UNLOCKING A LIFETIME **OF OPTIMAL HEALTH**

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Two men of the same age eat a diet low in fruits and vegetables and high in salt and saturated fat. One develops high-blood pressure, high cholesterol, and eventually heart disease, while the other lives a long life without chronic disease. In another example, two post-menopausal women consume similar diets low in choline. One develops liver dysfunction due to a choline deficiency, but the other does not.

Why these individuals experience different health outcomes despite following similar diets and living comparable lifestyles is a question that has been on the minds of nutrition and healthcare experts for decades. While professionals have suspected that genetics (the study of heredity) plays a critical role in determining how a person responds to dietary intake, research in nutrigenetics has only recently demonstrated this to be true. The result is that people can now receive personalised nutritional recommendations based on their genetic makeup to help prevent chronic illnesses down the road, such as cardiovascular disease and diabetes (known as polygenic diseases).

Nutrigenetics and personalised nutrition

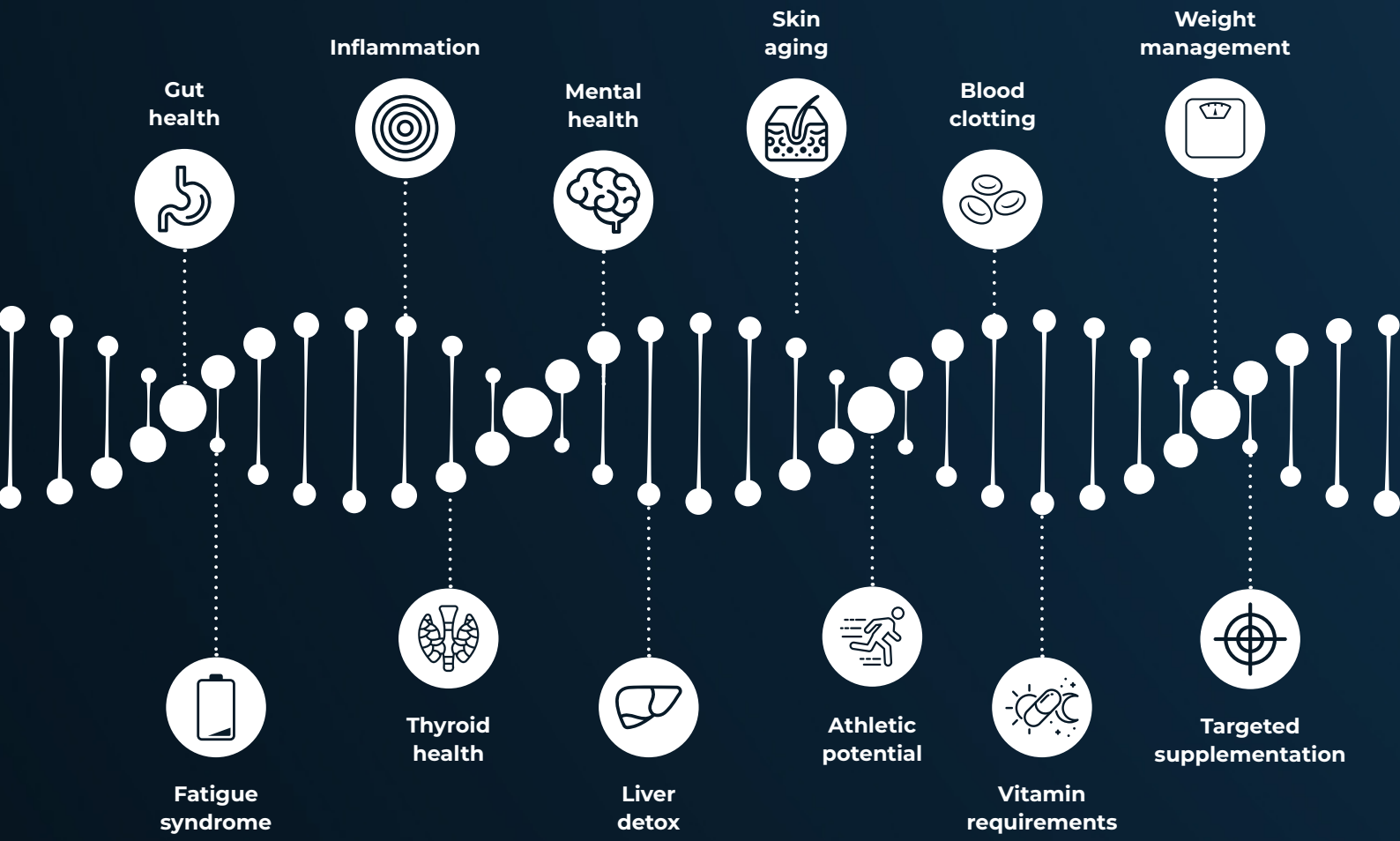
Nutrigenetics is the study of the relationships between genes, diet and health outcomes. Nutrigenomics, a related but distinct field, is the study of how genes and nutrients interact at the molecular level.

The field of nutrigenetics is relatively new and came about when, in 2003, the Human Genome Project completed its identification of all the genes in human DNA and determined the sequence of the three-billion chemical base pairs that make up human DNA. Knowing the sequences of the human genome opened the doors to examine the relationship between an individual's genetic makeup, dietary intake, and health outcomes. The excitement surrounding nutrigenetics stems from the idea that it forms the foundation of personalised nutrition.

While population-based dietary recommendations are helpful, they aren't adequate for all individuals because people respond differently to diets. Personalised nutrition bases dietary recommendations on genetic predisposition to disease. The idea is that once personalised nutrition is integrated into routine care, patients can be genotyped for specific genetic variations, made aware of their chronic disease risk and nutrient deficiencies, and given strategies to dramatically reduce their risk.

Interestingly, the genetic variation among individuals is minimal. Most people are approximately 99 percent genetically identical, with little variation in the roughly three-billion base pairs that comprise the human genome. However, this approximately one percent genetic variation leads to a wide variability in health outcomes – depending on dietary intake and other environmental exposures. Some of the genetic variation among individuals is in the form of single nucleotide polymorphisms (SNPs, pronounced 'snips').

A SNP is the variation in a single base pair in a gene, and the base pair a person has for that particular SNP varies. A gene is a sequence of DNA that codes for a protein. Humans have two copies of each gene: one copy is inherited from their mother and the other from their father. A SNP may or may not have an effect on an individual's health. Whether it does, depend on the gene associated with the SNP, the genotype of the SNP, and whether the person has one or two copies of a particular genotype of a SNP. In rare cases, a SNP can cause a disease, such as sickle-cell anaemia.



More often, SNPs affect health by increasing or decreasing the risk for chronic disease. Estimates show that there are more than 10 million SNPs in the human genome – each individual has his or her own number and pattern of SNPs. And some of these SNPs will influence a person's nutritional status. For example, a person's genetic sequence affects his or her nutrient requirements, energy use, appetite, taste and risk of chronic disease in response to diet.

Preventing chronic disease based on genotype

Chronic illnesses such as cardiovascular disease, diabetes, and cancer are prevalent in developed countries. They can be attributed, at least in part, to lifestyle and environmental factors, since our genes haven't changed appreciably over the last 100 years (although the incidence of chronic disease has skyrocketed). Furthermore, when individuals from cultures that have a low prevalence of chronic disease begin living in a culture with higher rates of chronic illness, their risk increases, suggesting that genetics aren't the sole risk factor.

Recent studies indicate that genetics affect whether a person will develop a chronic disease in response to diet and lifestyle. Fortunately, research also shows that people who are genetically predisposed to chronic disease won't necessarily develop the condition if they follow a specific preventive diet. In an age of personalised nutrition, individuals who are genetically predisposed to chronic disease will be advised to adhere to a preventive diet, since they'd be more likely to develop the disease.

Diabetes

The prevalence of Type 2 diabetes is increasing at a rapid pace. The role of diet and lifestyle in the prevention and management of diabetes has been established. Researchers have discovered several polymorphisms strongly associated with Type 2 diabetes risk and in some cases, this association may be modified by diet.

To date, a SNP in the transcription factor 7-like 2 protein gene has the strongest association with Type 2 diabetes. People with the higher-risk genotype for this SNP are approximately twice as likely to develop Type 2 diabetes than those with the lower risk genotype for the SNP.

Since the discovery of this association, several studies have demonstrated that the relationship between the genotype for the transcription factor 7-like 2 protein gene and diabetes outcome depends on diet and lifestyle. One study found that people with the higher risk genotype were more than two and a half times as likely to develop Type 2 diabetes when they consumed high glycaemic index foods and a high glycaemic load. Those who ate low glycaemic index foods and a low glycaemic load were more than one and a half times as likely to develop the disease. Therefore, people who are genetically predisposed to Type 2 diabetes may decrease their risk of the disease if they consume a low glycaemic load diet.

Obesity

Researchers found a gene associated with fat mass and obesity (FTO) to be significantly related to obesity in a series of studies that included almost 39,000 participants. Researchers found that a SNP within the FTO gene predisposed people to obesity.

Individuals who had both copies (one from each parent) of the higher-risk SNP in the FTO gene (16% of study participants) were four kilograms heavier and more than one and a half times likely to be obese compared with those who didn't have the higher-risk SNP. While this genetic association doesn't explain the obesity epidemic, it's the strongest genetic link to obesity risk.

Future Outlook

As you know, two people who have similar lifestyles and environmental exposures can have very different health outcomes. Research suggests our individual genetic composition influences our health outcome in response to lifestyle and environmental factors.

Researchers are making progress to understand the complex relationships between genes, diet, and disease risk. Personalised nutrition based on genetic composition is becoming more available and cost-effective and it takes a simple cheek swab to collect DNA, which is then sequenced for specific SNPs.