 101.
7. Fleming RM, Fleming DM, Gaede R (1994) Treatment of hyperlipidemic patients: Diet versus drug therapy. The council on arteriosclerosis for the 67th Scientific Sessions of the AHA, pp. 101.
8. Fleming RM, Ketcham K, Fleming D, Gaede R (1995) Controlling hypercholesterolemia by diet and drug therapy in the elderly. 1st Annual Scientific Session on Cardiovascular Disease in the Elderly.
9. Fleming RM (1995) Reducing cholesterol and triglyceride levels in both the young and elderly patient by dietary changes, with and without hyperlipidemic medications. 17th World Congress of the International Union of Angiology, Westminster, London, SWI, The Royal Society of Medicine.
10. Fleming RM, Ketchum K, Fleming DM, Gaede R (1995) Investigating differences in cholesterol and triglyceride levels as influenced by diet and hyperlipidemic medications. 42nd Annual World Assembly of the American College of Angiology.
11. Fleming RM, Ketchum K, Fleming DM, Gaede R (1995) Treating Hyperlipidemia in the elderly. Angiology 46: 1075-1083.
12. Fleming RM, Ketchum K, Fleming DM, Gaede R (1996) Effect of dietary counseling on hypercholesterolemia lost unless periodic counseling continues. The 69th Scientific Sessions of the AHA. Nov
13. Fleming RM, Ketchum K, Fleming DM, Gaede R (1996) Assessing the independent effect of dietary counseling and Hypolipidemic medications on serum lipids. Angiology 47: 831-840.
14. Fleming RM, Ketchum K, Fleming DM, Gaede R (1997) Intensive dietary counseling a necessary adjunct to significantly increase the effectiveness of hypolipidemic medications. 5th World Congress on Heart Failure-Mechanisms and Management. Washington DC.
15. Fleming RM (1998) Assessing PET myocardial perfusion and viability in 32 patients undergoing risk factor modification. XVI World Congress of the International Society of Heart Research, Rhodes, Greece.
16. Fleming RM (1998) The importance of FDG in the assessment of risk factor modification outcomes. XVI World Congress of the International Society of Heart Research, Rhodes, Greece.
17. Fleming RM (1998) Determining the outcomes of risk factor modification using positron emission tomography (PET) imaging. International College of Angiology. 40th Annual World Congress. Lisbon, Portugal.
18. Fleming RM, Boyd L, Forster M (2000) Unified Theory approach reduces heart disease and recovers viable myocardium. 42nd Annual World Congress International College of Angiology. San Diego, CA.
19. Lipsenthal L, Ornish D, Fleming RM (2000) Diets to reduce weight and CV risk: Hype or hope? Lower fat is better. 73rd AHA Scientific Sessions.
20. Fleming RM (2002) The effect of low-fat, moderate fat and high fat diets on weight loss and cardiovascular disease risk factors. The Asian-Pacific Scientific Forum. 42nd Annual Conference on Cardiovascular Disease Epidemiology and Prevention.
21. Fleming RM (2002) The effect of dieting on weight loss and cardiovascular disease. 49th Annually Congress of the American College of Angiology.
22. Fleming RM (2000) The clinical importance of risk factor modification: looking at both myocardial viability (MV) and myocardial perfusion imaging (MPI). Intern J Angiol 9: 55-69.
23. Fleming RM, Boyd L, Forster M (2000) Reversing heart disease in the new millennium-the Fleming Unified Theory. Angiology 51: 617-629.
24. Fleming RM (2000) The effect of high protein diets on coronary blood flow. Angiology 51: 817-826.
25. Fleming RM (2002) The effect of high, moderate and low-fat diets on weight loss and cardiovascular disease risk factors. Preventive Cardiology V: 110-118.
26. Fleming RM (2003) Caloric intake, not carbohydrate or fat consumption, determines weight loss. Amer J Med 114: 178.
27. Rouher R, Cantat A (1962) Anorectic drugs and retinal venous complications. Ann Oculaist 195: 667-668.
28. Takahashi T, Wagenvoort CA (1983) Density of muscularized arteries in the lung: its role in congenital heart disease and its clinical significance. Arch Pathol Lab Med 107: 23-28.
29. Gertsch M, Stucki P (1970) Weitgenhend reversibele primar vaskulare pulmonale hypertonie bei einem patienten mit menocil-einnahme. Z Kreislaufforsch 59: 902.

30. Stuart KL, Bras G (1957) Veno-occlusive disease of the liver. QJ Med 26: 291.

31. Connolly HM, Crary JL, McGoon MD, Schaff HV, Edwards WD (1997) Valvular heart disease associated with Fenfluramine-phentermine. NEJM 337: 581-588.

32. Khan MA, Herzog CA, St Peter JV (1998) The prevalence of cardiac valvular insufficiency assessed by transthoracic echocardiography in obese patients treated with appetite-suppressant drugs. NEJM 339: 713-718.

33. Jick H, Vasilakis C, Weinrauch LA (1998) A population-based study on appetite suppressant drugs and the risk of cardiac valve regurgitation. NEJM 339: 719-724.

34. Weissman NJ (1999) Appetite suppressant valvulopathy: a review of current data. CVR & R, pp. 146-155.

35. Burger AJ, Sherman HB, Charlamb MJ (1999) Low prevalence of valvular heart disease in 226 phentermine-fenfluramine protocol subjects prospectively followed for up to 30 months. JACC 34: 1153-1162.

36. Fleming RM (1999) Defining and treating heart failure. Chang JB editor. Textbook of Angiology. Springer-Verlag NY, pp. 407-418.

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