gynaecmap™

Women's wellness predictor

mapmygenome.in™

Know Yourself

SAMPLE REPORT

SNAPSHOT

Endocrine and Reproductive

CONDITION	INFERENCE
AGE AT MENARCHE	Slightly high likelihood for late menarche
HYPOTHYROIDISM	Baseline risk
PREMATURE MENOPAUSE	Typical likelihood for premature menopause
ENDOMETRIOSIS	Baseline risk

Bone Health

CONDITION	INFERENCE
BONE MINERAL DENSITY	Low risk for osteoporosis; regular bone mineral density

Lifestyle Traits

CONDITION	INFERENCE
OBESITY	Slightly high risk for obesity
DIET PATTERN	Normal diet recommended
ALCOHOLISM	Slightly high risk for alcoholism
POLY UNSATURATED FATTY ACIDS	Regular levels of omega-3 and omega-6.
SKIN DETOX	Slightly reduced antioxidant levels
VITAMIN B6	Reduced levels of Vitamin B6. Increased dietary intake recommended.
VITAMIN B9	Slightly reduced levels of active Vitamin B9 (folate)
VITAMIN D	Regular levels of Vitamin D
CAFFEINE CONSUMPTION	Low caffeine consumption

Cancers

CONDITION	INFERENCE
BREAST CANCER	Baseline risk
OVARIAN CANCER	Baseline risk
THIOPURINES	High enzyme activity. Low risk for drug induced toxicity.

Heart & Diabetes

RISK FACTOR	INFERENCE
HOMOCYSTEINE LEVELS	Slightly high risk for increased homocysteine levels
HDL CHOLESTEROL	Typical likelihood for optimal levels of HDL-C
LDL CHOLESTEROL	Typical likelihood for optimal LDL -C level

SAMPLE REPORT

AGE AT MENARCHE

Menarche is defined as the onset of menstrual cycle (menses) in females. A major milestone in pubertal development, menarche is the beginning of the reproductive lifespan of a woman.

Genetics

Menarche onset is heavily influenced by genetic factors with a heritability of 50-70%.
Genes related to estrogen and hormone pathways, metabolism and synthesis have been studied for their possible role in driving pubertal timing in females. Some of them include ESR1 (estrogen receptor alpha), PGR (progesterone receptor), CCR3 (chemokine receptor 3 for endometrial function) and LIN28B. The most significant association of menarcheal age has been found with LIN28B variants. This gene produces RNA binding protein that controls embryonic development and cellular growth. Mouse-based studies have exhibited a clear role of LIN28B gene expression in tissue development and aging. The polymorphisms reported here have smaller effect on their own, but are important determinants of the genetic component of puberty, when cumulatively measured.

Your Genetic Profile for Age at Menarche

Loci	Genotype	Inference
LIN28B	AG	Slightly high likelihood for late menarche
LIN28B	AG	
LIN28B	AC	

Note: There could be other variants, not screened by Mapmygenome.

Complications

Age at menarche influences key aspects such as body composition, bone mineral density, lipid profile and insulin levels.

Early menarche (<12 years)	Late menarche (>12 years)
• Obesity, type 2 diabetes risk • Breast, endometrial and ovarian cancer risk • Shorter height and greater BMI	• Osteoporosis risk • Cardiovascular disease risk • Lower BMI

Risk Factors

- **Estrogen levels:** Exposure to endogenous and exogenous estrogens and anti-androgen agents causes fluctuation in hormonal levels, that triggers the endometrium (uterine lining) to lose its thickness and start the first ''period'' (bleeding).
- **Nutritional status:** Greater body fat (childhood obesity) and protein-rich diet trigger earlier menarche in pre-pubescents..
- **Socio-economic status:** Girls belonging to middle- and higher-income families generally mature faster than those in the lower-income category.

SAMPLE REPORT

BONE HEALTH

BONE MINERAL DENSITY

Bone mineral density (BMD) or bone density measures how much calcium and other types of minerals are in an area of bone. BMD is used as an indicator of osteoporosis and predicts the risk of bone fracture.

Genetics of BMD

Several environmental and genetic factors trigger osteoporotic fractures or determine bone mineral density in individuals. Although bones turn fragile with increasing age, heritability factors also account for disease outcome, to a considerable extent.

There are over 20 genome wide association studies reporting nearly 100 genetic markers for osteoporosis related traits. However, the association of these markers with precise causative mechanism of reduction in bone mineral density is yet to be defined.

WNT4 – ZBTB40 loci on chromosome 1, RANKL region on chromosome 13 and LRP5 region on chromosome 11 are widely reported to be associated with a greater risk (over 1.2 times) of developing osteoporotic fractures.

Your genetic profile for BMD

Loci	Genotype	Inference
WNT4 – ZBTB40	GG	Low risk for osteoporosis; regular bone mineral density
RANKL	AG	
LRP5	GG	
LRP5	GG	

Note: There could be other variants, not screened by Mapmygenome.

Complications

Bones are made of a mineral and protein scaffold filled with bone cells. Bone is continually broken down and replaced. When the rate of bone loss outpaces the rate of replacement, bones weaken, eventually leading to osteoporosis and increased risk of fracture. Low bone density (Osteopenia) increases the risk of osteoporosis and fracture.

Risk Factors

Many factors, including age, menopausal status, smoking, physical activity, diet, coexisting diseases, and pharmacologic treatments influence the risk of osteoporosis; however, one of the most clinically important risk factors is a family history of the disorder. Many studies have suggested that genetic differences may account for more than half the variance in bone mineral density between people.

SAMPLE REPORT

LIFESTYLE TRAITS

BODY MASS INDEX / OBESITY

Body mass is measured using Body Mass Index (BMI), which is the best estimate of a person's body fat based on individuals weight and height. Body mass index is defined as the individual's body mass divided by the square of his or her height and expressed in units kg/m^2.

Genetics of BMI

Family studies, linkage analysis, twin studies and genome wide association data indicate that genetic framework of an individual contributes significantly to weight related phenotype like body mass index.
Hereditary factors account for nearly 60% of variability for obesity, globally.
Genes of appetite regulation and energy homeostasis like those involved in leptin-melanocortin pathway form direct correlation with body weight. However, genome wide association studies have tagged over 25 genetic variants related to obesity with a significant odds ratio of greater than 1.12.
'Melanocortin-4 receptor' (MC4R) gene located on chromosome 18, and 'fat mass and obesity-associated' (FTO) gene on chromosome 16 are often studied to determine the person's risk for obesity. The two genes were correlated with obesity in polycystic ovary syndrome and the latter is often associated with early onset obesity.

Your genetic profile for BMI

Loci	Genotype	Inference
FTO	AT	Slightly high risk for obesity
FTO	AA	
MC4R	AG	
MC4R	AG	

Note: There could be other variants, not screened by Mapmygenome.

Complications

The higher the BMI (above 30), the greater the risk of incurring diseases such as heart disease, high blood pressure, type 2 diabetes, gallstones, breathing problems, and certain cancers.

Risk Factors

Studies indicate that 40-70% of the difference between individuals with respect to body fatness can be attributed to genetics. Many studies suggest that the continuous interaction between the environment in which a person lives and his genetic makeup greatly impact the body weight. One should understand that presence of genes that impact body weight does not directly cause obesity, but increase susceptibility to weight gain when the person lives in an environment that supports eating calories in excess and/or limiting physical activity.
Many current findings have suggest that with, except few single gene mutations that cause severe obesity, multiple genes (over 100) and their interaction with one another affect susceptibility to this complex trait.

SAMPLE REPORT

CANCER

BREAST CANCER

Your risk	Population average	Your variant score
0.74x relative to average population	1 in 5	5 out of 8

Note: The markers screened here are specific to women. These may not be applicable to men. However, certain mutations are inherited and men could be carriers.

Breast cancer is a type of cancer that affects the breast tissue, and is the most common cancer in women. It is also the main cause of death from cancer among women worldwide. Although breast cancer mainly affects women, men can also develop breast cancer.

There are 2 common types of breast cancers based on the origin. However, in rare cases, breast cancer can start in other areas of the breast.

1. Ductal carcinoma starts in the tubes (ducts) that move milk from the breast to the nipple. Most breast cancers are of this type.
2. Lobular carcinoma starts in the parts of the breast, called lobules that produce milk.

Genetics

Apart from BRCA, other important genes also contribute to breast cancer risk. Cell repair and signalling associated pathways and their proteins have been extensively studied.

- A well established risk factor for breast cancer is the FGFR2 (fibroblast growth factor receptor 2) gene that plays a key role in cellular growth, differentiation and apoptosis (programmed cell death). Variants of FGFR2 cause downregulation of this protein, which is seen is ~67% of breast tumours.
- TERT (telomerase reverse transcriptase) is another cancer-associated gene that encodes an enzyme that regulates cellular longevity by shortening chromosomal length during the process of aging. In case of tumor cells, this gene is abnormally upregulated, causing uncontrolled cell division and growth (cancer).
- MAPK1 (mitogen activated protein kinase1) is a signalling protein that is also implicated in breast cancer risk.
- TOX3 or TNCR9 (trinucleotide repeat containing 9), TNP1 (transition protein 1), SLC4A7 (solute carrier family 4, sodium co-transporter that maintains intracellular pH) are other genes relevant to breast cancer risk.

The genetic content, DNA (deoxyribonucleic acid) of your sample was analyzed and we identified some 'mutations' or changes that are associated with this medical condition.

Details regarding 'mutations' or changes detected in your DNA are given below:

Gene	Chr. #	Risk Allele	Genotype
TERT	5	A	AG
FGFR2	10	G	AA
TNP1 - DIRC3	2	A	GG
SRRM1P1 - POU5F1B	8	A	AA
FGFR2	10	A	GG
TOX3 - CHD9	16	A	AG
SLC4A7	3	A	AA
RPL26P19 - MAP3K1	5	C	AC

SAMPLE REPORT

Note: Genetic component of an individual form a minor fraction of the equation and are not the absolute causative factors that determine the outcome. There could be several other influential elements acting simultaneously that decide the final outcome of the condition.

Note: There could be other variants, not screened by Mapmygenome.

Risk Factors

Many environmental, genetic and life style factors together contribute to development of breast cancer

- **Gender**: Being a woman is the most significant risk factor for developing breast cancer. Women's risk of developing breast cancer is 100 times more than that of men. The activity of female hormones estrogen and progesterone, which regulates the constantly changing and growing breast cells puts women at much greater risk for breast cancer.
- **Age**: The risk of developing breast cancer increases as a person grows older. The life time risk of breast cancer varies with the age, from age 30 to 39, the risk is 1 in 229, or 0.43%. which drastically increases to 1 in 26, or almost 4%, by the time one in is in her 60s.
- **Family history/Genetics of breast cancer**: Studies about inheritance of breast cancer have showed that approximately one-third of women with breast cancer have one or more first-degree relatives with breast cancer. Having a first-degree relative (mother, daughter, sister) who has or had breast cancer, or multiple relatives affected by breast or ovarian cancer (especially before they turned age 50), significantly increases the risk of getting breast cancer. People who have mutations in some genes like BRCA1 and BRCA2 are more susceptible to development of breast cancer. Women with one of these defects have up to an 80% chance of getting breast cancer sometime during their life.
- **Race**: White women are slightly more likely to develop breast cancer than are African American women. Asian, Hispanic, and Native American women have a lower risk of developing and dying from breast cancer.
- **Menstrual cycle**: Women who had their menarche early (before age 12) or went through menopause late (after age 55) have an increased risk for breast cancer.
- **Exposure to ionizing radiations**: Receiving radiation therapy to the chest area as a child or young adult as treatment for another cancer, especially during teen years while breasts are still developing, significantly increases breast cancer risk.
- **Alcohol consumption**: Taking more than 1-2 glasses of alcohol per day may increase the risk of development of breast cancer
- **Obesity (postmenopausal women only)**: Many studies have shown the association of obesity with an increased risk of breast cancer, especially among postmenopausal women.
- **Postmenopausal hormone replacement therapy**: Receiving hormone therapy with estrogen over long periods of time, without any breaks, can increase the risk of breast cancer.
- **Childbirth**: Women who have never had children (nulliparity) or who had them only after age 30 (late age at first birth) have an increased risk for breast cancer
- **Oral contraceptives**: (for longer than 10 years).

Breast and Ovarian Cancer in Correlation with BRCA Mutations

BRCA genes are popularly known as 'breast cancer, early onset' genes. They are tumor suppressor in nature. BRCA genes repair damages or double stranded breaks in DNA and restore genomic integrity of human cell. Hence they play a vital role in maintaining normal cell growth in the body.

The two genes BRCA1 and BRCA2 are present on chromosome 17 and chromosome 13 respectively. The former gene carries 24 exons, spanning around 83 kb long, encoding a 220 kD nuclear protein. The latter gene has 27 exons and is larger than former spanning around 86 kb long. It encodes for a protein of 385kD.

Harmful mutations or changes in the two genes have been associated with increased risk of cancers. Most common type of cancers with BRCA in women include breast and ovarian. Defective BRCA genes account for nearly 5-10% of breast cancer cases, while the rest are of sporadic in nature.

SAMPLE REPORT

Who Is at Risk

Not every individual with BRCA mutations gets cancer. Those individuals with BRCA mutations having the Hereditary or Familial history of malignancies – especially of breast/ovarian cancer are at a very high risk of getting affected. Hence inheriting BRCA mutations from cancer family lineage (at least 3 first degree relatives or second degree relatives, second degree – mainly in case of paternal inheritance, affected with breast or ovarian cancer in 2 successive generations at a young age of within 50 years) increases the lifetime risk for developing breast cancer by 65% and ovarian cancer by 40%, with BRCA1. The lifetime risk associated with BRCA2 may be lesser (45% Breast cancer, 11% Ovarian cancer) than BRCA1. Typically such a cancer occurs at an early age of less than 50 years; however, increasing age may enhance the risk. It is usually characterized by the presence of more than one primary tumor occurring bilaterally (both breasts or both ovaries).

However, the general population (without family history of breast/ovarian cancer) is estimated to have a lifetime risk of 12% up to 85 years, for getting breast cancer with defective BRCA genes.

Nevertheless, it is important to understand that BRCA mutations may not always mean the disease will occur.

Your Profile for BRCA Mutations

Markers for BRCA1:

Loci	Marker	Genotype	Inference
BRCA1	rs55770810	GG	You may have inherited 2 variants associated with increased risk for Breast or Ovarian Cancer
	rs1799950	AA	
	rs4986852	GG	
	rs16942	GG	
	rs1799966	GG	

Note: There may be other markers/variants of BRCA1 not screened here.

Markers for BRCA2:

Loci	Marker	Genotype	Inference
BRCA2	rs144848	CC	You may have inherited 1 variant associated with increased risk for Breast or Ovarian Cancer
	rs1801426	AA	

Note: There may be other markers/variants of BRCA2 not screened here.

Measures Adopted to Prevent Cancer

Many measures can be taken to reduce cancer risk. Living healthy lifestyle is on the forefront. Regular health check ups or routine mammography is mandatory. Prophylactic surgeries are generally done to those who have family history of breast and ovarian cancers and proven risk of BRCA mutations.

OVARIAN CANCER

Your risk	Population average	Your variant score
0.47x relative to average population	4 in 100	1 out of 8

Note: The markers screened here are specific to women. These may not be applicable to men. However, certain mutations are inherited and men could be carriers.

SAMPLE REPORT

Ovarian cancer is a type of gynecological/reproductive cancer that affects the ovaries. It is the fifth most common cancer in women.

The different types of ovarian cancer are,

- Ovarian epithelial carcinomas (cancer that develops in the cells on the surface of the ovary) — this is the most common type (90%) of ovarian cancer.
- Malignant germ cell tumors (cancer that begins in egg producing cells: 1–2% of the ovarian cancers are of this type).
- Stromal tumors (cancer that develops within the cells/connective tissues that hold the ovaries together).

Genetics

Changes in some genes predispose one to risk of developing Ovarian cancer. Studies have shown the association of BRCA1 and BRCA2 tumor suppressor gene mutations with ovarian and/or breast cancer. Studies have shown that lifetime risk for ovarian cancer for women who have a harmful BRCA1 or BRCA2 mutation is 15 to 40 percent (150–400 out of 1,000) when compared to general population where the lifetime risk is 1.4 percent (14 out of 1,000). Studies have also shown the involvement other genes like TP53, PTEN, CDH1, CHEK2, ATM, MLH1, MSH2, and STK11/LKB1 with ovarian and or breast cancer.

The genetic content, DNA (deoxyribonucleic acid) of your sample was analyzed and we identified some 'mutations' or changes that are associated with this medical condition.

Details regarding 'mutations' or changes detected in your DNA are given below:

Gene	Chr. #	Risk Allele	Genotype
PVT1 - GSDMC	8	G	AG
HOXD3 - HOXD1	2	A	CC
SKAP1	17	A	GG
ANKLE1	19	A	CC
TIPARP	3	G	AA
BNC2 - RPL31P42	9	A	GG
BABAM1	19	A	GG
SKAP1	17	G	AA

Note: Genetic component of an individual form a minor fraction of the equation and are not the absolute causative factors that determine the outcome. There could be several other influential elements acting simultaneously that decide the final outcome of the condition.

Note: There could be other variants, not screened by Mapmygenome.

Risk Factors

Many genetic and life style and environmental factors are involved in the development of ovarian cancer. Some of them are listed below

- **Family history**: Women having a first degree relative (mother or sister) who has or had ovarian, breast, or uterine cancer are at greater risk of developing ovarian cancer. Females with one affected relative has 4-5% risk, where as those with 2 affected members in a family has a 7% risk of developing cancer.
- **Age**: The risk of developing ovarian cancer increases with age. Women over 50 have the highest risk of developing ovarian cancer.
- **Childbirth and menopause**: Women who have never been pregnant/have not had children, never taken the contraceptive pill, who started menstruating at an early age (before age of 12) or whose went through menopause at later than average age (after 50 years) have a higher risk of developing ovarian cancer. Most ovarian cancers are diagnosed after the menopause.
- **Genetics**: Having certain genetic mutations also increases the risk of developing ovarian cancer in small no of cases. A woman risk of developing ovarian cancer increases by 23-54% (1 in every 500 women), if she has mutations in the BRCA1 or BRCA2 genes.

SAMPLE REPORT

HOMOCYSTEINE LEVELS

Homocysteine is a common amino acid found in the blood and is acquired mostly from eating methionine rich foods like meat, fish, and dairy products. Vitamin B6 (pyridoxine), vitamin B12, and folic acid are needed to help these reactions occur. Methionine undergoes a terminal methyl transfer reaction to form Homocysteine molecule. Ideally, optimal homocysteine levels are close to 7-8 µmol/L.

Genetics

A well established MTHFR variation known to cause an amino acid change of Ala222Val in the enzyme leads to reduced enzyme activity. This affects the conversion of homocysteines leading to the metabolite accumulation in the body.
The marker is associated with susceptibility to neural tube defects, colon cancer etc.
Many studies have reported a notable association of MTHFR gene variation to Hyperhomocysteinemia. However, its correlation with CVD is not statistically significant.

Your Genetic Profile for Homocysteine Levels

Loci	Genotype	Inference
MTHFR	AG	Slightly high risk for increased homocysteine levels

Note: There could be other variants, not screened by Mapmygenome.

Complications

Increased homocysteine levels in the body lead to a condition called Hyperhomocysteinemia. Studies indicate that hyperhomocysteinemia is associated with higher risk of heart diseases and stroke among general population. Enhanced homocysteine in the body can act as an independent risk factor for CVD. Associate evidences for hyperhomocysteinemia with cognitive dysfunctions (Alzheimer's disease, dementia, schizophrenia) are also reported.
Homocysteinuria is a condition where homocysteine level in blood and urine reaches up to 100-400 µmol/L. This is an extreme condition of metabolite accumulation and the patient is generally treated with heavy doses of vitamin therapy.
Enhanced homocysteine levels may increase the risk of osteoporosis, cerebrovascular disease or kidney diseases.

Risk Factors

Many studies have reported notable association of MTHFR gene variation to hyperhomocysteinemia. However, its correlation with CVD is not statistically significant.
Globally, South Asians and Europeans are known to have increased homocysteine levels.

SAMPLE REPORT

CHOLESTEROL LEVELS

Cholesterol is a fat-like substance found in all cells of the body and is required for membrane function and fluidity. Cholesterol serves as a precursor for the biosynthesis of steroid hormones, bile acids, and vitamin D. It travels through your bloodstream in small packages called lipoproteins.

Two kinds of lipoproteins carry cholesterol throughout our body, low-density lipoproteins (LDL) and high-density lipoproteins (HDL). Having healthy levels of both types of lipoproteins is important. High blood cholesterol is a condition in which one has too much cholesterol in blood.

Genetics of cholesterol levels

Genetic factors account for nearly 40% of cholesterol concentration in the body. External factors (environment and lifestyle) hold the rest of the proportion. Familial hypercholesterolemia is an inherited condition characterized by high levels of plasma cholesterol (particularly LDL) in the body.

Apolipoprotein family of genes such as 'APOB' and 'APOC1' are known to influence LDL-C concentration in the body. Genetic variations in these genes or their regulatory regions cause hypercholesterolemia and particularly increase the plasma LDL-C levels. These genes are also associated with higher risk for heart diseases.

On the other hand, variants on LDLR gene (low density lipoprotein receptor) correlate with decreased LDL-C level.

Specific beneficial variations on chromosome 8, 16 and 18 are known to increase HDL-C level in the body. It is identified that variants on chromosomes 8p21.3 and 18q21.1 are known to increase HDL-C level by 1.5mg/dl. Furthermore, variants of TBL2 gene on chromosome 7 also contribute to high HDL-C.

Your genetic profile for cholesterol levels

Markers for LDL cholesterol levels:

Loci	Genotype	Inference
APOC1	AA	Typical likelihood for optimal LDL -C level
LDLR	CC	

Markers for HDL cholesterol levels:

Loci	Genotype	Inference
8p21.3	AA	Typical likelihood for optimal levels of HDL-C
NUTF2	GG	
16q13	CC	
18q21.1	AG	
HNF4A	GG	

Note: There could be other variants, not screened by Mapmygenome.

Complications

Although cholesterol is important for human health, high levels of cholesterol in blood causes damage to arteries and cardiovascular disease. Higher the level of HDL cholesterol blood, the lower is the chance of getting heart disease. Higher the level of LDL cholesterol in blood, higher is the chance of getting heart disease.

Risk Factors

Research has proved that both genes and diet influence cholesterol levels. Familial hypercholesteraemia is an inherited condition, characterized by increased level of LDL - cholesterol.

SAMPLE REPORT

BACKGROUND

ABOUT US

Mapmygenome™ is an Indian genomics company whose vision is to "Provide better health to Indians using technology". Our immediate focus is to provide personal genomics services unique for Indian population. With more than a decade of successful projects in the field of genomics and related domains our foray into personal genomics was on anvil for quite some time, now with the knowledge we have gained through our research and from numerous research organizations around the world, we are confident to provide people with analyzed reports of their genetic makeup. The first is a comprehensive report called **Genomepatri™**. This product **Gynaecmap™**, which is now in your hands, is a sub-panel of **Genomepatri™**.

What do we do with your DNA

When we receive your sample, the first thing we do is to isolate and extract your DNA from the deposited sample. The extracted DNA is your genetic component and is used by us to identify potentially hazardous markers in your DNA, which have proven association with health conditions we cover. The markers we look for in your DNA are called SNPs or single nucleotide polymorphisms (explained below) and these are selected by our scientists after stringent scrutiny of their association for a given health condition.

What is genetic information?

Genome is the genetic content or hereditary information of an organism, which is made up of DNA (Deoxyribonucleic Acid) in humans and other higher organisms. DNA is made up of four bases Adenine, Thymine Guanine and Cytosine designated by four letters A, T, G, C, respectively. Although the genome of all the humans is almost same, a minor difference exists among individuals. This difference, which is called genetic variation is responsible for unique phenotype and difference in the health of each individual. In most of the cases, this difference or variation is passed on to the next generation (inheritance), which confers disease susceptibility in the offspring.

UNDERSTANDING YOUR RESULTS

Relative Risk

Relative Risk (RR) is the probability of an individual with a SNP developing a disease relative to an individual without that SNP developing the disease.

RR =P(disease with SNP) / P(disease without SNP)

RR ~1 -association between SNP and disease unlikely to exist.

RR > 1 -increased risk of disease among those with that SNP.

RR < 1 -decreased risk of disease among those with that SNP.

SAMPLE REPORT

Your Risk Assessment

What it means: It is an estimate of the likelihood of developing a medical condition. In other words, it indicates the probability threat value of your mutation leading to health condition.

For Example:

Your lifetime risk: 6.8%

What it means: Individuals with your genetic variants are estimated to develop this condition in 6.8 out of every 100 persons. These results indicate your odds of developing this condition.

Your average lifetime risk: 8%

What it means: Individuals from the average population are estimated to develop this condition in 8 out of every 100 persons.

Single Nucleotide Polymorphism (SNP)

Single nucleotide polymorphism or SNP is a type of genetic variation, where in a single letter difference occurs in the DNA sequence of an individual when compared to others.

Example: Sequence 1: ---AGCCTAATGGGC----

Sequence 2: ---AGCCTAAGGGGC----

Here, in the given example, the first sequence differs from the second sequence only by a single letter (nucleotideT/G). This single letter variation affects many phenotypic traits, disease susceptibility/resistance, response to drugs, chemicals, radiation, etc.

SNP Genotyping

Genotype is the genetic makeup of an organism, and genotyping (process by which the genotype sequence is decoded) is done to understand difference in the genetic makeup between different individuals. SNP genotyping helps to analyze the SNPs present in an individual.

What your report is and what it is not

The report what we provide you is not diagnostic in nature and should not be considered as one. What we report is your genetic predisposition towards any particular health condition. If you are reported to be on the higher risk for any of the health condition we cover, it does not mean that you have or you will contract the health condition and the same applies if you are reported to be on the lower risk.

When a person develops a health condition it may be due to their genetic predisposition, lifestyle, exposure to hazardous material, environmental conditions and many more. What we provide you should help in assessing your health status on genetic level and making the right choices for your health.

GENETIC PREDISPOSITION DOES NOT MEAN PREDETERMINATION

SAMPLE REPORT

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