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Impact of Diet and Lifestyle on Reversal of Cardiovascular Disease Markers-Diet and Lifestyle Change Primary Influencing Factor Compared to Genetic Predisposition

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Abstract

Cardio vascular disease (CVD) has remained the top cause of death across the globe for decades. It is absolutely normal to think that the CVD is genetic. But the research does not support the fact. There is a constellation of data which supports the fact that reduction in parameters like total and LDL cholesterol, weight reduction, sugar levels reduction in case of diabetics, blood pressure reduction in case of hypertensive and increased physical activity helps in prevention as well as treatment of such diseases. It has also been shown that interchanging the nucleus between two cells or removal of nucleus from a cell does not change the metabolism of the cell till it survives which means that the internal environment of the cell is more important than the DNA for its functions. Cultures where people lived for more than a hundred years with no signs of any heart disease now have their children dying at a tender age of 40's and 50's which shows clearly that it is not genetic. Change in diet has also shown reversal of the condition in patients suffering from such diseases. In this paper I have shown how changes in the diet and lifestyle in individuals have helped in improving parameters which as stated above can prevent and treat heart diseases, supporting the theory that diet and lifestyle change is primary influencing factor compared to genetics for CVD. Diet and lifestyle changes can reverse the parameters which predispose an individual to CVD.

Keywords: Cardiovascular disease; Cholesterol; Diet; Lifestyle

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Introduction

Cardio vascular diseases (CVD) are among the top reasons of death worldwide for a very long time. Research shows that various factors influenced the rates of a person suffering from CVDs. These factors also affected the survival and prognosis of the disease. Some modifiable risk factors include high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, and excessive alcohol consumption, among others [1].

Obesity, a fast growing problem worldwide which has reached epidemic levels is a risk factor for 41 adverse health conditions including CVD, heart failure and premature death for individuals including children [2-9]. Owing to the increasing rates of childhood obesity, the global life expectancy in the US will, for the first time in recent history, decline and the American heart association (AHA) has reclassified obesity as a 'major, modifiable risk factor' for coronary heart disease (CHD) [10,11]. Individuals with a central deposition of adipose tissue can experience elevated cardiovascular morbidity and mortality, including stroke, congestive heart failure, myocardial infarction and cardiovascular death, and this is independent of the association between obesity and other cardiovascular risk factors [12,13].

Patients with type 2 diabetes are at high risk of coronary heart disease (CHD) [14,15]. Furthermore patients with diabetes who experience a myocardial infarction have a poorer prognosis and a higher CHD mortality rate either immediately or in the long term that non diabetic patients with a prior myocardial infarction [16,17].

Men with higher levels of cholesterol were found to be 3 times more prone to heart diseases compared to those with slightly lower levels. High blood pressure also predisposes an individual to heart diseases [18]. Strong evidence indicates that weight loss in overweight and obese individuals reduces risk factors for diabetes and CVD [19].

Lowering elevated LDL cholesterol levels with statins has demonstrated significant reductions in cardiovascular events in patients with diabetes and CHD [20-23]. Physical activity improves glucose tolerance and sensitivity by improving non-insulin dependent glucose uptake; it improves the ratio between HDL and LDL cholesterol because it increases the activity of lipoprotein lipase; it decreases triacylglycerols, increases fibrinolysis, decreases platelet aggregation, improves oxygen uptake in the heart as well as in peripheral tissues, lowers the resting heart rate by increasing vagal tone, and lowers blood pressure. Physical activity also directly increases myocardial oxygen supply, improving myocardial contraction and electrical stability [24]. Studies have found that even people with advanced heart disease can avoid coronary bypass surgery and angioplasty by following the diet and making the other lifestyle changes [25].

Dr. Dean Ornish's lifestyle heart trial –A group of patients with advanced heart disease were put on a very low fat plant based whole food diet for a year and asked to stop smoking, take regular moderate exercise and other recreational activities. Meanwhile a control group was treated with a standard American heart association heart disease programme, which includes a significant amount of low fat animal products and utilises cholesterol lowering drugs.

Those patients who completed experimental programme achieved heretofore medically unprecedented improvements in health and vitality. On average their cholesterol dropped from 5.9 mmol/L to 4.5 mmol/L, and their LDL cholesterol dropped even more dramatically – from 4.0 mmol/L to 2.5 mmol/L. Furthermore the frequency, duration and severity of their chest pain plummented. The more closely the patients adhered to the lifestyle recommendations, the more their hearts healed. Their condition just didn't deteriorate but got better [26].

Women who ate at least 1 serving of whole grain a day had substantially lower risk of mortality, including mortality from cancer, CVD and other causes compared to those who ate less [27-30].

Japanese men living in Hawaai or California have a much higher blood cholesterol level and incidence of CHD than Japanese men living in Japan. Men living in japan had the same genetic makeup and also smoking habit was common. Cholesterol increased as their diet changed when they migrated [31,32]. The life expectancy of Okinawans who are known for living really long lives dropped 17 years on an average when they migrated [33,34]. The life expectancy changes as per the place they migrate to. With the change in diet and lifestyle the younger okinawans are also falling prey to the lifestyle diseases at young age if 40-50 years though they share the same genes of their healthy centenarian parents [33,34].

In an experiment scientists were able to show that when the nucleus of a normal cell and cancer cell are interchanged, the normal cell remains normal while the cancerous cell remained cancerous [35-37]. In another experiment it was shown that some cells could survive with normal metabolism even when their nucleus was removed altogether although they do not divide further. Enucleated cells are used as feeder cells for industrial production of virus vaccines [38-40].

Some scientists also contest with convincing facts that it is the cytoplasm which actually dictates the differentiation of cells right from the zygote state and the nucleus only helps in the process [41].

The above data shows that CVD depends on various factors which are modifiable by diet and lifestyle. The genetic predisposition or genetic immunity due to ancestral history of long and healthy life plays very little role in case of lifestyle diseases like CVD. The cell metabolism is dependent on the cellular and body environment rather than the genes in the nucleus of the cells. The most compelling and convincing experiment has been the lifestyle heart trial which for the first time successfully showed that even advance heart diseases could be reversed by changes in diet and lifestyle.

Methods

Three individuals coming from different cultural backgrounds having different food habits and lifestyle having high risk of CVD were identified. Factors predisposing them to CVD like weight, sugar levels, cholesterol and blood pressure were monitored before and after they made changes in their diet and lifestyle.

The individuals were put on a high fibre diet. The diet completely eliminated packaged foods, refined carbohydrates, sugars, fruit juices, dairy products, fried foods, refined oils while giving whole grains, whole seeds, nuts, whole pulses, whole legumes and millets. The individuals were asked to perform minimum possible exercise throughout the day every day. They were also advised to take sunbath for a few minutes every day. The progress and health was monitored on a daily basis by personal communication.

Results and Discussion

The markers of CVD like weight, total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, HbA1c (Sugar levels) before the start of the diet and follow up values after a few days were found to be as follows for 3 individual patients with completely different backgrounds **(Tables 1-3)**.

The results show a marked difference in the value of markers over a period of time. All the markers have improved. Total cholesterol reduced from over 200 mg/dL to less than 200 mg/dL which is considered as the safe range. HDL cholesterol considered as the good cholesterol has remained unchanged or even improved (increased) marginally. Similar results can be seen for weight, Sugar (HbA1c) and Triglycerides. The safe range for HbA1c is below 6 (<6.0). The diet and lifestyle changes show a modulating effect on HbA1c values bringing it close to 6.0 in cases where it is high and keeping it steady in case it is normal. Similar observation is noted for triglycerides values as well which should be below

Table 1 The markers of CVD before the start of the diet and follow up values after a few days in patient 1 (male, 40 years) Ht-6'2".

Patient 1 (Male, 40 years) Ht-6'2"				
	Markers	Day 1	Day 36	Day 226
1	Blood Glucose Fasting (mg/dL)	185	101	97
2	Total Cholesterol (mg/dL)	200.1	174	
3	HDL (mg/dL)	26.9	27	
4	LDL (mg/dL)	128.7	118	
5	Triglycerides (mg/dL)	222.2	146	
6	HbA1c (%)	6.4		6.1
7	Blood Pressure	180 / 110	140 / 90	120 / 80
8	Weight (kg)	115	99	95

Table 2 The markers of CVD before the start of the diet and follow up values after a few days in patient 2 (male, 35 years) Ht-5'11".

Patient 2 (Male, 35 years) Height 5'11"			
	Markers	Day 1	Day 41
1	Total Cholesterol (mg/dL)	221	150
2	HDL (mg/dL)	40	42
3	LDL (mg/dL)	164	95
4	Triglycerides (mg/dL)	91	66
5	HbA1c (%)	5.2	5.2
6	Blood Pressure	120/80	120 / 80
6	Weight (kg)	85	76

Table 3 The markers of CVD before the start of the diet and follow up values after a few days in patient 3 (female, 50 years) Ht- 5".

Patient 3 (Female, 50 years) Ht: 5"			
	Markers	Day 1	Day 36
1	Total Cholesterol (mg/dL)	213	181
2	HDL (mg/dL)	37	36
3	LDL (mg/dL)	144.8	114.4
4	Triglycerides (mg/dL)	156	153
5	HbA1c (%)	6.2	5.7
6	Blood Pressure	110/70	110/70
7	Weight (kg)	74	68

150 (<150). All the individuals had some family history of heart disease. But the changes in diet and lifestyle markedly improved the marker values improving their chances of preventing CVD.

Conclusion

These results confirm that the markers of CVD can be modified by diet and lifestyle. Keeping in mind these observed changes along with the various research cited in the paper it can be said that diet and lifestyle change is the primary influencing factor compared to genetics for CVD markers, as diet and lifestyle changes the internal environment of the body which primarily dictates the cell metabolism and body health overall.

References

1 Mendis S, Puska P, Norrving B (2011) Global Atlas on cardiovascular disease prevention and control. World Health Organisation, Geneva.

2 James PT, Rigby N, Leach R (2004) The obesity epidemic, metabolic syndrome and future prevention strategies. Eur J Cardiovasc Prev Rehabil 11: 3-8.

3 Adams KF, Schatzkin A, Harris TB, Kipnis V, Mouw T, et al. (2006) Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. N Engl J Med 355: 763-778.

4 Van Gaal LF, Mertens IL, De Block CE (2007) Mechanisms linking obesity with cardiovascular disease. Nature 444: 875-880.

5 Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, et al. (2004) Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. Lancet 364: 937-952.

6 Yusuf S, Vaz M, Pais P (2004) Tackling the challenge of cardiovascular disease burden in developing countries. Am Heart J 148: 1-4.

7 Ogden CL, Flegal KM, Carroll MD, Johnson CL (2002) Prevalence and trends in overweight among US children and adolescents, 1999-2000. JAMA 288: 1728-1732.

8 Teixeira PJ, Sardinha LB, Going SB, Lohman TG (2001) Total and regional fat and serum cardiovascular disease risk factors in lean and obese children and adolescents. Obes Res 9: 432-442.

9 Patterson RE, Frank LL, Kristal AR, White E (2004) A comprehensive examination of health conditions associated with obesity in older adults. Am J Prev Med 27: 385-90.

10 Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, et al. (2005) A potential decline in life expectancy in the United States in the 21st century. N Engl J Med 352: 1138-1145.

11 Eckel RH, Kahn R, Robertson RM, Rizza RA (2006) Preventing cardiovascular disease and diabetes: A call to action from the American Diabetes Association and the American Heart Association. Circulation 113: 2943-2946.

12 Lakka TA, Lakka HM, Salonen R, Kaplan GA, Salonen JT (2001) Abdominal obesity is associated with accelerated progression of carotid atherosclerosis in men. Atherosclerosis 154: 497-504.

13 Kenchaiah S, Evans JC, Levy D, Wilson PW, Benjamin EJ, et al. (2002) Obesity and the risk of heart failure. N Engl J Med 347: 305-313.

14 Almdal T, Scharling H, Jensen JS, Vestergaard H (2004) The independent effect of type 2 diabetes mellitus on ischemic heart disease, stroke, and death: a population based study of 13,000 men and women with 20 years of follow-up. Arch Intern Med 164: 1422-1426.

15 Stamler J, Vaccaro O, Neaton JD, Wentworth D (1993) Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. Diabetes Care 16: 434-444.

16 Haffner SM, Lehto S, Ronnemaa T, Pyorala K, Laakso M (1998) Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Engl J Med 339: 229-234.

17 Miettinen H, Lehto S, Salomaa V, Mahonen M, Niemela M, et al. (1998) Impact of diabetes on mortality after the first myocardial infarction: The FINMONICA Myocardial Infarction Register Study Group. Diabetes Care 21: 69-75.

18 Kannel WB, Dawber TR, Kagan A, Revotskie N, Stokes J (1961) Factors of risk in the development of Coronary Heart Disease – Six Year Follow up experience. The Framingham Study. Ann Internal Medi 55: 33-50.

19 Lavie CJ, Milani RV, Ventura HO (2009) Obesity and cardiovascular

disease: risk factor, paradox, and impact of weight loss. J Am Coll Cardiol 53: 1925-32.

20 Pyorala K, Pedersen TR, Kjekshus J, Faergeman O, Olsson AG, et al. (1997) Cholesterol lowering with simvastatin improves prognosis of diabetic patients with coronary heart disease: A subgroup analysis of the Scandinavian Simvastatin Survival Study (4S). Diabetes Care 20: 614-620.

21 Goldberg RB, Mellies MJ, Sacks FM, Moye LA, Howard BV, et al. (1998) Cardiovascular events and their reduction with pravastatin in diabetic and glucose intolerant myocardial infarction survivors with average cholesterol levels: subgroup analyses in the cholesterol and recurrent events (CARE) trial: The Care Investigators. Circulation 98: 2513-2519.

22 Keech A, Colquhoun D, Best J, Kirby A, Simes RJ, et al. (2003) Secondary prevention of cardiovascular events with long-term pravastatin in patients with diabetes or impaired fasting glucose: Results from the LIPID trial. Diabetes Care 26: 2713-2721.

23 Collins R, Armitage J, Parish S, Sleigh P, Peto R, et al. (2003) MRC/BHF heart protection study of cholesterol-lowering with simvastatinin 5963 people with diabetes: A randomised placebo- controlled trial. Lancet 361: 2005-2016.

24 Sandvik L, Erikssen J, Thaulow E, Erikssen G, Mundal R, et al. (1993) Physical fitness as a predictor of mortality among healthy, middle-aged Norwegian men. N Engl J Med 328: 533-537.

25 Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ (2005) Comparison of atkins, ornish, weight-watchers, and Zone diet for weight loss and heart disease risk resolution: A randomized trial. JAMA 293: 43-53.

26 Ornish D, Brown SE, Scherwitz LW, Billings JH, Armstrong WT, et al. (1990) Can lifestyle changes reverse coronary heart disease? The lifestyle heart trial. Lancet 336: 129-33.

27 Jacobs DR, Meyer KA, Kushi LH, Folsomet AR (1999) Is whole grain intake associated with reduced total and cause specific death rates in older women? The IOWA Women's Health Study. Am J Public Health 89: 322-329.

28 Liu S (2002) Intake of refined carbohydrates and whole grain foods in relation to risk of type 2 diabetes mellitus and coronary heart disease. J Am Coll Nutr 21: 298-306.

29 Jacobs DR, Meyer HE, Solvoll K (2001) Reduced mortality among whole grain bread eaters in men and women in the Norwegian County Study. Eur J Clin Nutr 55: 137-43.

30 Slavin JL, Jacobs D, Marquart L, Wiemer K (2001) The role of whole grains in disease prevention. J Am Diet Assoc 101: 780-785.

31 Kagan A, Harris BR, Winkelstein W, Johnson KG, Kato H, et al. (1974) Epidemiologic studies of Coronary Heart Disease and Stroke in Japanese men living in Japan, Hawaii and California. J Chron Dis 27: 345-364.

32 Kato H, Tillotson J, Nichaman MZ, Rhoads GG, Hamilton HB (1973) Epidemelogic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: Serum lipids and diets. Am J Epidemiol 97: 372-385.

33 Mizushima S, Nakada Y, Nara Y, Murakami K, Mimura G, et al. (1992) The relationship of dietary factors to CVD among Japanese in Okinawa and Japanese immigrants, originally from Okinawa, in Brazil. Hypertens Res 15: 45-55.

34 Daigaku AI (1999) Japanese Centenarians: Medical research for the final stages of human aging. In: Tauchi H, Sato T, Watanabe T (eds.). Institute for Medical Science of Aging, Aichi Medical University, Japan. pp: 85-94.

35 Israel BA, Schaeffer WI (1987) Cytoplasmic suppression of malignancy. In Vitro Cell Dev Biol 23: 627-32.

36 Israel BA, Schaeffer WI (1988) Cytoplasmic mediation of malignancy. In Vitro Cell Dev Biol 24: 487-90.

37 Shay JW, Yinong L, Werbin H (1988) Cytoplasmic suppression of tumor progression in reconstituted cells. Somat Cell Mol Genet 14: 345-50.

38 Goldman RD, Pollack R, Hopkins NH (1973) Preservation of normal behavior by enucleated cells in culture. Proc Nat Acad Si USA 70: 750-754.

39 Kojima MK (1984) Effects of D_2O on parthenogenetic activation and cleavage in the sea urchin egg. Develop Growth and Differ 26: 61-71.

40 Goldman RD (1975) Propertiesof enucleated cells. Experimental Cell Research 93: 175-83.

41 Kothari ML, Mehta LA (1984) The cytoplasmic basis of cellular differentiation--redressing the injustice done to the cytoplasm. J Postgrad Med 30: 199-206.