

# **Examining How Chronic Inflammation Impacts Colorectal Cancer Incidence in Atlantic Canada**

By

**Avery Cook**

Submitted in partial fulfillment of the degree requirements

for a

Bachelor of Science with Honours

April 2025

St. Francis Xavier University

Antigonish, Nova Scotia

---

Dr. Derrick Lee

---

Dr. Cory Bishop

---

Dr. Moira Galway

## Copyright Permission

Permission is herewith granted to St. Francis Xavier University to have copied for non-commercial purposes, at its discretion, this thesis upon request of individuals and institutions.

The author reserves other publication rights and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

---

Avery Cook

---

Dr. Derrick Lee

---

Date

# Table of Contents

<b>Abstract .....</b>	<b>3</b>
<b>Introduction .....</b>	<b>5</b>
<i>Review of Literature</i> .....	7
Inflammation .....	7
Inflammatory Bowel Diseases (IBD) .....	8
Colorectal Cancer .....	10
Inflammation, CRC and Atlantic Canada .....	14
<i>Study Context</i> .....	17
<b>Materials and Methods .....</b>	<b>18</b>
<i>Study Population</i> .....	18
<i>Study Design</i> .....	19
<i>Genotyping</i> .....	19
<i>Quality Control Procedures</i> .....	20
<i>Gene and SNP Selection</i> .....	20
<i>Statistical Analysis</i> .....	21
<b>Discussion .....</b>	<b>31</b>
<i>Demographic Statistics</i> .....	31
<i>Inflammatory Genes</i> .....	33
<i>Limitations</i> .....	36
<b>References .....</b>	<b>39</b>
<b>Appendix.....</b>	<b>47</b>

## Abstract

**Background:** Colorectal cancer (CRC) is the third most common cancer in Canada. Incidence of CRC in Canada is highest in the Atlantic provinces. This may be, in part, due to increased exposure to risk factors and common genetic susceptibilities in the region. Atlantic Canada is more ethnically homogenous than other parts of Canada, which may allow certain genetic mutations to be more prevalent. Chronic inflammation is a risk factor for cancer. Therefore, genetic variations in inflammatory pathways may be contributing to regional differences in CRC risk, due to their role in tumour development.

**Objectives:** This study aims to examine the associations between single nucleotide polymorphisms (SNPs) of inflammatory genes and CRC.

**Methods:** A case-control study was conducted using data from 2,500 participants from the Atlantic PATH and BC Generations Project cohorts of CanPath. Multivariable logistic regression was used to assess CRC risk among nearly 1,200 SNPs, while the Benjamini-Hochberg procedure was applied to correct for multiple testing.

**Results:** 87 SNPs were found to be significantly associated with CRC and, after adjusting for the false discovery rate, 4 remained significant. Three of the four significant SNPs also had allele distributions that differed significantly by region.

**Discussion:** These findings suggest that SNPs may contribute to the elevated CRC incidence in Atlantic Canada. Additionally, regional differences in allele distribution support the role of genetic factors in shaping disease risk. Understanding these associations could help identify populations with increased CRC risk and inform targeted prevention efforts in Atlantic Canada.

## **Acknowledgements**

I would like to thank my supervisor Dr. Derrick Lee for his support and guidance throughout this research. The knowledge I have gained and the experiences I have had from this project have been extremely valuable, and I am very grateful for the time and effort that has been required to properly support me in this endeavour. I would also like to thank my second reader Dr. Cory Bishop for his assistance with this project.

Additionally, I would like to express my gratitude to my funding sources, including the St. Francis Xavier Biology Department and Research Nova Scotia. Without their support, I would have faced constraints that could have limited my ability to fully engage with my research. This funding permitting me to focus on my research, encouraging me to grow as a researcher and reinforce my passion for biology.

Lastly, I would like to thank my family and friends for their continued love and encouragement. I would be unable to succeed as a student without assistance of my support system.

## Introduction

Cancer is one of the leading morbidities in Canada, with an estimated 2 in 5 Canadians being diagnosed with cancer during their lifetime (Canadian Cancer Statistics Advisory Committee, 2023). In 2024, colorectal cancer (CRC) is expected to account for 24,100 cancer diagnoses or almost 10% of all cases in Canada, making CRC the third most common cancer for men and women (Canadian Cancer Statistics Advisory Committee, 2023).

Rates of CRC vary regionally due to a variety of factors, including ethnicity, gender, and socioeconomic status; however, certain regions of Canada are disproportionately impacted by this disease (Saint-Jacques et al., 2023). The highest age-standardized incidence rate (ASIR) of CRC for Canadian men and women in 2023 was observed in Newfoundland and Labrador (NFLD), with an estimated 84 cases per 100,000, compared to the national ASIR of 52 cases per 100,000 (Canadian Cancer Statistics Advisory Committee, 2023). The other Atlantic provinces either see similarly elevated ASIRs; 65 cases per 100,000 in Nova Scotia (NS) and 57 cases per 100,000 in New Brunswick (NB), or rates comparable to the national average; 51 cases per 100,000 in Prince Edward Island (PEI) (Decker et al., 2023; Canadian Cancer Statistics, 2023).

While the exact cause for these high incidence rates is unknown, one possible theory is that a combination of genetic factors and environmental exposures influence risk is having a larger impact in the region than previously thought (Institute of Medicine (US), 2002). Atlantic Canada has low immigration rates and a culture strongly influenced by shared European heritage, resulting in a cohesive regional identity (Fang et al., 2023, StatCan, 2023). This has limited the region's demographic diversity compared to other provinces, which increases risk of genetic disease (Fang et al., 2023). Additionally, certain risk behaviours (e.g. high rates of alcohol consumption and a poor diet) are prevalent within this region, potentially contributing to

increased incidence as a result of gene-environment interactions (DeClercq et al., 2017, Government of Canada, 2020). Lastly, inflammatory bowel disease (IBD), which is risk factor for CRC development, is incredibly common among Atlantic Canadians (Peña-Sánchez et al., 2022). Inflammation is a hallmark of cancer pathogenesis, and the over activation of inflammatory pathways has been frequently associated with worsened CRC survival (Hanahan, 2022). IBD is prevalent in Atlantic Canada, as the incidence of IBD in Nova Scotia is not only the highest in the country (i.e. almost 55 cases per 100,000 compared to approximately 19 to 28 cases per 100,000 for other provinces) but also among some of the highest rates in the world (Kaplan et al., 2018, Zhao et al., 2021, Bernstein et al., 2006). Although data on IBD rates by province are limited, recent projections confirm similarly high and increasing IBD rates in Newfoundland which provides further supporting evidence of the impact of IBD in Atlantic Canada (Coward et al., 2024).

Given the high rates of CRC and IBD in this population, and the associations between chronic inflammation and these conditions, it is possible that there are unrecognized mutations in genes regulating inflammatory pathways that could be influencing CRC risk. Gaining a greater understanding of this community-specific risk, including genetic, environmental and cultural factors, would allow healthcare providers to better anticipate and prevent future colorectal cancer cases, standing to improve the region's overall health.

## Review of Literature

### Inflammation

#### *Inflammatory Response*

The inflammatory response is a key aspect of the innate immune system and is triggered by tissue damage as a response to infection and other exogenous exposures (Amerman, 2019). This response involves the release of inflammatory mediators by injured cells and the arrival of phagocytes to damaged areas (Amerman, 2019). Inflammatory mediators (e.g. cytokines, prostaglandin) produce signals that stimulate the migration of phagocytes (e.g. local macrophages, neutrophils and monocytes) to begin the process of healing (Amerman, 2019).

The inflammatory response can be classified as acute or chronic (Amerman, 2019). Acute inflammatory response, which occurs within the first few hours after exposure to an inflammatory stimulus, is short-lived and ends when the issue is resolved (Serhan et al., 2010, Afsar, 2011). Acute inflammation is a necessary and protective bodily response required for proper immune functioning (Fernandes et al., 2024). Conversely, chronic inflammation occurs when the acute inflammatory response continues to persist (Fernandes et al., 2024). Prolonged exposure to inflammatory mediators can disrupt DNA repair systems and cell cycle checkpoints, which can lead to the accumulation of mutations and improper protein function, resulting in severe damage to organs (Fernandes et al., 2024, Serhan et al., 2010). Unsurprisingly, chronic inflammation is associated with many life-threatening conditions and diseases, including cancer (Afsar, 2011, Fernandes et al., 2024).

## *Etiology of Chronic Inflammation*

Chronic inflammation can result from various factors including infectious agents, chronic health conditions and lifestyle factors (Pahwa et al., 2023). Chronic infectious diseases, such as hepatitis C and HIV have been known to cause chronic inflammation due to prolonged overactivation of immune cells that secrete proinflammatory compounds (Furman et al., 2019). Similarly, persistent health conditions, including rheumatoid arthritis, chronic obstructive pulmonary disease (COPD), allergies and inflammatory bowel disease (IBD), can result in chronic inflammation as a result of immune activation (Pahwa et al., 2023). Additionally, certain lifestyle factors, including low levels of physical activity and high consumption of alcohol, are associated with inflammation related pathologies because of increased cytokine production (Furman et al., 2019). Diets also plays an important role in the regulation of inflammation, as diets high in ultra-processed foods, trans fatty acids, and vitamin deficiency can increase the oxidative stress experienced by the intestines and prevent proper resolution of inflammation, leading to chronic inflammatory states (Furman et al., 2019).

## **Inflammatory Bowel Diseases (IBD)**

### *Anatomy and Physiology of the Large Intestine*

The gastrointestinal (GI) tract is the pathway by which food passes through the body (Amerman, 2019). It contains all structures connecting the mouth to the anus, including the esophagus, stomach, and the small and large intestine (Amerman, 2019). The large intestine contains three main sections: the colon, the rectum, and the anal canal. The colon and rectum are made up of four layers of tissue: the mucosa, which lines the lumen of the digestive tract; the submucosa, which is made up of fibrous connective tissue; the muscularis propria, which is a muscular layer responsible for contractions of the intestine; and the serosa, which surrounds the

outside of the organ (Bass, 2019). The large intestine also contains large numbers of symbiotic bacteria, known as the gut flora or microbiome, that serve many purposes in the human body, including stimulation of the immune system (Amerman, 2019).

### *Inflammatory Bowel Diseases*

Conditions involving the chronic inflammation of the large intestine, including Crohn's disease (CD) and Ulcerative Colitis (UC), are collectively known as inflammatory bowel diseases (IBD) (Rubin et al., 2012). IBD should not be confused with irritable bowel syndrome (IBS), which is a condition that impacts the functionality of the large intestine but is not associated with chronic inflammation (Azzouz & Sharma, 2023). CD is mainly associated with the ileum, which is the most distal segment of the small intestine, and the colon (Rubin et al., 2012). UC is a disease mainly of the colon and involves inflammation of the intestinal mucosa (Rubin et al., 2012, Fuss et al., 2004). The causes of IBD are not fully understood, but it is mainly attributed to a combination of genetic factors, overactivation of the intestinal immune response, exposure to certain environmental factors, and disruption to gut microflora (Salla et al., 2023).

### *Incidence and Risk Factors of IBD*

Global rates of IBD vary worldwide, with incidence rates ranging from 10 to 30 cases per 100,000; however, the Western world is disproportionately affected by this disease, and Canada has one of the highest rates of IBD in the world. (Kaplan, 2015). In 2023, an estimated 322,600 people in Canada were living with IBD, which is expected to increase to 470,000 by 2035 (Crohn's Colitis Canada, 2023). Moreover, IBD incidence in Canada, which was an estimated 30 cases per 100,000 in 2023, is projected to increase to 32.1 cases per 100,000 by 2035 (Coward et

al., 2023). Atlantic Canada also sees a relatively high incidence of IBD compared to other regions. Nova Scotia records the highest incidence of IBD that is well above the national average, with a reported 51.8 cases per 100,000 people (Kaplan et al., 2019). Discrepancies in IBD incidence may be due to differences in the distribution of risk factors, as they can vary geographically, as well as within different ethnic groups (Windsor, 2023).

## **Colorectal Cancer**

### *Overview of Cancer*

Cancer describes a group of diseases characterized by the unregulated growth and proliferation of cells (Amerman, 2019). It is the consequence of various genetic mutations that disrupt the normal regulation of cellular growth, reproduction, and apoptosis that can lead to tumour development (Amerman, 2019, Fernandez et al., 2024). Although cancers are very diverse and can develop in any region of the body, there are hallmark traits that remain consistent across all types (Hanahan, 2022). The hallmarks, including sustained proliferative signalling, evading growth suppressors, invasion, and replicative immortality, evolve progressively over time and lead to cancer metastasis (Hanahan, 2022). These hallmarks are driven by two enabling processes: genetic instability and inflammation (Hanahan, 2022).

### *Overview of Colorectal Cancer*

Colorectal Cancer (CRC) is a cancer of the colon and/or rectum (Duan et al., 2022). CRC initially forms in the mucosa and submucosa of the intestine and frequently forms in structures known as colon polyps (Sawicki et al., 2021). As the symptoms of CRC are relatively benign, the majority of patients are diagnosed in the latter stages of the disease when it has metastasized and infiltrated the lymphatic system or bloodstream (Bryan et al., 2018, Duan et al., 2022). Almost

half of CRC cases were identified at stage III or IV compared to 20% for breast cancer in 2018 (Blair & Datta, 2020). Although prognosis is positive if caught early (i.e. Stages I/II have a 5-year survival rate around 90%), late-stage diagnosis (i.e. Stage 4) has a 5-year survival of 11-13% (Ellison & Saint-Jacques, 2023). Various CRC screening methods are currently in use, including digital rectal exam, colonoscopy, and stool testing (Canadian Cancer Society, 2024). Improved screening behaviours and enhanced treatment options have increased overall survival; however, the rates of CRC in individuals under 50 (i.e. early onset colorectal cancer) have increased, which highlights the importance of screening programs in regions more prone to this disease (Miller et al., 2019, Brenner et al., 2017).

### *CRC Causes and Risk Factors*

Although the etiology of CRC is still not entirely understood, multiple risk factors and points of origin have been identified, many of which can be classified as modifiable or intrinsic factors. Given that 70-80% of CRC cases are sporadic, and therefore attributed to environmental causes, modifiable risk factors, including dietary choices, substance use patterns, and activity levels, can have a large effect (Yamagishi et al., 2016). Individuals who consume high amounts of red meats, ultra-processed foods, and low amounts of fruits and vegetables are at greater risk of developing CRC (Hang et al., 2023, Sawicki et al., 2021). Similarly, high rates of smoking, high rates or volume of alcohol consumption, sedentary lifestyle (i.e. low levels of physical activity), and some health conditions (e.g. IBD) can also elevate CRC risk (Sawicki et al., 2021). The association between individuals with IBD-induced chronic inflammation and increased risk of CRC is well-established, as individuals with IBD are 2-3 times more likely to develop CRC than individuals without IBD (Shah & Itzkowitz, 2022, Crohn's Colitis Canada, 2023). Intrinsic risk factors, such as certain demographic factors and genetic conditions, can also impact

susceptibility. Older age is a risk factor for cancer, as genetic mutations are more likely to occur and accumulate over time (Shah & Itzkowitz, 2022). Individuals with a lower socioeconomic status (e.g. low education and income) typically have poorer health and are therefore at greater risk of developing cancer (Nasiri et al., 2024). Moreover, approximately 5-10% of cases are associated with inherited genes that can significantly increase risk of CRC, including Lynch syndrome and Familial Adenomatous Polyposis Coli (FAP) (Kastrinos et al., 2020, Sawicki et al., 2021). Additionally, 35% of cases are related to familial forms of CRC, which may be attributed to shared environmental exposures or, more likely, from associations with unrecognized genes (Kastrinos et al., 2020).

### *Region Specific CRC Risk Factors*

In 2024, 24,100 new cases of CRC are expected to be diagnosed, which will make up 10% of cancer cases (Canadian Cancer Statistics Advisory Committee, 2023); however, Atlantic Canada has the highest incidence of CRC among any region of Canada, with all four provinces having incidence rates near to or above the national average age-standardized incidence rate (ASIR) (Canadian Cancer Statistics Advisory Committee, 2023). Although the reasons underlying the high incidence of CRC observed in Atlantic Canada are not yet well-defined, certain environmental and genetic risk factors may play important roles. Regional patterns of substance abuse, dietary habits, and limited access to healthcare are thought to modify the impact of CRC in this region. Alcohol consumption in the Atlantic provinces is both notably high and typically begins at an early age, with the onset of alcohol consumption in Nova Scotia being at, on average, 12 (Health Canada, 2021, McLean, 2025). A culture of alcohol abuse in these regions fosters habits of heavy and frequent use, which results in alcohol consumption rates much higher than other parts of Canada (McLean, 2025). Many Atlantic Canadians also have poor dietary

habits, with one study reporting the majority of participants in Atlantic Canada failed to meet recommended dietary guidelines (7-10 servings of fruits and vegetables), eating only 1-2 daily servings of fruits and vegetables (DeClercq et al., 2017). Additionally, Atlantic Canadians were found to source over half of their daily calories from ultra-processed foods (Hamel, et al., 2024). Lastly, individuals living in Atlantic Canada experience barriers to accessing healthcare, including a high turnover rate for family physicians (Curnew & Lukewich, 2018), which is exacerbated by the fact that a significant proportion of Atlantic Canadians currently live in rural communities, which are also underserved by specialist physicians (Government of Canada, 2022, Hippe et al., 2014). This results in longer wait times to access healthcare services and high travel costs to seek out adequate care (Hippe et al., 2014). This reduction in the quality of care Atlantic Canadians may receive can impact this disease both in terms of prognosis and treatment.

In addition to these environmental influences, there is likely a genetic component. Atlantic Canada is a culturally isolated region that experiences some of the lowest immigration rates in the country, with only 8% of immigrants settling there (Fang et al., 2023, StatCan, 2023). Moreover, the Atlantic provinces are made up of small communities, composed mainly of individuals of European descent (Fang et al., 2023). This has produced a population with relatively low diversity, as only 7% of the population is made up by visible minorities (Atlantic Economic Council, 2024). While only 5-10% of CRC cases are attributed to known genetic predispositions, in populations with reduced diversity, there is the potential for certain genetic susceptibilities to be much more frequent (Kastrinos et al., 2020). Therefore, it is possible that genetic variants unique to or more common with the region could be a contributing factor to elevated CRC risk.

## **Inflammation, CRC and Atlantic Canada**

### *The Intersection between CRC, IBD and Inflammation*

Chronic inflammation is key to the development of cancer as it produces an environment conducive to cellular proliferation and angiogenesis to facilitate tumor growth (Hanahan, 2022). Associations between chronic inflammation and various inflammatory mediators have linked CRC and IBD pathogenesis to malfunctions in inflammatory pathways. Mutations in the genes producing key inflammatory mediators can result in disruptions to the normal inflammatory process, producing chronic inflammatory states. Two proteins involved in the regulation of the inflammatory response, cyclooxygenase (COX) and nuclear factor kappa beta (NF- $\kappa$ B), in combination with the cytokine interleukin-6 (IL6) and the transcription factor tumour necrosis factor (TNF), have been shown to increase cancer risk by promoting tumour growth (Kraus & Arber, 2009). Additionally, NF- $\kappa$ B and TNF have been directly associated with the initiation of IBD pathogenesis (Jostins et al., 2012).

### *Key Inflammatory Markers*

*IL6* encodes a cytokine IL-6, which is involved in the regulation of innate immune and inflammatory responses (Yu et al., 2012). IL-6 upregulates components of a signal transduction pathway that regulates the homeostasis of intestinal mucosa cells, and inappropriate activation of this pathway has been associated with chronic inflammation, as well as carcinogenic cellular growth (Gui et al., 2020, Zhang & Qiao, 2022). Moreover, several genetic variants of *IL6* have been associated with increased risk of developing CRC (Caramujo-Balseiro et al., 2021, Yu et al., 2012).

Tumour necrosis factor alpha ( $TNF-\alpha$ ) is a cytokine involved in the regulation of cellular proliferation and differentiation, the innate immune response, and apoptosis (El-Tahan et al., 2012, Zhang & Qiao, 2022).  $TNF-\alpha$  triggers the  $NF-\kappa B$  and  $IL6$  pathways that produce various compounds, including growth-factors and proteases, that support normal immune system and inflammatory pathway functioning (Wei et al., 2023). Activity of  $TNF-\alpha$  is regulated by the promoter region of its gene, and genetic variants in this region can alter the rate of production (Bani et al., 2021). Several  $TNF-\alpha$  variants have been associated with higher levels of  $TNF-\alpha$  expression, particularly among CRC patients (El-Tahan et al., 2012, Zhang & Qiao, 2022).

Cyclooxygenases (COXs), also known as prostaglandin-H synthase enzymes (PTGSs), initiate the production of prostaglandins, which are necessary for the supporting inflammatory pathways and protect the GI tract (Berbecka et al., 2021). There are two main  $COX$  isoforms,  $COX-1$  and  $COX-2$  that are encoded by  $PTGS1$  and  $PTGS2$ , respectively (Makar et al., 2013, Zheng et al., 2024).  $COX-1$  and 2 are both associated with CRC and perform similar functions, but they are expressed during different conditions.  $COX-1$  has been associated with tumour development while  $COX-2$  plays an important role in CRC metastasis (Makar et al., 2013; Sheng et al., 2001).

Nuclear factor kappa beta (NF- $\kappa B$ ) is a family of transcription factors associated with inflammation and the immune responses (Zhang & Qiao, 2022). There are five genes that encode the proteins within this family:  $NFKB1$ ,  $NFKB2$ ,  $RELA$ ,  $REL$ ,  $RELB$  (Dolcet et al., 2005). NF- $\kappa B$  controls the expression of genes related to cell proliferation and, when functioning incorrectly, has been observed to inhibit apoptosis and stimulate angiogenesis, both of which are key attributes of cancerous tissue (Zhang & Qiao, 2022, Zhang et al., 2015). Overexpression of NF- $\kappa B$ , which has been associated with several forms of cancer, is found commonly in tissues of

CRC patients and mutations in related genes have been associated with an increased risk of CRC (Zhang & Qiao, 2022, Zhang et al., 2015).

### *Proinflammatory Factors Characteristic of Atlantic Canada*

Although certain proinflammatory environmental factors are likely playing an integral role in CRC development among Atlantic Canadians, genetic factors are possibly playing a role through gene-environment interactions, whereby genetic and environmental influences converge to affect the risk of disease development (Pekmezović, 2010). Many common lifestyle and demographic factors of Atlantic Canadians, such as high rates of alcohol consumption, poor dietary habits, and IBD pathogenesis, have harmful effects on health through the onset of chronic inflammation and can therefore increase risk of cancer. The varied impact of these factors, including their potential interactions with genes, could produce a further exacerbated state of chronic inflammation and contribute to CRC incidence.

## Study Context

As cancer development has a major genetic component, it is plausible that populations with high incidence of inflammation-associated conditions, such as IBD and CRC, could have common genetic mutations related to inflammatory pathways that would influence susceptibility. Given that Atlantic Canada is disproportionately affected by both IBD and CRC, and has reduced genetic diversity, this population is suited for investigating potentially pathogenic genetic mutations. I hypothesize that Atlantic Canadians are at greater risk of CRC due to the combined effects of inflammatory pathway mutations and exposure to pro-inflammatory environmental factors. Thus, the objectives of this study are as follows:

(1) Identify variants in genes associated with inflammatory pathways (e.g. IL6, TNF, COX, NF-kB) that may be unique to, or occur at higher frequencies in, Atlantic Canada, when compared to other populations and

(2) examine their potential role in gene-environment interactions (e.g., alcohol consumption), which may help explain the significantly higher CRC incidence rates observed within the region.

Learning more about these underlying risk factors will provide opportunities to learn more about CRC development and support the creation of specialized treatment strategies tailored to the needs of Atlantic Canadians.

## Materials and Methods

### Study Population

The data used for this research was collected by the Canadian Partnership for Tomorrow's Health (CanPath), which is a multi-centred, prospective, cohort study. CanPath was established in 2008 with the goal of following over 330,000 Canadians over the course of 30 years to learn more about the causes and risk factors for chronic diseases (Sweeney et al., 2017, CanPath, 2025). Originally, there were five regional cohorts associated with Can Path; the Atlantic Partnership for Tomorrow's Health (Atlantic PATH), Alberta's Tomorrow Project, the Ontario Health Study, the BC Generations Project (BCGP) and Quebec's CARTaGENE (Sweeney et al., 2017). The Manitoba Tomorrow Project and Healthy Future Sask were added to CanPath in 2020 and 2023, respectively, which completed CanPath's coverage of Canada (Sweeney et al., 2017). Atlantic PATH collects data on residents of the four Atlantic provinces (53% NS, 25% NB, 15% NFLD, 4% PEI) (Sweeney et al., 2017). Various information was collected from participants, including urine and blood samples, the latter of which were used for whole genome sequences, physical measurements, and data regarding lifestyle and socioeconomic factors, which provided a comprehensive dataset for studying the interactions between genetic, biological, environmental, and social determinants of health (CanPath, 2025). Eligible participants for this study are members of the Atlantic and British Columbian (BC) arms of CanPath. Data from the BC cohort was selected as a standard for comparison to Atlantic Canada, as BC has an incidence rate of CRC similar to the national average (Canadian Cancer Statistics Advisory Committee, 2023).

## **Study Design**

The study design is a nested case-control study involving both primary prevalent and incidence cases of CRC (i.e. participants identified as cases could have received a diagnosis of CRC prior to or during enrollment within the CanPath cohort study). Controls were participants with no previous history of cancer, apart from non-melanoma skin cancers. Non-melanoma skin cancers are unique in that they result from different risk factors than most other cancers (i.e. UV light exposure) and are less likely to metastasize, making them a less significant confounding factor in studies such as this (Canadian Cancer Society, 2014). CanPath previously genotyped 5,000 healthy participants from across the original 5 cohorts, which included 1,961 participants from the PATH and BCGP cohorts that were selected as controls for this study and, including the 559 CRC cases, a total of 2,520 participants were included in this study. Participants were restricted to those of European descent to account for population stratification and to match the selection criteria done by CanPath. Demographic information, including age, socioeconomic status, and medical history was consolidated to identify any cohort-specific differences in important characteristics, therefore increasing the reliability of the results and aiding in the assessment of their external validity.

## **Genotyping**

To examine the influence of genetics on CRC, a Genome Wide Association Study (GWAS) was conducted, which examines thousands of SNPs across many genomes for their association with disease development (Uffelmann et al., 2020). The UK Biobank Axiom Array was used for genotyping the CRC case data, which covered of a broad range of SNPs relevant to the European-based population. The procedures completed reflected those done by CanPath for the approximately 2,000 participants previously genotyped and genotyping among 559 CRC

cases was completed through Genome Quebec and the Genetic & Molecular Epidemiology Laboratory (McMaster University).

### **Quality Control Procedures**

Axiom™ Analysis Suite (Thermo Fisher, Waltham, Massachusetts, USA) using the specific UK Biobank Axiom Array type (Axiom\_UKG\_WCSG.r5) and the Best Practice Workflow (BPW; Affymetrix, 2020) was the method chosen to complete genotype quality control procedures to match those done by CanPath (CanPath, 2019). Genotyping occurs in batches (e.g. 96-samples per UK Biobank microarray plate), per product specifications, while the BPW is a two-step procedure that ensures only samples that will pass quality control steps (i.e. SNP call rate > 95%) will be genotyped in case there are data quality issues. Poor quality data can have a negative impact on the calling accuracy of better samples. The call rate is the proportion of individuals in the study for which the corresponding SNP information is not missing (CanPath, 2019).

### **Gene and SNP Selection**

After the quality control procedure was completed, a total of 771,055 SNPs were available for use in this study. Following the literature review, four general groups of genes, PTGS, TNF, NFKB and IL6, were selected for their role in inflammatory pathways of interest. Each group encompasses the genes encoding the inflammatory mediator of interest and their associated proteins. Identification of these genes of interest reduced the applicable SNPs to 2,050 across 53 individual genes. A further quality control procedure was completed on the refined sample data. Any SNPs that had a minor allele frequency (MAF) value of lower than 1%, which indicates that the minor allele is very rare in the population, were excluded. Low frequency alleles (i.e. those lower than 5% or 1%) are often removed as they are more likely to have been

impacted by genotyping errors and they lack statistical power (Anderson et al., 2010). In this study, a threshold of 1% was chosen, rather than the more conservative 5%, in order to retain low-frequency variants that may still hold biological significance and preserve variants with enough representation in the sample to contribute meaningful information to downstream analyses. This reduced the overall number of available SNPs for statistical analysis to 1,487 across 53 genes.

### **Statistical Analysis**

Statistical analysis and data cleaning for this study was completed using the programming language R. To determine if there were associations between CRC and variants of inflammatory genes, multivariable logistic regression was used to calculate odds ratios (OR) and 95% confidence intervals (CIs). The OR, which is a common metric for risk in case-control studies, measures the association between an event and exposure by comparing the odds of exposure between the cases and controls (Tenny and Hoffman, 2023). In the context of this study, OR were used to examine the the odds of a specific genotype or carrier of the minor allele among cases compare to controls. To determine the minor allele, the MAFs for each SNP was calculated, and the mode of inheritance (i.e. additive, recessive, or dominant) was selected based on the max-stat method.

To explore whether differences in the distribution of the risk alleles between regional cohorts exist, the chi-square test of association were applied to each SNP (Abramovs et al., 2020). Additionally, due to the high volume of hypothesis tests completed under this study design, there is an increased risk of observing false positives (i.e. committing a Type I error) and therefore potential to observe a relationship between some genetic variation and CRC when its association is merely due to random chance (Columbia University, 2023). To control for the

false-discovery rate (FDR), the Benjamini-Hochberg (BH) procedure, was implemented to calculate adjusted p-values. While there are alternative methods to adjust for the FDR, such as the Bonferroni correction, this method can reduce statistical power, as it is more conservative (Narum, 2006). The BH procedure provides a balance between the need for statistical power and modification to account for false positives, making it a common measure for large-scale genomic studies (Gui et al., 2012).

## Results

A total of 2,520 individuals, 559 cases and 1,961 controls, were included in this study. This included 1,238 participants (300 cases, 982 controls) from the BC cohort and 1,282 participants (259 cases, 979 controls) from the Atlantic Path cohort. Descriptive statistics for the study population, including factors that could modify an individual's risk of cancer development such as lifestyle choices, medical history, and level of social determinants, are consolidated in Table 1. A more detailed summary of population demographic information, including cohort-specific statistics, is available in Supplementary Table 1.

Among the demographic factors examined, age, sex, household income and colonoscopy/sigmoidoscopy history differed significantly between case and control groups. The average age of individuals with CRC (58.3) was higher than those in the control group (55.4), as the control group had a greater proportion of individuals under the age of 50 (controls: 27%, cases: 19%). Controls tended to have a higher proportion of females (59% vs 54%) and have a household income greater than \$50,000 (78% vs 66%) compared to cases. The most apparent difference occurred with history of colonoscopy and/or sigmoidoscopy, as more than 70% of cases had a history of the procedure, compared to only 40% of controls. No significant differences were observed among the other demographic variables.

Examining the difference by centre (i.e. PATH vs BCGP), similar trends were observed for most demographic variables with the exception of smoking and family history (see Supplementary Table 1). There was a greater number of cases who reported being an active or prior smoker compared to controls; however, for both cohorts, any difference in smoking history by case-control status was no longer apparent. For family history, there was no apparent difference with family history of at least 1 first-degree relative (e.g. parent, sibling, or child)

among the entire study population; however, when you stratify by centre, among the PATH cohort, there was a difference by case-control status.

Following quality control checks, 1,487 SNPs across 53 genes were involved in the statistical analysis (Supplementary Table 2). These genes can be grouped into four general categories based on the inflammatory mediator they are associated with: IL6, NF-kB, PTGS and TNF. At a threshold of significance of  $\alpha = 0.05$ , 87 SNPs across 36 genes were observed to be significantly associated with CRC risk (Supplementary Table 2). SNPs from every general category were observed to be significant, with the majority of genes (83%) coming from the TNF gene family. Among the 87 SNPs identified to be significant associated with CRC, 28 (32%) were classified as additive, 20 (23%) as dominant, and 39 (45%) as recessive, and MAF ranged between 0.029 and 0.405.

After adjusting for the False Discovery Rate using the Benjamini-Hochberg procedure, only four SNPs (Table 2) met our threshold for significance. Three of these SNPs (rs16972249, rs8073937, rs59119269) were associated with genes encoding TNF-associated proteins and one SNP was associated with IL6. The latter of the set does not have a dbSNP rsID number, which is a unique identifier used by researchers and databases to identify a specific SNP sequence. The unclassified ID, which had an internal ID (AX.94361920) unique to Thermofisher, may be a novel sequence not previously reported to the dbSNP database.

Using the four SNPs observed to be significant following adjustment, chi-square testing was used to determine if significant differences in the distribution of alleles by cohort. Of these, three SNPs (rs16972249, rs8073937, AX.94361920) showed significant variation in allele distribution between cohorts at a significance of  $\alpha = 0.05$  (Table 3). Among the SNPs with a

difference in allele distribution, the Atlantic Path cohort were observed to have a higher frequency of minor allele (Supplementary Table 4).

<b>Table 1: Summary Statistics of Study Population</b>			
<b>Variable</b>	<b>Cases (%)</b>	<b>Controls (%)</b>	<b>p-value</b>
<b>Centre</b>			
PATH	300 (53.7)	982 (50.1)	0.515
BCGP	259 (46.6)	979 (49.9)	
<b>Age</b>			
Mean, SD	57.12, 8.68	55.25, 7.86	-----
Under 50	110 (0.19)	537 (0.27)	<b>&lt;0.001</b>
50-59	220 (0.38)	762 (0.39)	
60-69	222 (0.39)	674 (0.34)	
70 and above	23 (0.04)	0 (0)	
<b>Sex</b>			
Female	313 (0.54)	1166 (0.59)	<b>0.047</b>
Male	263 (0.46)	807 (0.41)	
<b>Education</b>			
High school or less	121 (0.21)	395 (0.20)	0.155
Trade, Diploma or Certificate	234 (0.41)	764 (0.39)	
Bachelor's Degree	119 (0.21)	507 (0.26)	
Graduate or Professional Degree	80 (0.14)	297 (0.15)	
NA	22 (0.04)	11 (0.01)	
<b>Household income</b>			
Less than 50,000	141 (0.24)	331 (0.17)	<b>&lt;0.001</b>
50,000-74,999	127 (0.22)	402 (0.20)	
75,000-99,999	90 (0.16)	390 (0.20)	
100,000-149,999	104 (0.18)	454 (0.23)	
150,000 or more	59 (0.10)	288 (0.15)	
NA	55 (0.10)	109 (0.06)	
<b>Cigarette Smoker Status</b>			
Non-smoker	253 (0.44)	1006 (0.51)	
Previous	266 (0.46)	826 (0.42)	

<b>Table 1: Summary Statistics of Study Population</b>			
<b>Variable</b>	<b>Cases (%)</b>	<b>Controls (%)</b>	<b>p-value</b>
Occasional	9 (0.02)	35 (0.02)	<b>0.042</b>
Daily	27 (0.05)	87 (0.04)	
NA	21 (0.04)	20 (0.01)	
<b>Alcohol Usage</b>			
Never	44 (0.08)	146 (0.07)	0.145
Less than once a week	172 (0.30)	685 (0.35)	
1-5 per week	251 (0.44)	867 (0.44)	
6-7 per week	81 (0.14)	227 (0.11)	
NA	28 (0.05)	49 (0.02)	
<b>BMI</b>			
Mean, SD	27.62, 4.69	27.44, 5.07	0.575
Underweight/Normal	90 (0.16)	422 (0.21)	0.203
Overweight	121 (0.21)	452 (0.23)	
Obese	81 (0.14)	288 (0.15)	
NA	284 (0.49)	812 (0.41)	
<b>First-degree Family History of CRC</b>			
None	506 (0.88)	1790 (0.91)	0.055
One or greater	70 (0.12)	184 (0.09)	
<b>Colonoscopy/Sigmoidoscopy History</b>			
Ever	415 (0.72)	796 (0.40)	<b>&lt;0.001</b>
Never	142 (0.25)	1148 (0.58)	
NA	19 (0.03)	30 (0.02)	
<b>Crohn's Disease (CD) Diagnosis</b>			
Ever	12 (0.02)	20 (0.01)	0.067
Never	558 (0.97)	1944 (0.98)	
NA	6 (0.01)	10 (0.01)	
<b>Ulcerative Colitis (UC) Diagnosis</b>			
Ever	10 (0.02)	25 (0.01)	

<b>Table 1: Summary Statistics of Study Population</b>			
<b>Variable</b>	<b>Cases (%)</b>	<b>Controls (%)</b>	<b>p-value</b>
Never	557 (0.97)	1928 (0.98)	0.508
NA	9 (0.02)	21 (0.01)	

**Table 2:** SNPs significantly associated with a greater risk of developing CRC after BH adjustment at  $\alpha = 0.05$

Gene	dbSNP.RS.ID	OR	Minor Allele Frequency (MAF)	Major Allele Frequency	Adjusted p-value
TNFSF13B	rs16972249	4333.51	A = 0.156	T = 0.844	<0.001
TNFSF12	rs8073937	24645.00	A = 0.214	G = 0.786	<0.001
IL6	AX.94361920	0.016	C = 0.409	A = 0.591	<0.001
TNFRSF10B	rs59119269	12.42	T = 0.048	C = 0.952	0.0064

**Table 3:** Results of chi-square testing of allele distribution by cohort population. Significance level  $\alpha = 0.05$

Gene	SNP ID	p-value
TNFSF13B	rs16972249	<0.0001
TNFSF12	rs8073937	0.00259
IL6	AX.94361920	<0.0001
TNFRSF10B	rs59119269	0.274

## Discussion

### Demographic Statistics

Some of the demographic factors analyzed, including age, sex, household income and colonoscopy/sigmoidoscopy history differed between study groups. As these factors can have an effect on study outcomes, recognizing these differences can improve the accuracy of results. Although there is a significant difference between the cases and controls with respect to age, difference by disease status is being driven mainly by the 70 and older group. As age is a risk factor many diseases, including CRC, it is therefore an important consideration in case-control studies such as these (Niccoli & Partridge, 2012). Similarly, sex differences can affect disease susceptibility, symptom expression, and treatment outcomes, and therefore variation in sex distribution among study groups is an important variable to consider (Morrow, 2015). Furthermore, household income is a common surrogate for socioeconomic status and often impacts how, when, and if an individual can or does seek medical care, as well as the quality that they receive (van den Berg et al., 2020). Given that a greater proportion of cases reported a less than \$50,000 household income compared to controls, these individuals could be at greater risk of disease due to reduced access to medical care. Lastly, many those without a CRC diagnosis have not undergone colonoscopy or sigmoidoscopy testing, while the majority of CRC cases have. Given that these testing methods are some of the main ways CRC is diagnosed, it should not be surprising that there is a substantially higher rate of diagnostic usage among cases than controls. Although three of the variables mentioned are important risk factors for CRC that differ by case-control status, they are not associated with genotype and therefore are unlikely to confound the genotype-disease relationship. There are exceptions, particularly with respect to

gene-environment interactions and epigenetic studies, and therefore future analysis of these results would be beneficial to account for these factors.

After stratifying for centre, it was observed cases from the PATH cohort had a greater proportion of family members with history of CRC. As the risk of CRC is increased if a first-degree relative has been previously diagnosed, this could make this population more susceptible to CRC (American Cancer Society, 2023). Given that, generally, there are higher rates of CRC observed within Atlantic Canada, the difference observed within the Atlantic cohort was not surprising. Although overall there were no apparent difference among the entire study population, this consideration is still important.

Lastly an important variable for consideration in this study is prior diagnosis with an inflammatory bowel disease (IBD), such as ulcerative colitis (UC) or Crohn's disease (CD). As these diseases are independent risk factors for CRC, they could have an influence on the results observed from this study (Sato et al., 2023). Additionally, as the focus of this study was on inflammatory pathways, seeing a higher proportion of cases with an IBD diagnosis would be consistent with the hypothesis that SNPs of inflammatory genes could be modulating both CRC and IBD incidence. However, there were no significant differences observed between the proportion of cases and controls with a prior IBD diagnosis. Additionally, despite prior research indicating that the Atlantic provinces have the highest prevalence of IBD of anywhere in Canada, and although the prevalence of IBD was almost 50% higher in Atlantic Canadian population compared to the BCGP cohort, the differences were not significant.

## **Inflammatory Genes**

The results of this study indicate that there are associations between multiple inflammatory genes of interest and increased CRC risk (Table 2, Supplementary Table 2). This is consistent with previous literature, which has indicated that SNPs of inflammatory genes are associated with increased risk of intestinal diseases such as CRC and IBD (Bondurant et al., 2013, Imge Hulus et al., 2015). Supplementary Table 3 presents a 2x2 table illustrating the genotype distribution between cases and controls for one of the SNPs under investigation, rs16972249. This format allows for a visual interpretation of the measure of risk (OR) as it highlights the differences in genotype between cases and controls. The high numbers of cases carrying the minor allele, compared to the high number of controls carrying the major allele, helps contextualize the statistical significance of the OR for this SNP. Three of these SNPs (rs16972249, rs8073937, rs59119269) were associated with genes encoding TNF-associated proteins one SNP (AX.94361920) was associated with IL6.

The cytokines TNF and IL6 are key inflammatory mediators that, along with their related proteins, are necessary for the development of an inflammatory state. The TNF superfamily encompasses a broad family of cytokines and their receptors (Zhang & Qiao, 2022). They are involved in the regulation of the innate immune response and cell proliferation (Zhang & Qiao, 2022). TNF activation triggers multiple regulatory pathways, resulting in the production of transcription factors and enzymes that regulate apoptosis and inflammation (Arguello et al., 2021, Wei et al., 2023). The three TNF proteins of relevance to this study are TNFSF13B, TNFSF12 and TNFRSF10B. TNFSF13B is a cytokine expressed in B cells that regulates B cell proliferation and maturation (National Library of Medicine, 2025). Variants of TNFSF13B genes have been associated with chronic inflammatory diseases such as rheumatoid arthritis and lupus

(González-Serna et al., 2018). TNFSF12 is a cytokine with similar functions to TNF but a wider range of effect (National Library of Medicine, 2025). It is associated with many cell regulating functions such as angiogenesis, apoptosis and cellular growth (National Library of Medicine, 2025). TNFRSF10B, also known as DR5, is a TNF receptor (Arguello et al., 2021). It known as a death receptor (DR) for its role in the activation of the caspase-3 and -7 pathways, which results in the initiation of apoptosis (Arguello et al., 2021). Genes encoding DR5 have been observed to be upregulated in multiple forms of cancer and its overactivation has been seen to initiate metastasis in cancer cell lines (Arguello et al., 2021, Koornstra et al., 2003). IL6 is a cytokine with a variety of physiological functions, including supporting the inflammatory response and hematopoiesis (Candido et al., 2021, Tanaka et al., 2014). IL6 binds to its receptor (IL6R), which produces a signal transduction pathway that involves the transcriptional activator STAT3 and cytokine signalling repressor SOCS3 (Gui et al., 2020, Yue et al., 2020). The IL6/STAT3/SOCS3 pathway regulates homeostasis of intestinal mucosa cells, and faulty activation of this pathway has been associated with chronic inflammation, carcinogenic cellular growth and angiogenesis (Gui et al., 2020, Zhang & Qiao, 2022). As both TNF and IL6 play important roles in the regulation of the immune response and cell cycle, it is unsurprising that mutations in these pathways could result in inflammatory pathologies and environments ideal for cancer development. It is possible the TNF and IL6-associated SNPs identified in this study could be impacting the functionality of cell cycle regulation and result in increased risk of CRC.

The results of the chi-square test show that three of the four significant SNPs (rs16972249, rs8073937, AX.94361920) have allele distributions that differ significantly by population (see Table 3 and Supplementary Table 3). Among these SNPs, the minor allele was more prevalent in the Atlantic cohort compared to the BC cohort. These findings support the

hypothesis that there are potentially pathogenic variants of inflammatory genes that are more prevalent within the Atlantic Canadian population. For rs16972249 and rs8073937, the risk allele is more common among Atlantic Canadians, indicating the Atlantic Canadian population could be at greater risk of CRC due to these genetic predispositions, which would contribute to the high CRC incidence observed in this region. The IL6 SNP (AX.94361920), which displayed a protective effect (Table 2), saw significant distributional differences but unlike the prior two SNPs. Almost 90% of individuals in the BC cohort had this protective allele; however, every individual in the Atlantic cohort carried the major allele (i.e. the risk allele) for this SNP. Given that all Atlantic participants were carriers of this allele, no association between this SNP and CRC was observed in this cohort when we stratified, suggesting a greater risk due to the absence of this protective effect. Although there is strong evidence of an association with this SNP observed within the BC cohort, the somewhat contradictory results within the Atlantic cohort make it difficult to determine the broader significance of this relationship. However, these findings do suggest that this SNP, in combination with environmental or other genetic risk factors, may also play a key role in modulating CRC risk.

Out of the four SNPs found to be significantly associated with CRC, only one (rs8073937) was previously reported for clinical relevance in disease pathology based on our literature review. The rs8073937 SNP was found to be positively associated with risk of atrial fibrillation (Roselli et al., 2018), which demonstrates its potential medical applications. No studies have previously found associations between any of these SNPs with CRC. Therefore, these SNPs, particularly the IL6 associated SNP, may be novel colorectal cancer biomarkers that could be used in genetic screening programs. SNPs have previously been shown to be effective tools for recognizing individuals at greater risk of CRC (Jenkins et al., 2019). As two of the four

significant SNPs have risk alleles that are more prevalent in the Atlantic Canadian cohort, they have potential clinical relevance in a clearly vulnerable population. Evaluating these SNPs as potential biomarkers for CRC risk could offer valuable insights, particularly for the health of Atlantic Canadians.

Many environmental risk factors for CRC are common in Atlantic Canada and have the potential to be impacting susceptibility to cancer, in combination with these identified genetic factors. Therefore, investigating potential gene-environment interactions, as well as gene-gene interactions among these SNPs is an important future direction to better understand their role in colorectal cancer. By examining regional variations in lifestyle, diet, and healthcare access, it can be better understood how these environmental exposures may influence the effect of these genetic variants on inflammatory pathways.

## **Limitations**

While these results are promising, and indicate potential avenues for future research endeavours, it cannot be confirmed whether these mutations are truly modifying disease risk. Case-control studies are limited in that they can only evaluate associations between variables of interest and not elucidate causation (Tenny & Hoffman, 2023). Moreover, due to the use of a case-control study, there is risk of Neyman's bias. Neyman's bias, also known as prevalence-incidence bias, occurs when cases included in the study disproportionately represent survivors of a disease, rather than all individuals who are diagnosed (Tripepi et al., 2008). This bias can lead to an underestimation of disease severity because excluding fatal cases will distort associations with disease and given the high mortality rate associated with CRC, any risk of Neyman's bias is not negligible (Tripepi et al., 2008). Despite these study design issues, these SNPs have the potential to be used as a guide for future investigations, such as functional (e.g. gene expression)

studies, which could be used to better determine their role in CRC development. Additionally, genetic modification experiments, such as those using cell cultures or mouse models, allow researchers to examine a mutation's influence on gene expression and protein activity. These studies could be used to examine how these SNPs actually alter inflammatory signalling to determine whether these modifications contribute to the development of tumours (Brown, 2021).

Other limitations include the use of the max-statistics to determine the mode of inheritance for risk alleles. The max-statistic test or max-stat method is commonly applied in case-control genetic association studies to determine the maximum of the standardized version of the optimal tests of association based on either the dominant, additive, or recessive mode of inheritance (Freidlin et al., 2002). While this test provides more statistical power than merely assuming all SNPs follow an additive model, it is limited in that it ignores co-dominant or polygenic modes of inheritance (Freidlin et al., 2002), which could lead to inaccuracies in these results. However, without additional studies, it is unlikely that the true modes of inheritance can actually be determined. Additionally, the primary objective of this study was to identify SNPs that may have been involved in CRC development and the max-statistic highlights the mode of inheritance for the SNP with the strongest effect. Thus, while this max-statistic approach may be an oversimplification of inheritance patterns, it remains a useful tool for identifying SNPs with the greatest association with CRC.

As mentioned previously, the analysis for this study did not include gene-environment interactions or gene-gene interactions (e.g. epistasis or polygenic inheritance) which limits the extent of these results; however, these interactions will be explored in future analysis.

Additionally, statistical testing, such as that completed in this study, is unable to directly analyze the biological processes underlying why an association may exist. Therefore, further testing to

determine the biological significance of these SNPs would provide more information on how these SNPs could be used to positively affect health outcomes.

## References

- Abramovs, N., Brass, A., & Tassabehji, M. (2020). Hardy-Weinberg equilibrium in the large-scale genomic sequencing era. *Frontiers in Genetics, 11*(1664-8021). <https://doi.org/10.3389/fgene.2020.00210>
- Afsar, A. U. (2011). An overview of inflammation: mechanism and consequences. *Frontiers in Biology, 6*, 274-281. <https://doi.org/10.1007/s11515-011-1123-9>
- Affymetrix (2020). *Axiom analysis suite v5.1 user guide*. Thermo Fisher. [www.downloads.thermofisher.com](http://www.downloads.thermofisher.com).
- American Cancer Society. (2023, July 19). *Colorectal Cancer Risk Factors*. [www.cancer.org](http://www.cancer.org); American Cancer Society. <https://www.cancer.org/cancer/types/colon-rectal-cancer/causes-risks-prevention/risk-factors.html>
- Amerman, E. C. (2019). *Human Anatomy & Physiology*. Pearson.
- Anar Dhalla, McDonald, T., Gallagher, R. P., Spinelli, J. J., Brooks-Wilson, A., Lee, T. K., Lai, C., Borugian, M. J., Woods, R., Le, N. D., & Trevor J.B. Dummer. (2018). Cohort Profile: The British Columbia Generations Project (BCGP). *International Journal of Epidemiology, 48*(2), 377–378k. <https://doi.org/10.1093/ije/dyy160>
- Anderson, C. A., Pettersson, F. H., Clarke, G. M., Anderson, C. A., Pettersson, F. H., Clarke, G. M., Cardon, L. R., Morris, A. P., & Zondervan, K. T. (2010). Data quality control in genetic case-control association studies. *Nature Protocols, 5*(9), 1564–1573. <https://doi.org/10.1038/nprot.2010.116>
- Atlantic Economic Council. (2024). Spotlight on diversity, equity and inclusion in Atlantic Canada. Atlantic Economic Council. [https://cdn.ymaws.com/atlanticeconomiccouncil.ca/resource/collection/0EFB4B1F-5BB1-4991-9CC3-A09456A1D024/ATLANTIC\\_Diversity\\_Review\\_May\\_29,\\_2024\\_.pdf](https://cdn.ymaws.com/atlanticeconomiccouncil.ca/resource/collection/0EFB4B1F-5BB1-4991-9CC3-A09456A1D024/ATLANTIC_Diversity_Review_May_29,_2024_.pdf)
- Arguello, O., Haisma, H., Diaz Arguello, O. A., & Haisma, H. J. (2021). Apoptosis-Inducing TNF Superfamily Ligands for Cancer Therapy. *Cancers, 13*(7). <https://doi.org/10.3390/cancers13071543>
- Azzouz, L. L., & Sharma, S. (2023). *Physiology, Large Intestine*. Nih; StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK507857/>
- Bani, N., Moetamani-Ahmadi, M., Alidoust, M., ShahidSales, S., Khazaei, M., Esmaily, H., Joudi-Mashhad, M., Ferns, G. A., Gharib, M., & Avan, A. (2021). Association between the 308 G>A variant of the TNF- $\alpha$  gene and risk of colorectal cancer. *Meta Gene, 28*, 100878. <https://doi.org/10.1016/j.mgene.2021.100878>
- Bass, L. M., & Wershil, B. K. (2016). Anatomy, histology, embryology, and developmental anomalies of the small and large intestine. *Sleisenger and Fordtran's gastrointestinal and liver disease, 10th ed. Philadelphia, PA: Saunders, Elsevier Inc*, 1649.
- Bardelčíková, A., Šoltys, J., & Mojžiš, J. (2023). Oxidative Stress, Inflammation and Colorectal Cancer: An Overview. *Antioxidants, 12*(4). <https://doi.org/10.3390/antiox12040901>
- Berbecka, M., Forma, A., Baj, J., Furtak-Niczyporuk, M., Maciejewski, R., & Sitarz, R. (2021). A Systematic Review of the Cyclooxygenase-2 (COX-2) Expression in Rectal Cancer Patients Treated with Preoperative Radiotherapy or Radiochemotherapy. *Journal of Clinical Medicine, 10*(19). <https://doi.org/10.3390/jcm10194443>
- Bernstein, C. N., Wajda, A., Svenson, L. W., MacKenzie, A., Koehoorn, M., Jackson, M., Fedorak, R., Israel, D., & Blanchard, J. F. (2006). The epidemiology of inflammatory bowel disease in Canada: A population-based study. *The American Journal of Gastroenterology, 101*(7), 1559–1568. <https://doi.org/10.1111/j.1572-0241.2006.00603.x>

- Blair, A., & Datta, G. D. (2020). Associations between area-level deprivation, rural residence, physician density, screening policy and late-stage colorectal cancer in Canada. *Cancer Epidemiology*, 64. <https://doi.org/10.1016/j.canep.2019.101654>
- Bondurant, K. L., Lundgreen, A., Herrick, J. S., Kadlubar, S., Wolff, R. K., & Slattery, M. L. (2013). Interleukin genes and associations with colon and rectal cancer risk and overall survival. *International journal of cancer*, 132(4), 905–915. <https://doi.org/10.1002/ijc.27660>
- Brown S. D. M. (2021). Advances in mouse genetics for the study of human disease. *Human molecular genetics*, 30(R2), R274–R284. <https://doi.org/10.1093/hmg/ddab153>
- Bryan, S., Masoud, H., Weir, H. K., Woods, R., Lockwood, G., Smith, L., Brierley, J., Gospodarowicz, M., & Badets, N. (2018). Cancer in Canada: stage at diagnosis. *Health reports*, 29(12), 21–25.
- Burns, E. E., Mathias, H. M., Heisler, C., Cui, Y., Kits, O., Veldhuyzen van Zanten, S., & Jones, J. L. (2021). Access to inflammatory bowel disease speciality care: the primary healthcare physician perspective. *Family practice*, 38(4), 416–424. <https://doi.org/10.1093/fampra/cmab006>
- Canadian Cancer Society (2024). Colorectal Cancer. Canadian Cancer Society. <https://cancer.ca/en/cancer-information/cancer-types/colorectal>
- Canadian Cancer Society (2024). Cancer risk in families. Canadian Cancer Society. <https://cancer.ca/en/cancer-information/what-is-cancer/genes-and-cancer/cancer-risk-in-families>
- Canadian Cancer Society. (2014). *Non-melanoma skin cancer*. Canadian Cancer Society. <https://cancer.ca/en/cancer-information/cancer-types/skin-non-melanoma>
- Canadian Cancer Statistics Advisory Committee in collaboration with the Canadian Cancer Society, Statistics Canada and the Public Health Agency of Canada. (2023) Canadian Cancer Statistics 2023. Canadian Cancer Society. [cancer.ca/Canadian-Cancer-Statistics-2023-EN](https://cancer.ca/Canadian-Cancer-Statistics-2023-EN)
- CanPath (2019). *Canadian Partnership for Tomorrow's Health Genetic Information: Derived Data Report*. [https://portal.canpath.ca/ws/file-dl/harmonization-study/genotype/CanPath\\_DerivedDataReport\\_Genotype\\_March2020.pdf](https://portal.canpath.ca/ws/file-dl/harmonization-study/genotype/CanPath_DerivedDataReport_Genotype_March2020.pdf)
- Candido, S., Tomasello, B. M. R., Lavoro, A., Falzone, L., Gattuso, G., & Libra, M. (2021). Novel Insights into Epigenetic Regulation of IL6 Pathway: In Silico Perspective on Inflammation and Cancer Relationship. *International Journal of Molecular Sciences*, 22(18). <https://doi.org/10.3390/ijms221810172-023-01302-y>
- Caramujo-Balseiro, S., Faro, C., & Carvalho, L. (2021). Metabolic pathways in sporadic colorectal carcinogenesis: A new proposal. *Medical Hypotheses*, 148, 110512. <https://doi.org/10.1016/j.mehy.2021.110512>
- Caruana, E. J., Roman, M., Hernández-Sánchez, J., & Solli, P. (2015). Longitudinal studies. *Journal of thoracic disease*, 7(11), E537–E540. <https://doi.org/10.3978/j.issn.2072-1439.2015.10.63>
- Chávez-Sánchez, L., Espinosa-Luna, J. E., Chávez-Rueda, K., Legorreta-Haquet, M. V., Montoya-Díaz, E., & Blanco-Favela, F. (2014). Innate Immune System Cells in Atherosclerosis. *Archives of Medical Research*, 45(1), 1–14. <https://doi.org/10.1016/j.arcmed.2013.11.007>
- Columbia University. (2023). *False discovery rate*. Mailman School of Public Health. <https://www.publichealth.columbia.edu/research/population-health-methods/false-discovery-rate>
- Coward, S., Benchimol, E. I., Kuenzig, M. E., Windsor, J. W., Bernstein, C. N., Bitton, A., Jones, J. L., Lee, K., Murthy, S. K., Targownik, L. E., Peña-Sánchez, J.-N., Rohatinsky, N., Ghandeharian, S., Im, J. H., Davis,

- T., Weinstein, J., Goddard, Q., Gorospe, J., Bennett, J., ... Kaplan, G. G. (2023). The 2023 impact of inflammatory bowel disease in Canada: Epidemiology of IBD. *Journal of the Canadian Association of Gastroenterology*, 6(Supplement\_2). <https://doi.org/10.1093/jcag/gwad004>
- Crohn's and Colitis Foundation. (2019). Inflammatory bowel disease vs. irritable bowel syndrome. <https://www.crohnscolitisfoundation.org/sites/default/files/2019-10/ibd-and-IBS-brochure-final.pdf>
- Curnew, D. R., & Lukewich, J. (2018). Nursing Within Primary Care Settings in Atlantic Canada: A Scoping Review. *SAGE Open*, 8(2), 215824401877437. <https://doi.org/10.1177/2158244018774379>
- Darvishian, M., Moustaqim-Barrette, A., Awadalla, P., Bhatti, P., Broet, P., McDonald, K., Murphy, R. A., Skead, K., & Urquhart, R. (2023). Provincial variation in colorectal cancer screening adherence in Canada; evidence from the Canadian Partnership for Tomorrow's Health. *Frontiers in Oncology*, 13. <https://doi.org/10.3389/fonc.2023.1113907>
- Decker, K. M., Lambert, P., Bravo, J., Demers, A., & Singh, H. (2023). Time Trends in Colorectal Cancer Incidence From 1992 to 2016 and Colorectal Cancer Mortality From 1980 to 2018 by Age Group and Geography in Canada. *The American Journal of Gastroenterology*, 118(2), 338–344. <https://doi.org/10.14309/ajg.0000000000002058>
- DeClercq, V., Cui, Y., Forbes, C., Grandy, S., Keats, M., Parker, L., Sweeney, E., Yu, Z., & Dummer, T. (2017). Association between diet quality and adiposity in the Atlantic PATH Cohort. *Nutrients*, 9(10), 1155. <https://doi.org/10.3390/nu9101155>
- Dolcet, X., Llobet, D., Pallares, J., & Matias-Guiu, X. (2005). NF- $\kappa$ B in development and progression of human cancer. *Virchows Archiv*, 446(5), 475–482. <https://doi.org/10.1007/s00428-005-1264-9>
- Duan, B., Zhao, Y., Bai, J., Wang, J., Duan, X., Luo, X., Zhang, R., Pu, Y., Kou, M., Lei, J., & Yang, S. (2022). Colorectal cancer: An overview. *Gastrointestinal Cancers*, 1–12. <https://doi.org/10.36255/exon-publications-gastrointestinal-cancers-colorectal-cancer>
- Ellison, L. F., & Saint-Jacques, N. (2023). Five-year cancer survival by stage at diagnosis in Canada. *Statistics Canada - Health Reports*, 34. <https://www.doi.org/10.25318/82-003-x202300100001-eng>
- Fang, T., Zhang, T., & Hartley, J. (2023). Examining Determinants of Employers' Attitudes toward Hiring Immigrant Workers: Evidence from an Employer Survey. *Journal of Immigrant & Refugee Studies*, 1–16. <https://doi.org/10.1080/15562948.2023.2219634>
- Fernandes, Q., Inchakalody, V. P., Bedhiafi, T., Mestiri, S., Taib, N., Uddin, S., Merhi, M., & Dermime, S. (2024). Chronic inflammation and cancer; the two sides of a coin. *Life Sciences*, 338. <https://doi.org/10.1016/j.lfs.2023.122390>
- Freidlin, B., Zheng, G., Li, Z., & Gastwirth, J. L. (2002). Trend tests for case-control studies of genetic markers: power, sample size and robustness. *Human heredity*, 53(3), 146–152. <https://doi.org/10.1159/000064976>
- Furman, D., Campisi, J., Verdini, E., Carrera-Bastos, P., Targ, S., Franceschi, C., Ferrucci, L., Gilroy, D. W., Fasano, A., Miller, G. W., Miller, A. H., Mantovani, A., Weyand, C. M., Barzilai, N., Goronzy, J. J., Rando, T. A., Effros, R. B., Lucia, A., Kleinstreuer, N., & Slavich, G. M. (2019). Chronic inflammation in the etiology of disease across the life span. *Nature Medicine*, 25(12), 1822–1832. <https://doi.org/10.1038/s41591-019-0675-0>
- Fuss, I. J., Heller, F., Boirivant, M., Leon, F., Yoshida, M., Fichtner-Feigl, S., Yang, Z., Exley, M., Kitani, A., Blumberg, R. S., Mannon, P., & Strober, W. (2004). Nonclassical CD1D-restricted NK T cells that produce IL-13 characterize an atypical th2 response in ulcerative colitis. *Journal of Clinical Investigation*, 113(10), 1490–1497. <https://doi.org/10.1172/jci19836>

- González-Serna, D., Ortiz-Fernández, L., Vargas, S., García, A., Raya, E., Fernández-Gutierrez, B., López-Longo, F. J., Balsa, A., González-Álvaro, I., Narvaez, J., Gómez-Vaquero, C., Sabio, J. M., García-Portales, R., González-Escribano, M. F., Tolosa, C., Carreira, P., Kiemeny, L., Coenen, M. J. H., Witte, T., & Schneider, M. (2018). Association of a rare variant of the TNFSF13B gene with susceptibility to Rheumatoid Arthritis and Systemic Lupus Erythematosus. *Scientific Reports*, 8(1). <https://doi.org/10.1038/s41598-018-26573-4>
- Government of Canada. (2022, February 9). *Population growth in Canada's rural areas, 2016 to 2021*. [www12.Statcan.gc.ca. https://www12.statcan.gc.ca/census-recensement/2021/as-sa/98-200-x/2021002/98-200-x2021002-eng.cfm](https://www12.statcan.gc.ca/census-recensement/2021/as-sa/98-200-x/2021002/98-200-x2021002-eng.cfm)
- Gui, J., Tosteson, T. D., & Borsuk, M. (2012). Weighted multiple testing procedures for genomic studies. *BioData mining*, 5(1), 4. <https://doi.org/10.1186/1756-0381-5-4>
- Gui, X., Iacucci, M., Ghosh, S. (2020). Dysregulation of IL6/IL6R-STAT3-SOCS3 signaling pathway in IBD-associated colorectal dysplastic lesions as compared to sporadic colorectal adenomas in non-IBD patients. *Pathology - Research and Practice*, 216(11). <https://doi.org/10.1016/j.prp.2020.153211>.
- Hamel, V., Polsky, J. Y., Milena Nardocci, Kirkpatrick, S. I., Vanderlee, L., Hammond, D., Didier Garriguet, Shanks, C. B., Laura, M., Robitaille, É., & Jean-Claude Moubarac. (2024). Who is consuming ultra-processed food in Canada? A cross-sectional analysis of the 2018/2019 International Food Policy Study. *Applied Physiology Nutrition and Metabolism*. <https://doi.org/10.1139/apnm-2024-0218>
- Hanahan, D. (2022). Hallmarks of cancer: New dimensions. *Cancer Discovery*, 12(1), 31–46. <https://doi.org/10.1158/2159-8290.cd-21-1059>
- Hang, D., Wang, L., Fang, Z., Du, M., Wang, K., He, X., Khandpur, N., Rossato, S. L., Wu, K., Hu, Z., Shen, H., Ogino, S., Chan, A. T., Giovannucci, E. L., Zhang, F. F., & Song, M. (2023). Ultra-processed food consumption and risk of colorectal cancer precursors: results from 3 prospective cohorts. *Journal of the National Cancer Institute*, 115(2), 155–164. <https://doi.org/10.1093/jnci/djac221>
- Health Canada. (2021). *Canadian alcohol and drugs survey (CADS): Summary of results for 2019*. Government of Canada. <https://www.canada.ca/en/health-canada/services/canadian-alcohol-drugs-survey/2019-summary.html>
- Hippe, J., Maddalena, V., Heath, S., Jesso, B., McCahon, M., & Olson, K. (2014). Access to health services in Western Newfoundland, Canada: Issues, barriers and recommendations emerging from a community-engaged research project. *Gateways: International Journal of Community Research and Engagement*, 7(1). <https://doi.org/10.5130/ijcre.v7i1.3390>
- Imge Hulus, Gamazon, E. R., Skol, A. D., Xicola, R. M., Llor, X., Kenan Onel, Ellis, N. A., & Kupfer, S. S. (2015). Enrichment of inflammatory bowel disease and colorectal cancer risk variants in colon expression quantitative trait loci. *BMC Genomics*, 16(1). <https://doi.org/10.1186/s12864-015-1292-z>
- Impact of IBD in Canada Report. Crohn's Colitis Canada. (2023). [https://crohnsandcolitis.ca/Crohns\\_and\\_Colitis/documents/reports/2023-IBD-Report-English-LR.pdf?ext=.pdf](https://crohnsandcolitis.ca/Crohns_and_Colitis/documents/reports/2023-IBD-Report-English-LR.pdf?ext=.pdf)
- Jenkins, M. A., Win, A. K., Dowty, J. G., MacInnis, R. J., Makalic, E., Schmidt, D. F., Dite, G. S., Kapuscinski, M., Clendenning, M., Rosty, C., Winship, I. M., Emery, J. D., Saya, S., Macrae, F. A., Ahnen, D. J., Duggan, D., Figueiredo, J. C., Lindor, N. M., Haile, R. W., Potter, J. D., ... Hopper, J. L. (2019). Ability of known susceptibility SNPs to predict colorectal cancer risk for persons with and without a family history. *Familial cancer*, 18(4), 389–397. <https://doi.org/10.1007/s10689-019-00136-6>

- Jostins, L., Ripke, S., Weersma, R. *et al.* Host–microbe interactions have shaped the genetic architecture of inflammatory bowel disease. *Nature* **491**, 119–124 (2012). <https://doi.org/10.1038/nature11582>
- Kaplan, G. The global burden of IBD: from 2015 to 2025. *Nat Rev Gastroenterol Hepatol* **12**, 720–727 (2015). <https://doi.org/10.1038/nrgastro.2015.150>
- Kaplan, G. G., Bernstein, C. N., Coward, S., Bitton, A., Murthy, S. K., Nguyen, G. C., Lee, K., Cooke-Lauder, J., & Benchimol, E. I. (2018). The Impact of Inflammatory Bowel Disease in Canada: Epidemiology. *Journal of the Canadian Association of Gastroenterology*, 2(Supplement\_1), S6–S16. <https://doi.org/10.1093/jcag/gwy054>
- Kastrinos, F., Samadder, N. J., & Burt, R. W. (2020). Use of family history and genetic testing to determine risk of colorectal cancer. *Gastroenterology*, 158(2), 389–403. <https://doi.org/10.1053/j.gastro.2019.11.029>
- Kinoshita, T., & Goto, T. (n.d.). Links between Inflammation and Postoperative Cancer Recurrence. *Journal of Clinical Medicine*, 10(2), 228–240. <https://doi.org/10.3390/jcm10020228>
- Koornstra, J. J., Kleibeuker, J. H., MM, C., Fleur EM Rijcken, Hollema, H., GE, E., & Jong, S. de. (2003). Expression of TRAIL (TNF-related apoptosis-inducing ligand) and its receptors in normal colonic mucosa, adenomas, and carcinomas. *The Journal of Pathology*, 200(3), 327–335. <https://doi.org/10.1002/path.1364>
- Kraus, S., & Arber, N. (2009). Inflammation and colorectal cancer. *Current Opinion in Pharmacology*, 9(4), 405–410. <https://doi.org/10.1016/j.coph.2009.06.006>
- Leddin, D., Tamim, H., & Levy, A. R. (2014). Decreasing incidence of inflammatory bowel disease in eastern Canada: A population database study. *BMC Gastroenterology*, 14(1). <https://doi.org/10.1186/1471-230x-14-140>
- Libby, P. (2007). Inflammatory Mechanisms: the Molecular Basis of Inflammation and Disease. *Nutrition Reviews*, 65(3), S140–S146. <https://doi.org/10.1111/j.1753-4887.2007.tb00352.x>
- Lin, M., Zhong, H.-Y., Yim, R. L.-H., Chen, Q.-Y., Du, H., He, H., Lin, K., Zhao, P., Gao, R., Gao, F., & Zhang, M.-Y. (2022). Pan-cancer analysis of oncogenic TNFAIP2 identifying its prognostic value and immunological function in acute myeloid leukemia. *BMC Cancer*, 22(1). <https://doi.org/10.1186/s12885-022-10155-9>
- Makar, K. W., Poole, E. M., Resler, A. J., Seufert, B., Curtin, K., Kleinstein, S. E., Duggan, D., Kulmacz, R. J., Hsu, L., Whitton, J., Carlson, C. S., Rimorin, C. F., Caan, B. J., Baron, J. A., Potter, J. D., Slattery, M. L., & Ulrich, C. M. (2013). Cox-1 (PTGS1) and cox-2 (PTGS2) polymorphisms, NSAID interactions, and risk of colon and rectal cancers in two independent populations. *Cancer Causes & Control*, 24(12), 2059–2075. <https://doi.org/10.1007/s10552-013-0282-1>
- Mansouri, L., Noerenberg, D., Young, E., Mylonas, E., Abdulla, M., Frick, M., Asmar, F., Ljungström, V., Schneider, M., Yoshida, K., Skaftason, A., Pandzic, T., Gonzalez, B., Tasidou, A., Waldhueter, N., Rivas-Delgado, A., Angelopoulou, M., Ziepert, M., Arends, C. M., Couronné, L., ... Damm, F. (2016). Frequent NFKBIE deletions are associated with poor outcome in primary mediastinal B-cell lymphoma. *Blood*, 128(23), 2666–2670. <https://doi.org/10.1182/blood-2016-03-704528>
- McLean, K. (2025, January 2). *LibGuides: HealthyNS: Alcohol*. Library.nshealth.ca. <https://library.nshealth.ca/HealthyLiving/Alcohol>
- Miller, K. D., Nogueira, L., Mariotto, A. B., Rowland, J. H., Yabroff, K. R., Alfano, C. M., Jemal, A., Kramer, J. L., & Siegel, R. L. (2019). Cancer treatment and survivorship statistics, 2019. *CA: A Cancer Journal for Clinicians*, 69(5), 363–385. <https://doi.org/10.3322/caac.21565>

- Morrow, E.H. (2015). The evolution of sex differences in disease. *Biol Sex Differ* 6, 5. <https://doi.org/10.1186/s13293-015-0023-0>
- Nasiri, N., Hu, M., & Hajizadeh, M. (2024). Trends in socioeconomic inequalities in breast cancer mortality in Canada: 1992–2019. *Breast Cancer Research and Treatment.*, 205(3), 533–543. <https://doi.org/10.1007/s10549-024-07277-y>
- National Library of Medicine. (2025). *TNFSF13B TNF superfamily member 13b [Homo sapiens (human)] - Gene - NCBI.* Nih.gov. <https://www.ncbi.nlm.nih.gov/gene/10673>
- National Library of Medicine. (2025a, February 8). *TNFSF12 TNF superfamily member 12 [Homo sapiens (human)] - Gene - NCBI.* Nih.gov. <https://www.ncbi.nlm.nih.gov/gene/8742>
- Narum, S. R. (2006). Beyond Bonferroni: Less conservative analyses for conservation genetics. *Conservation Genetics*, 7(5), 783–787. <https://doi.org/10.1007/s10592-005-9056-y>
- Niccoli, T., & Partridge, L. (2012). Ageing as a risk factor for disease. *Current biology : CB*, 22(17), R741–R752. <https://doi.org/10.1016/j.cub.2012.07.024>
- Novelli, G., Ciccacci, C., Borgiani, P., Papaluca Amati, M., & Abadie, E. (2008). Genetic tests and genomic biomarkers: regulation, qualification and validation. Clinical cases in mineral and bone metabolism: the official journal of the Italian Society of Osteoporosis, Mineral Metabolism, and Skeletal Diseases, 5(2), 149–154.
- Pahwa, R., Jialal, I., & Goyal, A. (2023, August 7). *Chronic inflammation.* NIH.gov; StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK493173/>
- Pekmezović, T. (2010). Gene-environment interaction: a genetic-epidemiological approach. *Journal of Medical Biochemistry*, 29(3), 131–134. <https://doi.org/10.2478/v10011-010-0021-z>
- Peña-Sánchez, J. N., Osei, J. A., Marques Santos, J. D., Jennings, D., Andkhoie, M., Brass, C., Bukassa-Kazadi, G., Lu, X., Johnson-Jennings, M., Porter, L., Porter, R., Quintin, C. L., Sanderson, R., Teucher, U., & Fowler, S. (2022). Increasing Prevalence and Stable Incidence Rates of Inflammatory Bowel Disease Among First Nations: Population-Based Evidence From a Western Canadian Province. *Inflammatory bowel diseases*, 28(4), 514–522. <https://doi.org/10.1093/ibd/izab096>
- Prel, J.-B. du, Hommel, G., Röhrig, B., & Blettner, M. (2009). Confidence Interval or P-Value? Part 4 of a Series on Evaluation of Scientific Publications. *Deutsches Ärzteblatt Online*, 106(19). <https://doi.org/10.3238/arztebl.2009.0335>
- Rawla, P., Sunkara, T., & Barsouk, A. (2019). Epidemiology of Colorectal cancer: incidence, mortality, survival, and Risk Factors. *Gastroenterology Review*, 14(2). <https://doi.org/10.5114/pg.2018.81072>
- Rocchi, A., Benchimol, E. I., Bernstein, C. N., Bitton, A., Feagan, B., Panaccione, R., Glasgow, K. W., Fernandes, A., & Ghosh, S. (2012). Inflammatory bowel disease: A Canadian burden of Illness Review. *Canadian Journal of Gastroenterology*, 26(11), 811–817. <https://doi.org/10.1155/2012/984575>
- Roselli, C., Chaffin, M. D., Weng, L.-C., Aeschbacher, S., Ahlberg, G., Albert, C. M., Almgren, P., Alonso, A., Anderson, C. D., Aragam, K. G., Arking, D. E., Barnard, J., Bartz, T. M., Benjamin, E. J., Bihlmeyer, N. A., Bis, J. C., Bloom, H. L., Boerwinkle, E., Bottinger, E. B., & Brody, J. A. (2018). Multi-ethnic genome-wide association study for atrial fibrillation. *Nature Genetics*, 50(9), 1225–1233. <https://doi.org/10.1038/s41588-018-0133-9>
- Rubin, D. C., Shaker, A., & Levin, M. S. (2012). Chronic intestinal inflammation: inflammatory bowel disease and colitis-associated colon cancer. *Frontiers in Immunology*, 3(107), 1-6. <https://doi.org/10.3389/fimmu.2012.00107>

- Saint-Jacques, N., Brown, P. E., Purcell, J., Rainham, D. G., Terashima, M., & Dummer, T. J. (2023). The Nova Scotia Community Cancer Matrix: A geospatial tool to support cancer prevention. *Social Science & Medicine*, 330. <https://doi.org/10.1016/j.socscimed.2023.116038>
- Salla, M., Guo, J., Joshi, H., Gordon, M., Dooky, H., & Lai, J. (2023). Novel Biomarkers for Inflammatory Bowel Disease and Colorectal Cancer: An Interplay between Metabolic Dysregulation and Excessive Inflammation. *International Journal of Molecular Sciences*, 24(6). <https://doi.org/10.3390/ijms24065967>
- Sato, Y., Tsujinaka, S., Miura, T., Kitamura, Y., Suzuki, H., & Shibata, C. (2023). Inflammatory Bowel Disease and Colorectal Cancer: Epidemiology, Etiology, Surveillance, and Management. *Cancers*, 15(16), 4154. <https://doi.org/10.3390/cancers15164154>
- Sawicki, T., Ruskowska, M., Danielewicz, A., Niedzwiedska, E., Arlukowicz, T., & Przybylowicz, K. E. (2021). A Review of Colorectal Cancer in Terms of Epidemiology, Risk Factors, Development, Symptoms and Diagnosis. *Cancers*, 13(9), 2025-2048. <https://doi.org/10.3390/cancers13092025>
- Serhan, C. N., Ward, P. A., & Gilroy, D. W. (Eds.). (2010). *Fundamentals of Inflammation*. Cambridge University Press. [https://books.google.ca/books?id=cJq1RMPKEYkC&printsec=frontcover&source=gbs\\_ge\\_summary\\_r&cad=0#v=onepage&q&f=false](https://books.google.ca/books?id=cJq1RMPKEYkC&printsec=frontcover&source=gbs_ge_summary_r&cad=0#v=onepage&q&f=false)
- Shah, S., & Itzkowitz, S. (2022). Colorectal Cancer in Inflammatory Bowel Disease: Mechanisms and Management. *Reviews in Basic and Clinical Gastroenterology and Hepatology*, 162(3), 715-730. <https://doi.org/10.1053/j.gastro.2021.10.035>
- Sheng, H., Shao, J., Washington, M. K., DuBois, R. N., (2001). Prostaglandin E2 Increases Growth and Motility of Colorectal Carcinoma Cells. *Journal of Biological Chemistry*, 276 (21), 18075-18081. <https://doi.org/10.1074/jbc.M009689200>.
- StatCan. (September 27, 2023). Number of immigrants arriving in Canada in 2023, by province or territory of residence [Graph]. In *Statista*. Retrieved November 08, 2024, from <https://www.statista.com/statistics/444906/number-of-immigrants-in-canada/>
- Statistics Canada. (2020, February 18). *Household food security by living arrangement*. Statcan.gc.ca; Government of Canada, Statistics Canada. <https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1310038501>
- Strober, W., Fuss, I., & Mannon, P. (2007, March 1). The Fundamental Basis of Inflammatory Bowel Disease. *The Journal of Clinical Investigation*, 117(3), 514-521. <https://doi.org/10.1172/JCI30587>
- Sung, H., Ferlay, J., Siegel, R. L., Laversanne, M., Soerjomataram, I., Jemal, A., & Bray, F. (2021). Global cancer statistics 2020: Globocan estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA: A Cancer Journal for Clinicians*, 71(3), 209–249. <https://doi.org/10.3322/caac.21660>
- Tanaka, T., Narazaki, M., & Kishimoto, T. (2014). IL-6 in inflammation, immunity, and disease. *Cold Spring Harbor perspectives in biology*, 6(10), a016295. <https://doi.org/10.1101/cshperspect.a016295>
- Tenny, S., & Hoffman, M. R. (2023, March 27). *Case Control Studies*. Nih.gov; StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK448143/>
- Tran, K. B., Lang, J. J., Compton, K., Xu, R., Acheson, A. R., Henrikson, H. J., Kocarnik, J. M., Penberthy, L., Aali, A., Abbas, Q., Abbasi, B., Abbasi-Kangevari, M., Abbasi-Kangevari, Z., Abbastabar, H., Abdelmasseh, M., Abd-Elsalam, S., Abdelwahab, A. A., Abdoli, G., Abdulkadir, H. A., ... Murray, C. J. (2022). The global burden of cancer attributable to risk factors, 2010–19: A systematic analysis for the global burden of disease study 2019. *The Lancet*, 400(10352), 563–591. [https://doi.org/10.1016/s0140-6736\(22\)01438-6](https://doi.org/10.1016/s0140-6736(22)01438-6)

- Tripepi, G., Jager, K. J., Dekker, F. W., Wanner, C., & Zoccali, C. (2008). Bias in clinical research. *Kidney International*, 73(2), 148–153. <https://doi.org/10.1038/sj.ki.5002648>
- van den Berg, I., Buettner, S., Coebergh van den Braak, R. R. J., Ultee, K. H. J., Lingsma, H. F., van Vugt, J. L. A., & Ijzermans, J. N. M. (2020). Low socioeconomic status is associated with worse outcomes after curative surgery for colorectal cancer: Results from a large, multicenter study. *Journal of Gastrointestinal Surgery*, 24(11), 2628–2636. <https://doi.org/10.1007/s11605-019-04435-2>
- Waldum, H., & Fossmark, R. (2023). Inflammation and Digestive Cancer. *International Journal of Molecular Sciences*, 24(17), 13503. <https://doi.org/10.3390/ijms241713503>
- Wei, W., Wang, J., Huang, P., Gou, S., Yu, D., & Zong, L. (2023). Tumor necrosis factor- $\alpha$  induces proliferation and reduces apoptosis of colorectal cancer cells through STAT3 activation. *Immunogenetics*, 75(2), 161–169. <https://doi.org/10.1007/s00251-023-01302-y>
- Windsor, J. W., Kuenzig, M. E., Murthy, S. K., Bitton, A., Bernstein, C. N., Jones, J. L., Lee, K., Targownik, L. E., Peña-Sánchez, J.-N., Rohatinsky, N., Ghandeharian, S., Im, J. H., Davis, T., Weinstein, J., Goddard, Q., Gorospe, J., Benchimol, E. I., & Kaplan, G. G. (2023). The 2023 impact of inflammatory bowel disease in Canada: Executive summary. *Journal of the Canadian Association of Gastroenterology*, 6(Supplement\_2). <https://doi.org/10.1093/jcag/gwad003>
- Yamagishi, H., Kuroda, H., Imai, Y., & Hiraishi, H. (2016). Molecular pathogenesis of sporadic colorectal cancers. *Chinese journal of cancer*, 35, 4. <https://doi.org/10.1186/s40880-015-0066-y>
- Yu, Y., Liu, H., Jin, M., Zhang, M., Pan, Y., Zhang, S., Li, Q., & Chen, K. (2012). The Joint Association of rest and nfkb1 polymorphisms on the risk of colorectal cancer. *Annals of Human Genetics*, 76(4), 269–276. <https://doi.org/10.1111/j.1469-1809.2012.00709.x>
- Yue, Y., Zhang, Q., Wu, S., Wang, S., Cui, C., Yu, M., & Sun, Z. (2020). Identification of key genes involved in JAK/STAT pathway in colorectal cancer. *Molecular Immunology*, 128, 287–297. <https://doi.org/10.1016/j.molimm.2020.10.007>
- Zhang, F., & Qiao, S. (2022). Research Progress on the Relationship Between Inflammation and Colorectal Cancer. *Annals of Gastroenterological Surgery*, 6(2), 204–211. <https://doi.org/10.1002/ags3.12517>
- Zhang, M., Huang, J., Tan, X., Bai, J., Wang, H., Ge, Y., Xiong, H., Shi, J., Lu, W., Lv, Z., & Liang, C. (2015). Common polymorphisms in the NFKBIA gene and cancer susceptibility: A meta-analysis. *Medical Science Monitor*, 21, 3186–3196. <https://doi.org/10.12659/msm.895257>
- Zhao J, Zhu Y, Du M, Wang Y, Vallis J, Parfrey PS, McLaughlin JR, Qi X, Wang PP. (2022). Association between Dietary Fiber Intake and Mortality among Colorectal Cancer Survivors: Results from the Newfoundland Familial Colorectal Cancer Cohort Study and a Meta-Analysis of Prospective Studies. *Cancers*, 14(15), 3801. <https://doi.org/10.3390/cancers14153801>
- Zheng, W., Guo, Y., Kahar, A. et al. RUNX1-induced upregulation of PTGS2 enhances cell growth, migration and invasion in colorectal cancer cells. *Sci Rep* 14, 11670 (2024). <https://doi.org/10.1038/s41598-024-60296-z>
- Zinatizadeh, M. R., Schock, B., Chalbatani, G. M., Zarandi, P. K., Jalali, S. A., & Miri, S. R. (2021). The Nuclear Factor Kappa B (NF- $\kappa$ B) signaling in cancer development and immune diseases. *Genes & Diseases*, 8(3), 287–297. <https://doi.org/10.1016/j.gendis.2020.06.005>

## Appendix

**Supplementary Table 1:** Summary statistics of study population stratified by centre

Variable	Cases (%)	Controls (%)	p-value
<b>Centre</b>			
PATH	300 (53.7)	982 (50.1)	0.515
BCGP	259 (46.6)	979 (49.9)	
<b>Age</b>			
<i>Overall</i>			
Mean, SD	57.12, 8.68	55.25, 7.86	-----
Under 50	110 (19.1)	537 (27.2)	<0.001
50-59	220 (38.3)	762 (38.6)	
60-69	222 (38.6)	674 (34.2)	
70 and above	23 (4.0)	0 (0)	
<i>PATH</i>			
Mean, SD	56.07, 8.61	55.06, 7.62	-----
Under 50	67 (0.22)	261 (0.26)	0.068
50-59	122 (0.41)	409 (0.41)	
60-69	103 (0.34)	325 (0.33)	
70 and above	8 (0.03)	0 (0)	
<i>BCGP</i>			
Mean, SD	58.26, 8.64	55.44, 8.09	<0.001
Under 50	43 (0.16)	276 (0.28)	<0.001
50-59	98 (0.36)	353 (0.36)	
60-69	119 (0.43)	349 (0.36)	
70 and above	15 (0.05)	0 (0)	
<b>Sex</b>			
<i>Overall</i>			
Female	313 (0.54)	1166 (0.59)	0.047
Male	263 (0.46)	807 (0.41)	
<i>PATH</i>			
Female	171 (0.57)	658 (0.66)	0.005
Male	129 (0.43)	337 (0.34)	
<i>BCGP</i>			
Female	142 (0.51)	508 (0.52)	0.939
Male	134 (0.49)	470 (0.48)	
<b>Education</b>			
<i>Overall</i>			

Variable	Cases (%)	Controls (%)	p-value
High school or less	121 (0.21)	395 (0.20)	0.155
Trade, Diploma or Certificate	234 (0.41)	764 (0.39)	
Bachelor's Degree	119 (0.21)	507 (0.26)	
Graduate or Professional Degree	80 (0.14)	297 (0.15)	
<i>PATH</i>			
High school or less	57 (0.19)	189 (0.19)	0.342
Trade, Diploma or Certificate	119 (0.40)	384 (0.39)	
Bachelor's Degree	61 (0.20)	268 (0.27)	
Graduate or Professional Degree	45 (0.15)	153 (0.15)	
<i>BCGP</i>			
High school or less	64 (0.23)	206 (0.21)	0.461
Trade, Diploma or Certificate	115 (0.42)	380 (0.39)	
Bachelor's Degree	58 (0.21)	239 (0.24)	
Graduate or Professional Degree	35 (0.13)	144 (0.15)	
Household income			
<i>Overall</i>			
Less than 50,000	141 (0.24)	331 (0.17)	<0.001
50,000-74,999	127 (0.22)	402 (0.20)	
75,000-99,999	90 (0.16)	390 (0.20)	
100,000-149,999	104 (0.18)	454 (0.23)	
150,000 or more	59 (0.10)	288 (0.15)	
NA	55 (0.10)	109 (0.06)	
<i>PATH</i>			
Less than 50,000	69 (0.23)	199 (0.20)	<0.001
50,000-74,999	56 (0.19)	212 (0.21)	
75,000-99,999	49 (0.16)	188 (0.19)	
100,000-149,999	57 (0.19)	226 (0.23)	
150,000 or more	28 (0.09)	118 (0.12)	
NA	41 (0.14)	52 (0.05)	
<i>BCGP</i>			
Less than 50,000	72 (0.26)	132 (0.13)	<0.001
50,000-74,999	71 (0.26)	190 (0.19)	
75,000-99,999	41 (0.15)	202 (0.21)	
100,000-149,999	47 (0.17)	228 (0.23)	
150,000 or more	31 (0.11)	170 (0.17)	
NA	14 (0.05)	57 (0.06)	
Cigarette Smoker Status			
<i>Overall</i>			

Variable	Cases (%)	Controls (%)	p-value
Non-smoker	253 (0.44)	1006 (0.51)	<b>0.042</b>
Previous	266 (0.46)	826 (0.42)	
Occasional	9 (0.02)	35 (0.02)	
Daily	27 (0.05)	87 (0.04)	
NA	21 (0.04)	20 (0.01)	
<i>PATH</i>			
Non-smoker	126 (0.42)	477 (0.48)	0.566
Previous	131 (0.44)	427 (0.43)	
Occasional	7 (0.02)	23 (0.02)	
Daily	17 (0.06)	62 (0.06)	
NA	19 (0.06)	6 (0.01)	
<i>BCGP</i>			
Non-smoker	127 (0.46)	529 (0.54)	0.078
Previous	135 (0.49)	399 (0.41)	
Occasional	2 (0.01)	12 (0.01)	
Daily	10 (0.04)	25 (0.03)	
NA	2 (0.01)	14 (0.01)	
Alcohol Usage			
<i>Overall</i>			
Never	44 (0.08)	146 (0.07)	0.145
Less than once a week	172 (0.30)	685 (0.35)	
1-5 per week	251 (0.44)	867 (0.44)	
6-7 per week	81 (0.14)	227 (0.11)	
NA	28 (0.05)	49 (0.02)	
<i>PATH</i>			
Never	25 (0.08)	61 (0.06)	0.188
Less than once a week	98 (0.33)	386 (0.39)	
1-5 per week	117 (0.39)	412 (0.41)	
6-7 per week	33 (0.11)	91 (0.09)	
NA	27 (0.09)	45 (0.05)	
<i>BCGP</i>			
Never	19 (0.07)	85 (0.09)	0.279
Less than once a week	74 (0.27)	299 (0.31)	
1-5 per week	134 (0.49)	455 (0.46)	
6-7 per week	48 (0.17)	136 (0.14)	
NA	1 (0)	4 (0)	
BMI			
<i>Overall</i>			

Variable	Cases (%)	Controls (%)	p-value
Mean, SD	27.62, 4.69	27.44, 5.07	0.575
Underweight/Normal	90 (0.16)	422 (0.21)	0.203
Overweight	121 (0.21)	452 (0.23)	
Obese	81 (0.14)	288 (0.15)	
NA	284 (0.49)	812 (0.41)	
<i>PATH</i>			
Mean, SD	28.47, 4.17	29.05, 6.6	0.425
Underweight/Normal	12 (0.04)	52 (0.05)	0.323
Overweight	25 (0.08)	74 (0.07)	
Obese	24 (0.08)	57 (0.06)	
NA	239 (0.80)	812 (0.82)	
<i>BCGP</i>			
Mean, SD	27.39, 4.8	27.14, 4.68	0.475
Underweight/Normal	78 (0.28)	370 (0.38)	0.516
Overweight	96 (0.35)	378 (0.39)	
Obese	57 (0.21)	231 (0.24)	
NA	45 (0.16)	0 (0)	
First-degree Family History of CRC			
<i>Overall</i>			
None	506 (0.88)	1790 (0.91)	0.055
One or more	75 (0.12)	154 (0.09)	
<i>PATH</i>			
None	270 (0.90)	932 (0.94)	<b>0.042</b>
One or more	30 (0.10)	63 (0.06)	
<i>BCGP</i>			
None	236 (0.86)	858 (0.88)	0.349
One or more	45 (0.14)	131 (0.12)	
Colonoscopy/Sigmoidoscopy History			
<i>Overall</i>			
Ever	415 (0.72)	796 (0.40)	<b>&lt;0.001</b>
Never	142 (0.25)	1148 (0.58)	
NA	19 (0.03)	30 (0.02)	
<i>PATH</i>			
Ever	204 (0.68)	405 (0.41)	<b>&lt;0.001</b>
Never	78 (0.26)	570 (0.57)	
NA	18 (0.06)	20 (0.02)	
<i>BCGP</i>			
Ever	211 (0.76)	391 (0.40)	

<b>Variable</b>	<b>Cases (%)</b>	<b>Controls (%)</b>	<b>p-value</b>
Never	64 (0.23)	578 (0.59)	<b>&lt;0.001</b>
NA	1 (0)	10 (0.01)	
<b>Crohn's Disease (CD) Diagnosis</b>			
<i>Overall</i>			
Ever	12 (0.02)	20 (0.01)	0.067
Never	558 (0.97)	1944 (0.98)	
NA	6 (0.01)	10 (0.01)	
<i>PATH</i>			
Ever	8 (0.03)	13 (0.01)	0.164
Never	288 (0.96)	976 (0.98)	
NA	4 (0.01)	6 (0.01)	
<i>BCGP</i>			
Ever	4 (0.01)	7 (0.01)	0.2658
Never	270 (0.98)	986 (0.99)	
<b>Ulcerative Colitis (UC) Diagnosis</b>			
<i>Overall</i>			
Ever	10 (0.02)	25 (0.01)	0.508
Never	557 (0.97)	1928 (0.98)	
NA	9 (0.02)	21 (0.01)	
<i>PATH</i>			
Ever	5 (0.02)	14 (0.01)	0.952
Never	291 (0.97)	971 (0.98)	
NA	4 (0.01)	10 (0.01)	
<i>BCGP</i>			
Ever	5 (0.02)	11 (0.01)	0.354
Never	266 (0.96)	957 (0.98)	
NA	5 (0.02)	11 (0.01)	

**Supplementary Table 2:** Information Regarding SNPs and their Associated Genes, Inheritance Model and Minor and Major Allele Frequencies

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
IL6	rs1524100	Dominant	G = 0.287	A = 0.713	0.899
IL6	rs1524104	Recessive	G = 0.142	A = 0.858	0.800
IL6	rs2905341	Additive	T = 0.044	C = 0.956	0.793
IL6	rs4722177	Recessive	T = 0.44	C = 0.56	0.593
IL6	rs7801617	Recessive	A = 0.096	G = 0.904	0.637
IL6	rs13225099	Dominant	A = 0.256	G = 0.744	0.522
IL6	rs55733027	Dominant	G = 0.037	A = 0.963	0.924
IL6	rs117914483	Additive	C = 0.041	T = 0.959	0.968
IL6	rs34788132	Dominant	G = 0.22	A = 0.78	0.503
IL6	rs57561814	Dominant	C = 0.072	G = 0.928	0.677
IL6	rs73683960	Recessive	G = 0.266	A = 0.734	0.180
IL6	rs1800795	Dominant	C = 0.421	G = 0.579	0.725
IL6	rs2069843	Dominant	A = 0.022	G = 0.978	0.312
IL6	rs55941116	Dominant	T = 0.299	C = 0.701	0.640
IL6	rs73276345	Dominant	G = 0.054	A = 0.946	0.491
IL6	rs1996962	Additive	A = 0.492	G = 0.508	0.475
IL6	rs10225286	Additive	A = 0.418	G = 0.582	0.295
IL6	rs68130034	Recessive	C = 0.073	T = 0.927	0.273
IL6	rs60129508	Additive	A = 0.031	C = 0.969	0.968
<b>IL6</b>	<b>rs34413901</b>	<b>Recessive</b>	<b>A = 0.063</b>	<b>G = 0.937</b>	<b>0.030</b>
IL6	rs34929064	Dominant	C = 0.254	T = 0.746	0.749
IL6	rs56858483	Dominant	A = 0.045	G = 0.955	0.934
IL6	rs62449492	Additive	A = 0.077	G = 0.923	0.236
IL6	rs4719714	Recessive	T = 0.235	A = 0.765	0.347
IL6	rs62449495	Dominant	A = 0.203	G = 0.797	0.869
IL6	rs35610689	Recessive	G = 0.285	A = 0.715	0.070
IL6	rs62448267	Dominant	C = 0.208	T = 0.792	0.841
IL6	rs117659798	Additive	G = 0.015	A = 0.985	0.972
IL6	rs117814509	Additive	G = 0.03	A = 0.97	0.972
IL6	rs75783788	Additive	A = 0.018	C = 0.982	0.972
IL6	rs2961304	Additive	T = 0.05	C = 0.95	0.643
IL6	rs10249648	Dominant	T = 0.18	G = 0.82	0.794
IL6	rs1546762	Dominant	C = 0.476	T = 0.524	0.604
IL6	rs2069824	Dominant	C = 0.075	T = 0.925	0.956
IL6	rs2069827	Dominant	T = 0.085	G = 0.915	0.904
IL6	rs1474347	Dominant	C = 0.421	A = 0.579	0.731
IL6	rs1524107	Dominant	T = 0.053	C = 0.947	0.487
IL6	rs13243585	Recessive	C = 0.093	A = 0.907	0.677
IL6	rs1029741	Recessive	C = 0.4	T = 0.6	0.966

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
<b>IL6</b>	---	<b>Dominant</b>	<b>C = 0.409</b>	<b>A = 0.591</b>	<b>&lt; 0.0001</b>
IL6R	rs4129267	Additive	T = 0.41	C = 0.59	0.072
IL6R	rs12719998	Dominant	G = 0.301	A = 0.699	0.267
IL6R	rs2229238	Additive	T = 0.185	C = 0.815	0.054
IL6R	rs7549250	Recessive	C = 0.428	T = 0.572	0.317
IL6R	rs11265608	Additive	A = 0.095	G = 0.905	0.950
<b>IL6R</b>	<b>rs1386821</b>	<b>Dominant</b>	<b>G = 0.194</b>	<b>T = 0.806</b>	<b>0.005</b>
IL6R	rs115224285	Additive	T = 0.02	C = 0.98	0.972
IL6R	rs4240872	Additive	C = 0.231	T = 0.769	0.069
IL6R	rs4509570	Additive	G = 0.232	C = 0.768	0.067
IL6R	rs35717427	Dominant	A = 0.128	G = 0.872	0.877
IL6R	rs61812598	Dominant	A = 0.412	G = 0.588	0.088
IL6R	rs6698040	Additive	T = 0.231	C = 0.769	0.063
IL6R	rs115437600	Additive	A = 0.051	G = 0.949	0.968
IL6R	rs79794939	Recessive	T = 0.078	C = 0.922	0.939
IL6R	rs116141616	Additive	A = 0.027	G = 0.973	0.963
<b>IL6R</b>	<b>rs6672627</b>	<b>Dominant</b>	<b>A = 0.147</b>	<b>C = 0.853</b>	<b>0.048</b>
IL6R	rs1889313	Additive	A = 0.159	C = 0.841	0.526
IL6R	rs12121085	Additive	T = 0.446	G = 0.554	0.658
IL6R	rs4845625	Recessive	T = 0.424	C = 0.576	0.133
IL6R	rs4845626	Dominant	T = 0.164	G = 0.836	0.217
IL6R	---	Dominant	C = 0.411	A = 0.589	0.091
IL6R	rs4537545	Additive	T = 0.423	C = 0.577	0.088
IL6R	rs79219014	Additive	T = 0.026	G = 0.974	0.574
IL6R	rs115200978	Dominant	T = 0.018	C = 0.982	0.538
IL6ST	rs13357543	Recessive	C = 0.343	T = 0.657	0.866
IL6ST	rs34417936	Additive	T = 0.03	C = 0.97	0.972
IL6ST	rs6861772	Recessive	G = 0.141	A = 0.859	0.087
IL6ST	rs74904741	Additive	G = 0.028	A = 0.972	0.961
IL6ST	rs11574783	Recessive	C = 0.073	T = 0.927	0.422
IL6ST	rs2228043	Recessive	C = 0.127	G = 0.873	0.168
IL6ST	rs78443884	Dominant	C = 0.063	G = 0.937	0.282
IL6ST	rs78474013	Recessive	C = 0.068	T = 0.932	0.392
IL6ST	rs72765604	Dominant	A = 0.032	G = 0.968	0.361
NFKB1	rs10489114	Additive	C = 0.022	T = 0.978	0.961
NFKB1	rs10516494	Recessive	G = 0.391	A = 0.609	0.705
NFKB1	rs1598860	Recessive	T = 0.36	C = 0.64	0.134
NFKB1	rs2085548	Recessive	T = 0.297	C = 0.703	0.302
NFKB1	rs230488	Additive	A = 0.261	G = 0.739	0.667
NFKB1	rs3774934	Recessive	A = 0.092	G = 0.908	0.057
NFKB1	rs4648022	Additive	T = 0.081	C = 0.919	0.704
NFKB1	rs4648075	Recessive	A = 0.037	G = 0.963	0.623

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
NFKB1	rs17825737	Additive	G = 0.11	A = 0.89	0.233
NFKB1	rs34333163	Recessive	G = 0.079	A = 0.921	0.531
NFKB1	rs3774965	Dominant	C = 0.037	A = 0.963	0.387
NFKB1	rs4647972	Additive	T = 0.042	C = 0.958	0.961
NFKB1	rs93059	Additive	A = 0.456	G = 0.544	0.385
NFKB1	rs75772141	Additive	C = 0.049	T = 0.951	0.447
NFKB1	rs6843751	Recessive	T = 0.311	C = 0.689	0.856
NFKB1	rs7657947	Additive	A = 0.311	C = 0.689	0.716
NFKB1	rs72694157	Dominant	G = 0.1	A = 0.9	0.137
NFKB1	rs62328514	Dominant	A = 0.029	G = 0.971	0.867
NFKB1	rs72694174	Dominant	A = 0.063	C = 0.937	0.395
NFKB1	rs2205438	Dominant	A = 0.132	G = 0.868	0.644
NFKB1	rs230487	Dominant	C = 0.289	A = 0.711	0.832
NFKB1	rs62328535	Additive	C = 0.408	T = 0.592	0.200
NFKB1	rs747559	Recessive	G = 0.39	A = 0.61	0.265
NFKB1	rs1585213	Recessive	T = 0.383	C = 0.617	0.345
NFKB1	rs230529	Recessive	T = 0.393	C = 0.607	0.086
NFKB1	rs79651301	Dominant	A = 0.289	G = 0.711	0.793
NFKB1	rs7665090	Dominant	A = 0.495	G = 0.505	0.775
NFKB1	rs61325025	Additive	C = 0.024	T = 0.976	0.973
NFKB1	rs2169598	Recessive	C = 0.418	T = 0.582	0.457
NFKB1	rs2926005	Recessive	A = 0.074	G = 0.926	0.101
NFKB1	rs4648104	Additive	C = 0.047	G = 0.953	0.963
NFKB1	rs78288143	Dominant	T = 0.036	C = 0.964	0.929
NFKB1	rs80322712	Additive	T = 0.045	C = 0.955	0.965
NFKB1	rs77855727	Dominant	C = 0.031	T = 0.969	0.582
NFKB1	rs7695247	Dominant	C = 0.054	T = 0.946	0.654
NFKB1	---	Recessive	C = 0.298	T = 0.702	0.971
NFKB1	rs2169596	Dominant	T = 0.361	C = 0.639	0.612
NFKB1	rs4648038	Dominant	G = 0.069	A = 0.931	0.396
NFKB1	rs4648073	Additive	T = 0.015	G = 0.985	0.515
NFKB1	rs4648126	Additive	G = 0.024	A = 0.976	0.961
NFKB2	rs7076748	Dominant	C = 0.265	G = 0.735	0.252
NFKB2	rs11574852	Additive	C = 0.042	A = 0.958	0.991
NFKB2	rs117715797	Dominant	T = 0.021	C = 0.979	0.533
NFKBIA	rs1028593	Recessive	G = 0.302	A = 0.698	0.976
NFKBIA	rs10483453	Dominant	C = 0.162	T = 0.838	0.802
NFKBIA	rs10872893	Additive	T = 0.198	C = 0.802	0.475
NFKBIA	rs1177257	Dominant	T = 0.273	C = 0.727	0.552
NFKBIA	rs12431807	Recessive	T = 0.145	C = 0.855	0.556
NFKBIA	rs17103338	Dominant	A = 0.259	G = 0.741	0.902
NFKBIA	rs3138045	Recessive	C = 0.213	T = 0.787	0.270

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
NFKBIA	rs3138053	Recessive	C = 0.287	T = 0.713	0.273
NFKBIA	rs36052232	Additive	G = 0.036	A = 0.964	0.958
NFKBIA	rs7145650	Dominant	A = 0.41	G = 0.59	0.258
NFKBIA	rs8904	Additive	A = 0.367	G = 0.633	0.132
NFKBIA	rs9322955	Recessive	G = 0.415	A = 0.585	0.705
NFKBIA	rs10140996	Recessive	G = 0.204	A = 0.796	0.764
NFKBIA	rs10147603	Dominant	C = 0.257	T = 0.743	0.525
NFKBIA	rs116861132	Dominant	A = 0.031	G = 0.969	0.968
NFKBIA	rs8005252	Additive	C = 0.108	A = 0.892	0.868
NFKBIA	rs72666683	Recessive	T = 0.029	C = 0.971	0.105
NFKBIA	rs79929575	Dominant	A = 0.029	G = 0.971	0.431
NFKBIA	rs74780291	Recessive	A = 0.09	G = 0.91	0.701
NFKBIA	rs77057144	Additive	T = 0.034	C = 0.966	0.969
NFKBIA	rs80269636	Dominant	A = 0.047	C = 0.953	0.578
NFKBIA	rs2057338	Recessive	C = 0.214	T = 0.786	0.488
NFKBIA	rs36073684	Recessive	G = 0.383	A = 0.617	0.225
NFKBIA	rs61988300	Recessive	G = 0.197	A = 0.803	0.507
NFKBIA	rs75426604	Dominant	A = 0.129	C = 0.871	0.878
NFKBIA	rs73242919	Dominant	T = 0.037	C = 0.963	0.273
NFKBIA	rs4981289	Recessive	A = 0.17	G = 0.83	0.701
NFKBIA	rs12895150	Dominant	T = 0.373	C = 0.627	0.247
NFKBIA	rs60411253	Recessive	T = 0.067	C = 0.933	0.772
<b>NFKBIA</b>	<b>rs2233416</b>	<b>Recessive</b>	<b>A = 0.055</b>	<b>G = 0.945</b>	<b>0.008</b>
NFKBIA	rs2233415	Dominant	A = 0.251	G = 0.749	0.211
NFKBIA	rs1957106	Additive	A = 0.292	G = 0.708	0.615
NFKBIA	rs8018024	Additive	T = 0.366	C = 0.634	0.396
NFKBIA	rs8019113	Recessive	G = 0.493	A = 0.507	0.662
NFKBIA	rs66653785	Dominant	A = 0.156	G = 0.844	0.164
NFKBIA	rs79132480	Recessive	G = 0.094	T = 0.906	0.034
NFKBIA	rs11156885	Recessive	A = 0.058	G = 0.942	0.954
NFKBIA	rs74654111	Dominant	T = 0.035	C = 0.965	0.510
NFKBIA	rs191511168	Dominant	T = 0.013	G = 0.987	0.507
NFKBIA	rs111330169	Dominant	C = 0.102	T = 0.898	0.433
NFKBIA	rs78354812	Additive	T = 0.05	C = 0.95	0.959
NFKBIA	rs117002247	Additive	G = 0.019	A = 0.981	0.973
NFKBIA	rs17103212	Dominant	T = 0.245	C = 0.755	0.337
NFKBIA	rs4982259	Dominant	C = 0.415	T = 0.585	0.666
NFKBIA	rs17103237	Additive	C = 0.297	T = 0.703	0.600
NFKBIA	rs8016947	Additive	T = 0.452	G = 0.548	0.166
NFKBIA	rs11621391	Recessive	T = 0.18	C = 0.82	0.718
NFKBIA	rs7155714	Dominant	A = 0.143	G = 0.857	0.630
NFKBIA	rs12892002	Recessive	A = 0.023	G = 0.977	0.625

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
NFKBIA	rs4982269	Recessive	C = 0.351	T = 0.649	0.897
NFKBIA	rs1050851	Recessive	A = 0.229	G = 0.771	0.367
NFKBIA	rs17103274	Dominant	C = 0.083	T = 0.917	0.601
NFKBIA	rs8018407	Dominant	T = 0.365	C = 0.635	0.265
NFKBIA	rs28372224	Recessive	T = 0.056	G = 0.944	0.976
NFKBIA	rs10141358	Dominant	T = 0.154	C = 0.846	0.431
NFKBIA	rs12433910	Recessive	T = 0.032	C = 0.968	0.628
NFKBIB	rs12979755	Dominant	T = 0.392	C = 0.608	0.407
NFKBIB	rs4803006	Dominant	A = 0.391	G = 0.609	0.370
NFKBIB	rs11879872	Dominant	C = 0.243	T = 0.757	0.045
NFKBIB	rs3136644	Dominant	G = 0.154	A = 0.846	0.178
NFKBID	rs60869994	Dominant	G = 0.035	A = 0.965	0.624
NFKBID	rs80265681	Recessive	C = 0.023	T = 0.977	0.299
NFKBID	rs7251879	Recessive	G = 0.045	A = 0.955	0.196
NFKBID	rs8113704	Recessive	G = 0.045	A = 0.955	0.114
NFKBID	rs6510512	Dominant	T = 0.017	G = 0.983	0.493
NFKBIE	rs12211448	Additive	A = 0.136	C = 0.864	0.158
NFKBIE	rs3997540	Dominant	G = 0.216	A = 0.784	0.294
NFKBIE	rs28385698	Additive	A = 0.034	G = 0.966	0.429
NFKBIE	rs2233424	Dominant	T = 0.045	C = 0.955	0.629
NFKBIE	rs28385699	Dominant	T = 0.049	C = 0.951	0.358
NFKBIE	rs730775	Recessive	G = 0.423	A = 0.577	0.975
NFKBIE	rs2233423	Dominant	A = 0.023	G = 0.977	0.335
NFKBIE	rs513688	Dominant	G = 0.363	T = 0.637	0.747
NFKBIE	rs2233433	Dominant	A = 0.046	G = 0.954	0.748
NFKBIL1	rs13192469	Dominant	C = 0.279	T = 0.721	0.960
NFKBIL1	rs13215091	Dominant	A = 0.038	G = 0.962	0.946
NFKBIL1	rs2071590	Recessive	A = 0.363	G = 0.637	0.314
NFKBIL1	rs2071591	Recessive	A = 0.347	G = 0.653	0.873
NFKBIL1	rs2523500	Recessive	G = 0.363	A = 0.637	0.256
NFKBIL1	rs2844484	Recessive	A = 0.399	G = 0.601	0.468
NFKBIL1	rs2857602	Recessive	G = 0.4	A = 0.6	0.537
NFKBIL1	rs2857708	Dominant	T = 0.133	C = 0.867	0.798
NFKBIL1	rs3093539	Additive	A = 0.017	G = 0.983	0.968
NFKBIL1	rs6916921	Dominant	T = 0.084	C = 0.916	0.798
NFKBIL1	rs6929796	Recessive	A = 0.156	G = 0.844	0.827
NFKBIL1	rs7762619	Additive	G = 0.015	T = 0.985	0.972
NFKBIL1	rs928815	Recessive	T = 0.4	G = 0.6	0.518
NFKBIL1	---	Dominant	G = 0.038	C = 0.962	0.958
NFKBIL1	rs114820425	Dominant	A = 0.024	G = 0.976	0.468
NFKBIL1	rs2857709	Recessive	A = 0.143	G = 0.857	0.867
NFKBIL1	rs2009658	Recessive	G = 0.158	C = 0.842	0.823

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
NFKBIL1	rs115623520	Additive	G = 0.02	A = 0.98	0.972
NFKBIL1	rs72847263	Additive	G = 0.025	C = 0.975	0.972
NFKBIL1	rs72847264	Additive	G = 0.025	A = 0.975	0.980
NFKBIL1	rs3219184	Recessive	G = 0.063	A = 0.937	0.112
NFKBIL1	rs2255798	Dominant	C = 0.134	G = 0.866	0.668
NFKBIL1	rs2857605	Recessive	C = 0.211	T = 0.789	0.640
NFKBIL1	rs2239707	Recessive	C = 0.322	T = 0.678	0.605
NFKBIL1	rs2230365	Dominant	T = 0.147	C = 0.853	0.511
NFKBIL1	rs2844482	Recessive	T = 0.16	C = 0.84	0.843
NFKBIL1	rs4947324	Recessive	T = 0.093	C = 0.907	0.706
NFKBIL1	rs17200740	Additive	C = 0.015	A = 0.985	0.972
NFKBIZ	rs11718446	Recessive	A = 0.284	G = 0.716	0.192
NFKBIZ	rs616597	Dominant	A = 0.224	C = 0.776	0.175
NFKBIZ	rs7644388	Recessive	T = 0.248	C = 0.752	0.748
NFKBIZ	rs1626282	Dominant	A = 0.466	G = 0.534	0.869
NFKBIZ	rs77121775	Recessive	C = 0.051	T = 0.949	0.869
NFKBIZ	rs677011	Dominant	G = 0.322	A = 0.678	0.926
NFKBIZ	rs74951263	Additive	T = 0.218	C = 0.782	0.516
NFKBIZ	rs75645148	Additive	G = 0.077	A = 0.923	0.386
NFKBIZ	rs72949505	Dominant	T = 0.071	C = 0.929	0.515
NFKBIZ	rs2860329	Dominant	G = 0.397	A = 0.603	0.978
NFKBIZ	rs1456202	Recessive	G = 0.403	A = 0.597	0.603
NFKBIZ	rs80099440	Recessive	A = 0.055	G = 0.945	0.198
NFKBIZ	rs2926538	Recessive	G = 0.458	A = 0.542	0.790
NFKBIZ	rs77044702	Recessive	G = 0.052	C = 0.948	0.333
NFKBIZ	rs62280827	Additive	C = 0.13	G = 0.87	0.629
NFKBIZ	rs4683950	Recessive	G = 0.184	A = 0.816	0.976
NFKBIZ	rs76769018	Additive	T = 0.029	C = 0.971	0.964
NFKBIZ	rs76200452	Additive	G = 0.023	A = 0.977	0.071
NFKBIZ	rs16844348	Recessive	T = 0.053	C = 0.947	0.506
NFKBIZ	rs1672385	Dominant	G = 0.169	A = 0.831	0.709
PTGS1	rs10306122	Dominant	C = 0.061	T = 0.939	0.961
PTGS1	rs10513401	Recessive	G = 0.303	A = 0.697	0.969
PTGS1	rs11521582	Dominant	T = 0.141	C = 0.859	0.149
PTGS1	rs12353214	Dominant	T = 0.105	C = 0.895	0.516
PTGS1	rs16911650	Additive	C = 0.075	T = 0.925	0.443
PTGS1	rs2778636	Dominant	A = 0.316	G = 0.684	0.914
<b>PTGS1</b>	<b>rs5789</b>	<b>Recessive</b>	<b>A = 0.03</b>	<b>C = 0.97</b>	<b>0.004</b>
PTGS1	rs883485	Additive	G = 0.071	A = 0.929	0.593
<b>PTGS1</b>	<b>rs59379461</b>	<b>Additive</b>	<b>G = 0.031</b>	<b>A = 0.969</b>	<b>0.005</b>
<b>PTGS1</b>	<b>rs1234909</b>	<b>Dominant</b>	<b>G = 0.164</b>	<b>A = 0.836</b>	<b>0.011</b>
PTGS1	rs1332408	Dominant	A = 0.229	C = 0.771	0.382

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
PTGS1	rs76267794	Additive	T = 0.011	C = 0.989	0.484
PTGS1	rs10760221	Dominant	T = 0.034	C = 0.966	0.866
PTGS1	rs72769720	Dominant	T = 0.06	C = 0.94	0.405
PTGS1	rs10818682	Dominant	A = 0.136	G = 0.864	0.966
PTGS1	rs72769722	Recessive	G = 0.055	T = 0.945	0.960
PTGS1	rs76865668	Recessive	A = 0.088	C = 0.912	0.352
PTGS1	rs10306114	Additive	G = 0.072	A = 0.928	0.589
PTGS1	rs1213265	Recessive	C = 0.081	T = 0.919	0.225
PTGS1	rs35119072	Recessive	G = 0.139	T = 0.861	0.930
PTGS1	rs72769736	Recessive	T = 0.024	G = 0.976	0.361
PTGS1	rs72769741	Additive	G = 0.021	A = 0.979	0.972
PTGS1	rs79671731	Additive	T = 0.03	C = 0.97	0.717
PTGS1	rs7049102	Recessive	C = 0.112	T = 0.888	0.310
PTGS1	rs1330344	Dominant	C = 0.215	T = 0.785	0.847
PTGS1	rs10306163	Dominant	G = 0.193	A = 0.807	0.066
<b>PTGS1</b>	<b>rs12551233</b>	<b>Additive</b>	<b>G = 0.065</b>	<b>A = 0.935</b>	<b>0.036</b>
PTGS1	rs9332460	Recessive	G = 0.192	A = 0.808	0.866
PTGS1	rs181790703	Recessive	G = 0.122	A = 0.878	0.698
PTGS1	rs117593391	Additive	C = 0.025	T = 0.975	0.972
PTGS1	rs77329588	Dominant	T = 0.029	C = 0.971	0.463
PTGS1	rs3842787	Additive	T = 0.072	C = 0.928	0.515
PTGS2	rs10911902	Dominant	T = 0.182	C = 0.818	0.758
PTGS2	rs34070874	Additive	C = 0.021	T = 0.979	0.816
PTGS2	rs5275	Additive	G = 0.344	A = 0.656	0.590
PTGS2	rs16825516	Dominant	A = 0.084	G = 0.916	0.434
PTGS2	rs11578436	Additive	A = 0.136	G = 0.864	0.094
PTGS2	rs6694957	Recessive	A = 0.263	G = 0.737	0.136
PTGS2	rs1569879	Additive	T = 0.134	C = 0.866	0.131
PTGS2	rs34249733	Dominant	T = 0.128	C = 0.872	0.581
PTGS2	rs477416	Dominant	A = 0.027	C = 0.973	0.365
<b>PTGS2</b>	<b>rs79745692</b>	<b>Additive</b>	<b>G = 0.026</b>	<b>T = 0.974</b>	<b>0.013</b>
PTGS2	rs6425034	Dominant	T = 0.164	C = 0.836	0.059
PTGS2	rs71634175	Recessive	C = 0.127	T = 0.873	0.639
PTGS2	rs76941944	Dominant	A = 0.087	G = 0.913	0.787
PTGS2	rs79032296	Dominant	A = 0.081	C = 0.919	0.200
PTGS2	rs10911903	Additive	T = 0.137	G = 0.863	0.070
PTGS2	rs115997934	Recessive	T = 0.036	G = 0.964	0.104
PTGS2	rs78114044	Dominant	G = 0.024	A = 0.976	0.771
PTGS2	rs115577537	Dominant	T = 0.033	C = 0.967	0.712
PTGS2	rs114949270	Additive	T = 0.027	G = 0.973	0.961
PTGS2	rs505366	Dominant	G = 0.047	A = 0.953	0.965
PTGS2	rs79322388	Additive	T = 0.035	C = 0.965	0.975

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNF	rs1800610	Dominant	A = 0.082	G = 0.918	0.874
TNF	rs3093664	Additive	G = 0.071	A = 0.929	0.455
TNF	rs1799964	Recessive	C = 0.209	T = 0.791	0.969
TNF	rs1800750	Additive	A = 0.017	G = 0.983	0.961
TNF	rs1800629	Recessive	A = 0.188	G = 0.812	0.892
TNF	rs3093662	Recessive	G = 0.069	A = 0.931	0.926
TNF	rs3093668	Additive	C = 0.036	G = 0.964	0.968
TNF	rs3093671	Additive	A = 0.025	G = 0.975	0.961
TNF	rs3093559	Dominant	T = 0.022	C = 0.978	0.275
TNF	rs1800630	Recessive	A = 0.171	C = 0.829	0.616
<b>TNF</b>	<b>rs3093665</b>	<b>Dominant</b>	<b>C = 0.021</b>	<b>A = 0.979</b>	<b>0.036</b>
TNF	rs769177	Additive	T = 0.024	C = 0.976	0.972
TNFAIP1	rs3093720	Recessive	A = 0.116	C = 0.884	0.611
TNFAIP1	rs2234116	Recessive	T = 0.061	C = 0.939	0.371
TNFAIP2	rs4906280	Additive	T = 0.1	C = 0.9	0.627
TNFAIP2	rs8014278	Recessive	A = 0.181	G = 0.819	0.634
TNFAIP2	rs72706644	Dominant	G = 0.024	A = 0.976	0.481
TNFAIP2	rs3759571	Additive	A = 0.214	G = 0.786	0.225
TNFAIP2	---	Recessive	T = 0.111	G = 0.889	0.127
TNFAIP2	rs8011815	Additive	G = 0.292	A = 0.708	0.708
TNFAIP2	rs55865473	Additive	T = 0.059	C = 0.941	0.963
TNFAIP2	rs115051200	Dominant	T = 0.047	C = 0.953	0.612
TNFAIP2	rs112200265	Recessive	G = 0.031	T = 0.969	0.362
TNFAIP2	rs71417843	Dominant	T = 0.026	C = 0.974	0.664
TNFAIP2	rs118140944	Recessive	G = 0.037	A = 0.963	0.197
TNFAIP2	rs116958646	Additive	G = 0.073	T = 0.927	0.452
TNFAIP2	rs7145735	Additive	G = 0.16	A = 0.84	0.633
TNFAIP2	rs2297065	Additive	G = 0.028	C = 0.972	0.961
TNFAIP2	rs2149674	Recessive	T = 0.2	C = 0.8	0.876
TNFAIP2	rs10137213	Dominant	G = 0.375	T = 0.625	0.712
TNFAIP2	rs4457930	Additive	A = 0.162	G = 0.838	0.296
TNFAIP2	rs4906287	Dominant	G = 0.321	T = 0.679	0.656
TNFAIP2	rs2183195	Dominant	C = 0.396	T = 0.604	0.677
TNFAIP3	rs11756669	Recessive	A = 0.079	G = 0.921	0.893
TNFAIP3	rs12203234	Dominant	T = 0.201	C = 0.799	0.749
TNFAIP3	rs12210441	Recessive	A = 0.286	G = 0.714	0.295
TNFAIP3	rs12524275	Dominant	G = 0.095	A = 0.905	0.700
TNFAIP3	rs13213035	Additive	A = 0.263	C = 0.737	0.129
TNFAIP3	rs1545092	Dominant	C = 0.404	T = 0.596	0.992
TNFAIP3	rs1628976	Additive	T = 0.078	C = 0.922	0.969
TNFAIP3	rs17067054	Dominant	A = 0.181	C = 0.819	0.627
TNFAIP3	rs17724708	Additive	C = 0.043	T = 0.957	0.934

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFAIP3	rs34218444	Dominant	C = 0.076	T = 0.924	0.951
TNFAIP3	rs34375712	Recessive	G = 0.153	A = 0.847	0.061
TNFAIP3	rs34744522	Dominant	A = 0.024	G = 0.976	0.817
TNFAIP3	rs4289679	Additive	A = 0.117	G = 0.883	0.111
TNFAIP3	rs4895499	Additive	A = 0.069	G = 0.931	0.763
TNFAIP3	rs4895504	Recessive	C = 0.081	T = 0.919	0.787
TNFAIP3	rs610604	Dominant	G = 0.322	T = 0.678	0.875
TNFAIP3	rs6570201	Additive	A = 0.451	G = 0.549	0.085
TNFAIP3	rs9321637	Dominant	C = 0.109	T = 0.891	0.224
TNFAIP3	rs9385816	Dominant	C = 0.33	T = 0.67	0.853
TNFAIP3	rs9402932	Recessive	G = 0.252	A = 0.748	0.091
TNFAIP3	rs9399229	Recessive	C = 0.252	T = 0.748	0.138
TNFAIP3	rs112308288	Additive	C = 0.02	T = 0.98	0.972
TNFAIP3	rs72980776	Recessive	G = 0.146	A = 0.854	0.644
TNFAIP3	rs76010350	Dominant	T = 0.077	G = 0.923	0.908
TNFAIP3	rs113164383	Additive	A = 0.01	C = 0.99	0.961
TNFAIP3	rs6928190	Additive	T = 0.356	C = 0.644	0.748
TNFAIP3	rs72982708	Dominant	A = 0.139	G = 0.861	0.566
TNFAIP3	rs9373202	Recessive	A = 0.196	G = 0.804	0.320
TNFAIP3	rs66489050	Recessive	C = 0.362	T = 0.638	0.239
TNFAIP3	rs77979875	Additive	T = 0.041	G = 0.959	0.961
TNFAIP3	rs9373204	Additive	G = 0.194	A = 0.806	0.388
TNFAIP3	rs59395111	Dominant	G = 0.024	A = 0.976	0.119
TNFAIP3	rs59298293	Dominant	A = 0.169	G = 0.831	0.101
<b>TNFAIP3</b>	<b>rs75202629</b>	<b>Recessive</b>	<b>C = 0.05</b>	<b>T = 0.95</b>	<b>0.038</b>
TNFAIP3	rs55822717	Dominant	T = 0.156	C = 0.844	0.465
TNFAIP3	rs62432767	Dominant	T = 0.049	G = 0.951	0.425
TNFAIP3	rs6570205	Dominant	A = 0.379	G = 0.621	0.553
TNFAIP3	rs34705709	Additive	A = 0.083	G = 0.917	0.417
TNFAIP3	rs117949324	Dominant	T = 0.103	C = 0.897	0.637
TNFAIP3	rs13217392	Recessive	A = 0.149	G = 0.851	0.228
TNFAIP3	rs56950323	Dominant	A = 0.041	G = 0.959	0.362
TNFAIP3	rs78058696	Recessive	A = 0.03	G = 0.97	0.134
TNFAIP3	rs9321647	Recessive	A = 0.433	G = 0.567	0.916
TNFAIP3	rs5029948	Recessive	T = 0.047	C = 0.953	0.484
TNFAIP3	rs35168417	Additive	G = 0.06	A = 0.94	0.957
TNFAIP3	rs34598468	Recessive	C = 0.424	T = 0.576	0.112
TNFAIP3	rs7748243	Dominant	T = 0.433	C = 0.567	0.054
TNFAIP3	rs72982725	Additive	C = 0.057	G = 0.943	0.500
TNFAIP3	rs6907149	Additive	G = 0.342	C = 0.658	0.090
TNFAIP3	rs6570199	Dominant	C = 0.477	T = 0.523	0.799
TNFAIP3	rs28548425	Recessive	A = 0.136	G = 0.864	0.941

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFAIP3	rs77196747	Recessive	C = 0.061	G = 0.939	0.114
TNFAIP3	rs733868	Recessive	A = 0.152	G = 0.848	0.052
TNFAIP3	rs6926508	Recessive	G = 0.215	A = 0.785	0.630
TNFAIP3	rs35191620	Additive	C = 0.075	T = 0.925	0.539
TNFAIP3	rs12530033	Dominant	A = 0.059	G = 0.941	0.966
TNFAIP3	rs74592588	Additive	C = 0.028	G = 0.972	0.096
<b>TNFAIP3</b>	<b>rs77320369</b>	<b>Dominant</b>	<b>A = 0.033</b>	<b>G = 0.967</b>	<b>0.011</b>
TNFAIP3	rs118188393	Additive	T = 0.025	C = 0.975	0.972
TNFAIP3	rs117391422	Dominant	C = 0.026	A = 0.974	0.374
TNFAIP3	rs9376307	Recessive	T = 0.26	C = 0.74	0.257
TNFAIP3	rs12205708	Additive	A = 0.018	G = 0.982	0.972
TNFAIP6	rs10204335	Recessive	C = 0.144	T = 0.856	0.085
TNFAIP6	rs1046668	Recessive	G = 0.134	A = 0.866	0.227
TNFAIP6	rs17805045	Recessive	T = 0.118	C = 0.882	0.588
TNFAIP6	rs4664356	Recessive	A = 0.391	G = 0.609	0.324
TNFAIP6	rs6743034	Dominant	G = 0.053	A = 0.947	0.342
TNFAIP6	rs16829969	Dominant	G = 0.306	A = 0.694	0.491
TNFAIP6	rs6708492	Dominant	C = 0.493	T = 0.507	0.741
TNFAIP6	rs72858636	Additive	C = 0.055	A = 0.945	0.554
<b>TNFAIP6</b>	<b>rs73967537</b>	<b>Dominant</b>	<b>A = 0.1</b>	<b>C = 0.9</b>	<b>0.034</b>
TNFAIP6	rs4664025	Additive	T = 0.402	C = 0.598	0.508
TNFAIP6	rs3771893	Recessive	C = 0.133	T = 0.867	0.145
TNFAIP6	rs113089339	Recessive	T = 0.061	G = 0.939	0.101
<b>TNFAIP6</b>	<b>rs10178027</b>	<b>Dominant</b>	<b>T = 0.406</b>	<b>C = 0.594</b>	<b>0.042</b>
TNFAIP6	rs60474976	Additive	A = 0.028	G = 0.972	0.327
TNFAIP6	rs4664021	Dominant	C = 0.259	T = 0.741	0.558
TNFAIP6	rs115060686	Dominant	G = 0.024	A = 0.976	0.280
TNFAIP6	rs116741739	Additive	T = 0.047	C = 0.953	0.964
TNFAIP6	rs113837987	Additive	A = 0.028	G = 0.972	0.973
TNFAIP6	rs78557188	Dominant	A = 0.042	G = 0.958	0.941
TNFAIP6	rs76139707	Dominant	T = 0.046	C = 0.954	0.659
TNFAIP6	rs10167886	Recessive	A = 0.373	G = 0.627	0.831
TNFAIP6	rs16829962	Additive	A = 0.04	G = 0.96	0.539
<b>TNFAIP6</b>	<b>rs114469854</b>	<b>Dominant</b>	<b>G = 0.02</b>	<b>A = 0.98</b>	<b>0.010</b>
TNFAIP8	rs10478419	Dominant	C = 0.058	G = 0.942	0.239
TNFAIP8	rs1504977	Additive	C = 0.337	T = 0.663	0.168
TNFAIP8	rs17145063	Recessive	C = 0.105	T = 0.895	0.367
TNFAIP8	rs17145265	Dominant	C = 0.264	T = 0.736	0.956
TNFAIP8	rs1966479	Recessive	A = 0.337	G = 0.663	0.461
TNFAIP8	rs250303	Additive	G = 0.287	A = 0.713	0.817
TNFAIP8	rs34500219	Recessive	A = 0.046	G = 0.954	0.830
TNFAIP8	rs35695978	Additive	A = 0.021	G = 0.979	0.972

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFAIP8	rs467076	Recessive	C = 0.247	T = 0.753	0.119
TNFAIP8	rs4895364	Recessive	G = 0.272	A = 0.728	0.625
<b>TNFAIP8</b>	<b>rs7722580</b>	<b>Additive</b>	<b>C = 0.087</b>	<b>T = 0.913</b>	<b>0.025</b>
<b>TNFAIP8</b>	<b>rs10053596</b>	<b>Recessive</b>	<b>G = 0.144</b>	<b>A = 0.856</b>	<b>0.012</b>
TNFAIP8	rs4895187	Additive	G = 0.167	A = 0.833	0.080
<b>TNFAIP8</b>	<b>rs78450065</b>	<b>Dominant</b>	<b>T = 0.057</b>	<b>C = 0.943</b>	<b>0.021</b>
TNFAIP8	rs62375087	Dominant	T = 0.025	C = 0.975	0.197
<b>TNFAIP8</b>	<b>rs2112323</b>	<b>Recessive</b>	<b>T = 0.114</b>	<b>C = 0.886</b>	<b>0.004</b>
TNFAIP8	rs6888116	Additive	A = 0.378	C = 0.622	0.304
TNFAIP8	rs7704398	Dominant	T = 0.067	C = 0.933	0.677
TNFAIP8	rs80327020	Additive	C = 0.019	A = 0.981	0.972
TNFAIP8	rs3813304	Additive	G = 0.055	A = 0.945	0.540
TNFAIP8	rs55682871	Recessive	A = 0.056	G = 0.944	0.101
<b>TNFAIP8</b>	<b>rs62375120</b>	<b>Additive</b>	<b>G = 0.032</b>	<b>A = 0.968</b>	<b>0.037</b>
<b>TNFAIP8</b>	<b>rs75810953</b>	<b>Recessive</b>	<b>A = 0.064</b>	<b>G = 0.936</b>	<b>0.024</b>
<b>TNFAIP8</b>	<b>rs73790861</b>	<b>Additive</b>	<b>C = 0.019</b>	<b>T = 0.981</b>	<b>0.046</b>
TNFAIP8	rs10900729	Dominant	A = 0.241	G = 0.759	0.599
TNFAIP8	rs35956442	Recessive	T = 0.027	G = 0.973	0.103
TNFAIP8	rs62375110	Additive	G = 0.238	C = 0.762	0.768
TNFAIP8	rs62375113	Recessive	T = 0.27	C = 0.73	0.103
TNFAIP8	rs62375115	Dominant	C = 0.022	G = 0.978	0.267
TNFAIP8	rs6860034	Dominant	G = 0.063	A = 0.937	0.906
TNFAIP8	rs3920174	Additive	G = 0.449	A = 0.551	0.129
TNFAIP8	rs10477585	Additive	T = 0.449	A = 0.551	0.081
<b>TNFAIP8</b>	<b>rs4895191</b>	<b>Dominant</b>	<b>A = 0.486</b>	<b>G = 0.514</b>	<b>0.005</b>
TNFAIP8	rs35575384	Additive	A = 0.084	G = 0.916	0.487
TNFAIP8	rs114790657	Dominant	G = 0.036	A = 0.964	0.132
TNFAIP8	rs114814118	Additive	C = 0.025	T = 0.975	0.548
TNFAIP8	rs116051841	Dominant	G = 0.034	A = 0.966	0.182
TNFAIP8	rs115136626	Additive	C = 0.016	T = 0.984	0.972
TNFAIP8	rs77390892	Additive	T = 0.021	C = 0.979	0.973
TNFAIP8	rs17145135	Additive	A = 0.052	G = 0.948	0.964
TNFAIP8	rs17450512	Recessive	T = 0.072	C = 0.928	0.784
TNFAIP8	rs6893184	Additive	G = 0.309	A = 0.691	0.145
TNFAIP8	rs116055231	Dominant	T = 0.035	C = 0.965	0.361
TNFAIP8L1	rs115052410	Dominant	T = 0.015	C = 0.985	0.966
TNFAIP8L1	rs62112609	Additive	G = 0.041	A = 0.959	0.831
TNFAIP8L1	rs73920854	Dominant	C = 0.041	G = 0.959	0.491
TNFAIP8L1	rs79344859	Recessive	T = 0.072	C = 0.928	0.635
TNFAIP8L1	rs11880372	Additive	T = 0.019	C = 0.981	0.351
TNFAIP8L1	rs59264408	Dominant	C = 0.185	A = 0.815	0.176
TNFAIP8L1	rs36033258	Dominant	G = 0.47	A = 0.53	0.943

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFAIP8L1	rs34497844	Recessive	T = 0.317	C = 0.683	0.736
TNFAIP8L1	rs73919172	Additive	T = 0.022	C = 0.978	0.212
TNFAIP8L1	rs4611581	Recessive	G = 0.136	A = 0.864	0.054
TNFAIP8L1	rs11880042	Recessive	G = 0.415	A = 0.585	0.222
TNFAIP8L1	rs61669641	Dominant	A = 0.108	G = 0.892	0.763
TNFAIP8L1	rs35961994	Dominant	G = 0.268	A = 0.732	0.417
TNFAIP8L1	rs75415216	Dominant	G = 0.116	C = 0.884	0.980
TNFAIP8L1	rs12971564	Recessive	C = 0.496	T = 0.504	0.625
TNFAIP8L1	rs117882665	Dominant	T = 0.012	C = 0.988	0.639
TNFAIP8L1	rs12151291	Dominant	C = 0.173	T = 0.827	0.413
TNFAIP8L1	rs11878789	Dominant	T = 0.302	C = 0.698	0.507
TNFAIP8L1	---	Dominant	T = 0.262	G = 0.738	0.519
TNFAIP8L1	rs117508147	Recessive	T = 0.027	C = 0.973	0.298
TNFAIP8L1	rs117498468	Dominant	T = 0.038	C = 0.962	0.488
TNFAIP8L1	rs117666290	Dominant	T = 0.016	G = 0.984	0.777
TNFAIP8L1	rs8103778	Dominant	A = 0.45	C = 0.55	0.224
<b>TNFAIP8L1</b>	<b>rs17434614</b>	<b>Additive</b>	<b>C = 0.212</b>	<b>T = 0.788</b>	<b>0.011</b>
TNFAIP8L1	rs4806993	Recessive	C = 0.362	G = 0.638	0.519
TNFAIP8L1	rs9917028	Additive	A = 0.354	G = 0.646	0.427
TNFAIP8L1	rs10417957	Dominant	C = 0.238	T = 0.762	0.409
TNFAIP8L1	rs10426502	Dominant	A = 0.05	G = 0.95	0.811
TNFAIP8L2-SCNM1	rs6587557	Recessive	A = 0.324	G = 0.676	0.683
TNFAIP8L2-SCNM1	rs72708455	Dominant	T = 0.049	C = 0.951	0.351
TNFAIP8L3	rs17522120	Dominant	C = 0.207	T = 0.793	0.423
TNFAIP8L3	rs7169770	Recessive	A = 0.308	G = 0.692	0.168
TNFAIP8L3	rs9972359	Dominant	C = 0.424	T = 0.576	0.812
TNFAIP8L3	rs8025456	Additive	C = 0.212	T = 0.788	0.146
TNFAIP8L3	rs78897873	Dominant	G = 0.046	T = 0.954	0.055
TNFAIP8L3	rs115944733	Dominant	A = 0.04	G = 0.96	0.722
TNFAIP8L3	rs1438924	Additive	C = 0.355	T = 0.645	0.429
TNFAIP8L3	rs17600885	Additive	G = 0.032	A = 0.968	0.961
TNFAIP8L3	rs12443062	Additive	T = 0.22	C = 0.78	0.494
<b>TNFAIP8L3</b>	<b>rs56369578</b>	<b>Recessive</b>	<b>A = 0.049</b>	<b>G = 0.951</b>	<b>0.022</b>
TNFAIP8L3	rs78898628	Dominant	T = 0.181	C = 0.819	0.748
TNFAIP8L3	rs62020061	Dominant	T = 0.198	G = 0.802	0.918
TNFAIP8L3	rs117209414	Additive	A = 0.016	G = 0.984	0.961
TNFAIP8L3	rs75067433	Dominant	C = 0.047	A = 0.953	0.655
TNFAIP8L3	rs79044878	Additive	A = 0.025	G = 0.975	0.972
TNFAIP8L3	rs17600375	Dominant	T = 0.172	C = 0.828	0.681
TNFAIP8L3	rs11631783	Dominant	T = 0.208	C = 0.792	0.454

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFAIP8L3	rs11070834	Dominant	A = 0.244	G = 0.756	0.765
TNFAIP8L3	rs7179084	Dominant	C = 0.16	T = 0.84	0.249
TNFRSF10A	rs11775256	Additive	T = 0.267	C = 0.733	0.178
TNFRSF10A	rs20576	Recessive	G = 0.202	T = 0.798	0.692
TNFRSF10A	rs3808530	Recessive	G = 0.235	A = 0.765	0.140
TNFRSF10A	rs4872077	Recessive	C = 0.304	T = 0.696	0.893
TNFRSF10A	rs7832037	Recessive	G = 0.248	A = 0.752	0.166
TNFRSF10A	rs62501123	Dominant	T = 0.276	G = 0.724	0.748
TNFRSF10A	rs75858659	Dominant	T = 0.042	C = 0.958	0.623
<b>TNFRSF10A</b>	<b>rs75359040</b>	<b>Recessive</b>	<b>T = 0.08</b>	<b>C = 0.92</b>	<b>0.004</b>
TNFRSF10A	rs6982233	Recessive	C = 0.234	T = 0.766	0.168
TNFRSF10A	rs11135706	Recessive	A = 0.306	G = 0.694	0.313
TNFRSF10A	rs75857301	Dominant	T = 0.026	C = 0.974	0.210
TNFRSF10A	rs75071042	Additive	G = 0.037	A = 0.963	0.961
TNFRSF10A	rs4871853	Additive	C = 0.373	T = 0.627	0.271
TNFRSF10A	rs20575	Recessive	G = 0.467	C = 0.533	0.198
TNFRSF10A	rs11777928	Dominant	T = 0.319	C = 0.681	0.656
TNFRSF10A	rs17620	Recessive	C = 0.469	T = 0.531	0.184
TNFRSF10A	rs2230229	Dominant	C = 0.15	T = 0.85	0.961
TNFRSF10B	---	Dominant	A = 0.105	G = 0.895	0.193
TNFRSF10B	rs13269530	Dominant	A = 0.255	G = 0.745	0.737
TNFRSF10B	rs7834281	Additive	T = 0.184	C = 0.816	0.504
TNFRSF10B	rs1001792	Recessive	C = 0.322	T = 0.678	0.962
<b>TNFRSF10B</b>	<b>rs59119269</b>	<b>Recessive</b>	<b>T = 0.048</b>	<b>C = 0.952</b>	<b>&gt;0.0001</b>
TNFRSF10B	rs78198802	Dominant	G = 0.013	A = 0.987	0.553
TNFRSF10B	rs78158454	Dominant	A = 0.192	G = 0.808	0.696
TNFRSF10B	rs35974498	Recessive	C = 0.06	G = 0.94	0.299
TNFRSF10B	rs79042829	Additive	C = 0.034	A = 0.966	0.825
TNFRSF10B	rs76826036	Dominant	T = 0.174	C = 0.826	0.950
TNFRSF10B	rs7825044	Dominant	G = 0.108	A = 0.892	0.304
TNFRSF10B	rs7817787	Dominant	C = 0.061	T = 0.939	0.677
TNFRSF10B	rs12541497	Additive	C = 0.342	T = 0.658	0.529
<b>TNFRSF10B</b>	<b>rs79649969</b>	<b>Additive</b>	<b>C = 0.05</b>	<b>T = 0.95</b>	<b>0.031</b>
TNFRSF10B	rs6997701	Recessive	C = 0.161	A = 0.839	0.069
TNFRSF10B	rs117947202	Additive	A = 0.016	G = 0.984	0.782
<b>TNFRSF10B</b>	<b>rs1047266</b>	<b>Recessive</b>	<b>A = 0.07</b>	<b>G = 0.93</b>	<b>0.040</b>
TNFRSF10B	rs13252794	Recessive	C = 0.372	T = 0.628	0.369
TNFRSF10B	rs1129424	Additive	G = 0.409	A = 0.591	0.139
TNFRSF10C	rs12681965	Dominant	G = 0.205	T = 0.795	0.410
TNFRSF10C	rs7009522	Dominant	G = 0.222	A = 0.778	0.805
TNFRSF10C	rs9644029	Additive	G = 0.198	A = 0.802	0.258

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
<b>TNFRSF10C</b>	<b>rs4872052</b>	<b>Recessive</b>	<b>C = 0.096</b>	<b>T = 0.904</b>	<b>0.041</b>
TNFRSF10C	rs76666473	Dominant	A = 0.097	G = 0.903	0.420
TNFRSF10C	rs11135699	Dominant	G = 0.071	A = 0.929	0.948
TNFRSF10C	rs11774127	Additive	G = 0.044	A = 0.956	0.971
TNFRSF10C	rs62501084	Additive	T = 0.025	C = 0.975	0.112
TNFRSF10C	rs6995015	Dominant	C = 0.32	T = 0.68	0.408
TNFRSF10C	rs78875129	Additive	C = 0.053	T = 0.947	0.158
TNFRSF10C	rs77395967	Dominant	A = 0.048	G = 0.952	0.299
<b>TNFRSF10C</b>	<b>rs62501087</b>	<b>Recessive</b>	<b>A = 0.425</b>	<b>G = 0.575</b>	<b>0.044</b>
TNFRSF10C	rs77837771	Recessive	G = 0.066	C = 0.934	0.492
TNFRSF10C	rs117646277	Recessive	A = 0.038	G = 0.962	0.362
TNFRSF10C	rs117325737	Dominant	A = 0.032	G = 0.968	0.966
TNFRSF10C	rs116908180	Recessive	T = 0.013	G = 0.987	0.360
TNFRSF10C	rs12546238	Additive	T = 0.093	C = 0.907	0.885
TNFRSF10C	rs7843602	Dominant	T = 0.211	C = 0.789	0.619
TNFRSF10C	rs9644063	Recessive	T = 0.174	C = 0.826	0.855
TNFRSF10D	rs34941234	Recessive	C = 0.081	T = 0.919	0.576
TNFRSF10D	rs4242391	Recessive	T = 0.395	C = 0.605	0.906
TNFRSF10D	rs4278155	Recessive	C = 0.172	A = 0.828	0.053
TNFRSF10D	rs1133782	Dominant	A = 0.396	G = 0.604	0.629
TNFRSF10D	rs55636833	Dominant	T = 0.05	C = 0.95	0.907
TNFRSF10D	rs62501099	Additive	A = 0.062	G = 0.938	0.470
TNFRSF10D	rs10089572	Dominant	G = 0.128	A = 0.872	0.391
TNFRSF10D	rs73222565	Recessive	A = 0.119	G = 0.881	0.891
TNFRSF10D	rs11135703	Recessive	A = 0.123	G = 0.877	0.611
TNFRSF10D	rs117906816	Additive	T = 0.019	C = 0.981	0.961
TNFRSF10D	rs78918796	Dominant	A = 0.04	C = 0.96	0.966
TNFRSF10D	rs7816142	Additive	G = 0.244	A = 0.756	0.428
TNFRSF10D	rs13257094	Recessive	A = 0.266	G = 0.734	0.416
TNFRSF10D	rs34407287	Recessive	C = 0.315	T = 0.685	0.070
TNFRSF10D	rs34866525	Dominant	G = 0.393	A = 0.607	0.956
TNFRSF11A	rs10503071	Recessive	A = 0.111	G = 0.889	0.204
TNFRSF11A	rs12970081	Recessive	A = 0.234	G = 0.766	0.513
TNFRSF11A	rs17069958	Recessive	C = 0.14	T = 0.86	0.351
TNFRSF11A	rs1805034	Dominant	C = 0.471	T = 0.529	0.553
TNFRSF11A	rs2957139	Recessive	C = 0.148	T = 0.852	0.173
TNFRSF11A	rs3018362	Additive	A = 0.359	G = 0.641	0.488
TNFRSF11A	rs34950264	Recessive	G = 0.069	A = 0.931	0.729
TNFRSF11A	rs4524035	Recessive	G = 0.133	A = 0.867	0.442
TNFRSF11A	rs7237982	Additive	G = 0.233	A = 0.767	0.473

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF11A	rs7239261	Dominant	A = 0.426	C = 0.574	0.927
TNFRSF11A	rs12458117	Dominant	A = 0.14	G = 0.86	0.701
TNFRSF11A	rs12607917	Dominant	C = 0.318	T = 0.682	0.731
TNFRSF11A	rs2957128	Dominant	A = 0.413	G = 0.587	0.968
TNFRSF11A	rs3017353	Dominant	C = 0.09	T = 0.91	0.675
TNFRSF11A	rs4375763	Recessive	T = 0.321	C = 0.679	0.613
TNFRSF11A	rs4574025	Dominant	C = 0.446	T = 0.554	0.772
TNFRSF11A	rs8086340	Recessive	C = 0.443	G = 0.557	0.688
TNFRSF11A	rs17069845	Recessive	C = 0.069	T = 0.931	0.092
<b>TNFRSF11A</b>	<b>rs58112300</b>	<b>Additive</b>	<b>C = 0.013</b>	<b>T = 0.987</b>	<b>0.017</b>
TNFRSF11A	rs17069898	Additive	G = 0.369	A = 0.631	0.029
TNFRSF11A	rs117978262	Additive	G = 0.022	A = 0.978	0.972
TNFRSF11A	rs4426449	Recessive	T = 0.334	C = 0.666	0.373
TNFRSF11A	rs78459945	Recessive	G = 0.105	A = 0.895	0.265
TNFRSF11A	rs61091371	Recessive	T = 0.029	C = 0.971	0.105
TNFRSF11A	rs56145860	Recessive	A = 0.232	G = 0.768	0.056
TNFRSF11A	rs878663	Additive	G = 0.335	A = 0.665	0.324
TNFRSF11A	rs60426222	Dominant	T = 0.154	C = 0.846	0.987
TNFRSF11A	rs672217	Dominant	G = 0.128	A = 0.872	0.158
TNFRSF11A	rs694419	Dominant	C = 0.475	T = 0.525	0.474
TNFRSF11A	rs77192711	Additive	T = 0.029	C = 0.971	0.961
TNFRSF11A	rs80336853	Additive	C = 0.035	T = 0.965	0.576
TNFRSF11A	rs72937606	Additive	G = 0.021	A = 0.979	0.961
TNFRSF11A	rs8087616	Dominant	C = 0.319	T = 0.681	0.845
TNFRSF11A	rs74938001	Recessive	T = 0.19	C = 0.81	0.592
TNFRSF11A	rs72933609	Recessive	C = 0.068	T = 0.932	0.174
TNFRSF11A	rs34256674	Additive	C = 0.479	T = 0.521	0.143
TNFRSF11A	rs12455323	Recessive	C = 0.279	G = 0.721	0.107
TNFRSF11A	rs12960638	Recessive	T = 0.304	G = 0.696	0.083
TNFRSF11A	rs111798464	Recessive	A = 0.031	G = 0.969	0.105
TNFRSF11A	rs117001936	Additive	G = 0.012	C = 0.988	0.168
TNFRSF11A	rs34170196	Recessive	T = 0.286	C = 0.714	0.518
TNFRSF11A	rs117653594	Dominant	C = 0.029	G = 0.971	0.869
TNFRSF11A	rs74396114	Recessive	A = 0.05	G = 0.95	0.363
TNFRSF11A	rs10163810	Recessive	T = 0.219	C = 0.781	0.884
TNFRSF11A	rs17069840	Additive	G = 0.065	A = 0.935	0.613
TNFRSF11A	rs34739845	Additive	G = 0.119	A = 0.881	0.688
<b>TNFRSF11A</b>	<b>rs17665986</b>	<b>Recessive</b>	<b>A = 0.132</b>	<b>G = 0.868</b>	<b>0.035</b>
TNFRSF11A	rs7231380	Dominant	G = 0.24	A = 0.76	0.415
TNFRSF11A	rs1618130	Additive	A = 0.056	C = 0.944	0.429
TNFRSF11A	rs12962662	Recessive	T = 0.157	C = 0.843	0.712
TNFRSF11A	rs9951790	Dominant	A = 0.094	C = 0.906	0.786

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF11A	rs17070008	Recessive	T = 0.03	C = 0.97	0.105
TNFRSF11B	---	Recessive	T = 0.068	C = 0.932	0.392
TNFRSF11B	rs11573931	Additive	A = 0.035	G = 0.965	0.471
TNFRSF11B	rs11995824	Recessive	C = 0.451	G = 0.549	0.426
TNFRSF11B	rs12679857	Recessive	G = 0.299	A = 0.701	0.237
TNFRSF11B	rs1485296	Additive	G = 0.186	A = 0.814	0.432
TNFRSF11B	rs1493929	Additive	G = 0.058	C = 0.942	0.773
TNFRSF11B	rs16891419	Recessive	C = 0.071	A = 0.929	0.762
TNFRSF11B	rs2073618	Recessive	C = 0.451	G = 0.549	0.763
TNFRSF11B	rs2450058	Additive	C = 0.214	T = 0.786	0.172
TNFRSF11B	rs3103985	Dominant	G = 0.119	A = 0.881	0.637
TNFRSF11B	rs36080380	Additive	A = 0.033	C = 0.967	0.968
TNFRSF11B	---	Recessive	G = 0.471	A = 0.529	0.524
TNFRSF11B	rs4373549	Recessive	C = 0.377	T = 0.623	0.992
TNFRSF11B	rs4380972	Recessive	T = 0.129	C = 0.871	0.454
TNFRSF11B	rs4541956	Dominant	A = 0.065	G = 0.935	0.543
TNFRSF11B	rs4876429	Recessive	C = 0.135	T = 0.865	0.644
TNFRSF11B	rs6469804	Recessive	G = 0.448	A = 0.552	0.104
TNFRSF11B	rs6993813	Recessive	T = 0.465	C = 0.535	0.199
TNFRSF11B	rs3103977	Dominant	C = 0.218	T = 0.782	0.233
TNFRSF11B	rs11988997	Recessive	T = 0.079	C = 0.921	0.804
TNFRSF11B	rs12334995	Dominant	T = 0.198	C = 0.802	0.807
TNFRSF11B	rs56308944	Dominant	A = 0.04	G = 0.96	0.968
TNFRSF11B	rs78904105	Additive	C = 0.04	T = 0.96	0.218
TNFRSF11B	rs7822190	Recessive	C = 0.079	T = 0.921	0.885
TNFRSF11B	rs79724366	Recessive	C = 0.169	T = 0.831	0.279
TNFRSF11B	rs80294347	Recessive	A = 0.075	G = 0.925	0.879
TNFRSF11B	rs75332815	Dominant	G = 0.014	T = 0.986	0.686
TNFRSF11B	rs76124971	Additive	A = 0.059	G = 0.941	0.591
TNFRSF11B	rs17751221	Additive	G = 0.095	C = 0.905	0.533
TNFRSF11B	rs73315533	Recessive	T = 0.085	C = 0.915	0.346
TNFRSF11B	rs4242590	Additive	T = 0.065	C = 0.935	0.541
TNFRSF11B	rs76936579	Recessive	A = 0.071	G = 0.929	0.520
TNFRSF11B	rs76879888	Additive	A = 0.017	G = 0.983	0.972
TNFRSF11B	rs11573856	Dominant	T = 0.079	C = 0.921	0.387
<b>TNFRSF11B</b>	<b>rs77601444</b>	<b>Recessive</b>	<b>A = 0.044</b>	<b>G = 0.956</b>	<b>0.025</b>
TNFRSF11B	rs117576079	Additive	C = 0.023	T = 0.977	0.972
TNFRSF11B	rs78399257	Dominant	A = 0.038	G = 0.962	0.965
TNFRSF11B	rs78736097	Additive	T = 0.052	C = 0.948	0.773
TNFRSF11B	rs75749655	Additive	A = 0.116	G = 0.884	0.754
TNFRSF11B	rs11573927	Recessive	C = 0.136	T = 0.864	0.079
TNFRSF11B	rs2465380	Dominant	G = 0.212	A = 0.788	0.493

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF11B	rs117524446	Additive	C = 0.032	T = 0.968	0.969
TNFRSF11B	rs77417190	Dominant	A = 0.018	G = 0.982	0.625
TNFRSF11B	rs10808502	Additive	G = 0.068	C = 0.932	0.537
TNFRSF11B	rs117117000	Dominant	G = 0.029	A = 0.971	0.931
TNFRSF11B	rs116931319	Additive	T = 0.028	C = 0.972	0.484
TNFRSF11B	rs117863403	Dominant	T = 0.019	C = 0.981	0.360
TNFRSF11B	rs10505338	Dominant	A = 0.267	G = 0.733	0.423
TNFRSF11B	rs3134079	Recessive	T = 0.228	C = 0.772	0.199
TNFRSF11B	rs3134069	Additive	C = 0.05	A = 0.95	0.643
TNFRSF11B	rs1586275	Dominant	A = 0.055	G = 0.945	0.793
TNFRSF11B	rs139237057	Additive	T = 0.012	C = 0.988	0.975
TNFRSF11B	rs1493936	Dominant	T = 0.015	C = 0.985	0.567
TNFRSF11B	rs77578017	Dominant	A = 0.025	C = 0.975	0.966
TNFRSF11B	rs17683646	Dominant	T = 0.035	G = 0.965	0.839
TNFRSF11B	rs117522264	Dominant	T = 0.014	C = 0.986	0.966
TNFRSF11B	rs2062377	Recessive	T = 0.437	A = 0.563	0.291
TNFRSF12A	rs74758164	Additive	T = 0.045	C = 0.955	0.971
TNFRSF12A	rs8052002	Recessive	C = 0.237	G = 0.763	0.889
TNFRSF13B	rs35612151	Dominant	A = 0.045	G = 0.955	0.470
TNFRSF13B	rs4578698	Recessive	T = 0.421	G = 0.579	0.435
TNFRSF13B	rs4985726	Recessive	G = 0.106	C = 0.894	0.825
TNFRSF13B	rs4985780	Recessive	C = 0.14	T = 0.86	0.171
TNFRSF13B	rs6416870	Dominant	C = 0.4	T = 0.6	0.750
TNFRSF13B	rs9894818	Additive	T = 0.434	C = 0.566	0.604
TNFRSF13B	rs9908528	Recessive	A = 0.158	G = 0.842	0.082
TNFRSF13B	rs12051726	Additive	C = 0.099	T = 0.901	0.084
<b>TNFRSF13B</b>	<b>rs4312350</b>	<b>Additive</b>	<b>C = 0.258</b>	<b>T = 0.742</b>	<b>0.049</b>
TNFRSF13B	rs4985724	Dominant	A = 0.288	G = 0.712	0.924
TNFRSF13B	rs11651972	Additive	C = 0.106	A = 0.894	0.580
TNFRSF13B	rs12953270	Recessive	C = 0.157	T = 0.843	0.387
TNFRSF13B	rs56172143	Dominant	T = 0.016	C = 0.984	0.360
TNFRSF13B	rs8064661	Recessive	C = 0.199	T = 0.801	0.477
TNFRSF13B	rs4985762	Dominant	G = 0.306	A = 0.694	0.949
TNFRSF13B	rs11867934	Recessive	T = 0.214	C = 0.786	0.263
TNFRSF13B	rs117968761	Dominant	T = 0.081	C = 0.919	0.225
TNFRSF13B	rs11655172	Dominant	G = 0.103	A = 0.897	0.835
TNFRSF13B	rs56324271	Additive	G = 0.262	A = 0.738	0.128
TNFRSF13B	rs9908870	Additive	A = 0.478	G = 0.522	0.357
TNFRSF13B	rs72637375	Additive	C = 0.022	T = 0.978	0.972
TNFRSF13B	rs62065416	Dominant	C = 0.04	T = 0.96	0.361
TNFRSF13B	rs56369903	Dominant	T = 0.216	C = 0.784	0.985
TNFRSF13B	rs56396775	Dominant	T = 0.211	G = 0.789	0.893

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF13B	rs56399647	Dominant	T = 0.029	C = 0.971	0.078
TNFRSF13B	rs34562254	Recessive	A = 0.103	G = 0.897	0.821
TNFRSF13B	rs28613542	Dominant	C = 0.099	T = 0.901	0.247
TNFRSF13B	rs55825380	Dominant	A = 0.356	G = 0.644	0.118
TNFRSF13B	rs57096150	Recessive	T = 0.155	C = 0.845	0.869
TNFRSF13B	rs55984928	Additive	A = 0.029	G = 0.971	0.972
TNFRSF13B	rs9630716	Dominant	A = 0.129	G = 0.871	0.845
TNFRSF13B	rs111882157	Dominant	T = 0.061	C = 0.939	0.786
TNFRSF13B	rs113556432	Recessive	A = 0.027	G = 0.973	0.526
TNFRSF13B	rs9910011	Recessive	G = 0.111	T = 0.889	0.063
TNFRSF13B	rs72837920	Dominant	C = 0.021	A = 0.979	0.845
TNFRSF13B	rs191763772	Recessive	G = 0.103	A = 0.897	0.599
TNFRSF13B	rs77303927	Dominant	G = 0.028	A = 0.972	0.769
TNFRSF13B	rs113436029	Additive	G = 0.043	A = 0.957	0.976
TNFRSF13B	rs12942194	Additive	G = 0.093	A = 0.907	0.508
TNFRSF13B	rs3751993	Additive	T = 0.322	C = 0.678	0.436
TNFRSF13B	rs3751990	Dominant	A = 0.028	G = 0.972	0.966
TNFRSF13B	rs8065836	Dominant	A = 0.43	G = 0.57	0.464
TNFRSF13B	rs7225344	Dominant	G = 0.262	A = 0.738	0.981
TNFRSF13B	rs11656868	Additive	A = 0.065	G = 0.935	0.219
TNFRSF13B	rs4273077	Additive	G = 0.101	A = 0.899	0.893
TNFRSF13B	rs9893198	Recessive	A = 0.463	G = 0.537	0.890
TNFRSF13B	---	Dominant	G = 0.15	C = 0.85	0.984
TNFRSF13B	rs7214091	Recessive	C = 0.384	T = 0.616	0.362
TNFRSF13B	rs9890795	Additive	C = 0.072	T = 0.928	0.078
TNFRSF13B	rs7501848	Dominant	G = 0.472	C = 0.528	0.373
<b>TNFRSF13B</b>	<b>rs11653100</b>	<b>Recessive</b>	<b>T = 0.065</b>	<b>C = 0.935</b>	<b>&gt;0.0001</b>
TNFRSF13B	rs9907308	Dominant	A = 0.316	G = 0.684	0.764
TNFRSF13C	rs7290134	Dominant	G = 0.224	A = 0.776	0.985
<b>TNFRSF14</b>	<b>rs2257763</b>	<b>Recessive</b>	<b>A = 0.493</b>	<b>C = 0.507</b>	<b>0.032</b>
TNFRSF14	rs2234163	Dominant	A = 0.028	G = 0.972	0.961
TNFRSF14	rs2234167	Recessive	A = 0.14	G = 0.86	0.105
<b>TNFRSF14</b>	<b>rs4870</b>	<b>Recessive</b>	<b>G = 0.482</b>	<b>A = 0.518</b>	<b>0.030</b>
TNFRSF17	rs3743591	Additive	G = 0.049	A = 0.951	0.273
TNFRSF17	rs451354	Dominant	G = 0.137	A = 0.863	0.069
TNFRSF17	rs74449077	Dominant	A = 0.044	G = 0.956	0.239
<b>TNFRSF17</b>	<b>rs11862958</b>	<b>Recessive</b>	<b>T = 0.394</b>	<b>C = 0.606</b>	<b>0.008</b>
<b>TNFRSF17</b>	<b>rs11647499</b>	<b>Recessive</b>	<b>G = 0.459</b>	<b>A = 0.541</b>	<b>0.033</b>
TNFRSF17	rs12445244	Recessive	C = 0.13	G = 0.87	0.313
TNFRSF17	rs9922891	Additive	A = 0.075	G = 0.925	0.306
TNFRSF17	rs34166367	Additive	C = 0.018	A = 0.982	0.356
TNFRSF17	rs12597429	Additive	C = 0.34	T = 0.66	0.207

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF17	rs7193792	Additive	T = 0.025	C = 0.975	0.961
TNFRSF18	rs3753350	Additive	C = 0.034	T = 0.966	0.968
TNFRSF18	rs35038461	Dominant	A = 0.044	G = 0.956	0.552
TNFRSF18	rs114946555	Dominant	G = 0.013	A = 0.987	0.834
<b>TNFRSF19</b>	<b>rs1104905</b>	<b>Additive</b>	<b>C = 0.321</b>	<b>T = 0.679</b>	<b>0.011</b>
TNFRSF19	rs11616958	Recessive	C = 0.222	T = 0.778	0.989
TNFRSF19	rs12428914	Additive	T = 0.06	C = 0.94	0.458
TNFRSF19	rs12876874	Recessive	A = 0.267	G = 0.733	0.967
TNFRSF19	rs1572072	Additive	T = 0.404	G = 0.596	0.418
TNFRSF19	---	Recessive	G = 0.091	C = 0.909	0.635
TNFRSF19	rs17389605	Dominant	C = 0.072	A = 0.928	0.469
TNFRSF19	rs2095185	Dominant	A = 0.4	C = 0.6	0.049
TNFRSF19	rs4770455	Dominant	A = 0.301	C = 0.699	0.523
TNFRSF19	rs6490805	Recessive	C = 0.299	T = 0.701	0.460
<b>TNFRSF19</b>	<b>rs7324557</b>	<b>Recessive</b>	<b>A = 0.338</b>	<b>G = 0.662</b>	<b>0.029</b>
TNFRSF19	rs7338414	Dominant	C = 0.072	T = 0.928	0.175
TNFRSF19	rs7987909	Recessive	G = 0.116	A = 0.884	0.488
TNFRSF19	rs932880	Dominant	T = 0.317	C = 0.683	0.844
TNFRSF19	rs9510756	Recessive	G = 0.097	A = 0.903	0.879
TNFRSF19	rs9510759	Recessive	T = 0.163	C = 0.837	0.494
TNFRSF19	rs9510787	Recessive	G = 0.289	A = 0.711	0.428
TNFRSF19	rs9510815	Dominant	C = 0.126	T = 0.874	0.669
TNFRSF19	rs17078840	Recessive	A = 0.394	G = 0.606	0.519
<b>TNFRSF19</b>	<b>rs56252934</b>	<b>Recessive</b>	<b>T = 0.1</b>	<b>C = 0.9</b>	<b>0.006</b>
TNFRSF19	rs73154699	Dominant	T = 0.189	A = 0.811	0.224
TNFRSF19	rs10507332	Recessive	T = 0.094	G = 0.906	0.091
TNFRSF19	rs78248755	Additive	T = 0.037	C = 0.963	0.961
TNFRSF19	rs9553004	Recessive	A = 0.096	G = 0.904	0.359
TNFRSF19	rs9510784	Dominant	T = 0.42	C = 0.58	0.468
TNFRSF19	rs9553020	Dominant	A = 0.396	G = 0.604	0.535
TNFRSF19	rs61756242	Dominant	T = 0.022	C = 0.978	0.548
TNFRSF19	rs79547453	Dominant	A = 0.029	G = 0.971	0.624
TNFRSF19	rs113198775	Additive	C = 0.028	G = 0.972	0.961
TNFRSF19	rs75921120	Additive	A = 0.035	G = 0.965	0.330
TNFRSF19	rs7333845	Recessive	A = 0.088	G = 0.912	0.642
TNFRSF19	rs76131936	Recessive	T = 0.093	C = 0.907	0.148
TNFRSF19	rs71425107	Recessive	A = 0.075	C = 0.925	0.166
TNFRSF19	rs55882269	Recessive	C = 0.098	T = 0.902	0.116
TNFRSF19	rs913563	Additive	A = 0.082	G = 0.918	0.069
TNFRSF19	rs61946394	Dominant	G = 0.02	A = 0.98	0.844
TNFRSF19	rs78482434	Dominant	G = 0.101	C = 0.899	0.508
TNFRSF19	rs117306125	Additive	A = 0.025	G = 0.975	0.972

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF19	rs74947725	Dominant	A = 0.065	C = 0.935	0.629
TNFRSF19	rs111163809	Dominant	A = 0.071	G = 0.929	0.961
TNFRSF19	rs78841617	Recessive	T = 0.084	C = 0.916	0.362
TNFRSF19	rs77907216	Additive	C = 0.033	T = 0.967	0.959
TNFRSF19	rs118083020	Additive	G = 0.061	A = 0.939	0.456
TNFRSF19	rs75784613	Dominant	C = 0.014	G = 0.986	0.741
<b>TNFRSF19</b>	<b>rs7319524</b>	<b>Recessive</b>	<b>A = 0.095</b>	<b>G = 0.905</b>	<b>&lt;0.0001</b>
TNFRSF19	rs4770474	Recessive	G = 0.35	A = 0.65	0.969
TNFRSF19	rs12430043	Dominant	C = 0.32	A = 0.68	0.661
<b>TNFRSF19</b>	<b>rs753955</b>	<b>Recessive</b>	<b>A = 0.353</b>	<b>G = 0.647</b>	<b>0.032</b>
TNFRSF19	rs118050973	Additive	C = 0.02	T = 0.98	0.974
TNFRSF19	rs117089013	Additive	T = 0.026	C = 0.974	0.972
<b>TNFRSF1A</b>	<b>---</b>	<b>Additive</b>	<b>T = 0.018</b>	<b>C = 0.982</b>	<b>0.003</b>
TNFRSF1A	rs4149586	Additive	A = 0.015	C = 0.985	0.973
TNFRSF1A	rs4149574	Recessive	T = 0.088	C = 0.912	0.146
TNFRSF1A	rs4149572	Dominant	A = 0.036	C = 0.964	0.952
TNFRSF1A	rs73049962	Additive	T = 0.02	G = 0.98	0.961
TNFRSF1A	rs36205633	Dominant	A = 0.068	G = 0.932	0.391
<b>TNFRSF1A</b>	<b>rs1800693</b>	<b>Dominant</b>	<b>C = 0.397</b>	<b>T = 0.603</b>	<b>0.002</b>
<b>TNFRSF1A</b>	<b>rs1860545</b>	<b>Dominant</b>	<b>A = 0.387</b>	<b>G = 0.613</b>	<b>0.015</b>
TNFRSF1A	rs4149570	Recessive	A = 0.407	C = 0.593	0.638
TNFRSF1B	rs653667	Recessive	G = 0.359	T = 0.641	0.769
TNFRSF1B	rs116095144	Additive	T = 0.056	C = 0.944	0.973
TNFRSF1B	rs11587549	Additive	T = 0.026	C = 0.974	0.961
TNFRSF1B	rs114444558	Additive	A = 0.046	G = 0.954	0.963
TNFRSF1B	rs11583360	Dominant	G = 0.375	T = 0.625	0.278
TNFRSF1B	rs816063	Recessive	T = 0.481	C = 0.519	0.462
TNFRSF1B	rs75160808	Additive	A = 0.013	G = 0.987	0.972
TNFRSF1B	rs17883094	Additive	A = 0.019	G = 0.981	0.972
TNFRSF1B	rs1148458	Dominant	A = 0.069	G = 0.931	0.202
TNFRSF1B	rs616645	Additive	G = 0.21	T = 0.79	0.462
TNFRSF1B	rs17882988	Additive	A = 0.157	G = 0.843	0.772
TNFRSF1B	rs17883122	Dominant	G = 0.053	C = 0.947	0.775
TNFRSF1B	rs17884378	Additive	A = 0.041	C = 0.959	0.394
TNFRSF1B	rs5746062	Dominant	G = 0.336	A = 0.664	0.785
TNFRSF1B	rs11588511	Additive	T = 0.024	C = 0.976	0.969
TNFRSF1B	rs12563166	Recessive	C = 0.04	T = 0.96	0.196
TNFRSF1B	rs76802657	Additive	A = 0.026	G = 0.974	0.972
TNFRSF1B	rs1148467	Dominant	A = 0.075	G = 0.925	0.733
TNFRSF1B	rs631272	Additive	G = 0.481	A = 0.519	0.093
TNFRSF1B	rs630542	Recessive	G = 0.448	T = 0.552	0.368
TNFRSF1B	rs816060	Recessive	G = 0.411	A = 0.589	0.991

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF1B	rs590314	Recessive	A = 0.421	G = 0.579	0.599
TNFRSF1B	rs816050	Dominant	A = 0.197	G = 0.803	0.942
TNFRSF1B	rs6697733	Dominant	C = 0.245	T = 0.755	0.790
TNFRSF1B	rs5746012	Additive	A = 0.036	C = 0.964	0.968
TNFRSF1B	rs5746026	Additive	A = 0.035	G = 0.965	0.968
TNFRSF1B	rs2275416	Additive	A = 0.198	G = 0.802	0.496
TNFRSF1B	rs5746059	Recessive	G = 0.242	A = 0.758	0.556
<b>TNFRSF1B</b>	<b>rs1061628</b>	<b>Recessive</b>	<b>T = 0.393</b>	<b>C = 0.607</b>	<b>0.048</b>
TNFRSF1B	rs5746073	Additive	T = 0.05	C = 0.95	0.959
TNFRSF1B	rs5746074	Recessive	A = 0.041	G = 0.959	0.330
TNFRSF1B	rs4846100	Additive	G = 0.423	C = 0.577	0.165
TNFRSF1B	rs7552664	Additive	A = 0.278	C = 0.722	0.237
TNFRSF1B	rs1148461	Dominant	A = 0.469	G = 0.531	0.075
TNFRSF1B	rs1061622	Dominant	G = 0.233	T = 0.767	0.994
TNFRSF21	rs10807350	Recessive	G = 0.161	A = 0.839	0.638
TNFRSF21	rs11753173	Dominant	T = 0.022	C = 0.978	0.816
TNFRSF21	rs12201254	Dominant	G = 0.128	A = 0.872	0.609
TNFRSF21	rs1226542	Dominant	G = 0.041	A = 0.959	0.583
TNFRSF21	rs13208869	Dominant	T = 0.044	G = 0.956	0.867
TNFRSF21	rs1490296	Recessive	G = 0.125	A = 0.875	0.216
TNFRSF21	rs16875836	Recessive	C = 0.102	T = 0.898	0.595
TNFRSF21	rs17216940	Dominant	G = 0.116	A = 0.884	0.309
TNFRSF21	rs17217010	Dominant	C = 0.078	T = 0.922	0.880
TNFRSF21	rs1884042	Additive	C = 0.144	T = 0.856	0.097
TNFRSF21	rs4236092	Recessive	A = 0.44	G = 0.56	0.583
TNFRSF21	rs4374821	Recessive	A = 0.075	G = 0.925	0.148
TNFRSF21	rs4482973	Additive	T = 0.089	G = 0.911	0.416
TNFRSF21	rs6919892	Additive	C = 0.356	T = 0.644	0.462
TNFRSF21	rs7751646	Recessive	A = 0.456	G = 0.544	0.037
TNFRSF21	rs7761581	Additive	A = 0.479	G = 0.521	0.349
TNFRSF21	rs7768789	Dominant	T = 0.06	C = 0.94	0.118
TNFRSF21	rs926774	Additive	G = 0.149	A = 0.851	0.068
<b>TNFRSF21</b>	<b>rs9369675</b>	<b>Recessive</b>	<b>A = 0.079</b>	<b>C = 0.921</b>	<b>0.027</b>
TNFRSF21	rs9369682	Recessive	C = 0.46	A = 0.54	0.551
TNFRSF21	rs9369686	Recessive	T = 0.18	C = 0.82	0.150
TNFRSF21	rs9381536	Recessive	T = 0.082	C = 0.918	0.273
TNFRSF21	rs10948341	Additive	C = 0.358	A = 0.642	0.318
TNFRSF21	rs6900354	Recessive	C = 0.417	T = 0.583	0.937
TNFRSF21	rs16875536	Additive	G = 0.027	C = 0.973	0.972
TNFRSF21	rs116473941	Recessive	A = 0.053	C = 0.947	0.196
<b>TNFRSF21</b>	<b>rs10498772</b>	<b>Additive</b>	<b>T = 0.069</b>	<b>G = 0.931</b>	<b>0.007</b>
TNFRSF21	rs55652946	Recessive	T = 0.168	C = 0.832	0.829

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF21	rs1387880	Dominant	G = 0.397	A = 0.603	0.811
TNFRSF21	rs75721724	Recessive	T = 0.081	C = 0.919	0.588
TNFRSF21	rs1387885	Additive	A = 0.332	G = 0.668	0.389
TNFRSF21	rs1687122	Dominant	T = 0.072	C = 0.928	0.543
TNFRSF21	rs1630504	Dominant	T = 0.452	C = 0.548	0.933
TNFRSF21	rs6908323	Dominant	T = 0.218	C = 0.782	0.202
TNFRSF21	rs1016244	Additive	A = 0.067	G = 0.933	0.184
TNFRSF21	rs13213075	Additive	G = 0.024	A = 0.976	0.961
TNFRSF21	rs73471691	Recessive	C = 0.072	A = 0.928	0.654
TNFRSF21	rs910554	Recessive	G = 0.23	A = 0.77	0.274
TNFRSF21	rs80272453	Dominant	G = 0.021	A = 0.979	0.966
TNFRSF21	rs6458556	Additive	G = 0.264	A = 0.736	0.547
TNFRSF21	---	Additive	T = 0.026	G = 0.974	0.972
TNFRSF21	rs76875310	Dominant	T = 0.089	G = 0.911	0.272
TNFRSF21	rs9283915	Dominant	T = 0.268	C = 0.732	0.799
TNFRSF21	rs6458559	Additive	T = 0.032	C = 0.968	0.825
TNFRSF21	rs9381534	Recessive	A = 0.044	G = 0.956	0.330
TNFRSF21	rs11755026	Recessive	A = 0.103	G = 0.897	0.427
TNFRSF21	rs9381537	Recessive	A = 0.32	G = 0.68	0.241
TNFRSF21	rs77522145	Dominant	T = 0.041	C = 0.959	0.735
TNFRSF21	rs78189219	Dominant	T = 0.019	C = 0.981	0.701
<b>TNFRSF21</b>	<b>rs115865729</b>	<b>Recessive</b>	<b>G = 0.133</b>	<b>A = 0.867</b>	<b>0.020</b>
TNFRSF21	rs4301296	Recessive	A = 0.45	G = 0.55	0.838
TNFRSF21	rs4711874	Recessive	T = 0.447	C = 0.553	0.959
TNFRSF21	rs1931832	Recessive	G = 0.355	A = 0.645	0.901
TNFRSF21	rs13207334	Recessive	G = 0.192	A = 0.808	0.259
TNFRSF21	rs76642149	Additive	A = 0.036	G = 0.964	0.987
TNFRSF21	rs7774615	Recessive	A = 0.487	G = 0.513	0.795
TNFRSF21	rs9473117	Recessive	C = 0.265	A = 0.735	0.500
TNFRSF21	rs12190006	Dominant	T = 0.089	G = 0.911	0.607
TNFRSF21	rs1762312	Dominant	T = 0.305	C = 0.695	0.385
TNFRSF21	rs9463292	Additive	G = 0.033	A = 0.967	0.963
TNFRSF21	rs3030592	Recessive	C = 0.143	T = 0.857	0.171
TNFRSF21	rs72860430	Additive	A = 0.038	G = 0.962	0.961
TNFRSF21	rs6458543	Additive	C = 0.039	T = 0.961	0.969
TNFRSF21	rs73471655	Dominant	C = 0.036	T = 0.964	0.062
TNFRSF21	rs2207225	Additive	A = 0.486	G = 0.514	0.193
TNFRSF21	rs4715001	Dominant	A = 0.44	C = 0.56	0.521
TNFRSF21	rs36081806	Additive	C = 0.053	T = 0.947	0.752
TNFRSF21	rs2281445	Dominant	T = 0.119	C = 0.881	0.422
TNFRSF21	rs13205450	Additive	T = 0.05	C = 0.95	0.989
TNFRSF21	rs73475487	Additive	T = 0.025	C = 0.975	0.702

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF21	rs6458558	Additive	G = 0.359	T = 0.641	0.702
TNFRSF21	rs12210021	Dominant	A = 0.073	G = 0.927	0.216
TNFRSF21	rs4422623	Recessive	T = 0.111	C = 0.889	0.506
TNFRSF21	rs9367278	Recessive	T = 0.484	C = 0.516	0.882
TNFRSF21	rs12173608	Dominant	G = 0.499	A = 0.501	0.200
TNFRSF21	rs62410670	Dominant	C = 0.062	A = 0.938	0.522
TNFRSF21	rs6906157	Dominant	A = 0.428	G = 0.572	0.956
TNFRSF21	rs111966938	Dominant	A = 0.032	C = 0.968	0.936
TNFRSF21	rs115056638	Dominant	A = 0.046	G = 0.954	0.057
TNFRSF21	rs114841434	Dominant	C = 0.024	G = 0.976	0.052
TNFRSF21	rs114770961	Additive	A = 0.028	G = 0.972	0.132
TNFRSF21	rs75614139	Dominant	G = 0.058	C = 0.942	0.051
TNFRSF21	rs115512876	Dominant	G = 0.021	C = 0.979	0.414
TNFRSF21	rs116512558	Dominant	T = 0.031	C = 0.969	0.415
TNFRSF21	rs116683092	Additive	T = 0.031	C = 0.969	0.027
TNFRSF21	rs78794444	Additive	T = 0.025	G = 0.975	0.961
TNFRSF21	rs115133271	Additive	G = 0.023	A = 0.977	0.972
TNFRSF21	rs77293470	Dominant	A = 0.033	C = 0.967	0.933
TNFRSF21	rs76718319	Dominant	T = 0.016	C = 0.984	0.935
<b>TNFRSF21</b>	<b>rs9395228</b>	<b>Recessive</b>	<b>A = 0.142</b>	<b>G = 0.858</b>	<b>0.026</b>
<b>TNFRSF21</b>	<b>rs17289051</b>	<b>Additive</b>	<b>C = 0.035</b>	<b>T = 0.965</b>	<b>0.048</b>
<b>TNFRSF21</b>	<b>rs9473038</b>	<b>Recessive</b>	<b>A = 0.052</b>	<b>G = 0.948</b>	<b>0.001</b>
TNFRSF21	rs6936025	Additive	A = 0.041	C = 0.959	0.693
TNFRSF21	rs16875825	Recessive	C = 0.069	T = 0.931	0.364
TNFRSF21	rs2103868	Recessive	G = 0.09	A = 0.91	0.646
TNFRSF21	rs6918220	Recessive	T = 0.047	C = 0.953	0.241
TNFRSF25	rs3170675	Additive	C = 0.054	T = 0.946	0.062
TNFRSF25	rs3007416	Additive	G = 0.059	A = 0.941	0.964
<b>TNFRSF25</b>	<b>rs2986758</b>	<b>Recessive</b>	<b>C = 0.155</b>	<b>A = 0.845</b>	<b>0.014</b>
TNFRSF25	rs11800462	Recessive	C = 0.072	T = 0.928	0.111
TNFRSF4	rs78479912	Additive	A = 0.038	G = 0.962	0.773
<b>TNFRSF6B</b>	<b>rs2738787</b>	<b>Dominant</b>	<b>A = 0.082</b>	<b>G = 0.918</b>	<b>0.043</b>
TNFRSF6B	rs55765053	Additive	T = 0.08	C = 0.92	0.201
TNFRSF6B	rs1291206	Recessive	A = 0.231	G = 0.769	0.322
TNFRSF8	rs11121855	Recessive	G = 0.103	A = 0.897	0.673
TNFRSF8	rs1201113	Additive	A = 0.129	G = 0.871	0.416
TNFRSF8	rs12042105	Additive	C = 0.24	T = 0.76	0.409
TNFRSF8	rs12409087	Additive	C = 0.028	T = 0.972	0.968
TNFRSF8	rs2230622	Dominant	T = 0.234	C = 0.766	0.824
TNFRSF8	rs4846094	Additive	A = 0.036	G = 0.964	0.220
TNFRSF8	rs59346161	Additive	A = 0.278	T = 0.722	0.122
TNFRSF8	rs72641094	Recessive	A = 0.082	G = 0.918	0.229

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF8	rs1208992	Recessive	A = 0.049	G = 0.951	0.897
TNFRSF8	rs74052932	Dominant	C = 0.068	T = 0.932	0.706
TNFRSF8	rs6541013	Recessive	T = 0.211	C = 0.789	0.502
TNFRSF8	rs6662910	Recessive	T = 0.068	G = 0.932	0.828
TNFRSF8	rs12143590	Recessive	G = 0.323	A = 0.677	0.082
TNFRSF8	rs11121853	Recessive	G = 0.207	A = 0.793	0.471
TNFRSF8	rs6684143	Dominant	G = 0.424	A = 0.576	0.431
<b>TNFRSF8</b>	<b>rs1201114</b>	<b>Recessive</b>	<b>A = 0.433</b>	<b>G = 0.567</b>	<b>0.017</b>
TNFRSF8	rs72641068	Dominant	G = 0.149	C = 0.851	0.391
TNFRSF8	rs4846091	Recessive	A = 0.323	G = 0.677	0.619
TNFRSF8	rs67622403	Recessive	G = 0.083	A = 0.917	0.112
TNFRSF8	rs551062	Recessive	C = 0.2	T = 0.8	0.052
TNFRSF8	rs12401286	Additive	C = 0.099	T = 0.901	0.065
TNFRSF8	rs603879	Dominant	C = 0.499	T = 0.501	0.079
TNFRSF8	rs816046	Additive	G = 0.361	T = 0.639	0.078
TNFRSF8	rs11590366	Dominant	C = 0.097	T = 0.903	0.139
TNFRSF8	rs2486316	Dominant	T = 0.154	G = 0.846	0.528
TNFRSF8	rs8179353	Additive	C = 0.429	T = 0.571	0.746
TNFRSF8	rs664654	Recessive	A = 0.08	G = 0.92	0.688
TNFRSF8	rs2230625	Dominant	G = 0.02	A = 0.98	0.065
TNFRSF8	rs11569922	Recessive	A = 0.03	G = 0.97	0.077
TNFRSF8	rs535068	Additive	G = 0.302	A = 0.698	0.496
TNFRSF8	rs3766735	Additive	A = 0.09	G = 0.91	0.074
TNFRSF8	rs671106	Recessive	G = 0.495	A = 0.505	0.273
TNFRSF8	---	Dominant	A = 0.014	G = 0.986	0.681
TNFRSF9	rs226476	Dominant	T = 0.038	G = 0.962	0.793
TNFRSF9	rs227163	Additive	C = 0.413	T = 0.587	0.748
TNFRSF9	rs228703	Dominant	G = 0.391	A = 0.609	0.082
TNFRSF9	rs2453021	Dominant	T = 0.357	C = 0.643	0.790
TNFRSF9	rs680522	Dominant	A = 0.496	G = 0.504	0.637
TNFRSF9	rs11586689	Additive	G = 0.16	A = 0.84	0.168
<b>TNFRSF9</b>	<b>rs228636</b>	<b>Additive</b>	<b>C = 0.44</b>	<b>T = 0.56</b>	<b>0.036</b>
TNFRSF9	rs79866090	Dominant	A = 0.019	G = 0.981	0.622
TNFRSF9	rs170628	Dominant	T = 0.217	C = 0.783	0.245
TNFRSF9	rs4908705	Dominant	T = 0.193	C = 0.807	0.550
TNFRSF9	rs763234	Dominant	A = 0.106	C = 0.894	0.360
TNFRSF9	rs11121046	Additive	G = 0.398	T = 0.602	0.392
TNFRSF9	rs170550	Dominant	T = 0.352	C = 0.648	0.440
TNFRSF9	rs227157	Dominant	G = 0.173	A = 0.827	0.865
TNFRSF9	rs10864320	Dominant	A = 0.014	G = 0.986	0.966
<b>TNFRSF9</b>	<b>rs76198826</b>	<b>Additive</b>	<b>T = 0.074</b>	<b>C = 0.926</b>	<b>0.010</b>
TNFRSF9	rs11582983	Additive	C = 0.13	T = 0.87	0.085

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFRSF9	rs707476	Dominant	T = 0.335	C = 0.665	0.118
TNFRSF9	rs664673	Additive	C = 0.404	T = 0.596	0.076
TNFRSF9	rs161826	Additive	G = 0.024	A = 0.976	0.828
TNFRSF9	rs9658041	Additive	T = 0.021	C = 0.979	0.973
TNFRSF9	rs161811	Dominant	T = 0.02	C = 0.98	0.966
TNFRSF9	rs161803	Additive	G = 0.024	A = 0.976	0.448
TNFSF10	rs12185945	Dominant	C = 0.431	T = 0.569	0.606
TNFSF10	rs12638147	Recessive	C = 0.276	G = 0.724	0.260
TNFSF10	rs16845868	Dominant	C = 0.107	A = 0.893	0.733
TNFSF10	rs17536035	Dominant	A = 0.081	G = 0.919	0.957
TNFSF10	rs17608639	Additive	T = 0.047	C = 0.953	0.971
TNFSF10	rs231997	Recessive	T = 0.136	C = 0.864	0.070
TNFSF10	rs233998	Recessive	A = 0.209	G = 0.791	0.257
TNFSF10	rs234030	Recessive	A = 0.098	G = 0.902	0.156
TNFSF10	rs234049	Recessive	C = 0.406	T = 0.594	0.238
TNFSF10	rs234071	Dominant	A = 0.1	C = 0.9	0.957
TNFSF10	rs3819773	Recessive	A = 0.328	G = 0.672	0.351
TNFSF10	rs41309770	Additive	C = 0.037	A = 0.963	0.521
<b>TNFSF10</b>	<b>rs6445064</b>	<b>Additive</b>	<b>A = 0.325</b>	<b>G = 0.675</b>	<b>0.028</b>
TNFSF10	rs6805167	Dominant	A = 0.367	G = 0.633	0.274
TNFSF10	rs9872004	Dominant	C = 0.053	T = 0.947	0.770
TNFSF10	rs11706116	Recessive	C = 0.233	A = 0.767	0.818
TNFSF10	rs13079726	Recessive	T = 0.465	C = 0.535	0.115
TNFSF10	rs179777	Dominant	C = 0.133	T = 0.867	0.727
TNFSF10	rs13061442	Recessive	C = 0.257	A = 0.743	0.468
TNFSF10	rs12488654	Dominant	A = 0.172	G = 0.828	0.288
TNFSF10	rs75278014	Dominant	G = 0.057	A = 0.943	0.905
TNFSF10	rs16845803	Dominant	G = 0.141	A = 0.859	0.220
TNFSF10	rs79729232	Dominant	A = 0.035	G = 0.965	0.759
TNFSF10	rs2286983	Dominant	A = 0.231	G = 0.769	0.087
TNFSF10	rs234062	Recessive	G = 0.284	A = 0.716	0.536
TNFSF10	rs234058	Recessive	T = 0.453	C = 0.547	0.327
TNFSF10	---	Recessive	A = 0.272	G = 0.728	0.238
TNFSF10	rs35307518	Additive	G = 0.177	C = 0.823	0.073
TNFSF10	rs437197	Recessive	C = 0.341	G = 0.659	0.384
TNFSF10	rs77457335	Dominant	A = 0.027	C = 0.973	0.298
TNFSF10	rs34988670	Additive	C = 0.225	A = 0.775	0.381
TNFSF10	rs231996	Recessive	C = 0.247	A = 0.753	0.890
TNFSF10	rs11922343	Dominant	C = 0.367	T = 0.633	0.769
TNFSF10	rs60792873	Dominant	A = 0.232	G = 0.768	0.604
TNFSF10	rs66991667	Additive	T = 0.046	C = 0.954	0.969
TNFSF10	rs115898535	Dominant	A = 0.026	C = 0.974	0.256

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF10	rs115984079	Additive	A = 0.023	G = 0.977	0.972
TNFSF10	rs75389921	Recessive	T = 0.04	C = 0.96	0.828
TNFSF10	rs115574784	Dominant	A = 0.029	G = 0.971	0.777
TNFSF10	rs3136597	Recessive	T = 0.222	G = 0.778	0.063
TNFSF10	rs3136595	Dominant	A = 0.014	G = 0.986	0.390
TNFSF10	rs6779133	Additive	T = 0.248	C = 0.752	0.399
TNFSF10	rs398778	Additive	C = 0.043	A = 0.957	0.023
TNFSF10	rs3136581	Dominant	A = 0.216	G = 0.784	0.124
TNFSF11	rs10507512	Additive	G = 0.03	A = 0.97	0.972
TNFSF11	rs11620123	Recessive	A = 0.03	G = 0.97	0.104
TNFSF11	rs12430303	Recessive	T = 0.485	C = 0.515	0.146
TNFSF11	rs1325794	Recessive	A = 0.046	G = 0.954	0.648
TNFSF11	rs1325802	Dominant	A = 0.035	G = 0.965	0.701
TNFSF11	rs17536635	Additive	A = 0.018	G = 0.982	0.961
TNFSF11	rs17537183	Additive	C = 0.029	T = 0.971	0.961
TNFSF11	rs17600227	Additive	T = 0.029	C = 0.971	0.115
TNFSF11	rs2062305	Recessive	G = 0.484	A = 0.516	0.106
TNFSF11	rs417768	Additive	G = 0.339	A = 0.661	0.092
TNFSF11	rs9533087	Recessive	T = 0.146	C = 0.854	0.634
TNFSF11	rs9533176	Recessive	G = 0.426	A = 0.574	0.919
TNFSF11	rs9533185	Recessive	T = 0.16	G = 0.84	0.786
TNFSF11	rs9594738	Dominant	T = 0.477	C = 0.523	0.427
TNFSF11	rs9594759	Additive	C = 0.465	T = 0.535	0.465
TNFSF11	rs36040075	Recessive	G = 0.254	A = 0.746	0.443
TNFSF11	rs17521782	Dominant	C = 0.155	T = 0.845	0.096
<b>TNFSF11</b>	<b>rs9566963</b>	<b>Recessive</b>	<b>A = 0.422</b>	<b>C = 0.578</b>	<b>0.014</b>
TNFSF11	rs9533068	Recessive	C = 0.102	T = 0.898	0.940
TNFSF11	rs78287951	Additive	T = 0.035	C = 0.965	0.972
TNFSF11	rs238252	Recessive	G = 0.16	C = 0.84	0.497
TNFSF11	rs71429414	Additive	A = 0.21	G = 0.79	0.389
TNFSF11	rs9525617	Recessive	T = 0.195	G = 0.805	0.941
TNFSF11	rs73172251	Dominant	A = 0.025	G = 0.975	0.966
TNFSF11	rs17595883	Additive	C = 0.045	T = 0.955	0.972
TNFSF11	rs71428876	Additive	A = 0.044	G = 0.956	0.973
TNFSF11	rs12874142	Additive	T = 0.228	C = 0.772	0.477
TNFSF11	rs79984659	Additive	T = 0.026	C = 0.974	0.276
TNFSF11	rs78059020	Recessive	G = 0.051	T = 0.949	0.171
TNFSF11	rs17457881	Additive	G = 0.241	A = 0.759	0.360
TNFSF11	rs117800509	Dominant	G = 0.027	A = 0.973	0.918
TNFSF11	rs77972371	Dominant	A = 0.034	G = 0.966	0.360
TNFSF11	rs4941432	Recessive	A = 0.266	G = 0.734	0.466
TNFSF11	rs78161200	Additive	T = 0.015	C = 0.985	0.972

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF11	rs1021188	Additive	C = 0.182	T = 0.818	0.574
TNFSF11	rs12721447	Additive	G = 0.016	T = 0.984	0.961
TNFSF11	rs73174448	Additive	A = 0.078	G = 0.922	0.168
TNFSF11	rs76824117	Dominant	C = 0.017	G = 0.983	0.966
TNFSF11	rs78667121	Additive	A = 0.031	G = 0.969	0.972
TNFSF11	rs184243428	Dominant	C = 0.029	T = 0.971	0.368
<b>TNFSF11</b>	<b>rs10507514</b>	<b>Recessive</b>	<b>G = 0.316</b>	<b>A = 0.684</b>	<b>0.042</b>
TNFSF11	rs73176302	Additive	C = 0.04	T = 0.96	0.430
TNFSF11	rs2104274	Recessive	G = 0.293	C = 0.707	0.805
TNFSF11	rs12867100	Recessive	T = 0.089	C = 0.911	0.097
TNFSF11	rs55985600	Recessive	A = 0.283	G = 0.717	0.183
TNFSF11	rs9590709	Additive	A = 0.046	G = 0.954	0.031
TNFSF11	rs73178050	Recessive	A = 0.039	G = 0.961	0.112
TNFSF11	rs2296115	Additive	A = 0.207	G = 0.793	0.866
TNFSF11	rs1535688	Recessive	G = 0.275	A = 0.725	0.464
TNFSF11	rs117275014	Additive	C = 0.022	T = 0.978	0.973
TNFSF11	rs9315953	Recessive	G = 0.254	A = 0.746	0.844
TNFSF11	rs61960884	Dominant	T = 0.151	C = 0.849	0.166
TNFSF11	rs9533084	Additive	A = 0.015	G = 0.985	0.563
TNFSF11	rs116857535	Dominant	G = 0.019	C = 0.981	0.450
TNFSF11	rs79499151	Additive	A = 0.015	G = 0.985	0.972
TNFSF11	rs9594768	Recessive	C = 0.487	A = 0.513	0.111
TNFSF11	rs2296533	Dominant	C = 0.445	T = 0.555	0.158
TNFSF11	rs9533159	Dominant	G = 0.429	T = 0.571	0.092
TNFSF11	rs35091986	Dominant	C = 0.107	T = 0.893	0.814
TNFSF11	rs73174493	Dominant	A = 0.02	G = 0.98	0.521
TNFSF11	rs117697199	Recessive	T = 0.027	C = 0.973	0.624
TNFSF11	rs77855024	Recessive	A = 0.084	G = 0.916	0.057
TNFSF11	rs77979811	Recessive	T = 0.043	C = 0.957	0.105
TNFSF11	rs118118307	Recessive	C = 0.032	T = 0.968	0.077
TNFSF11	rs113785233	Additive	G = 0.031	A = 0.969	0.972
TNFSF11	rs74583751	Dominant	T = 0.012	C = 0.988	0.256
TNFSF11	rs79475191	Additive	T = 0.047	C = 0.953	0.541
TNFSF11	rs116959176	Additive	T = 0.046	C = 0.954	0.539
TNFSF11	rs7998154	Recessive	C = 0.028	T = 0.972	0.105
TNFSF11	rs10507507	Additive	C = 0.229	T = 0.771	0.376
<b>TNFSF11</b>	<b>rs180848</b>	<b>Additive</b>	<b>T = 0.022</b>	<b>C = 0.978</b>	<b>0.002</b>
TNFSF11	rs1038435	Recessive	A = 0.044	G = 0.956	0.488
TNFSF11	rs1325798	Dominant	T = 0.427	C = 0.573	0.089
TNFSF11	rs346578	Recessive	A = 0.084	G = 0.916	0.155
TNFSF11	rs10507511	Dominant	T = 0.022	C = 0.978	0.938
TNFSF11	rs1535681	Recessive	C = 0.349	T = 0.651	0.876

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF11	rs79596148	Additive	G = 0.056	A = 0.944	0.622
TNFSF11	rs912100	Recessive	C = 0.343	T = 0.657	0.340
TNFSF11	rs117346132	Additive	G = 0.023	A = 0.977	0.155
TNFSF11	rs9533090	Recessive	T = 0.467	C = 0.533	0.450
TNFSF12	rs62059777	Dominant	A = 0.203	G = 0.797	0.080
TNFSF12	rs78002826	Additive	T = 0.11	C = 0.89	0.074
TNFSF12	rs11078691	Dominant	C = 0.188	T = 0.812	0.743
<b>TNFSF12</b>	<b>rs11078692</b>	<b>Dominant</b>	<b>C = 0.389</b>	<b>T = 0.611</b>	<b>0.013</b>
TNFSF12	rs11870720	Recessive	T = 0.147	C = 0.853	0.996
TNFSF12	rs62059792	Dominant	G = 0.236	A = 0.764	0.100
TNFSF12	rs62059794	Additive	A = 0.112	G = 0.888	0.149
TNFSF12	rs34445439	Recessive	T = 0.287	C = 0.713	0.613
<b>TNFSF12</b>	<b>rs9892297</b>	<b>Dominant</b>	<b>G = 0.334</b>	<b>A = 0.666</b>	<b>0.050</b>
TNFSF12	rs9902027	Dominant	C = 0.228	T = 0.772	0.744
TNFSF12	rs4968200	Additive	C = 0.134	G = 0.866	0.023
TNFSF12	rs9899183	Recessive	C = 0.256	T = 0.744	0.889
TNFSF12	rs3803796	Additive	G = 0.109	C = 0.891	0.067
TNFSF12	rs4968210	Additive	A = 0.405	G = 0.595	0.667
TNFSF12	rs3803800	Recessive	A = 0.209	G = 0.791	0.616
TNFSF12	rs6608	Additive	T = 0.172	C = 0.828	0.135
TNFSF12	rs11552708	Dominant	A = 0.117	G = 0.883	0.155
<b>TNFSF12</b>	<b>rs8073937</b>	<b>Dominant</b>	<b>A = 0.214</b>	<b>G = 0.786</b>	<b>&gt;0.001</b>
TNFSF12	rs11654632	Dominant	C = 0.389	T = 0.611	0.966
TNFSF12	rs62059793	Dominant	G = 0.421	A = 0.579	0.970
TNFSF13B	rs1012967	Recessive	C = 0.085	T = 0.915	0.972
TNFSF13B	rs10508198	Recessive	C = 0.34	G = 0.66	0.546
TNFSF13B	rs1224141	Recessive	G = 0.215	T = 0.785	0.979
TNFSF13B	rs12861715	Dominant	T = 0.343	C = 0.657	0.663
TNFSF13B	rs12873468	Dominant	T = 0.046	G = 0.954	0.933
TNFSF13B	rs12874404	Dominant	G = 0.057	A = 0.943	0.701
TNFSF13B	rs1326407	Additive	A = 0.027	G = 0.973	0.969
TNFSF13B	rs16972216	Additive	A = 0.161	G = 0.839	0.625
TNFSF13B	rs16972412	Dominant	G = 0.118	A = 0.882	0.964
TNFSF13B	rs17498440	Recessive	G = 0.124	A = 0.876	0.316
TNFSF13B	rs17498545	Recessive	C = 0.175	T = 0.825	0.499
TNFSF13B	rs1886195	Recessive	C = 0.45	T = 0.55	0.699
TNFSF13B	rs2893321	Additive	G = 0.22	A = 0.78	0.876
TNFSF13B	rs41275064	Additive	T = 0.052	C = 0.948	0.771
TNFSF13B	rs7329911	Dominant	C = 0.326	T = 0.674	0.468
TNFSF13B	rs7334250	Dominant	G = 0.364	A = 0.636	0.639
TNFSF13B	rs7338062	Recessive	A = 0.283	G = 0.717	0.475
TNFSF13B	rs9514827	Dominant	C = 0.312	T = 0.688	0.836

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF13B	rs9514828	Dominant	T = 0.477	C = 0.523	0.919
TNFSF13B	rs9520835	Dominant	A = 0.297	G = 0.703	0.988
TNFSF13B	rs9520862	Dominant	G = 0.171	T = 0.829	0.778
TNFSF13B	rs9520874	Additive	T = 0.019	C = 0.981	0.972
TNFSF13B	rs9520908	Dominant	T = 0.022	C = 0.978	0.070
TNFSF13B	rs9520911	Recessive	G = 0.461	A = 0.539	0.739
TNFSF13B	rs9559300	Dominant	G = 0.263	A = 0.737	0.086
TNFSF13B	rs9587544	Recessive	A = 0.093	G = 0.907	0.186
TNFSF13B	rs11617359	Recessive	C = 0.207	T = 0.793	0.655
TNFSF13B	rs1224096	Dominant	G = 0.429	A = 0.571	0.849
TNFSF13B	rs1924349	Recessive	C = 0.433	T = 0.567	0.373
TNFSF13B	rs9284240	Recessive	A = 0.092	G = 0.908	0.735
TNFSF13B	rs9520945	Dominant	G = 0.453	A = 0.547	0.211
TNFSF13B	rs9559317	Dominant	C = 0.193	T = 0.807	0.932
TNFSF13B	rs116953323	Recessive	A = 0.034	G = 0.966	0.104
TNFSF13B	rs73609093	Dominant	T = 0.025	C = 0.975	0.146
TNFSF13B	rs17499386	Recessive	C = 0.137	T = 0.863	0.408
TNFSF13B	rs61092568	Additive	T = 0.022	C = 0.978	0.262
TNFSF13B	rs79277237	Dominant	C = 0.058	T = 0.942	0.297
<b>TNFSF13B</b>	<b>rs16972249</b>	<b>Recessive</b>	<b>A = 0.156</b>	<b>T = 0.844</b>	<b>&gt;0.0001</b>
TNFSF13B	rs78300486	Dominant	G = 0.017	A = 0.983	0.853
TNFSF13B	rs78069397	Dominant	T = 0.02	C = 0.98	0.948
TNFSF13B	rs17465647	Dominant	G = 0.251	C = 0.749	0.985
TNFSF13B	rs56205602	Dominant	G = 0.134	A = 0.866	0.363
TNFSF13B	rs11069734	Recessive	T = 0.304	C = 0.696	0.738
TNFSF13B	rs77177017	Additive	T = 0.021	C = 0.979	0.973
TNFSF13B	rs7992286	Dominant	T = 0.083	C = 0.917	0.786
TNFSF13B	rs9559329	Recessive	T = 0.096	C = 0.904	0.954
TNFSF13B	rs61973465	Recessive	T = 0.103	C = 0.897	0.306
TNFSF13B	rs61973466	Recessive	T = 0.08	C = 0.92	0.090
TNFSF13B	rs77193142	Additive	T = 0.035	C = 0.965	0.961
TNFSF13B	rs61973471	Additive	C = 0.221	A = 0.779	0.721
TNFSF13B	rs7333206	Recessive	T = 0.208	C = 0.792	0.284
TNFSF13B	rs3886718	Additive	C = 0.362	T = 0.638	0.051
TNFSF13B	rs78987146	Recessive	T = 0.049	G = 0.951	0.625
TNFSF13B	rs7323926	Additive	G = 0.25	C = 0.75	0.674
TNFSF13B	rs118082599	Additive	A = 0.026	G = 0.974	0.972
TNFSF13B	rs17885582	Additive	G = 0.174	A = 0.826	0.785
TNFSF13B	rs74399908	Additive	T = 0.038	C = 0.962	0.969
TNFSF13B	rs1224167	Additive	A = 0.265	G = 0.735	0.447
TNFSF13B	rs72660892	Additive	C = 0.094	T = 0.906	0.719
TNFSF13B	rs72660894	Recessive	T = 0.027	C = 0.973	0.360

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF13B	rs79286499	Additive	G = 0.018	A = 0.982	0.968
TNFSF13B	rs9634570	Recessive	C = 0.087	G = 0.913	0.027
TNFSF13B	rs72662912	Dominant	C = 0.019	A = 0.981	0.340
TNFSF13B	rs77232105	Additive	C = 0.02	T = 0.98	0.550
TNFSF13B	rs72638424	Recessive	T = 0.076	G = 0.924	0.842
TNFSF13B	rs72638425	Dominant	T = 0.184	C = 0.816	0.870
TNFSF13B	rs66704083	Dominant	G = 0.058	A = 0.942	0.531
TNFSF13B	rs72662954	Dominant	T = 0.237	C = 0.763	0.837
TNFSF13B	rs67264860	Recessive	C = 0.067	T = 0.933	0.680
TNFSF13B	rs957038	Dominant	T = 0.133	C = 0.867	0.312
TNFSF13B	rs35218525	Dominant	G = 0.211	A = 0.789	0.593
TNFSF13B	rs56074835	Recessive	A = 0.042	G = 0.958	0.812
TNFSF13B	rs72662994	Additive	A = 0.02	G = 0.98	0.972
TNFSF13B	rs72664913	Additive	G = 0.053	A = 0.947	0.972
TNFSF13B	rs75249955	Dominant	C = 0.043	T = 0.957	0.333
TNFSF13B	rs79909975	Additive	C = 0.019	T = 0.981	0.972
TNFSF13B	rs79925237	Dominant	C = 0.039	A = 0.961	0.216
TNFSF13B	rs118186472	Recessive	T = 0.033	C = 0.967	0.105
TNFSF13B	rs78106682	Recessive	G = 0.061	A = 0.939	0.396
TNFSF13B	rs116898958	Additive	T = 0.042	C = 0.958	0.968
TNFSF13B	rs77835274	Additive	A = 0.023	G = 0.977	0.284
TNFSF13B	rs113307941	Recessive	T = 0.079	G = 0.921	0.793
TNFSF13B	rs77012415	Additive	G = 0.044	C = 0.956	0.961
TNFSF13B	rs77346221	Additive	T = 0.035	C = 0.965	0.961
TNFSF13B	rs78987550	Dominant	A = 0.043	G = 0.957	0.966
TNFSF13B	rs113519879	Recessive	G = 0.02	A = 0.98	0.361
TNFSF13B	rs36206505	Recessive	G = 0.064	A = 0.936	0.994
TNFSF13B	rs9301289	Dominant	A = 0.236	G = 0.764	0.376
TNFSF13B	rs11620183	Dominant	G = 0.135	A = 0.865	0.745
TNFSF13B	rs17381647	Additive	G = 0.194	A = 0.806	0.619
TNFSF13B	rs12861739	Additive	G = 0.035	A = 0.965	0.963
TNFSF13B	rs17468013	Recessive	T = 0.074	C = 0.926	0.657
TNFSF13B	rs16972513	Dominant	A = 0.109	G = 0.891	0.931
TNFSF13B	rs17384372	Additive	T = 0.065	C = 0.935	0.785
TNFSF13B	---	Additive	A = 0.056	G = 0.944	0.389
TNFSF13B	rs1924351	Dominant	T = 0.256	C = 0.744	0.701
TNFSF13B	rs7337447	Dominant	G = 0.254	A = 0.746	0.037
TNFSF14	rs344560	Dominant	T = 0.055	C = 0.945	0.812
TNFSF14	rs344569	Recessive	A = 0.173	G = 0.827	0.348
TNFSF14	rs344570	Recessive	T = 0.116	C = 0.884	0.746
TNFSF14	rs344577	Recessive	T = 0.186	C = 0.814	0.117
TNFSF14	rs379527	Dominant	C = 0.305	A = 0.695	0.381

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF14	rs408453	Recessive	G = 0.461	A = 0.539	0.692
TNFSF14	rs7254538	Recessive	G = 0.469	C = 0.531	0.143
TNFSF14	rs2279627	Additive	C = 0.258	G = 0.742	0.819
TNFSF14	rs72984514	Recessive	G = 0.205	T = 0.795	0.126
TNFSF14	rs10410643	Additive	A = 0.321	C = 0.679	0.194
TNFSF14	rs8104977	Additive	A = 0.022	G = 0.978	0.972
TNFSF14	rs447135	Recessive	C = 0.48	T = 0.52	0.987
TNFSF14	rs392751	Dominant	G = 0.321	A = 0.679	0.281
TNFSF14	rs1077667	Dominant	T = 0.211	C = 0.789	0.228
TNFSF14	rs35333884	Recessive	T = 0.304	C = 0.696	0.949
TNFSF14	rs404061	Recessive	A = 0.147	C = 0.853	0.646
TNFSF14	rs72988314	Dominant	G = 0.04	A = 0.96	0.803
TNFSF14	rs396352	Dominant	T = 0.321	C = 0.679	0.538
TNFSF14	rs433312	Dominant	A = 0.485	C = 0.515	0.200
TNFSF14	rs12972736	Recessive	T = 0.259	C = 0.741	0.730
TNFSF14	rs62123254	Recessive	C = 0.303	T = 0.697	0.805
TNFSF14	rs2291668	Dominant	A = 0.172	G = 0.828	0.185
TNFSF14	rs74441035	Additive	T = 0.048	C = 0.952	0.459
TNFSF14	rs344590	Dominant	T = 0.137	C = 0.863	0.852
TNFSF14	rs11666589	Dominant	C = 0.135	T = 0.865	0.484
TNFSF14	rs17516272	Recessive	T = 0.086	G = 0.914	0.148
TNFSF14	rs4807886	Dominant	A = 0.024	G = 0.976	0.944
TNFSF14	rs447573	Additive	A = 0.321	G = 0.679	0.298
TNFSF15	rs10982441	Dominant	T = 0.12	C = 0.88	0.589
TNFSF15	rs35843739	Recessive	C = 0.065	T = 0.935	0.644
TNFSF15	rs3810936	Recessive	T = 0.327	C = 0.673	0.104
TNFSF15	rs4263839	Additive	A = 0.331	G = 0.669	0.045
TNFSF15	rs4978609	Recessive	G = 0.116	A = 0.884	0.847
<b>TNFSF15</b>	<b>rs6478108</b>	<b>Additive</b>	<b>C = 0.346</b>	<b>T = 0.654</b>	<b>0.016</b>
TNFSF15	rs6478109	Additive	A = 0.335	G = 0.665	0.023
TNFSF15	rs7864084	Recessive	G = 0.156	A = 0.844	0.792
TNFSF15	rs10114224	Recessive	C = 0.142	T = 0.858	0.143
TNFSF15	rs10759734	Recessive	G = 0.249	A = 0.751	0.837
TNFSF15	rs2006996	Dominant	C = 0.042	T = 0.958	0.370
TNFSF15	rs4979467	Dominant	C = 0.448	T = 0.552	0.226
TNFSF15	rs17816047	Dominant	C = 0.086	A = 0.914	0.934
TNFSF15	rs72754566	Recessive	G = 0.038	A = 0.962	0.358
TNFSF15	rs13293384	Dominant	T = 0.337	C = 0.663	0.662
TNFSF15	rs76632088	Dominant	A = 0.175	G = 0.825	0.926
TNFSF15	rs4246905	Additive	T = 0.295	C = 0.705	0.103
TNFSF15	rs1407308	Recessive	G = 0.498	T = 0.502	0.335
TNFSF15	rs77648435	Additive	A = 0.023	G = 0.977	0.270

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF15	rs4979451	Dominant	G = 0.047	A = 0.953	0.872
TNFSF15	rs56404767	Additive	G = 0.03	A = 0.97	0.968
TNFSF15	rs62578666	Additive	T = 0.087	C = 0.913	0.525
TNFSF15	rs56000964	Additive	T = 0.017	C = 0.983	0.243
TNFSF15	rs80271384	Recessive	T = 0.153	A = 0.847	0.252
TNFSF15	rs56069985	Dominant	G = 0.053	A = 0.947	0.886
TNFSF15	rs55808543	Additive	G = 0.028	A = 0.972	0.968
TNFSF15	rs56323622	Dominant	A = 0.032	G = 0.968	0.624
TNFSF15	rs55637274	Dominant	A = 0.041	G = 0.959	0.758
TNFSF15	rs111954497	Recessive	T = 0.022	C = 0.978	0.231
TNFSF15	rs117819581	Recessive	T = 0.025	C = 0.975	0.016
TNFSF15	rs116927665	Additive	G = 0.046	T = 0.954	0.771
TNFSF15	rs78309793	Additive	C = 0.033	T = 0.967	0.968
TNFSF15	rs7868007	Dominant	G = 0.479	A = 0.521	0.651
TNFSF15	rs2183019	Additive	T = 0.389	C = 0.611	0.625
TNFSF15	rs10982388	Additive	A = 0.088	G = 0.912	0.642
TNFSF15	rs12342775	Additive	A = 0.02	G = 0.98	0.620
TNFSF15	rs150474498	Dominant	A = 0.015	G = 0.985	0.524
TNFSF18	rs10489272	Additive	A = 0.334	G = 0.666	0.391
TNFSF18	rs10912482	Dominant	C = 0.303	T = 0.697	0.114
TNFSF18	rs10912505	Dominant	G = 0.428	A = 0.572	0.738
TNFSF18	rs1116616	Recessive	C = 0.087	T = 0.913	0.831
TNFSF18	rs11582085	Recessive	G = 0.084	A = 0.916	0.246
TNFSF18	rs11801183	Recessive	T = 0.18	C = 0.82	0.650
TNFSF18	rs12033800	Dominant	A = 0.211	G = 0.789	0.216
TNFSF18	rs12035082	Recessive	C = 0.408	T = 0.592	0.129
TNFSF18	rs12060864	Dominant	C = 0.486	T = 0.514	0.212
TNFSF18	rs12118303	Dominant	C = 0.169	T = 0.831	0.886
TNFSF18	rs12127501	Recessive	G = 0.041	A = 0.959	0.063
TNFSF18	rs1234292	Recessive	C = 0.488	T = 0.512	0.835
TNFSF18	rs1234318	Dominant	G = 0.356	A = 0.644	0.884
TNFSF18	rs17300100	Additive	G = 0.113	T = 0.887	0.500
TNFSF18	rs17372135	Recessive	G = 0.416	A = 0.584	0.074
TNFSF18	rs35135173	Recessive	C = 0.092	A = 0.908	0.748
TNFSF18	rs4081544	Dominant	T = 0.267	C = 0.733	0.877
TNFSF18	rs4916288	Recessive	G = 0.376	T = 0.624	0.567
TNFSF18	rs6425030	Recessive	T = 0.065	C = 0.935	0.679
TNFSF18	rs6425203	Recessive	C = 0.267	T = 0.733	0.571
TNFSF18	rs704842	Dominant	T = 0.178	C = 0.822	0.459
TNFSF18	rs7517810	Additive	T = 0.242	C = 0.758	0.422
TNFSF18	rs859655	Dominant	A = 0.407	G = 0.593	0.292
TNFSF18	rs9286879	Dominant	G = 0.246	A = 0.754	0.594

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF18	rs972100	Recessive	C = 0.19	T = 0.81	0.807
TNFSF18	rs16845430	Recessive	A = 0.228	C = 0.772	0.994
TNFSF18	rs17279525	Recessive	C = 0.124	T = 0.876	0.437
TNFSF18	rs2236876	Recessive	A = 0.255	G = 0.745	0.885
TNFSF18	rs6665826	Additive	G = 0.218	A = 0.782	0.818
TNFSF18	rs6673681	Additive	G = 0.038	A = 0.962	0.961
TNFSF18	rs859666	Recessive	C = 0.331	G = 0.669	0.670
TNFSF18	rs1492907	Recessive	T = 0.303	C = 0.697	0.954
TNFSF18	rs75603433	Additive	C = 0.044	T = 0.956	0.034
TNFSF18	rs859651	Recessive	C = 0.354	T = 0.646	0.815
TNFSF18	rs78329887	Recessive	G = 0.111	T = 0.889	0.988
TNFSF18	rs859626	Recessive	G = 0.437	A = 0.563	0.544
TNFSF18	rs61828367	Recessive	G = 0.491	A = 0.509	0.860
TNFSF18	rs56204807	Dominant	A = 0.016	G = 0.984	0.409
TNFSF18	rs113922700	Dominant	A = 0.033	G = 0.967	0.599
TNFSF18	rs55961352	Dominant	A = 0.054	G = 0.946	0.930
TNFSF18	rs116637993	Additive	G = 0.031	A = 0.969	0.969
TNFSF18	rs72714665	Dominant	T = 0.035	C = 0.965	0.557
TNFSF18	rs41514049	Dominant	A = 0.401	G = 0.599	0.515
TNFSF18	rs72716582	Recessive	C = 0.054	A = 0.946	0.772
TNFSF18	rs116482992	Dominant	G = 0.013	A = 0.987	0.894
TNFSF18	rs10798251	Dominant	C = 0.372	T = 0.628	0.408
TNFSF18	rs76520075	Recessive	G = 0.057	A = 0.943	0.400
TNFSF18	rs115299430	Recessive	C = 0.043	T = 0.957	0.281
TNFSF18	rs114298675	Recessive	A = 0.029	G = 0.971	0.623
TNFSF18	rs72722820	Recessive	A = 0.035	G = 0.965	0.624
TNFSF18	rs72722823	Dominant	A = 0.039	G = 0.961	0.138
TNFSF18	rs73034192	Dominant	A = 0.03	G = 0.97	0.622
TNFSF18	rs76460432	Dominant	T = 0.037	C = 0.963	0.593
TNFSF18	rs12068671	Dominant	C = 0.17	T = 0.83	0.787
TNFSF18	rs859639	Dominant	T = 0.478	C = 0.522	0.247
TNFSF18	rs12143438	Dominant	T = 0.14	G = 0.86	0.690
TNFSF18	rs61828388	Additive	A = 0.027	G = 0.973	0.972
TNFSF18	rs12751388	Additive	A = 0.017	G = 0.983	0.961
TNFSF18	rs57440247	Dominant	A = 0.034	G = 0.966	0.952
TNFSF18	rs12132387	Recessive	G = 0.373	T = 0.627	0.514
TNFSF18	rs72718930	Dominant	G = 0.017	A = 0.983	0.244
TNFSF18	rs79618756	Recessive	C = 0.123	G = 0.877	0.930
TNFSF18	rs1234307	Dominant	A = 0.018	G = 0.982	0.626
TNFSF18	rs11806687	Recessive	G = 0.179	A = 0.821	0.570
TNFSF18	rs113681974	Additive	C = 0.049	T = 0.951	0.961
TNFSF18	rs80122300	Recessive	A = 0.067	G = 0.933	0.831

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF18	rs114054677	Additive	C = 0.048	A = 0.952	0.974
TNFSF18	rs116070574	Dominant	T = 0.034	C = 0.966	0.365
TNFSF18	rs74339030	Additive	A = 0.021	G = 0.979	0.972
TNFSF18	rs116096003	Recessive	T = 0.04	C = 0.96	0.063
TNFSF18	rs114355210	Additive	C = 0.039	T = 0.961	0.894
TNFSF18	rs79803765	Additive	A = 0.018	G = 0.982	0.972
TNFSF18	rs2422270	Additive	C = 0.07	G = 0.93	0.444
TNFSF18	rs4250	Recessive	C = 0.052	T = 0.948	0.958
TNFSF18	rs41478644	Dominant	T = 0.075	C = 0.925	0.620
TNFSF4	rs10489270	Recessive	A = 0.129	G = 0.871	0.679
TNFSF4	rs2142822	Additive	C = 0.026	T = 0.974	0.990
TNFSF4	rs3861953	Recessive	T = 0.082	C = 0.918	0.400
TNFSF4	rs7527564	Additive	G = 0.033	T = 0.967	0.297
TNFSF4	rs844644	Dominant	A = 0.461	C = 0.539	0.680
TNFSF4	rs844658	Dominant	C = 0.049	T = 0.951	0.866
TNFSF4	rs2205960	Additive	T = 0.225	G = 0.775	0.231
TNFSF4	rs12138547	Recessive	C = 0.111	T = 0.889	0.327
TNFSF4	rs12730413	Dominant	A = 0.062	G = 0.938	0.674
TNFSF4	rs77884178	Recessive	T = 0.03	C = 0.97	0.359
TNFSF4	rs10912572	Additive	A = 0.031	G = 0.969	0.935
TNFSF4	rs4081545	Recessive	C = 0.334	T = 0.666	0.800
TNFSF8	rs10817686	Recessive	C = 0.439	T = 0.561	0.088
TNFSF8	rs10982466	Dominant	C = 0.043	T = 0.957	0.866
TNFSF8	rs10982476	Dominant	C = 0.208	T = 0.792	0.079
TNFSF8	rs17292115	Additive	C = 0.294	A = 0.706	0.480
TNFSF8	rs17817825	Recessive	C = 0.262	A = 0.738	0.882
TNFSF8	rs35234842	Dominant	C = 0.038	A = 0.962	0.829
TNFSF8	rs7026683	Recessive	G = 0.479	A = 0.521	0.341
TNFSF8	rs7853354	Dominant	C = 0.205	T = 0.795	0.569
TNFSF8	rs3181354	Dominant	G = 0.266	A = 0.734	0.839
TNFSF8	rs4979476	Recessive	G = 0.09	A = 0.91	0.732
TNFSF8	rs62578687	Recessive	T = 0.077	C = 0.923	0.800
TNFSF8	rs73567432	Additive	T = 0.053	C = 0.947	0.964
TNFSF8	rs59961803	Recessive	T = 0.165	C = 0.835	0.836
TNFSF8	rs17817489	Recessive	T = 0.152	C = 0.848	0.141
TNFSF8	rs4979485	Recessive	T = 0.192	C = 0.808	0.594
TNFSF8	rs72758616	Dominant	T = 0.034	C = 0.966	0.289
TNFSF8	rs17239726	Recessive	A = 0.184	G = 0.816	0.873
TNFSF8	rs74503538	Additive	T = 0.068	C = 0.932	0.962
TNFSF8	rs78520368	Dominant	T = 0.046	C = 0.954	0.321
TNFSF8	rs72756571	Additive	T = 0.076	G = 0.924	0.458
TNFSF8	rs117803009	Dominant	T = 0.028	C = 0.972	0.221

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF8	rs34673934	Recessive	G = 0.216	T = 0.784	0.517
TNFSF8	rs1885384	Dominant	A = 0.155	G = 0.845	0.705
TNFSF8	rs76819793	Dominant	C = 0.024	T = 0.976	0.366
TNFSF8	rs35239785	Additive	G = 0.147	A = 0.853	0.107
TNFSF8	rs62578740	Dominant	T = 0.101	C = 0.899	0.919
TNFSF8	rs1107372	Additive	A = 0.229	G = 0.771	0.391
TNFSF8	rs117198845	Additive	A = 0.015	G = 0.985	0.972
TNFSF8	rs117157109	Additive	A = 0.071	C = 0.929	0.960
TNFSF8	rs117592852	Dominant	T = 0.024	C = 0.976	0.966
TNFSF8	rs1006027	Dominant	C = 0.4	T = 0.6	0.469
<b>TNFSF8</b>	<b>rs10118244</b>	<b>Additive</b>	<b>T = 0.029</b>	<b>C = 0.971</b>	<b>0.027</b>
TNFSF8	rs13288168	Additive	T = 0.036	C = 0.964	0.972
TNFSF8	rs12377943	Recessive	G = 0.446	A = 0.554	0.936
TNFSF8	rs10982475	Additive	T = 0.091	C = 0.909	0.138
TNFSF8	rs4979487	Recessive	G = 0.295	A = 0.705	0.618
TNFSF8	rs2094792	Additive	T = 0.016	C = 0.984	0.961
TNFSF8	rs1330362	Additive	T = 0.056	C = 0.944	0.255
TNFSF8	rs7030090	Additive	T = 0.453	C = 0.547	0.465
TNFSF9	rs10415368	Dominant	C = 0.065	T = 0.935	0.637
TNFSF9	rs10422845	Additive	A = 0.447	G = 0.553	0.640
TNFSF9	rs2642212	Recessive	T = 0.392	C = 0.608	0.757
TNFSF9	rs348347	Dominant	A = 0.338	G = 0.662	0.120
TNFSF9	rs348373	Additive	T = 0.1	C = 0.9	0.308
TNFSF9	rs367527	Additive	A = 0.05	G = 0.95	0.106
TNFSF9	rs3865466	Recessive	G = 0.494	A = 0.506	0.134
TNFSF9	rs6510908	Recessive	A = 0.158	G = 0.842	0.348
TNFSF9	rs7508584	Dominant	A = 0.305	C = 0.695	0.865
TNFSF9	rs2910434	Dominant	G = 0.124	T = 0.876	0.964
TNFSF9	rs11085177	Dominant	A = 0.483	G = 0.517	0.737
TNFSF9	rs62106960	Additive	A = 0.061	C = 0.939	0.592
TNFSF9	rs62106961	Additive	T = 0.163	C = 0.837	0.230
TNFSF9	rs80345212	Additive	C = 0.045	A = 0.955	0.959
TNFSF9	rs72993079	Recessive	T = 0.065	C = 0.935	0.330
TNFSF9	rs12974682	Dominant	T = 0.48	C = 0.52	0.729
TNFSF9	rs76439627	Additive	T = 0.058	C = 0.942	0.228
TNFSF9	rs7257358	Additive	T = 0.062	C = 0.938	0.414
TNFSF9	rs72987334	Recessive	A = 0.03	G = 0.97	0.062
TNFSF9	rs76771492	Dominant	G = 0.047	T = 0.953	0.362
TNFSF9	rs11085178	Dominant	A = 0.349	G = 0.651	0.677
TNFSF9	rs348375	Dominant	G = 0.469	T = 0.531	0.267
TNFSF9	rs17703837	Recessive	C = 0.347	A = 0.653	0.535
TNFSF9	rs8104184	Additive	T = 0.044	C = 0.956	0.078

Genes	dbSNP.RS.ID	Mode of Inheritance	Minor Allele Frequency (MAF)	Major Allele Frequency	p-value
TNFSF9	rs2562020	Additive	C = 0.308	G = 0.692	0.135
TNFSF9	rs68037426	Additive	A = 0.083	G = 0.917	0.215
TNFSF9	rs10418366	Dominant	A = 0.392	C = 0.608	0.915
TNFSF9	rs117779462	Dominant	T = 0.021	C = 0.979	0.500
TNFSF9	rs78565612	Recessive	T = 0.026	C = 0.974	0.359
TNFSF9	rs348337	Dominant	T = 0.164	C = 0.836	0.214
TNFSF9	rs3097297	Additive	T = 0.052	C = 0.948	0.772
TNFSF9	rs348389	Dominant	T = 0.368	C = 0.632	0.229
TNFSF9	rs348376	Dominant	A = 0.453	G = 0.547	0.860
TNFSF9	rs2547448	Recessive	C = 0.382	T = 0.618	0.385
TNFSF9	rs7259857	Dominant	C = 0.493	T = 0.507	0.987
TNFSF9	rs441751	Dominant	T = 0.442	C = 0.558	0.837
<b>TNFSF9</b>	<b>rs1808398</b>	<b>Recessive</b>	<b>C = 0.282</b>	<b>T = 0.718</b>	<b>0.015</b>

**Supplementary Table 3:** Distribution of rs16972249 genotypes of cases and controls from all cohorts.

	Cases	Controls
Homozygous minor (AA)	344	7
Heterozygous (TA)	15	5
Homozygous major (TT)	7	1935

**Supplementary Table 4: MAF values of significant SNPs stratified by centre**

SNP ID	Cohort	Odds Ratio (OR)	Minor Allele Frequency (MAF)	Major Allele Frequency
rs16972249	PATH	37560.63	A = 0.214	T = 0.786
	BCGP	1295.24	A = 0.088	T = 0.912
rs8073937	PATH	1161504.00	A = 0.232	G = 0.768
	BCGP	64,386.27	A = 0.194	G = 0.806
AX.94361920	PATH	0.302	A = 1	C = 0
	BCGP	40,237.71	A = 0.128	C = 0.872
rs59119269	PATH	0.719	T = 0.086	C = 0.914
	BCGP	0.916	T = 0.094	C = 0.906