

Diet and Genetics

A popular area of research is to determine genetic causes of obesity, diabetes, heart disease, autoimmune disease, depression and any other illness or condition that is plaguing our society. Proposed genetic solutions will result in expensive and profit-driven procedures that do not solve the primary cause of the problem.

This area of research ignores the fact that *often* our genetic code does not determine health outcomes and it will not solve the problems of our society's rapidly failing health.

Frequently the problem is not that complicated.

PPARG, ADRB2 and FABP2 Genes - DIETFITS Trial

Gardner and colleagues chose three genes: PPARG (PPAG-gamma), ADRB2 and FABP2. They claimed that variations in these genes result in a *low-fat responsive genotype* and a *low-carbohydrate responsive genotype*.¹

PPARG² is associated with fat cell differentiation. Mutations in PPARG may be a cause of type 2 diabetes and hypertension.³

A variation of the ADRB2⁴ gene is associated with a reduced ability to breakdown fatty acids, and in women, a reduced ability to oxidise fat.⁵ A hypothesis is that this variation of the ADRB2 gene "may be an important factor in the development or progression of obesity and obesity-related disorders".

The FABP2⁶ gene encodes a protein that is involved in long-chained fatty acid metabolism and transport. Some authors suggest that variations of this protein can have an effect on fatty acid assimilation resulting in an increase fat oxidation and a reduction in insulin resistance.

The *Diet Intervention Examining The Factors Interacting with Treatment Success* (DIETFITS) study was a clinical trial including 609 adults aged 18 to 50 years without diabetes with a body mass index between 28 and 40.⁷

The objective was to determine if a healthy low-fat (HLF) diet compared to a healthy low-carbohydrate (HLC) diet affected weight and if this was impacted by the genotype pattern.

The authors claim that the participants were healthy despite being overweight and obese. Obese is defined as BMI is 30 or greater. Overweight is 25 or greater but less than 30. The concept of being overweight or obese and being healthy is not valid.⁸

The following information given about the diets in the DIETFITS study was given at the baseline and at 3, 6 and 12 months. The information below is at the completion of the study at 12 months.

- the amount of energy consumed
- amount of carbohydrate
- fat
- protein
- saturated fat
- fibre
- sugar

Some conclusions can be made from these figures but it is not sufficient to determine how healthy a diet is. What foods were they consuming?

Important A healthy diet is the result of everything that we eat.

Criteria	Units	Low-Fat Diet 12 months	Low-Carb Diet 12 months
Energy Intake	kcal	1716	1697
Carbohydrate	g	213	132
Carbohydrate	% kcal	48	30
Fat	g	57	86
Fat	% kcal	29	45
Protein	g	85	93
Protein	% kcal	21	23
Saturated Fat	g	18	28
Saturated Fat	% kcal	9	15
Fibre	g	23	19
Added Sugar	% kcal	33	23
Sugar	g / 1000 kcal	19	13

The energy intake is much lower than the usual intake. The participants would have been miserable which accounts for the high attrition rate. This was 17% for the low-fat diet and 13% for the low-carbohydrate diet. This does not include those who were lost to follow-up. 24 participants in the low-fat diet and 29 in the low-carbohydrate diet could not be contacted.

The authors conclusion was:

In the 12-month study, there was no significant difference in weight change between a healthy low-fat diet compared with a healthy low-carbohydrate diet.

Neither of the 2 hypothesized predisposing factors [genotypes] was helpful in identifying which diet was better for whom.

During the trial, there were 7 serious adverse events, all requiring hospitalisation. According to the authors, 2 of these could have been related to the study (kidney stones and diverticulitis requiring surgery). There were 18 adverse events or serious adverse events that were evenly distributed across the 2 diet groups.

At baseline, both groups consumed a comparable percentage of daily calories from fat: 34.8% for the low-fat group and 36.0% for the low-carbohydrate group.

After 12 months, the percentage of daily calories from fat was 28.7% in the low-fat group and 44.6% in the low-carbohydrate group.

A diet of 28.7% in the low-fat group is a high-fat diet NOT a low-fat diet.

Many studies attempt to define how healthy a diet is simply based on the levels of macro-nutrients. Foods are much more than the sum of fats, protein and carbohydrates. Whole-food, plant-based diets automatically removes added sugars and oils. This results in a diet high in fibre, complex carbohydrates, micronutrients, antioxidants, vitamins, minerals and thousands of phytonutrients such carotenoids and flavonoids. Individual components do not work in isolation but work synergistically.

Note the prevalence of metabolic syndrome after completion of the study. Even participants free of the condition at the start of the study managed to acquire metabolic syndrome by the study's end. Given the seriousness of the adverse effects and the lack evidence for the two dietary genotypes the only conclusion from the study is that both diets are equal appalling under all conditions.

Criteria	Units	Baseline	12 months	Low-fat diet at 12 months	Low-carb diet at 12 months
Metabolic syndrome	%	Yes	No	11.8	11.8
Metabolic syndrome	%	Yes	Yes	12.8	11.8
Metabolic syndrome	%	No	No	42.0	45.1
Metabolic syndrome	%	No	Yes	4.3	3.6

[DIETFITS Study - Data Supplement](#)

Another paper that was attempting to find a link with variations in the ADRB2 gene has concluded that the ADRB2 gene polymorphisms studied do not contribute in any important way to the risk of essential hypertension or heart attacks in subjects of European ancestry.⁹

FABP2 Gene

Pima people (The River People) are native Americans from New Mexico, central and southern Arizona and NW Mexico.

The Pima Indians of Arizona reportedly have the highest prevalence of type 2 diabetes of any population which afflicts more than half of the population over 35. ¹⁰

A group of overweight, non-diabetic participants from Arizona was examined for the presence of 3 FABP2 alleles. An allele is a different version of the same gene. The 3 alleles are referred to AA, AT and TT. Average weight for the three alleles were 99kg, 96kg and 103kg respectively.

Details of their diet was not disclosed.

There was no significant correlation between the presence of the three alleles and the weight of the participants. However, the AT and TT alleles “may potentially be more deleterious in individuals who consume a diet high in saturated long-chain fatty acids, which is characteristic of the modern, ‘Westernized’ diet.”

Therefore, the variation in the FABP2 alleles is not associated with an increase in body weight but “may be” associated with greater health risks in those who are obese.

Two Japanese studies did not find any association with FABP2 gene in health outcomes.

The first paper stated there was “no evidence for the role of the common FABP2 A54T gene polymorphism in increased plasma levels of asymmetric dimethylarginine in patients with carotid stenosis.” ¹¹

The second paper stated “Our data suggested that Ala54Thr polymorphism of the FABP2 gene is not a major contributing factor for obesity and obesity with dyslipidemia in Japanese children.” ¹²

The conclusion is that in normal-weight individuals, the FABP2 gene does not affect health outcomes.

Japanese and Hawaiian Migration Studies

During 1955 and 1956, Ancel and Margaret Keys, along with Brian Bronte-Stewart from Oxford, Noburo Kimuro and Akira Kusukawa from Japan, and Nils Larsen from Honolulu were involved in a study involving Japanese men living in Japan, Hawaii and Los Angeles

They studied Japanese living in Fukuoka, on the southern most island of Kyushu, Americans living on the air base in Fukuoka, Japanese men in Hawaii and Los Angeles.

Heart attacks were non-existent in Fukuoka, despite Japanese having a high smoking rate. Levels of cancer, high blood pressure and stroke were relatively high. One factor was the high level of sodium in the diet. This was due to salt being used to preserve food, particularly in the north and the use of mono sodium glutamate (MSG) in soy sauce.

Below is a table showing the consumption of fats in the diet of Japanese and the serum cholesterol levels.

Place	% Fat (calories)	Cholesterol (mg/dL)	Cholesterol (mmol/L)
Fukuoka	13	120.3	3.1
Honolulu	32	183.0	4.7
Los Angeles	40	212.7	5.5

Below is a table showing the percentage of calories from fats from various food sources for different populations of Japanese and Caucasians.

Source	Japanese			Caucasian	
	Fukuoka	Honolulu	LA	Honolulu	LA
Meat, eggs and dairy products	3.3	20.2	28.1	33.7	28.0
Fish and other marine animals	5.9	1.4	0.8	0.8	0.5
Vegetable sources	2.8	10.2	10.2	6.7	13.9
Total	12.0	31.8	39.1	41.2	42.4

This supports the idea that genetics plays an insignificant role in heart disease. When Japanese move and change their diets to reflect their new location, their cholesterol level changes as does the exposure to heart disease - it is not due to their genetics.

Except for the rare condition of familiar hypercholesterolaemia (FH), genetics does not play a significant role in heart disease. FH is caused by a defect of a single gene. The cells are unable to produce a protein that allows LDL cholesterol to be removed from the body. This

results in a significant build up of cholesterol in the blood. Children as young as 6 may suffer heart attacks as a result. It is much more prevalent in places such as Norway and north-west India where marriage between cousins was more prevalent.

Seven-day Adventist's Studies

A strong commitment to health has been a part of Adventist's tradition since its founding in the 1840s. There has been three large Adventist cohort studies in the United States and Canada. These studies have generated hundreds of papers, which give a valuable insight to diet and the implication for our health.

Data from the AHS-2 study shows that Adventists smoke much less frequently than the general American population (males - 1.2%, females - 1.0%) and drink less alcohol (6.6% drink alcohol). Diet is also significantly different.¹³

The consumption of meat is much less than the standard American diet, even for those who consume meat. There is a much greater prevalence of people consuming vegan and vegetarian diets.

The AHS-1 study showed 30-year-old Adventist males lives 7.3 years longer than the average 30-year-old white Californian male and with females living 4.4 years longer than the average Californian white female. For vegetarians, it is 9.5 years longer for men and 6.1 years longer for women.¹⁴

Much publicity is given to the longevity of the people of Japan and Okinawa (an archipelago that stretches from southern Japan to Taiwan). However, the population with the longest lifespan and the highest levels of health on the planet is the vegan Californian Seventh-day Adventists¹⁵

There is more than sufficient evidence indicating that whole-food, plant-based diets, based on whole grains, fruit, vegetables and legumes, are not only optimal for our health but are also the best for the environment and for the animals we share the earth with.

Such diets will be low in fats, saturated fats, sugars and high in complex carbohydrates, fibre and antioxidants.

There is no need or benefit in over-complicating our current health issues by searching for complicated and expensive genetic solutions that at present offer only theoretical solutions

to problems that frequently have a much simpler solution.

Related articles

[Wheat and the Distorted Views of William Davis](#)

[Robert Lustig and The Men Who Made Us Fat](#)

[Taiwanese Buddhist Study](#)

[Seventh-day Adventists and Health](#)

Footnotes

1. Gardner, C. D. et al. (2018) Effect of Low-Fat vs Low-Carbohydrate Diet on 12-Month Weight Loss in Overweight Adults and the Association With Genotype Pattern or Insulin Secretion: The DIETFITS Randomized Clinical Trial. *Journal of American Medical Association*. 319 (7), 667.
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 15. Fraser, G. E. & Shavlik, D. J. (2001) Ten Years of Life - Is It a Matter of Choice? *Archives of Internal Medicine*. 161 (13), 1645-1652.