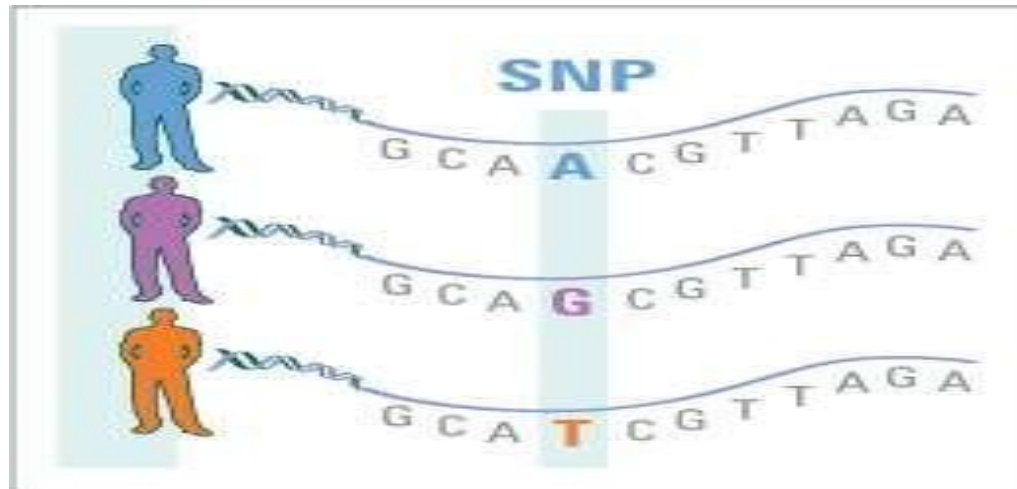




**CONCEPT OF PERSONALIZED
FOOD WITH SPECIAL
REFERENCES TO ATHLETES**

SINGLE NUCLEOTIDE POLYMORPHISM (SNP)



- Most of the genes have small sequence differences – polymorphisms- that vary among individuals
- SNP are the most common type of variation
- Specific genetic polymorphisms in human populations change their metabolic response to diet and influence the risk patterns of disease
- Some SNPs change the recipe for the gene so that either a different quantity of the protein is produced or the structure of the protein molecule is altered

NUTRITION-GENE INTERACTION

- 1. Direct interactions:** Nutrients after interacting with a receptor, behave as transcription factors that can bind to DNA and induce gene expression
- 2. Epigenetic interactions:** Nutrients can alter the structure of DNA so that gene expression is altered
- 3. Genetic variation:** Common genetic variations such as single-nucleotide polymorphisms (SNPs) can alter the expression or functionality of genes.

GENE DIET DISEASE INTERACTION

- Nutrigenetic diseases 97 per cent of the genes have known to be associated with human diseases result in monogenic diseases.
- Modifying the dietary intake can prevent some monogenetic diseases e.g., in phenylketonuria (PKU) food containing the amino acid phenylalanine,
--including high protein food such as fish, chicken, eggs, milk, cheese, dried beans, nuts and tofu must be avoided
- In case of defective aldehyde dehydrogenase enzyme, alcohol must be avoided
- Patients having galactosemia (lack of a liver enzyme to digest galactose) should avoid diets which contain lactose or galactose, including milk and milk products

NUTRIGENOMICS AND OBESITY

A study by Williams *et al.* (Am J Clin Nutr, 82: 181-7, 2005) attempted the contribution of genes to body weight and lipoprotein response

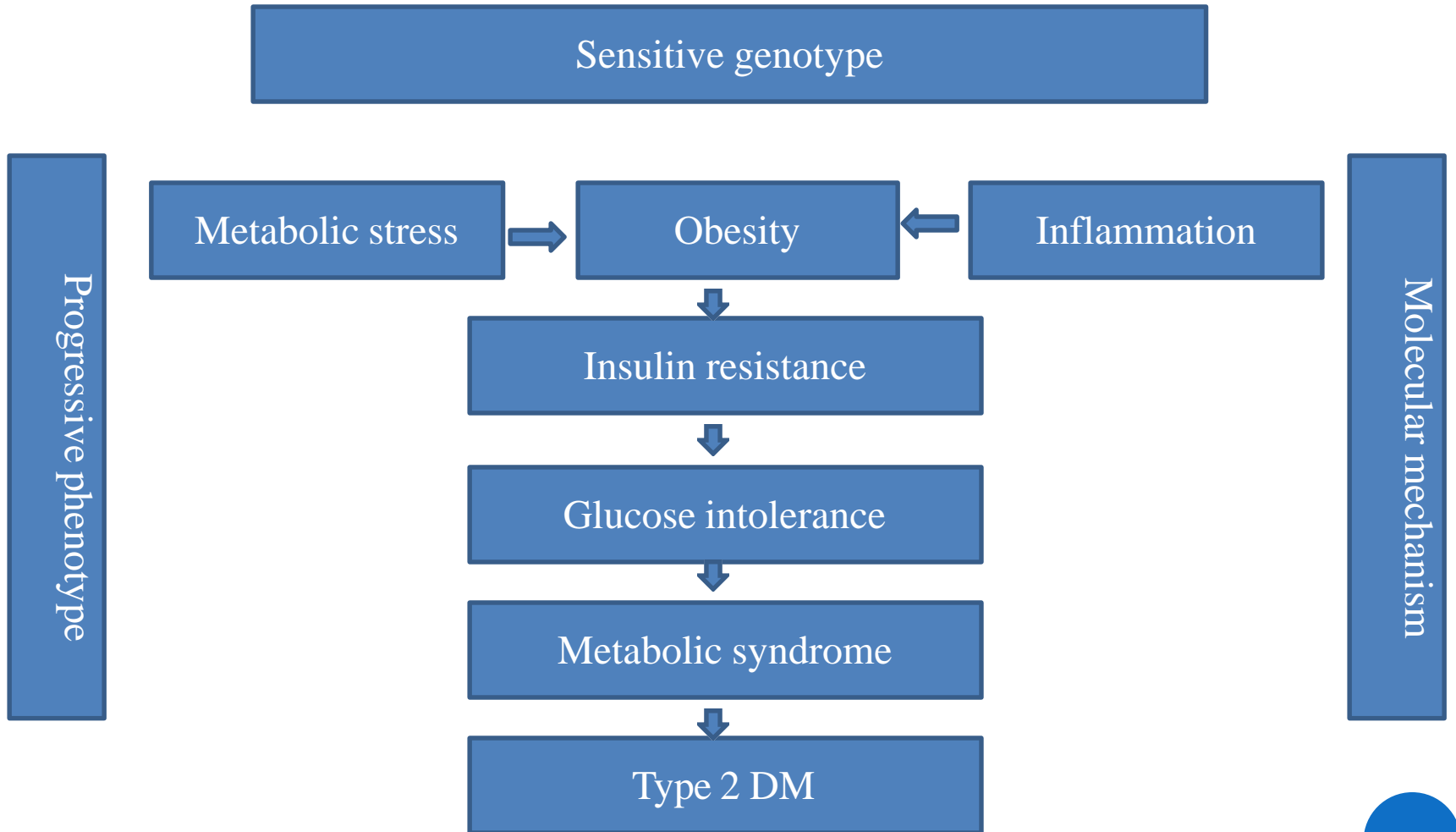
When 28 pairs of monozygotic twins are a high carbohydrate diet for six weeks then switched to high fat diet

In the study, one twin was sedentary and the other run an average of 50km/wk more than the sedentary twin which allowed researchers to assess the interaction of diet and exercise on genetic response

Results showed that decreasing dietary fat intake significantly decreased HDL and plasma Apo A-I levels in both the sedentary and running twin. LDL particles also increased significantly.

“The lipoprotein responses to the diets were not significantly different between the running and sedentary twins” which indicates that genetic rather than environmental influences may have greater impact on blood lipids.

NUTRIGENOMICS AND T2DM



NUTRIGENOMICS AND CVD

- Some of these variants are susceptible for dietary intervention, for example:

Individuals with the E4 allele in the apolipoprotein E gene show higher LDL levels with increased dietary fat intake compared with those with other (E1, E2 and E3) alleles receiving equivalent amounts of dietary fat.

- One single nucleotide polymorphism (-75G/A) in the *apoprotein A1 gene* in women is associated with an increase in HDL levels with the increase in the dietary intake of PUFA.

CONTI...

- Individuals with the *A variant* showed an increase in the protective HDL levels following increased consumption of PUFA compared with those with the *G Variant* taking similar amount of PUFA.
- One polymorphism (-515 CC) in the hepatic lipase gene is associated with an increase in protective HDL levels compared with the TT genotype (common in certain ethnic groups such as African-Americans) in response to high fat diet.

HYPERTENSION

- However, no evidence of the interactions between polymorphic variants of these genes and dietary factors are available.
- On the other hand sodium transport/metabolism- related genes such as those encoding epithelial sodium channel (ENaC) subunits, adducin, and 11
B-hydroxysteroid dehydrogenase are certainly of interest, given well-proven association between dietary salt intake and hypertension.

NUTRIGENOMICS AND CANCER

- Inherited mutations in genes can increase one's susceptibility for cancer. The risk of developing cancer can be markedly increased if there is a gene diet interaction.
- Studies of twins show that the likelihood of identical twins developing the same cancer is less than 10%, indicating that the environment plays an important role in cancer Susceptibility.
- It is clear that carcinogen metabolism-affecting polymorphisms may modify probability of contact between carcinogens and target cells, thus acting at the stage of cancer initiation.

DIET AND INCREASED RISK OF CANCER

- There is an increase risk of colorectal cancer with high consumption of red meat.
- Specific dietary irritants, such as salt and preservatives have been suggested as being carcinogens for gastric cancer.

- It was reported that a stronger relationship existed between the risk of developing hepatocellular carcinoma in Sudanese population and consumption of peanut butter with aflatoxins with the glutathione S-transferase M1 null genotype compared to those lacking the genotype.
- It is now accepted that a person's state-of-health arises from the dynamic interaction of environmental factors with his or her genetic uniqueness. These factors can positively or negatively impact the extent to which people will realize their genetic potential