ProMedicDNA

Colon Health Sensor

Jane Doe
DEMO_DS

ProGenom
www.progenom.com

COVER LETTER

Dear Ms. Doe,

Your sample for the analysis arrived on in the laboratory and was evaluated according to the highest laboratory quality standards. The results were evaluated and released by two independent geneticists and molecular biologists. After obtaining the results, your personal report was compiled. We hereby convey the results to you in the format of your choice.

We would like to thank you for your trust and hope that you are satisfied with our service. We are always open to questions and suggestions. Please do not hesitate to contact us. We value your feedback. This is the only way we can continuously improve our services.

We hope the analysis meets your expectations.

Kind regards,

Dr. Daniel Wallerstorfer BSc.
Laboratory Director

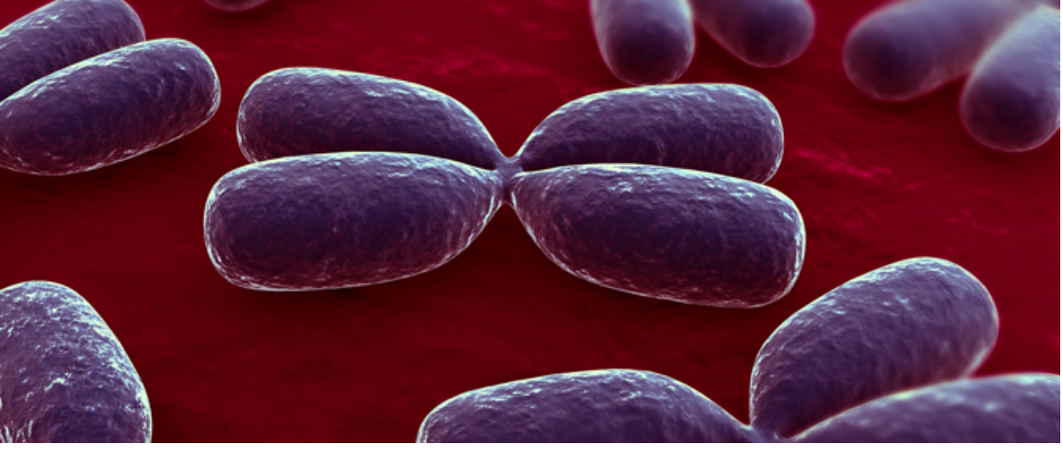
Florian Schneebauer, MSc.
Laboratory Manager

Colon Health Sensor

Personal analysis results for:
Jane Doe | Date of birth: 01/01/1990

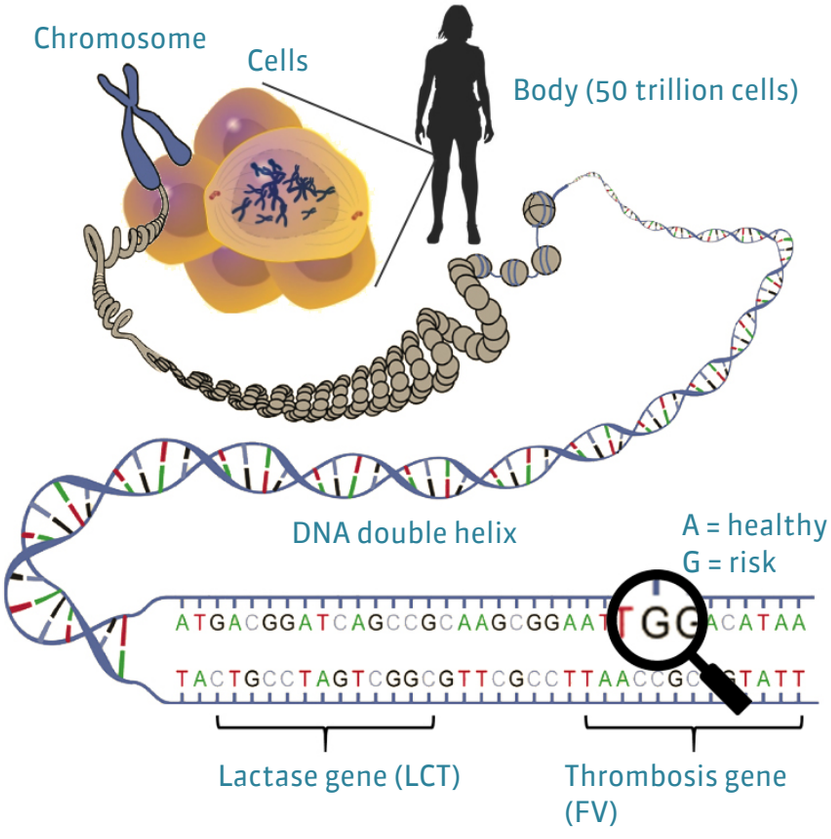
Order number:
DEMO_DS

This report contains personal medical information that is highly confidential. Data protection must be ensured.



How genes influence our health

The human body consists of about 50 trillion individual cells. Most of these cells have a nucleus, which contains 46 chromosomes. A chromosome consists of a very closely wound thread, the DNA "double helix."



DNA, the genetic code, is the blueprint of the human body. This genetic code consists of approximately 3.1 billion molecules, which are each represented by a letter. About 1% of this code makes up the genes. Each gene is an instruction for the body, usually with a single function. For example, some genes tell the body how to colour the iris and differences in these genes produce different eye colors. Every function of the body is controlled by one or more genes, including the way we break down food or medication.

Our genes are not completely error-free. The genes of each person are altered slightly by environmental effects. Most of these changes have no effect but a small number have a harmful effect. An even tinier number can produce a beneficial effect. Parents pass these changes, including defects, to their children. Thus most of our genetic defects are inherited from our parents.

In addition, our genes evolved to help us live in a completely different world, and some of our genetic traits can interact with our modern environment to create negative effects on the body. For example, the genetic predisposition to store dietary fat quickly and lose it slowly is beneficial for people who go through times when food is scarce: they have a better chance of surviving because their bodies use fat efficiently and store it for later. However, in the modern world, this trait is harmful because it programs the body to gain weight quickly and lose weight

slowly. Genes increase our risk of heart attacks, trigger asthma and allergies, cause lactose intolerance, and many other disorders.

Genetic traits can affect our health. While some genetic defects cause disease in all cases, most genetic traits just increase our risk of developing a disease. For example, a person may have genes that increase their risk for diabetes. However, not everyone at risk for diabetes actually develops the disease. Furthermore, even people with a high risk of diabetes can lower their risk with the right diet and exercise plan. Other genetic traits only cause illness when they are triggered by a specific environmental feature. For example, lactose intolerance is a genetic condition that causes a person who drinks milk to have digestive issues. A lactose-intolerant person who never drinks milk will not have any symptoms.

Thanks to the latest technologies, it is now possible to test specific genes to determine if you have genetic traits that are linked to various diseases. Based on the results of the analysis, we can develop a prevention program that significantly reduces your personal disease risk and helps you stay healthy.

A healthy lifestyle will decrease your risk of many diseases whether or not you have specific information about your genetic traits. However, we provide you with additional information that may point out other changes to your lifestyle that are not part of the standard medical advice. There are many examples, but one of the traits we test for is a gene that increases your body's ability to absorb iron. If you have this trait, you must not take iron supplements as the iron would accumulate and cause a life-threatening disease called haemochromatosis.

Experts estimate that every person carries about 2,000 genetic defects, which may affect their health, and in some cases, cause illnesses. A variety of factors can cause changes in our genes (also called mutations). In a few cases, these mutations can benefit us. However, the vast majority either have no effect or have a negative impact on our health. The best-known cause of mutations is radioactivity. Radioactive rays and particles actually impact the DNA in our cells and physically alter our genes. They mostly go unnoticed or cause deadly diseases, such as cancer, or congenital abnormality in newborns. Mutations are also caused by substances in burned food. The substances enter the cells and damage our genes, which can lead to colon cancer, among other forms of cancer. UV radiation from the sun can also damage our genes and cause diseases, such as skin cancer.

External influences can affect individual genes and disrupt their function, but the majority of our defective genes are inherited from our parents. Each embryo receives half of its genes from the father and half from the mother, resulting in a new human being with some characteristics of each parent. Whether a genetic defect is passed on, is determined randomly, and it may be that some of the children carry the defective gene and others do not.

Each person is the unique product of generations of accumulation and combination of different genetic traits. Some of those traits have negative effects on our health. With the latest technology, it is now finally possible to examine genes and determine personal health risks and strengths. In many cases, taking advantage of this knowledge, and following some precautionary measures, the diseases may be prevented. This is the next step in preventive medicine and a new generation of health care.

Action index

Discuss risks marked in orange or red with your doctor. All other results do not require any further attention assuming there are no current medical conditions.

Colon Health Sensor

PHARMACO GENETICS
Not ordered

ONCOLOGY

CARDIOVASCULAR SYSTEM
Not ordered

NEUROLOGY
Not ordered

METABOLISM
Not ordered

MOVEMENT
Not ordered

DIGESTION
Not ordered

OPHTHALMOLOGY
Not ordered

ODONTOLOGY
Not ordered

OTHERS
Not ordered

SCIENCE

ADDITIONAL INFORMATION

Colon Health Sensor

Effective prevention and early detection of colon cancer

Colon cancer

Colon cancer is one of the most common cancers in the Western world, affecting more than 6% of the population during the course of their lifetime. The chances of recovery are relatively good, provided early detection. Regular colonoscopy screening and the removal of benign intestinal polyps (which can turn into malignant cancer at a later stage) often helps detect cancer early on or prevent it altogether.

Men are more likely to be affected by colon cancer than females, with the ratio being around 60:40. About 90% of colon cancer cases occur after the age of 50. Statistics show that about 1 in 100 people between the ages of 45 and 75 has an undetected cancer, while about 3 in 100 have benign polyps in the gut that should be removed as a precautionary measure.

Risk factors

A number of environmental risk factors can increase the likelihood of colon cancer. In general, older people are increasingly affected. Vitamin D deficiency and the presence of colon polyps, as well as genetic predispositions or various disorders of the intestine (ulcerative colitis or Crohn's disease) are further factors that promote the development of the disease.

Diet is a major factor in colon cancer: the daily consumption of red meat increases the risk by 49% per 100g. The risk increases by about 70% per 100g of sausage. The risk can be reduced by 40% by increasing fibre intake.

Obesity, years of smoking, and a lack of sunlight that causes vitamin D deficiency, are also known risk factors for the onset of the disease.

Symptoms of colon cancer

Colon cancer usually remains unrecognized in the early stages, which makes early diagnosis and timely treatment much more difficult. The following symptoms may occur in advanced stages of colon cancer:

- Blood or mucus in the stool
- Intestinal cramps
- Pencil or goat kettle shaped stools
- Diarrhoea and constipation
- Flatulence
- Anemia (due to blood loss)
- Performance degradation
- Fatigue and general weakness
- Weight-loss

Genes relevant to colon cancer

Several genetic variations have been identified that are known to have an impact on the development of colon cancer. If considering these genetic variations as a whole, they can have a significant impact on the likelihood of developing a disease. The analysis of the relevant genetic variations allowed for the following conclusions:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
CASC8	rs6983267	T>G	G/G
CASC8	rs10505477	G>A	G/G
CASC8	rs10808555	A>G	A/A
CASC8	rs7837328	G>A	G/G
CASC8	rs7014346	G>A	G/G
CCND1	rs9344	G>A	G/G
CDH1	rs16260	C>A	A/A
COLCA1/COLCA2	rs3802842	A>C	A/A
CYP1A1	rs1048943	A>G	A/A
DNMT3B	rs1569686	T>G	G/T
GREM1	rs10318	C>T	C/C
IL8/CXCL8	rs4073	T>A	T/A
IL10	rs1800872	C>A	C/A
MTHFR	rs1801133	C>T	C/C
MTRR	rs1801394	A>G	G/G
SMAD7	rs12953717	C>T	C/C
TGFB1	rs1800469	A>G	A/A

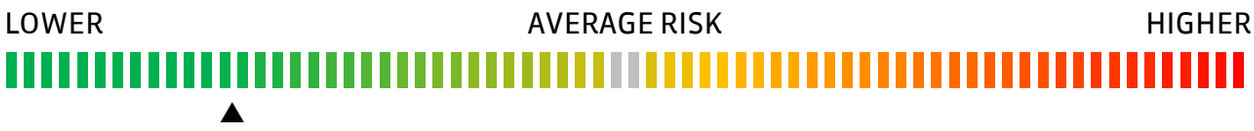
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

Here is a summary of the impact genetic variations have on your health:

- Your risk of developing colon cancer is lower than that of the population average.

Risk of colon cancer



Prevention

You do not have a genetically increased risk of developing colon cancer, therefore typical prevention and screening measures are sufficient for you. No special measures beyond the general rules of a healthy lifestyle are required.

Recommendations for your diet and lifestyle

According to the latest available information, the following nutritional recommendations apply as prevention against colon cancer:

- Reach and maintain a normal body weight.
- Ensure a balanced diet.
- Foods with large amounts of fat and sugar should only be consumed occasionally and in small quantities.
- Eat foods that are high in fibre.
- Reduce your consumption of red meat.
- Eat fish regularly.
- Alcohol should only be consumed in small quantities.
- Make sure you have enough vitamin D3.
- Do sports regularly.

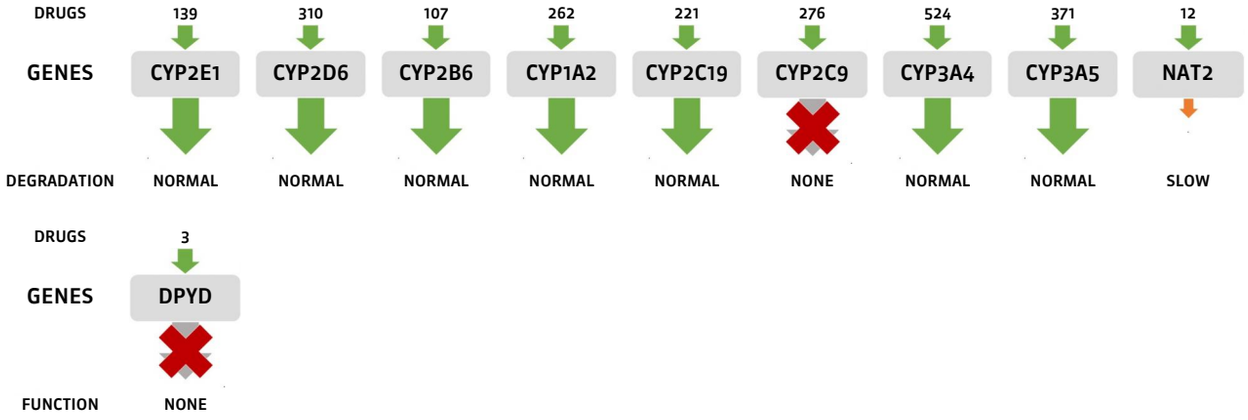
Early diagnosis

Early detection is critical to the successful treatment of any type of cancer. If the cancer is detected early on, it can usually be treated quite well and effectively.

The recommended screening program consists of the following examinations:

- CHEMICAL STOOL TEST: Annually (recommended from the age of 50)
- IMMUNOLOGICAL STOOL TEST: Annually (recommended from the age of 50)
- MAJOR COLONOSCOPY: Every 10 years (recommended from the age of 55)
- MINOR COLONOSCOPY: Every 5 years (recommended from the age of 55)

Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Alfentanil	✓	↑	↑
Buprenorphine	✓	↑	↑
Codeine	✓	✓	✓
Enflurane	✓	✓	✓
Halothane	✓	✓	✓
Isoflurane	✓	✓	✓
Methadone	✓	↑	↑
Metoclopramide	✓	✓	✓
Paracetamol	✓	✓	✓
Sevoflurane	✓	✓	✓
Trifluridine	✓	✓	✓

	Effect	Breakdown	Dose
Aprepitant	✓	↑	↑
Capecitabine	✓	✗	✗
Dolasetron	✓	✓	✓
Fentanyl	✓	↑	↑
Hydrocodone	✓	✓	✓
Levacetylmethadol	✓	↑	↑
Methotrexate	✓	✓	✓
Oxaliplatin	✓	✓	✓
Phenacetin	✓	✓	✓
Tegafur	✓	✗	✗
Zolmitriptan	✓	✓	✓

	Effect	Breakdown	Dose
Bevacizumab	✓	✓	✓
Cetuximab	✓	✓	✓
Domperidone	✓	✓	✓
Fluorouracil	✓	✗	✗
Irinotecan	✓	↑	↓
Lidocain	✓	✓	✓
Methoxyflurane	✓	✓	✓
Oxycodone	✓	↑	✓
Ropivacaine	✓	✓	✓
Tramadol	✓	↑	✓

Please note: The right choice and dose of medication is always the responsibility of the doctor. Never make your own decision on whether to stop taking a medication or changing its dose!

Legend:

Effect: Normal. Degredation: Normal. Recommendation: Normal dosage.

Effect: Normal. Degradation: Slower. Recommendation: Reduce the dosage.

Effect: Normal. Degradation: None. Recommendation: Alternative drug.

Effect: Lower. Degradation: Normal. Recommendation: Normal dosage.

Effect: Lower. Breakdown: Lower. Recommendation: Reduce the dosage.

Effect: Stronger. Degradation: Stronger. Recommendation: Normal dosage.

PHARMACO GENETICS
Not ordered

ONCOLOGY

CARDIOVASCULAR SYSTEM
Not ordered

NEUROLOGY
Not ordered

METABOLISM
Not ordered

MOVEMENT
Not ordered

DIGESTION
Not ordered

OPHTHALMOLOGY
Not ordered

ODONTOLOGY
Not ordered

OTHERS
Not ordered

SCIENCE

ADDITIONAL INFORMATION

SCIENCE

This chapter shows the science behind the test.

Colon Health Sensor

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs6983267)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
X	G/G	41%	Increased risk of colon cancer (OR: 1.51)
	G/T	39%	Increased risk of colon cancer (OR: 1.20)
	T/T	20%	No increased risk of colon cancer

References

Montazeri Z et al. Systematic meta-analyses and field synopsis of genetic association studies in colorectal adenomas. Int J Epidemiol. 2016 Feb,45(1):186-205.

Poynter JN et al. Variants on 9p24 and 8q24 are associated with risk of colorectal cancer: results from the Colon Cancer Family Registry. Cancer Res. 2007 Dec 1,67(23):11128-32.

Berndt SI et al. Pooled analysis of genetic variation at chromosome 8q24 and colorectal neoplasia risk. Hum Mol Genet. 2008 Sep 1,17(17):2665-72.

Nan H et al. Aspirin use, 8q24 single nucleotide polymorphism rs6983267, and colorectal cancer according to CTNNB1 alterations. J Natl Cancer Inst. 2013 Dec 18,105(24):1852-61.

Schafmayer C et al. Investigation of the colorectal cancer susceptibility region on chromosome 8q24.21 in a large German case-control sample. Int J Cancer. 2009 Jan 1,124(1):75-80.

Matsuo K et al. Association between an 8q24 locus and the risk of colorectal cancer in Japanese. BMC Cancer. 2009 Oct 26,9:379.

Xiong F et al. Risk of genome-wide association study-identified genetic variants for colorectal cancer in a Chinese population. Cancer Epidemiol Biomarkers Prev. 2010 Jul,19(7):1855-61.

Abulí A et al. Susceptibility genetic variants associated with colorectal cancer risk correlate with cancer phenotype. Gastroenterology. 2010 Sep,139(3):788-96, 796.e1-6.

Cui R et al. Common variant in 6q26-q27 is associated with distal colon cancer in an Asian population. Gut. 2011 Jun,60(6):799-805.

Haerian MS et al. Association of 8q24.21 loci with the risk of colorectal cancer: a systematic review and meta-analysis. J Gastroenterol Hepatol. 2011 Oct,26(10):1475-84.

Curtin K et al. Meta association of colorectal cancer confirms risk alleles at 8q24 and 18q21. Cancer Epidemiol Biomarkers Prev. 2009 Feb,18(2):616-21.

Cicek MS et al. Functional and clinical significance of variants localized to 8q24 in colon cancer. Cancer Epidemiol Biomarkers Prev. 2009 Sep,18(9):2492-500.

von Holst S et al. Association studies on 11 published colorectal cancer risk loci. Br J Cancer. 2010 Aug 10,103(4):575-80.

He J et al. Generalizability and epidemiologic characterization of eleven colorectal cancer GWAS hits in multiple populations. Cancer Epidemiol Biomarkers Prev. 2011 Jan,20(1):70-81.

Li M et al. Genetic variants on chromosome 8q24 and colorectal neoplasia risk: a case-control study in China and a meta-analysis of the published literature. PLoS One. 2011 Mar 24,6(3):e18251.

Yao K et al. Correlation Between CASC8, SMAD7 Polymorphisms and the Susceptibility to Colorectal Cancer: An Updated Meta-Analysis Based on GWAS Results. Medicine (Baltimore). 2015 Nov,94(46):e1884.

Hutter CM et al. Characterization of the association between 8q24 and colon cancer: gene-environment exploration and meta-analysis. BMC Cancer. 2010 Dec 4,10:670.

Li L et al. Association of 8q23-24 region (8q23.3 loci and 8q24.21 loci) with susceptibility to colorectal cancer: a systematic and updated meta-analysis. Int J Clin Exp Med. 2015 Nov 15,8(11):21001-13. eCollection 2015.

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs10505477)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
X	C/C	21%	No increased risk of colon cancer
	C/T	42%	Increased risk of colon cancer (OR: 1.13)
	T/T	37%	Increased risk of colon cancer (OR: 1.28)

References

Schafmayer C et al. Investigation of the colorectal cancer susceptibility region on chromosome 8q24.21 in a large German case-control sample. Int J Cancer. 2009 Jan 1,124(1):75-80.

Real LM et al. A colorectal cancer susceptibility new variant at 4q26 in the Spanish population identified by genome-wide association analysis. PLoS One. 2014 Jun 30,9(6):e101178.

Yao K et al. Correlation Between CASC8, SMAD7 Polymorphisms and the Susceptibility to Colorectal Cancer: An Updated Meta-Analysis Based on GWAS Results. Medicine (Baltimore). 2015 Nov,94(46):e1884.

Hutter CM et al. Characterization of the association between 8q24 and colon cancer: gene-environment exploration and meta-analysis. BMC Cancer. 2010 Dec 4,10:670.

Gruber SB et al. Genetic variation in 8q24 associated with risk of colorectal cancer. Cancer Biol Ther. 2007 Jul,6(7):1143-7.

Li L et al. Association of 8q23-24 region (8q23.3 loci and 8q24.21 loci) with susceptibility to colorectal cancer: a systematic and updated meta-analysis. Int J Clin Exp Med. 2015 Nov 15,8(11):21001-13. eCollection 2015.

Tan C et al. Risk of eighteen genome-wide association study-identified genetic variants for colorectal cancer and colorectal adenoma in Han Chinese. Oncotarget. 2016 Nov 22,7(47):77651-77663.

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs10808555)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
	G/G	11%	Increased risk of colon cancer (OR: 1.28)
	G/A	45%	Increased risk of colon cancer (OR: 1.13)
X	A/A	44%	No increased risk of colon cancer

References

Berndt SI et al. Pooled analysis of genetic variation at chromosome 8q24 and colorectal neoplasia risk. Hum Mol Genet. 2008 Sep 1,17(17):2665-72.

Tan C et al. Risk of eighteen genome-wide association study-identified genetic variants for colorectal cancer and colorectal adenoma in Han Chinese. Oncotarget. 2016 Nov 22,7(47):77651-77663.

Yang B et al. Genetic variants at chromosome 8q24, colorectal epithelial cell proliferation, and risk for incident, sporadic colorectal adenomas. Mol Carcinog. 2014 Feb,53 Suppl 1:E187-92.

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs7837328)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
	A/A	23%	Increased risk of colon cancer (OR: 1.37)
	A/G	45%	Increased risk of colon cancer (OR: 1.17)
X	G/G	32%	No increased risk of colon cancer

References

Berndt SI et al. Pooled analysis of genetic variation at chromosome 8q24 and colorectal neoplasia risk. Hum Mol Genet. 2008 Sep 1,17(17):2665-72.

Cui R et al. Common variant in 6q26-q27 is associated with distal colon cancer in an Asian population. Gut. 2011 Jun,60(6):799-805.

Yao K et al. Correlation Between CASC8, SMAD7 Polymorphisms and the Susceptibility to Colorectal Cancer: An Updated Meta-Analysis Based on GWAS Results. Medicine (Baltimore). 2015 Nov,94(46):e1884.

Li L et al. Association of 8q23-24 region (8q23.3 loci and 8q24.21 loci) with susceptibility to colorectal cancer: a systematic and updated meta-analysis. Int J Clin Exp Med. 2015 Nov 15,8(11):21001-13. eCollection 2015.

Tan C et al. Risk of eighteen genome-wide association study-identified genetic variants for colorectal cancer and colorectal adenoma in Han Chinese. Oncotarget. 2016 Nov 22,7(47):77651-77663.

Yang B et al. Genetic variants at chromosome 8q24, colorectal epithelial cell proliferation, and risk for incident, sporadic colorectal adenomas. Mol Carcinog. 2014 Feb,53 Suppl 1:E187-92.

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs7014346)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
X	G/G	44%	No increased risk of colon cancer
	G/A	45%	Increased risk of colon cancer (OR: 1.12)
	A/A	11%	Increased risk of colon cancer (OR: 1.25)

References

Kupfer SS et al. Genetic heterogeneity in colorectal cancer associations between African and European americans. Gastroenterology. 2010 Nov,139(5):1677-85, 1685.e1-8.

Tenesa A et al. Genome-wide association scan identifies a colorectal cancer susceptibility locus on 11q23 and replicates risk loci at 8q24 and 18q21. Nat Genet. 2008 May,40(5):631-7.

Yao K et al. Correlation Between CASC8, SMAD7 Polymorphisms and the Susceptibility to Colorectal Cancer: An Updated Meta-Analysis Based on GWAS Results. Medicine (Baltimore). 2015 Nov,94(46):e1884.

Li L et al. Association of 8q23-24 region (8q23.3 loci and 8q24.21 loci) with susceptibility to colorectal cancer: a systematic and updated meta-analysis. Int J Clin Exp Med. 2015 Nov 15,8(11):21001-13. eCollection 2015.

Tan C et al. Risk of eighteen genome-wide association study-identified genetic variants for colorectal cancer and colorectal adenoma in Han Chinese. Oncotarget. 2016 Nov 22,7(47):77651-77663.

CCND1 - Cyclin D1 (rs9344)

Cyclin D1, encoded by the CCND1 gene located on 11q13, plays an important role in the progression of the cell cycle. Cyclins function as regulators of CDK kinases and is required for progression through the G1 phase.

RES	Genotype	POP	Possible results
X	G/G	37%	No increased risk of colon cancer
	G/A	43%	Increased risk of colon cancer (OR: 1.13)
	A/A	20%	Increased risk of colon cancer (OR: 1.17)

References

Qiu H et al. Investigation of cyclin D1 rs9344 G>A polymorphism in colorectal cancer: a meta-analysis involving 13,642 subjects. Onco Targets Ther. 2016 Oct 27,9:6641-6650. eCollection 2016.

Xu XM et al. CCND1 G870A polymorphism and colorectal cancer risk: An updated meta-analysis. Mol Clin Oncol. 2016 Jun,4(6):1078-1084. Epub 2016 Apr 4.

Zahary MN et al. Polymorphisms of cell cycle regulator genes CCND1 G870A and TP53 C215G: Association with colorectal cancer susceptibility risk in a Malaysian population. Oncol Lett. 2015 Nov,10(5):3216-3222. Epub 2015 Sep 18.

Yang Y et al. Cyclin D1 G870A polymorphism contributes to colorectal cancer susceptibility: evidence from a systematic review of 22 case-control studies. PLoS One. 2012,7(5):e36813.

Yang J et al. CCND1 G870A polymorphism is associated with increased risk of colorectal cancer, especially for sporadic colorectal cancer and in Caucasians: a meta-analysis. Clin Res Hepatol Gastroenterol. 2012 Apr,36(2):169-77.

Zhang LQ et al. Cyclin D1 G870A polymorphism and colorectal cancer susceptibility: a meta-analysis of 20 populations. Int J Colorectal Dis. 2011 Oct,26(10):1249-55.

Grünhage F et al. Association of familial colorectal cancer with variants in the E-cadherin (CDH1) and cyclin D1 (CCND1) genes. Int J Colorectal Dis. 2008 Feb,23(2):147-54. Epub 2007 Oct 25.

Probst-Hensch NM et al. The effect of the cyclin D1 (CCND1) A870G polymorphism on colorectal cancer risk is modified by glutathione-S-transferase polymorphisms and isothiocyanate intake in the Singapore Chinese Health Study. Carcinogenesis. 2006 Dec,27(12):2475-82. Epub 2006 Jul 8.

Zhang W et al. Cyclin D1 and epidermal growth factor polymorphisms associated with survival in patients with advanced colorectal cancer treated with Cetuximab. Pharmacogenet Genomics. 2006 Jul,16(7):475-83.

Le Marchand L et al. Association of the cyclin D1 A870G polymorphism with advanced colorectal cancer. JAMA. 2003 Dec 3,290(21):2843-8.

Porter TR et al. Contribution of cyclin d1 (CCND1) and E-cadherin (CDH1) polymorphisms to familial and sporadic colorectal cancer. Oncogene. 2002 Mar 14,21(12):1928-33.

CDH1 - Cadherin 1 (rs16260)

Cadherin-1, a tumour suppressor gene, provides the genetic code for making a protein called epithelial cadherin. E-cadherin is one of the most important molecules in cell-cell adhesion in epithelial tissues.

RES	Genotype	POP	Possible results
	C/C	58%	No protection against colon cancer
	C/A	36%	Protection against colon cancer (OR: 0.92)
X	A/A	6%	Protection against colon cancer (OR: 0.92)

References

Grünhage F et al. Association of familial colorectal cancer with variants in the E-cadherin (CDH1) and cyclin D1 (CCND1) genes. Int J Colorectal Dis. 2008 Feb,23(2):147-54. Epub 2007 Oct 25.

Pittman AM et al. The CDH1-160C>A polymorphism is a risk factor for colorectal cancer. Int J Cancer. 2009 Oct 1,125(7):1622-5.

Wang Y et al. E-cadherin (CDH1) gene promoter polymorphism and the risk of colorectal cancer : a meta-analysis. Int J Colorectal Dis. 2012 Feb,27(2):151-8.

COLCA - Colorectal cancer associated (rs3802842)

Colorectal cancer associated protein 1 localizes in granular structures and has been associated with colorectal cancer.

RES	Genotype	POP	Possible results
X	A/A	52%	No increased risk of colon cancer
	A/C	39%	Increased risk of colon cancer (OR: 1.15)
	C/C	9%	Increased risk of colon cancer (OR: 1.32)

References

Xiong F et al. Risk of genome-wide association study-identified genetic variants for colorectal cancer in a Chinese population. Cancer Epidemiol Biomarkers Prev. 2010 Jul,19(7):1855-61.

Talseth-Palmer BA et al. Colorectal cancer susceptibility loci on chromosome 8q23.3 and 11q23.1 as modifiers for disease expression in Lynch syndrome. J Med Genet. 2011 Apr,48(4):279-84.

Tenesa A et al. Genome-wide association scan identifies a colorectal cancer susceptibility locus on 11q23 and replicates risk loci at 8q24 and 18q21. Nat Genet. 2008 May,40(5):631-7.

Niittymäki I et al. Low-penetrance susceptibility variants in familial colorectal cancer. Cancer Epidemiol Biomarkers Prev. 2010 Jun,19(6):1478-83.

He J et al. Generalizability and epidemiologic characterization of eleven colorectal cancer GWAS hits in multiple populations. Cancer Epidemiol Biomarkers Prev. 2011 Jan,20(1):70-81.

Li FX et al. Single-nucleotide polymorphism associations for colorectal cancer in southern chinese population. Chin J Cancer Res. 2012 Mar,24(1):29-35.

Giráldez MD et al. Susceptibility genetic variants associated with early-onset colorectal cancer. Carcinogenesis. 2012 Mar,33(3):613-9.

CYP1A1 - Cytochrome P450 family 1 subfamily A member 1 (rs1048943)

The haeme protein cytochrome P450-1A2 (CYP1A2) belongs to the group of cytochrome P450 enzymes and metabolizes various xenobiotic substances (including caffeine), medications and oestrogen.

RES	Genotype	POP	Possible results
X	A/A	77%	No increased risk of colon cancer
	A/G	19%	Increased risk of colon cancer (OR: 1.26)
	G/G	4%	Increased risk of colon cancer (OR: 1.54)

References

Yeh CC et al. Association between polymorphisms of biotransformation and DNA-repair genes and risk of colorectal cancer in Taiwan. J Biomed Sci. 2007 Mar,14(2):183-93. Epub 2006 Dec 27.

Zhu X et al. Associations between CYP1A1 rs1048943 A > G and rs4646903 T > C genetic variations and colorectal cancer risk: Proof from 26 case-control studies. Oncotarget. 2016 Aug 9,7(32):51365-51374.

Xu L et al. Association between CYP1A1 2454A > G polymorphism and colorectal cancer risk: A meta-analysis. J Cancer Res Ther. 2015 Oct-Dec,11(4):760-4.

Gil J et al. CYP1A1 Ile462Val polymorphism and colorectal cancer risk in Polish patients. Med Oncol. 2014 Jul,31(7):72.

Zheng Y et al. Association between CYP1A1 polymorphism and colorectal cancer risk: a meta-analysis. Mol Biol Rep. 2012 Apr,39(4):3533-40.

Jin JQ et al. CYP1A1 Ile462Val polymorphism contributes to colorectal cancer risk: a meta-analysis. World J Gastroenterol. 2011 Jan 14,17(2):260-6.

Pande M et al. Genetic variation in genes for the xenobiotic-metabolizing enzymes CYP1A1, EPHX1, GSTM1, GSTT1, and GSTP1 and susceptibility to colorectal cancer in Lynch syndrome. Cancer Epidemiol Biomarkers Prev. 2008 Sep,17(9):2393-401.

Pereira Serafim PV et al. Relationship between genetic polymorphism of CYP1A1 at codon 462 (Ile462Val) in colorectal cancer. Int J Biol Markers. 2008 Jan-Mar,23(1):18-23.

Hou L et al. CYP1A1 Val462 and NQO1 Ser187 polymorphisms, cigarette use, and risk for colorectal adenoma. Carcinogenesis. 2005 Jun,26(6):1122-8. Epub 2005 Feb 24.

Kiss I et al. Colorectal cancer risk in relation to genetic polymorphism of cytochrome P450 1A1, 2E1, and glutathione-S-transferase M1 enzymes. Anticancer Res. 2000 Jan-Feb,20(1B):519-22.

DNMT3B - DNA methyltransferase 3 beta (rs1569686)

The DNA methyltransferase 3-beta protein belongs to the group of DNA methyltransferases that can transfer methyl groups to nucleic bases of DNA. The DNA methyltransferase 3-beta protein is able to methylate cytosine de novo, which is particularly important in early embryonic development.

RES	Genotype	POP	Possible results
	G/G	13%	Protection against colon cancer (OR: 0.84)
X	G/T	30%	Protection against colon cancer (OR: 0.84)
	T/T	57%	No protection against colon cancer

References

Ho V et al. Genetic and epigenetic variation in the DNMT3B and MTHFR genes and colorectal adenoma risk. Environ Mol Mutagen. 2016 May,57(4):261-8.

Khoram-Abadi KM et al. DNMT3B -149 C>T and -579 G>T Polymorphisms and Risk of Gastric and Colorectal Cancer: a Meta-analysis. Asian Pac J Cancer Prev. 2016,17(6):3015-20.

Zhang Y et al. Association of DNMT3B -283 T > C and -579 G > T polymorphisms with decreased cancer risk: evidence from a meta-analysis. Int J Clin Exp Med. 2015 Aug 15,8(8):13028-38. eCollection 2015.

Duan F et al. Systematic evaluation of cancer risk associated with DNMT3B polymorphisms. J Cancer Res Clin Oncol. 2015 Jul,141(7):1205-20.

Bao Q et al. Correlation between polymorphism in the promoter of DNA methyltransferase-3B and the risk of colorectal cancer. Zhonghua Yu Fang Yi Xue Za Zhi. 2012 Jan,46(1):53-7.

Daraei A et al. DNA-methyltransferase 3B 39179 G > T polymorphism and risk of sporadic colorectal cancer in a subset of Iranian population. J Res Med Sci. 2011 Jun,16(6):807-13.

Zhu S et al. DNMT3B polymorphisms and cancer risk: a meta analysis of 24 case-control studies. Mol Biol Rep. 2012 Apr,39(4):4429-37.

Bao Q et al. Genetic variation in the promoter of DNMT3B is associated with the risk of colorectal cancer. Int J Colorectal Dis. 2011 Sep,26(9):1107-12.

Guo X et al. Association of the DNMT3B polymorphism with colorectal adenomatous polyps and adenocarcinoma. Mol Biol Rep. 2010 Jan,37(1):219-25.

Fan H et al. Promoter polymorphisms of DNMT3B and the risk of colorectal cancer in Chinese: a case-control study. J Exp Clin Cancer Res. 2008 Jul 28,27:24.

Hong YS et al. DNMT3b 39179GT polymorphism and the risk of adenocarcinoma of the colon in Koreans. Biochem Genet. 2007 Apr,45(3-4):155-63. Epub 2007 Feb 23.

GREM1 - Gremlin 1, DAN family BMP antagonist (rs10318)

Gremlin1 is a protein that inhibits the TGF-beta signaling pathway. It plays a role in the regulation of organogenesis, body patterns and tissue differentiation.

RES	Genotype	POP	Possible results
X	C/C	63%	No increased risk of colon cancer
	C/T	28%	Increased risk of colon cancer (OR: 1.13)
	T/T	9%	Increased risk of colon cancer (OR: 1.28)

References

Kupfer SS et al. Genetic heterogeneity in colorectal cancer associations between African and European americans. Gastroenterology. 2010 Nov,139(5):1677-85, 1685.e1-8.

Kupfer SS et al. Shared and independent colorectal cancer risk alleles in TGFβ-related genes in African and European Americans. Carcinogenesis. 2014 Sep,35(9):2025-30.

Tu L et al. Common genetic variants (rs4779584 and rs10318) at 15q13.3 contributes to colorectal adenoma and colorectal cancer susceptibility: evidence based on 22 studies. Mol Genet Genomics. 2015 Jun,290(3):901-12.

IL8 - Interleukin 8 (rs4073)

CXCL8 (interleukin-8) is a member of the chemokine family and produced by macrophages and other cell types. It acts on chemokine receptors CXCR1 and CXCR2, and is an important mediator in the immune reaction of the innate immune system response.

RES	Genotype	POP	Possible results
	A/A	31%	Increased risk of colon cancer (OR: 1.21)
X	A/T	42%	Increased risk of colon cancer (OR: 1.21)
	T/T	27%	No increased risk of colon cancer

References

Gunter MJ et al. Inflammation-related gene polymorphisms and colorectal adenoma. Cancer Epidemiol Biomarkers Prev. 2006 Jun,15(6):1126-31.

Küry S et al. Low-penetrance alleles predisposing to sporadic colorectal cancers: a French case-controlled genetic association study. BMC Cancer. 2008 Nov 7,8:326.

Bondurant KL et al. Interleukin genes and associations with colon and rectal cancer risk and overall survival. Int J Cancer. 2013 Feb 15,132(4):905-15.

Walczak A et al. The IL-8 and IL-13 gene polymorphisms in inflammatory bowel disease and colorectal cancer. DNA Cell Biol. 2012 Aug,31(8):1431-8.

IL10 - Interleukin 10 (rs1800872)

Interleukin-10 is a cytokine with multiple effects in immunoregulation and inflammation. Studies suggested the function of this cytokine as an immunoregulator in the intestinal tract.

RES	Genotype	POP	Possible results
	A/A	21%	Increased risk of colon cancer (OR: 1.25)
X	A/C	44%	Increased risk of colon cancer (OR: 1.25)
	C/C	35%	No increased risk of colon cancer

References

Yu Y et al. Polymorphisms of inflammation-related genes and colorectal cancer risk: a population-based case-control study in China. Int J Immunogenet. 2014 Aug,41(4):289-97.

Zhang YM et al. Meta-analysis of epidemiological studies of association of two polymorphisms in the interleukin-10 gene promoter and colorectal cancer risk. Genet Mol Res. 2012 Sep 25,11(3):3389-97.

Cai J et al. An Analysis of IL-10/IL-10R Genetic Factors Related to Risk of Colon Cancer and Inflammatory Bowel Disease in a Han Chinese Population. Clin Lab. 2016,62(6):1147-54.

Shi YH et al. The association of three promoter polymorphisms in interleukin-10 gene with the risk for colorectal cancer and hepatocellular carcinoma: A meta-analysis. Sci Rep. 2016 Aug 4,6:30809.

Cacev T et al. Influence of interleukin-8 and interleukin-10 on sporadic colon cancer development and progression. Carcinogenesis. 2008 Aug,29(8):1572-80.

MTRR - 5-methyltetrahydrofolate-homocysteine methyltransferase reductase (rs1801394)

Methionine is an essential, sulphur-containing proteinogenic amino acid. The synthesis of methionine is catalyzed by the methionine synthase enzyme, which in turn requires homocysteine. The protein encoded by the MTRR gene (methionine synthase reductase) regenerates the inactive methionine synthase through methylation.

RES	Genotype	POP	Possible results
	A/A	43%	No increased risk of colon cancer
	A/G	41%	Increased risk of colon cancer (OR: 1.11)
X	G/G	16%	Increased risk of colon cancer (OR: 1.23)

References

Zhou D et al. The polymorphisms in methylenetetrahydrofolate reductase, methionine synthase, methionine synthase reductase, and the risk of colorectal cancer. Int J Biol Sci. 2012,8(6):819-30.

Guimarães JL et al. Gene polymorphisms involved in folate and methionine metabolism and increased risk of sporadic colorectal adenocarcinoma. Tumour Biol. 2011 Oct,32(5):853-61.

Pardini B et al. MTHFR and MTRR genotype and haplotype analysis and colorectal cancer susceptibility in a case-control study from the Czech Republic. Mutat Res. 2011 Mar 18,721(1):74-80.

Matsuo K et al. Methionine Synthase Reductase Gene A66G Polymorphism is Associated with Risk of Colorectal Cancer. Asian Pac J Cancer Prev. 2002,3(4):353-359.

Wu PP et al. A meta-analysis of MTRR A66G polymorphism and colorectal cancer susceptibility. J BUON. 2015 May-Jun,20(3):918-22.

Han D et al. Methionine synthase reductase A66G polymorphism contributes to tumor susceptibility: evidence from 35 case-control studies. Mol Biol Rep. 2012 Feb,39(2):805-16.

SMAD7 - SMAD family member 7 (rs12953717)

SMAD Family Member 7 is an antagonist of the transforming growth factor β (TGF-β) signaling and plays an important role in modulating a large array of biological processes. Dysregulation of Smad7 is associated with a variety of human diseases.

RES	Genotype	POP	Possible results
X	C/C	50%	No increased risk of colon cancer
	C/T	39%	Increased risk of colon cancer (OR: 1.16)
	T/T	11%	Increased risk of colon cancer (OR: 1.35)

References

Thompson CL et al. Association of common genetic variants in SMAD7 and risk of colon cancer. Carcinogenesis. 2009 Jun,30(6):982-6.

Yao K et al. Correlation Between CASC8, SMAD7 Polymorphisms and the Susceptibility to Colorectal Cancer: An Updated Meta-Analysis Based on GWAS Results. Medicine (Baltimore). 2015 Nov,94(46):e1884.

Li X et al. A risk-associated single nucleotide polymorphism of SMAD7 is common to colorectal, gastric, and lung cancers in a Han Chinese population. Mol Biol Rep. 2011 Nov,38(8):5093-7.

Ho JW et al. Replication study of SNP associations for colorectal cancer in Hong Kong Chinese. Br J Cancer. 2011 Jan 18,104(2):369-75.

Jiang X et al. Genetic variations in SMAD7 are associated with colorectal cancer risk in the colon cancer family registry. PLoS One. 2013,8(4):e60464.

MTHFR - Methylenetetrahydrofolate reductase (NAD(P)H) (rs1801133)

Methylenetetrahydrofolate reductase (MTHFR) is involved in many metabolic pathways in the human body. It is responsible for the degradation of homocysteine to methionine in homocysteine metabolism.

RES	Genotype	POP	Possible results
X	C/C	59%	No protection against colon cancer
	C/T	33%	No protection against colon cancer
	T/T	8%	Protection against colon cancer (OR: 0.93)

References

Shiao SP et al. Meta-Prediction of MTHFR Gene Polymorphism Mutations and Associated Risk for Colorectal Cancer. Biol Res Nurs. 2016 Jul,18(4):357-69.

Xie SZ et al. Association between the MTHFR C677T polymorphism and risk of cancer: evidence from 446 case-control studies. Tumour Biol. 2015 Nov,36(11):8953-72.

Zhao Met al. Association of methylenetetrahydrofolate reductase C677T and A1298C polymorphisms with colorectal cancer risk: A meta-analysis. Biomed Rep. 2013 Sep,1(5):781-791. Epub 2013 Jul 15.

Guo XP et al. Association of MTHFR C677T polymorphisms and colorectal cancer risk in Asians: evidence of 12,255 subjects. Clin Transl Oncol. 2014 Jul,16(7):623-9.

Yousef AM et al. Allele and genotype frequencies of the polymorphic methylenetetrahydrofolate reductase and colorectal cancer among Jordanian population. Asian Pac J Cancer Prev. 2013,14(8):4559-65.

Teng Z et al. The 677C>T (rs1801133) polymorphism in the MTHFR gene contributes to colorectal cancer risk: a meta-analysis based on 71 research studies. PLoS One. 2013,8(2):e55332.

Delgado-Plasencia L et al. Impact of the MTHFR C677T polymorphism on colorectal cancer in a population with low genetic variability. Int J Colorectal Dis. 2013 Sep,28(9):1187-93.

Zhong S et al. Quantitative assessment of the association between MTHFR C677T polymorphism and colorectal cancer risk in East Asians. Tumour Biol. 2012 Dec,33(6):2041-51.

Yang Z et al. MTHFR C677T polymorphism and colorectal cancer risk in Asians, a meta-analysis of 21 studies. Asian Pac J Cancer Prev. 2012,13(4):1203-8.

Sheng X et al. MTHFR C677T polymorphism contributes to colorectal cancer susceptibility: evidence from 61 case-control studies. Mol Biol Rep. 2012 Oct,39(10):9669-79.

Zhou D et al. The polymorphisms in methylenetetrahydrofolate reductase, methionine synthase, methionine synthase reductase, and the risk of colorectal cancer. Int J Biol Sci. 2012,8(6):819-30.

Kim J et al. Dietary intake of folate and alcohol, MTHFR C677T polymorphism, and colorectal cancer risk in Korea. Am J Clin Nutr. 2012 Feb,95(2):405-12.

Li H et al. Methylenetetrahydrofolate reductase genotypes and haplotypes associated with susceptibility to colorectal cancer in an eastern Chinese Han population. Genet Mol Res. 2011 Dec 14,10(4):3738-46.

Pardini B et al. MTHFR and MTRR genotype and haplotype analysis and colorectal cancer susceptibility in a case-control study from the Czech Republic. Mutat Res. 2011 Mar 18,721(1):74-80.

Fernández-Peralta AM et al. Association of polymorphisms MTHFR C677T and A1298C with risk of colorectal cancer, genetic and epigenetic characteristic of tumors, and response to chemotherapy. Int J Colorectal Dis. 2010 Feb,25(2):141-51.

Gallegos-Arreola MP et al. Association of the 677C -->T polymorphism in the MTHFR gene with colorectal cancer in Mexican patients. Cancer Genomics Proteomics. 2009 May-Jun,6(3):183-8.

Koushik A et al. Nonsynonymous polymorphisms in genes in the one-carbon metabolism pathway and associations with colorectal cancer. Cancer Epidemiol Biomarkers Prev. 2006 Dec,15(12):2408-17.

Hubner RA et al. MTHFR C677T and colorectal cancer risk: A meta-analysis of 25 populations. Int J Cancer. 2007 Mar 1,120(5):1027-35.

Huang Y et al. Different roles of MTHFR C677T and A1298C polymorphisms in colorectal adenoma and colorectal cancer: a meta-analysis. J Hum Genet. 2007,52(1):73-85. Epub 2006 Nov 7.

Miao XP et al. Association between genetic variations in methylenetetrahydrofolate reductase and risk of colorectal cancer in a Chinese population. Zhonghua Yu Fang Yi Xue Za Zhi. 2005 Nov,39(6):409-11.

Le Marchand L et al. The MTHFR C677T polymorphism and colorectal cancer: the multiethnic cohort study. Cancer Epidemiol Biomarkers Prev. 2005 May,14(5):1198-203.

Ulvik A et al. Colorectal cancer and the methylenetetrahydrofolate reductase 677C -> T and methionine synthase 2756A -> G polymorphisms: a study of 2,168 case-control pairs from the JANUS cohort. Cancer Epidemiol Biomarkers Prev. 2004 Dec,13(12):2175-80.

Yin G et al. Methylenetetrahydrofolate reductase C677T and A1298C polymorphisms and colorectal cancer: the Fukuoka Colorectal Cancer Study. Cancer Sci. 2004 Nov,95(11):908-13.

Jiang Q et al. Diets, polymorphisms of methylenetetrahydrofolate reductase, and the susceptibility of colon cancer and rectal cancer. Cancer Detect Prev. 2005,29(2):146-54.

TGFB1 - Transforming growth factor beta 1 (rs1800469)

Transforming growth factor beta 1 is a cytokine which is involved in many cellular functions, including the control of cell growth, proliferation, differentiation, apoptosis, and plays an important role in controlling the immune system.

RES	Genotype	POP	Possible results
	C/C	42%	Increased risk of colon cancer (OR: 1.36)
	C/T	43%	Increased risk of colon cancer (OR: 1.18)
X	T/T	15%	No increased risk of colon cancer

References

Wang Y et al. An updated meta-analysis on the association of TGF-β1 gene promoter -509C/T polymorphism with colorectal cancer risk. Cytokine. 2013 Jan,61(1):181-7.

Liu Y et al. Meta-analyses of the associations between four common TGF-β1 genetic polymorphisms and risk of colorectal tumor. Tumour Biol. 2012 Aug,33(4):1191-9.

Fang F et al. TGFB1 509 C/T polymorphism and colorectal cancer risk: a meta-analysis. Med Oncol. 2010 Dec,27(4):1324-8.

Amirghofran Z et al. Genetic polymorphism in the transforming growth factor beta1 gene (-509 C/T and -800 G/A) and colorectal cancer. Cancer Genet Cytogenet. 2009 Apr 1,190(1):21-5.

Zhang Y et al. Genetic polymorphisms of transforming growth factor-beta1 and its receptors and colorectal cancer susceptibility: a population-based case-control study in China. Cancer Lett. 2009 Mar 8,275(1):102-8.

Chung SJ et al. Transforming growth factor-[beta]1 -509T reduces risk of colorectal cancer, but not adenoma in Koreans. Cancer Sci. 2007 Mar,98(3):401-4.

Slattery ML et al. Genetic variation in the TGF-β signaling pathway and colon and rectal cancer risk. Cancer Epidemiol Biomarkers Prev. 2011 Jan,20(1):57-69.

LEGEND: RES = your personal analysis result (marked with an X), GENOTYPE = different variations of the gene (called alleles),
POP = percent of the general population that have this genetic result,
POSSIBLE RESULTS = influence of the genetic variation.

PHARMACO GENETICS
Not ordered

ONCOLOGY

CARDIOVASCULAR SYSTEM
Not ordered

NEUROLOGY
Not ordered

METABOLISM
Not ordered

MOVEMENT
Not ordered

DIGESTION
Not ordered

OPHTHALMOLOGY
Not ordered

ODONTOLOGY
Not ordered

OTHERS
Not ordered

SCIENCE

ADDITIONAL INFORMATION

ADDITIONAL INFORMATION

In this chapter you will receive useful information

Certifications

Our laboratory is one of the most modern and automated laboratories in Europe and has numerous certifications and quality assurance systems that meet, and even exceed, international standards. The various areas of business are certified separately to the highest standards.

Laboratory diagnostics, manufacturing & sales

Quality management system in accordance with ISO 9001:2015

qualityaustria
SYSTEM CERTIFIED
ISO 9001:2015 No.14365/0

Licensed for medical genetics

Approved by the Federal Ministry of Health, Austria

BUNDESMINISTERIUM FÜR GESUNDHEIT

Cosmetic/genetic diagnostics and cosmetics manufacturing

Good manufacturing practice (GMP) in accordance with ISO 22716:2007

qualityaustria
SYSTEM CERTIFIED
EN ISO 22716:2007 No.00010/0

Food supplement manufacturing

Management system for food safety in accordance with ISO 22000:2018

qualityaustria
SYSTEM CERTIFIED
ISO 22000:2018 No.00521/0

Customer Service

Questions or comments about our service?

Our customer service team is happy to help with any enquiries or problems. You can contact us in the following ways:

- Phone +41 (0) 41 525 100.1
- office.ch@progenom.com

Our team is looking forward to your call. Customer satisfaction is our first priority. If you are not fully satisfied with our service, please let us know. We will do our best to help find a satisfactory solution to your problem.

Contact | Impressum
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

Technical details

Order number
DEMO_DS

Date of birth
01/01/1990

Established analysis methods
qRT-PCR, DNA sequencing, fragment length analysis, CNV assay, GC-MS, Immunocap ISAC, Cytolisa

Report generated
19/03/2021 17:59:50

Product codes
M7COL

Current version
V538

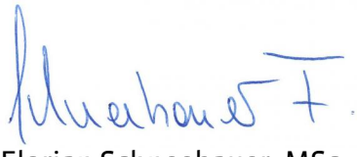
Ordering company
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

Analyzing company
DNA Plus - Zentrum für Humangenetik
Georg Wrede Strasse 13
83395 Freilassing
Deutschland

Laboratory Director

Dr. Daniel Wallerstorfer Bsc.

Laboratory Manager



Florian Schneebauer, MSc.

NOTES:


0000000000000

ProMedicDNA

Colon Health Sensor

Jane Doe
DEMO_DS

0000000000000

ProGenom
www.progenom.com