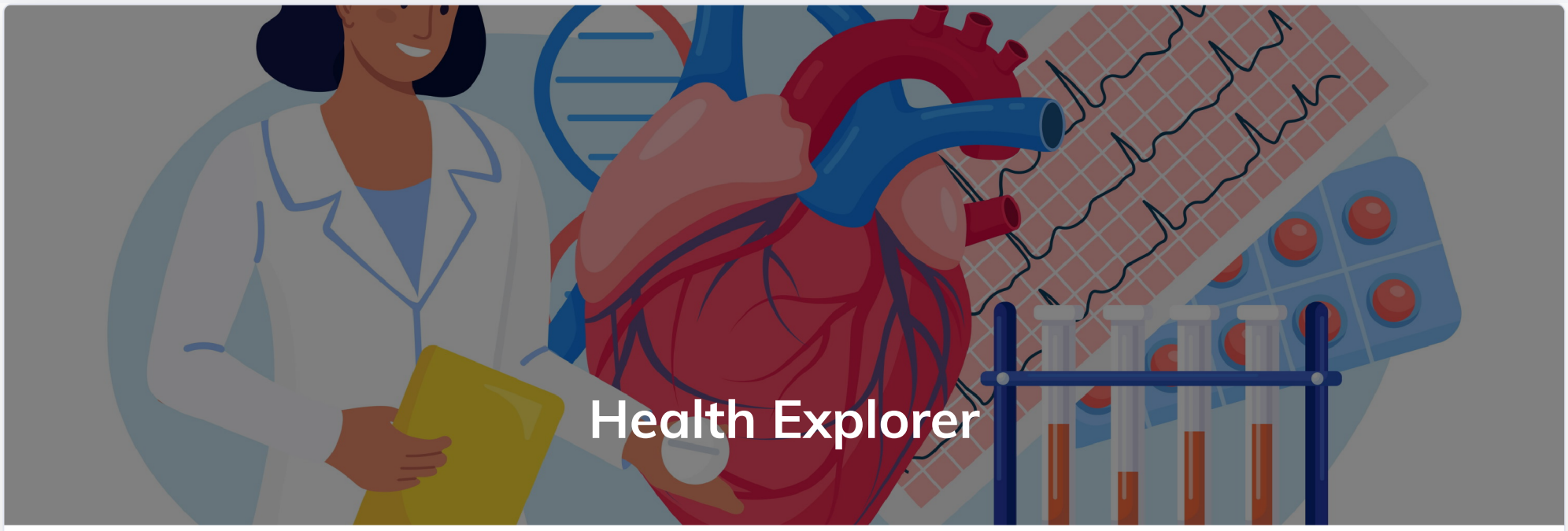


DEMO REPORT

Circulatory

Each body's arteries and veins transport blood in and out of the heart. Blood leaves the heart through the arteries and returns via the veins. Oxygen, nutrients, and hormones are delivered to cells through the circulatory system, eliminating waste materials like carbon dioxide. The heart (cardiovascular system), the lungs (pulmonary system), and the arteries, veins, coronary arteries, and portal arteries make up the circulatory system (systemic). Your circulatory system consists of the following components:

- The muscular heart is responsible for distributing blood throughout the body.
- The arteries, veins, and capillaries that make up your circulatory system.
- Platelets, plasma, and plasma-derived haemoglobin make up the majority of blood.

Disclaimer This report is intended as educational information. It is not intended to provide medical advice or be used solely by the customer in the diagnosis, cure, mitigation, treatment or prevention of disease. If you have any serious medical condition(s), including but not limited to, being over or under weight, or having diabetes or heart disease, you should not make any changes to your diet or exercise without consulting your doctor. Under no circumstances, should you make changes to your medication or other medical care without consulting your physician

Find more information at this link

Return to the health dashboard

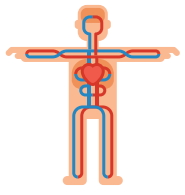
27 entries

Full View

Sort by risk | Sort by name

Abdominal aortic aneurysm

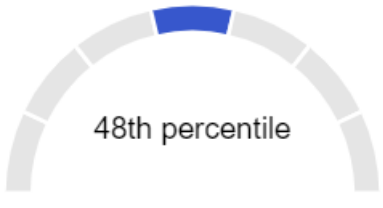
This result is based on **12 genetic variants** associated with **"Abdominal aortic aneurysm"** analyzed in the scientific paper **(01/20/2017 - Jones GT)**



Circulatory

Your results

Average genetic predisposition



Your genetic predisposition is lower than the 52% average person from your genetic population

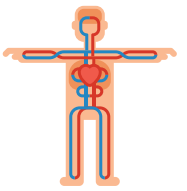
Study description

Abdominal aortic aneurysm (AAA) is a complex disease with both genetic and environmental risk factors. Together, 6 previously identified risk loci only explain a small proportion of the heritability of AAA.

Learn more

Atrial fibrillation

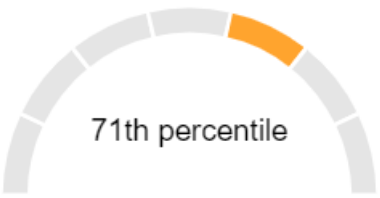
This result is based on **142 genetic variants** associated with **"Atrial fibrillation"** analyzed in the scientific paper **(2018 Sep - Nielsen JB)**



Circulatory

Your results

Above average genetic predisposition



Your genetic predisposition is higher than the 71% average person from your genetic population

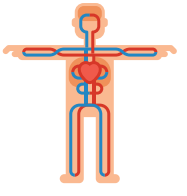
Study description

To identify genetic variation underlying atrial fibrillation, the most common cardiac arrhythmia, we performed a genome-wide association study of >1,000,000 people, including 60,620 atrial fibrillation cases and 970,216 controls. We identified 142 independent risk variants at 111 loci and prioritized 151 functional candidate genes likely to be involved in atrial fibrillation. Many of the identified risk variants fall near genes where more deleterious mutations have been reported to cause serious heart defects in humans (GATA4, MYH6, NKX2-5, PITX2, TBX5)1, or near genes important for striated muscle function and integrity (for example, CFL2, MYH7, PKP2, RBM20, SGCG, SSPN). Pathway and functional enrichment analyses also suggested that many of the putative atrial fibrillation genes act via cardiac structural remodeling, potentially in the form of an 'atrial cardiomyopathy'2, either during fetal heart development or as a response to stress in the adult heart.

Learn more

Behcet's disease

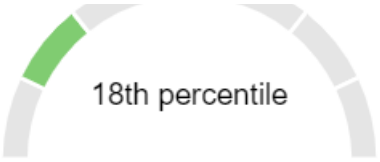
This result is based on **5 genetic variants** associated with **"Behcet's disease"** analyzed in the scientific paper **(2013 Feb - Kirino Y)**



Circulatory

Your results

Low genetic predisposition



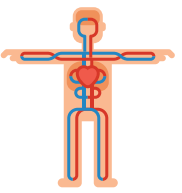
Your genetic predisposition is lower than the 82% average person from your genetic population

Study description

Individuals with Behçet's disease suffer from episodic inflammation often affecting the orogenital mucosa, skin and eyes. To discover new susceptibility loci for Behçet's disease, we performed a genome-wide association study (GWAS) of 779,465 SNPs with imputed genotypes in 1,209 Turkish individuals with Behçet's disease and 1,278 controls. We identified new associations at CCR1, STAT4 and KLRC4. Additionally, two SNPs in ERAP1, encoding ERAP1 p.Asp575Asn and p.Arg725Gln alterations, recessively conferred disease risk. These findings were replicated in 1,468 independent Turkish and/or 1,352 Japanese samples (combined meta-analysis P < 2 × 10(-9)). We also found evidence for interaction between HLA-B*51 and ERAP1 (P = 9 × 10(-4)). The CCR1 and STAT4 variants were associated with gene expression differences. Three risk loci shared with ankylosing spondylitis and psoriasis (the MHC class I region, ERAP1 and IL23R and the MHC class I-ERAP1 interaction), as well as two loci shared with inflammatory bowel disease (IL23R and IL10) implicate shared pathogenic pathways in the spondyloarthritides and Behçet's disease.

Learn more

Blood pressure

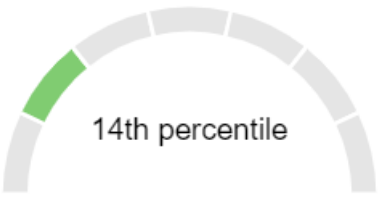


This result is based on **22 genetic variants** associated with **"Blood pressure"** analyzed in the scientific paper [(07/03/2014 - Simino J)]

Circulatory

Your results

Low genetic predisposition



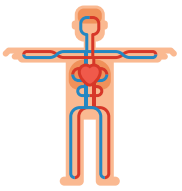
Your genetic predisposition is lower than the 86% average person from your genetic population

Study description

Although age-dependent effects on blood pressure (BP) have been reported, they have not been systematically investigated in large-scale genome-wide association studies (GWASs). We leveraged the infrastructure of three well-established consortia (CHARGE, GBPgen, and ICBP) and a nonstandard approach (age stratification and metaregression) to conduct a genome-wide search of common variants with age-dependent effects on systolic (SBP), diastolic (DBP), mean arterial (MAP), and pulse (PP) pressure. In a two-staged design using 99,241 individuals of European ancestry, we identified 20 genome-wide significant (p ≤ 5 × 10(-8)) loci by using joint tests of the SNP main effect and SNP-age interaction. Nine of the significant loci demonstrated nominal evidence of age-dependent effects on BP by tests of the interactions alone. Index SNPs in the EHBP1L1 (DBP and MAP), CASZ1 (SBP and MAP), and GOSR2 (PP) loci exhibited the largest age interactions, with opposite directions of effect in the young versus the old. The changes in the genetic effects over time were small but nonnegligible (up to 1.58 mm Hg over 60 years). The EHBP1L1 locus was discovered through gene-age interactions only in whites but had DBP main effects replicated (p = 8.3 × 10(-4)) in 8,682 Asians from Singapore, indicating potential interethnic heterogeneity. A secondary analysis revealed 22 loci with evidence of age-specific effects (e.g., only in 20 to 29-year-olds). Age can be used to select samples with larger genetic effect sizes and more homogenous phenotypes, which may increase statistical power. Age-dependent effects identified through novel statistical approaches can provide insight into the biology and temporal regulation underlying BP associations.

Learn more

Cardiovascular disease risk factors

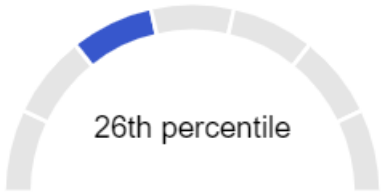


Circulatory

This result is based on **17 genetic variants** associated with **"Cardiovascular disease risk factors"** analyzed in the scientific paper [(09/24/2011 - Middelberg RP)]

Your results

Below average genetic predisposition



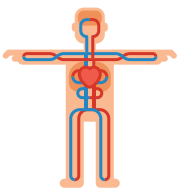
Your genetic predisposition is lower than the 74% average person from your genetic population

Study description

Genome-wide association studies (GWAS) have become a major strategy for genetic dissection of human complex diseases. Analysing multiple phenotypes jointly may improve both our ability to detect genetic variants with multiple effects and our understanding of their common features. Allelic associations for multiple biochemical traits (serum alanine aminotransferase, aspartate aminotransferase, butrylycholinesterase (BCHE), C-reactive protein (CRP), ferritin, gamma glutamyltransferase (GGT), glucose, high-density lipoprotein cholesterol (HDL), insulin, low-density lipoprotein cholesterol (LDL), triglycerides and uric acid), and body-mass index, were examined.

Learn more

Diastolic blood pressure

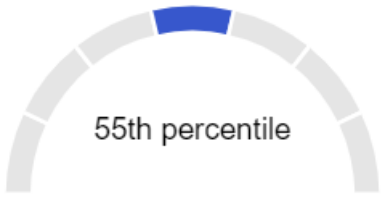


This result is based on **192 genetic variants** associated with **"Diastolic blood pressure"** analyzed in the scientific paper [(2018 Oct - Evangelou E)]

Circulatory

Your results

Average genetic predisposition

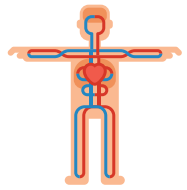


Your genetic predisposition is higher than the 55% average person from your genetic population

Study description

High blood pressure is a highly heritable and modifiable risk factor for cardiovascular disease. We report the largest genetic association study of blood pressure traits (systolic, diastolic and pulse pressure) to date in over 1 million people of European ancestry. We identify 535 novel blood pressure loci that not only offer new biological insights into blood pressure regulation but also highlight shared genetic architecture between blood pressure and lifestyle exposures. Our findings identify new biological pathways for blood pressure regulation with potential for improved cardiovascular disease prevention in the future.

Learn more



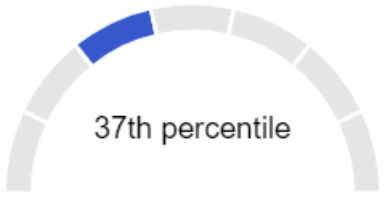
Circulatory

End-stage coagulation

This result is based on **23 genetic variants** associated with **"End-stage coagulation"** analyzed in the scientific paper [(2013 Jan - Williams FM)]

Your results

Below average genetic predisposition



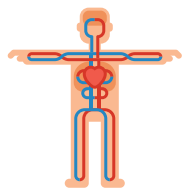
Your genetic predisposition is lower than the 63% average person from your genetic population

Study description

End-stage coagulation and the structure/function of fibrin are implicated in the pathogenesis of ischemic stroke. We explored whether genetic variants associated with end-stage coagulation in healthy volunteers account for the genetic predisposition to ischemic stroke and examined their influence on stroke subtype.

Learn more

Factor VIII levels

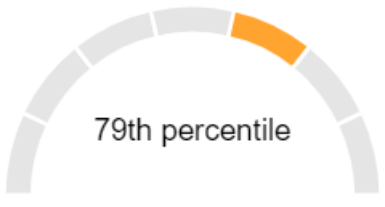


This result is based on **38 genetic variants** associated with **"Factor VIII levels"** analyzed in the scientific paper [(01/29/2019 - Sabater-Lleal M)]

Circulatory

Your results

Above average genetic predisposition



Your genetic predisposition is higher than the 79% average person from your genetic population

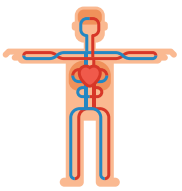
Study description

Factor VIII (FVIII) and its carrier protein von Willebrand factor (VWF) are associated with risk of arterial and venous thrombosis and with hemorrhagic disorders. We aimed to identify and functionally test novel genetic associations regulating plasma FVIII and VWF.

Learn more

Heart rate

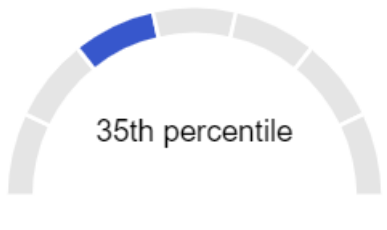
This result is based on **32 genetic variants** associated with **"Heart rate"** analyzed in the scientific paper (2013 Jun - den Hoed M)



Circulatory

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 65% average person from your genetic population

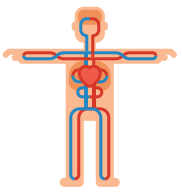
Study description

Elevated resting heart rate is associated with greater risk of cardiovascular disease and mortality. In a 2-stage meta-analysis of genome-wide association studies in up to 181,171 individuals, we identified 14 new loci associated with heart rate and confirmed associations with all 7 previously established loci. Experimental downregulation of gene expression in Drosophila melanogaster and Danio rerio identified 20 genes at 11 loci that are relevant for heart rate regulation and highlight a role for genes involved in signal transmission, embryonic cardiac development and the pathophysiology of dilated cardiomyopathy, congenital heart failure and/or sudden cardiac death. In addition, genetic susceptibility to increased heart rate is associated with altered cardiac conduction and reduced risk of sick sinus syndrome, and both heart rate-increasing and heart rate-decreasing variants associate with risk of atrial fibrillation. Our findings provide fresh insights into the mechanisms regulating heart rate and identify new therapeutic targets.

Learn more

Hypertension

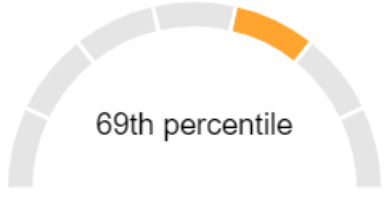
This result is based on **2 genetic variants** associated with **"Hypertension"** analyzed in the scientific paper (06/07/2007 -)



Circulatory

Your results

Above average genetic predisposition



Your genetic predisposition is higher than the 69% average person from your genetic population

Study description

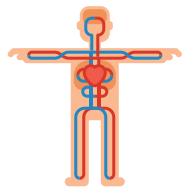
There is increasing evidence that genome-wide association (GWA) studies represent a powerful approach to the identification of genes involved in common human diseases. We describe a joint GWA study (using the Affymetrix

GeneChip 500K Mapping Array Set) undertaken in the British population, which has examined approximately 2,000 individuals for each of 7 major diseases and a shared set of approximately 3,000 controls. Case-control comparisons identified 24 independent association signals at P < 5 x 10(-7): 1 in bipolar disorder, 1 in coronary artery disease, 9 in Crohn's disease, 3 in rheumatoid arthritis, 7 in type 1 diabetes and 3 in type 2 diabetes. On the basis of prior findings and replication studies thus-far completed, almost all of these signals reflect genuine susceptibility effects. We observed association at many previously identified loci, and found compelling evidence that some loci confer risk for more than one of the diseases studied. Across all diseases, we identified a large number of further signals (including 58 loci with single-point P values between 10(-5) and 5 x 10(-7)) likely to yield additional susceptibility loci. The importance of appropriately large samples was confirmed by the modest effect sizes observed at most loci identified. This study thus represents a thorough validation of the GWA approach. It has also demonstrated that careful use of a shared control group represents a safe and effective approach to GWA analyses of multiple disease phenotypes; has generated a genome-wide genotype database for future studies of common diseases in the British population; and shown that, provided individuals with non-European ancestry are excluded, the extent of population stratification in the British population is generally modest. Our findings offer new avenues for exploring the pathophysiology of these important disorders. We anticipate that our data, results and software, which will be widely available to other investigators, will provide a powerful resource for human genetics research.

Learn more

Mean arterial pressure

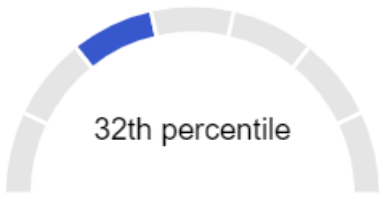
This result is based on **146 genetic variants** associated with **"Mean arterial pressure"** analyzed in the scientific paper **(11/28/2018 - Takeuchi F)**



Circulatory

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 68% average person from your genetic population

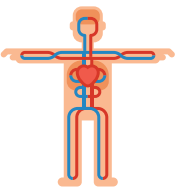
Study description

Blood pressure (BP) is a major risk factor for cardiovascular disease and more than 200 genetic loci associated with BP are known. Here, we perform a multi-stage genome-wide association study for BP (max N = 289,038) principally in East Asians and meta-analysis in East Asians and Europeans. We report 19 new genetic loci and ancestry-specific BP variants, conforming to a common ancestry-specific variant association model. At 10 unique loci, distinct non-rare ancestry-specific variants colocalize within the same linkage disequilibrium block despite the significantly discordant effects for the proxy shared variants between the ethnic groups. The genome-wide transethnic correlation of causal-variant effect-sizes is 0.898 and 0.851 for systolic and diastolic BP, respectively. Some of the ancestry-specific association signals are also influenced by a selective sweep. Our results provide new evidence for the role of common ancestry-specific variants and natural selection in ethnic differences in complex traits such as BP.

Learn more

Metabolic syndrome

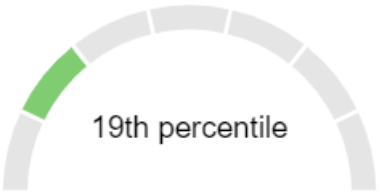
This result is based on **93 genetic variants** associated with **"Metabolic syndrome"** analyzed in the scientific paper [(2019 Dec - Lind L)]



Circulatory

Your results

Low genetic predisposition



Your genetic predisposition is lower than the 81% average person from your genetic population

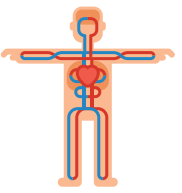
Study description

Background: The metabolic syndrome (MetS) is a description of a clustering of cardiometabolic risk factors in the same individual. Previous genome-wide association studies (GWASs) have identified 29 independent genetic loci linked to MetS as a binary trait. This study used data from UK biobank to search for additional loci. Methods: Using data from 291,107 individuals in the UK biobank, a GWAS was performed versus the binary trait MetS (harmonized NCEP criteria). Results: In a GWAS of MetS (binary) we found 93 independent loci with P < 5 × 10-8, of which 80 were not identified in previous GWASs of MetS. However, the majority of those loci have previously been associated with one or more of the five MetS components. Of particular interest are the genes being related to MetS (binary) in this study, but not to any of the MetS components in past studies, such as WDR48, KLF14, NAADL1, GADD45G, and OR5R1, as well as the two loci that have been associated with all five MetS components in past studies, SNX10 and C5orf67. A pathway analysis of the 93 independent loci showed the immune system, transportation of small molecules, and metabolism to be enriched. Conclusion: This GWAS of the MetS in UK biobank identified several new loci being associated with MetS. Most of those have previously been found to be associated with different components of MetS, but several loci were found not previously linked to cardiometabolic disease.

Learn more

Mitral valve prolapse

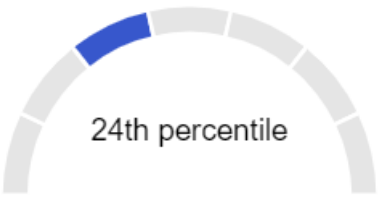
This result is based on **8 genetic variants** associated with **"Mitral valve prolapse"** analyzed in the scientific paper [(2015 Oct - Dina C)]



Circulatory

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 76% average person from your genetic population

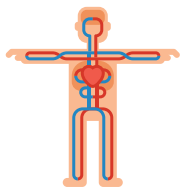
Study description

Nonsyndromic mitral valve prolapse (MVP) is a common degenerative cardiac valvulopathy of unknown etiology that predisposes to mitral regurgitation, heart failure and sudden death. Previous family and pathophysiological studies suggest a complex pattern of inheritance. We performed a meta-analysis of 2 genome-wide association studies in 1,412 MVP cases and 2,439 controls. We identified 6 loci, which we replicated in 1,422 cases and 6,779 controls, and provide functional evidence for candidate genes. We highlight LMCD1 (LIM and cysteine-rich domains 1), which encodes a transcription factor and for which morpholino knockdown of the ortholog in zebrafish resulted in atrioventricular valve regurgitation. A similar zebrafish phenotype was obtained with knockdown of the ortholog of TNS1, which encodes tensin

1, a focal adhesion protein involved in cytoskeleton organization. We also showed expression of tensin 1 during valve morphogenesis and describe enlarged posterior mitral leaflets in Tns1(-/-) mice. This study identifies the first risk loci for MVP and suggests new mechanisms involved in mitral valve regurgitation, the most common indication for mitral valve repair.

Learn more

P wave duration

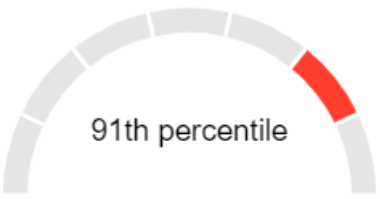


Circulatory

This result is based on **15 genetic variants** associated with **"P wave duration"** analyzed in the scientific paper **(2017 Aug - Christophersen IE)**

Your results

High genetic predisposition



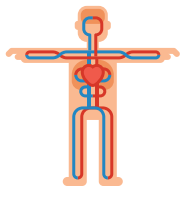
Your genetic predisposition is higher than the 91% average person from your genetic population

Study description

The P wave on an ECG is a measure of atrial electric function, and its characteristics may serve as predictors for atrial arrhythmias. Increased mean P-wave duration and P-wave terminal force traditionally have been used as markers for left atrial enlargement, and both have been associated with increased risk of atrial fibrillation. Here, we explore the genetic basis of P-wave morphology through meta-analysis of genome-wide association study results for P-wave duration and P-wave terminal force from 12 cohort studies.

Learn more

Peripheral artery disease

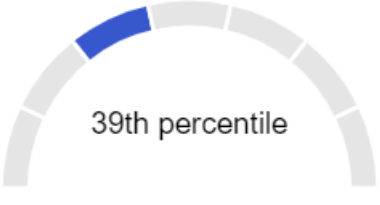


Circulatory

This result is based on **20 genetic variants** associated with **"Peripheral artery disease"** analyzed in the scientific paper **(2019 Aug - Klarin D)**

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 61% average person from your genetic population

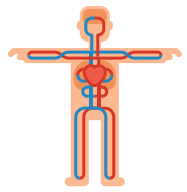
Study description

Peripheral artery disease (PAD) is a leading cause of cardiovascular morbidity and mortality; however, the extent to which genetic factors increase risk for PAD is largely unknown. Using electronic health record data, we performed a genome-wide association study in the Million Veteran Program testing ~32 million DNA sequence variants with PAD (31,307 cases and 211,753 controls) across veterans of European, African and Hispanic ancestry. The results were replicated in an independent sample of 5,117 PAD cases and 389,291 controls from the UK Biobank. We identified 19 PAD loci, 18 of which have not been previously reported. Eleven of the 19 loci were associated with disease in three vascular beds (coronary, cerebral, peripheral), including LDLR, LPL and LPA, suggesting that therapeutic modulation of low-density lipoprotein cholesterol, the lipoprotein lipase pathway or circulating lipoprotein(a) may be efficacious for multiple atherosclerotic disease phenotypes. Conversely, four of the variants appeared to be specific for PAD, including F5 p.R506Q, highlighting the pathogenic role of thrombosis in the peripheral vascular bed and providing genetic support for Factor Xa inhibition as a therapeutic strategy for PAD. Our results highlight mechanistic similarities and differences among coronary, cerebral and peripheral atherosclerosis and provide therapeutic insights.

Learn more

PR interval

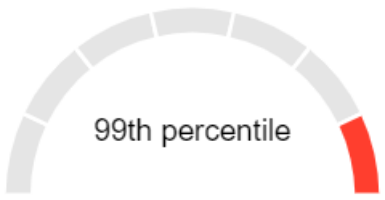
This result is based on **266 genetic variants** associated with **"PR interval"** analyzed in the scientific paper (05/21/2020 - Ntalla I)



Circulatory

Your results

Very high genetic predisposition



Your genetic predisposition is higher than the 99% average person from your genetic population

Study description

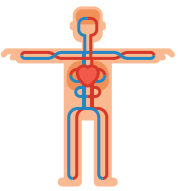
The electrocardiographic PR interval reflects atrioventricular conduction, and is associated with conduction abnormalities, pacemaker implantation, atrial fibrillation (AF), and cardiovascular mortality. Here we report a multi-ancestry (N = 293,051) genome-wide association meta-analysis for the PR interval, discovering 202 loci of which 141 have not previously been reported. Variants at identified loci increase the percentage of heritability explained, from 33.5% to 62.6%. We observe enrichment for cardiac muscle developmental/contractile and cytoskeletal genes, highlighting key regulation processes for atrioventricular conduction. Additionally, 8 loci not previously reported harbor genes underlying inherited arrhythmic syndromes and/or cardiomyopathies suggesting a role for these genes in cardiovascular pathology in the general population. We show that polygenic predisposition to PR interval duration is an endophenotype for cardiovascular disease, including distal conduction disease, AF, and atrioventricular pre-excitation. These findings advance our understanding of the polygenic basis of cardiac conduction, and the genetic relationship between PR interval duration and cardiovascular disease.

Learn more

Pulse pressure

Pulse pressure

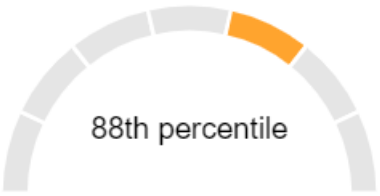
This result is based on **149 genetic variants** associated with **"Pulse pressure"** analyzed in the scientific paper [(2018 Oct - Evangelou E)]



Circulatory

Your results

Above average genetic predisposition



Your genetic predisposition is higher than the 88% average person from your genetic population

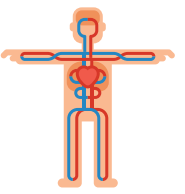
Study description

High blood pressure is a highly heritable and modifiable risk factor for cardiovascular disease. We report the largest genetic association study of blood pressure traits (systolic, diastolic and pulse pressure) to date in over 1 million people of European ancestry. We identify 535 novel blood pressure loci that not only offer new biological insights into blood pressure regulation but also highlight shared genetic architecture between blood pressure and lifestyle exposures. Our findings identify new biological pathways for blood pressure regulation with potential for improved cardiovascular disease prevention in the future.

Learn more

QT dynamics during exercise

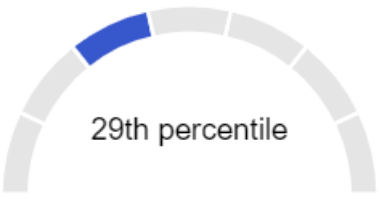
This result is based on **19 genetic variants** associated with **"QT dynamics during exercise"** analyzed in the scientific paper [(2020 Aug - van Duijvenboden S)]



Circulatory

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 71% average person from your genetic population

Study description

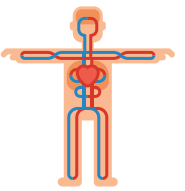
Abnormal QT interval responses to heart rate (QT dynamics) is an independent risk predictor for cardiovascular disease in patients, but its genetic basis and prognostic value in a population-based cohort have not been investigated.

Learn more

QT interval

QT interval

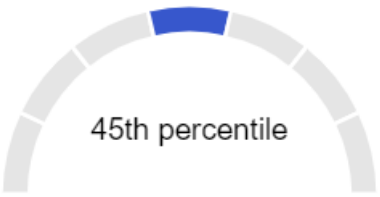
This result is based on **61 genetic variants** associated with **"QT interval"** analyzed in the scientific paper [(12/06/2017 - Méndez-Giráldez R)]



Circulatory

Your results

Average genetic predisposition



Your genetic predisposition is lower than the 55% average person from your genetic population

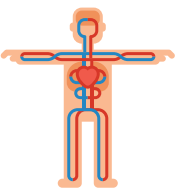
Study description

QT interval prolongation is a heritable risk factor for ventricular arrhythmias and can predispose to sudden death. Most genome-wide association studies (GWAS) of QT were performed in European ancestral populations, leaving other groups uncharacterized. Herein we present the first QT GWAS of Hispanic/Latinos using data on 15,997 participants from four studies. Study-specific summary results of the association between 1000 Genomes Project (1000G) imputed SNPs and electrocardiographically measured QT were combined using fixed-effects meta-analysis. We identified 41 genome-wide significant SNPs that mapped to 13 previously identified QT loci. Conditional analyses distinguished six secondary signals at NOS1AP (n = 2), ATP1B1 (n = 2), SCN5A (n = 1), and KCNQ1 (n = 1). Comparison of linkage disequilibrium patterns between the 13 lead SNPs and six secondary signals with previously reported index SNPs in 1000G super populations suggested that the SCN5A and KCNE1 lead SNPs were potentially novel and population-specific. Finally, of the 42 suggestively associated loci, AJAP1 was suggestively associated with QT in a prior East Asian GWAS; in contrast BVES and CAP2 murine knockouts caused cardiac conduction defects. Our results indicate that whereas the same loci influence QT across populations, population-specific variation exists, motivating future trans-ethnic and ancestrally diverse QT GWAS.

Learn more

Resting heart rate

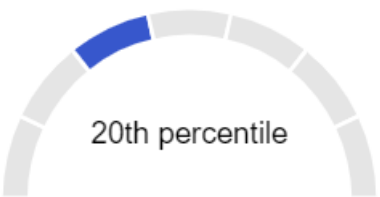
This result is based on **30 genetic variants** associated with **"Resting heart rate"** analyzed in the scientific paper [(05/16/2018 - Ramírez J)]



Circulatory

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 80% average person from your genetic population

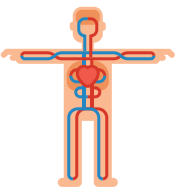
Study description

Impaired capacity to increase heart rate (HR) during exercise (ΔHRex), and a reduced rate of recovery post-exercise (ΔHRrec) are associated with higher cardiovascular mortality rates. Currently, the genetic basis of both phenotypes remains to be elucidated. We conduct genome-wide association studies (GWASs) for ΔHRex and ΔHRrec in ~40,000 individuals, followed by replication in ~27,000 independent samples, all from UK Biobank. Six and seven single-nucleotide polymorphisms for ΔHRex and ΔHRrec, respectively, formally replicate. In a full data set GWAS, eight further loci for ΔHRex and nine for ΔHRrec are genome-wide significant ($P \leq 5 \times 10\text{-}8$). In total, 30 loci are discovered, 8 being common

across traits. Processes of neural development and modulation of adrenergic activity by the autonomic nervous system are enriched in these results. Our findings reinforce current understanding of HR response to exercise and recovery and could guide future studies evaluating its contribution to cardiovascular risk prediction.

Learn more

Small vessel stroke

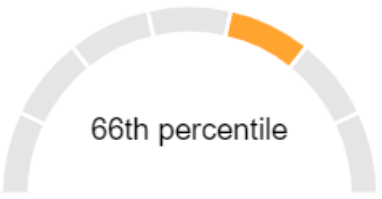


Circulatory

This result is based on **7 genetic variants** associated with **"Small vessel stroke"** analyzed in the scientific paper **(11/09/2017 - Lee TH)**

Your results

Above average genetic predisposition



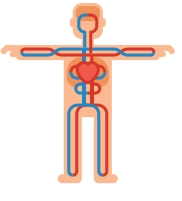
Your genetic predisposition is higher than the 66% average person from your genetic population

Study description

Genome-wide association studies (GWAS) can serve as strong evidence in correlating biological pathways with human diseases. Although ischemic stroke has been found to be associated with many biological pathways, the genetic mechanism of ischemic stroke is still unclear. Here, we performed GWAS for a major subtype of stroke-small-vessel occlusion (SVO)-to identify potential genetic factors contributing to ischemic stroke. GWAS were conducted on 342 individuals with SVO stroke and 1,731 controls from a Han Chinese population residing in Taiwan. The study was replicated in an independent Han Chinese population comprising an additional 188 SVO stroke cases and 1,265 controls. Three SNPs (rs2594966, rs2594973, rs4684776) clustered at 3p25.3 in ATG7 (encoding Autophagy Related 7), with P values between 2.52 × 10-6 and 3.59 × 10-6, were identified. Imputation analysis also supported the association between ATG7 and SVO stroke. To our knowledge, this is the first GWAS to link stroke and autophagy. ATG7, which has been implicated in autophagy, could provide novel insights into the genetic basis of ischemic stroke.

Learn more

Stroke

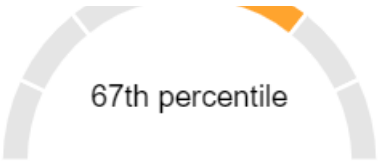


Circulatory

This result is based on **20 genetic variants** associated with **"Stroke"** analyzed in the scientific paper **(2015 Aug - Carty CL)**

Your results

Above average genetic predisposition



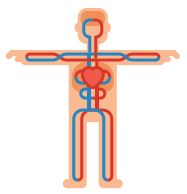
Your genetic predisposition is higher than the 67% average person from your genetic population

Study description

Background and The majority of genome-wide association studies (GWAS) of stroke have focused on European-ancestry populations; however, none has been conducted in African Americans, despite the disproportionately high burden of stroke in this population. The Consortium of Minority Population Genome-Wide Association Studies of Stroke (COMPASS) was established to identify stroke susceptibility loci in minority populations.

Learn more

Systolic blood pressure

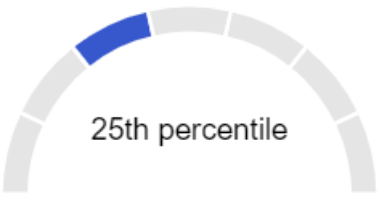


Circulatory

This result is based on **188 genetic variants** associated with **"Systolic blood pressure"** analyzed in the scientific paper **(2018 Oct - Evangelou E)**

Your results

Below average genetic predisposition



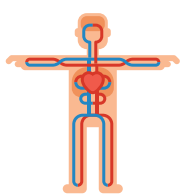
Your genetic predisposition is lower than the 75% average person from your genetic population

Study description

High blood pressure is a highly heritable and modifiable risk factor for cardiovascular disease. We report the largest genetic association study of blood pressure traits (systolic, diastolic and pulse pressure) to date in over 1 million people of European ancestry. We identify 535 novel blood pressure loci that not only offer new biological insights into blood pressure regulation but also highlight shared genetic architecture between blood pressure and lifestyle exposures. Our findings identify new biological pathways for blood pressure regulation with potential for improved cardiovascular disease prevention in the future.

Learn more

Takayasu arteritis

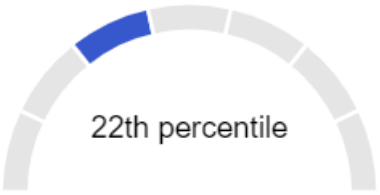


Circulatory

This result is based on **7 genetic variants** associated with **"Takayasu arteritis"** analyzed in the scientific paper **(12/18/2018 - Terao C)**

Your results

Below average genetic predisposition



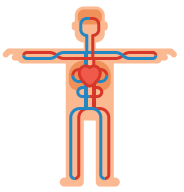
Your genetic predisposition is lower than the 78% average person from your genetic population

Study description

Takayasu arteritis (TAK) is a systemic vasculitis with severe complications that affects the aorta and its large branches. HLA-B*52 is an established susceptibility locus to TAK. To date, there are still only a limited number of reports concerning non-HLA susceptibility loci to TAK. We conducted a genome-wide association study (GWAS) and a follow-up study in a total of 633 TAK cases and 5,928 controls. A total of 510,879 SNPs were genotyped, and 5,875,450 SNPs were imputed together with HLA-B*52. Functional annotation of significant loci, enhancer enrichment, and pathway analyses were conducted. We identified four unreported significant loci, namely rs2322599, rs103294, rs17133698, and rs1713450, in PTK2B, LILRA3/LILRB2, DUSP22, and KLHL33, respectively. Two additional significant loci unreported in non-European GWAS were identified, namely HSPA6/FCGR3A and chr21q.22. We found that a single variant associated with the expression of MICB, a ligand for natural killer (NK) cell receptor, could explain the entire association with the HLA-B region. Rs2322599 is strongly associated with the expression of PTK2B Rs103294 risk allele in LILRA3/LILRB2 is known to be a tagging SNP for the deletion of LILRA3, a soluble receptor of HLA class I molecules. We found a significant epistasis effect between HLA-B*52 and rs103294 (P = 1.2 × 10-3). Enhancer enrichment analysis and pathway analysis suggested the involvement of NK cells (P = 8.8 × 10-5, enhancer enrichment). In conclusion, four unreported TAK susceptibility loci and an epistasis effect between LILRA3 and HLA-B*52 were identified. HLA and non-HLA regions suggested a critical role for NK cells in TAK.

Learn more

Tetralogy of Fallot

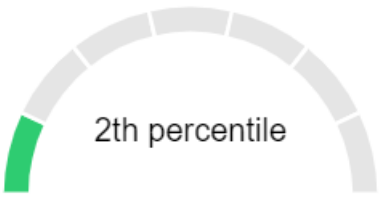


Circulatory

This result is based on **6 genetic variants** associated with **"Tetralogy of Fallot"** analyzed in the scientific paper **(04/01/2013 - Cordell HJ)**

Your results

Very low genetic predisposition



Your genetic predisposition is lower than the 98% average person from your genetic population

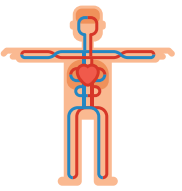
Study description

We conducted a genome-wide association study to search for risk alleles associated with Tetralogy of Fallot (TOF), using a northern European discovery set of 835 cases and 5159 controls. A region on chromosome 12q24 was associated (P = 1.4 × 10(-7)) and replicated convincingly (P = 3.9 × 10(-5)) in 798 cases and 2931 controls [per allele odds ratio (OR) = 1.27 in replication cohort, P = 7.7 × 10(-11) in combined populations]. Single nucleotide polymorphisms in the glypican 5 gene on chromosome 13q32 were also associated (P = 1.7 × 10(-7)) and replicated convincingly (P = 1.2 × 10(-5)) in 789 cases and 2927 controls (per allele OR = 1.31 in replication cohort, P = 3.03 × 10(-11) in combined populations). Four additional regions on chromosomes 10, 15 and 16 showed suggestive association accompanied by nominal replication. This study, the first genome-wide association study of a congenital heart malformation phenotype, provides evidence that common genetic variation influences the risk of TOF.

Learn more

Learn more

Thrombosis

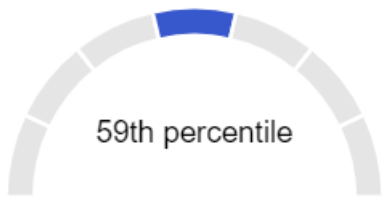


Circulatory

This result is based on **13 genetic variants** associated with **"Thrombosis"** analyzed in the scientific paper **(05/01/2016 - Hinds DA)**

Your results

Average genetic predisposition



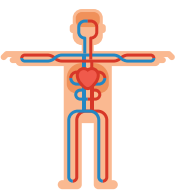
Your genetic predisposition is higher than the 59% average person from your genetic population

Study description

Thrombotic diseases are among the leading causes of morbidity and mortality in the world. To add insights into the genetic regulation of thrombotic disease, we conducted a genome-wide association study (GWAS) of 6135 self-reported blood clots events and 252 827 controls of European ancestry belonging to the 23andMe cohort of research participants. Eight loci exceeded genome-wide significance. Among the genome-wide significant results, our study replicated previously known venous thromboembolism (VTE) loci near the F5, FGA-FGG, F11, F2, PROCR and ABO genes, and the more recently discovered locus near SLC44A2 In addition, our study reports for the first time a genome-wide significant association between rs114209171, located upstream of the F8 structural gene, and thrombosis risk. Analyses of expression profiles and expression quantitative trait loci across different tissues suggested SLC44A2, ILF3 and AP1M2 as the three most plausible candidate genes for the chromosome 19 locus, our only genome-wide significant thrombosis-related locus that does not harbor likely coagulation-related genes. In addition, we present data showing that this locus also acts as a novel risk factor for stroke and coronary artery disease (CAD). In conclusion, our study reveals novel common genetic risk factors for VTE, stroke and CAD and provides evidence that self-reported data on blood clots used in a GWAS yield results that are comparable with those obtained using clinically diagnosed VTE. This observation opens up the potential for larger meta-analyses, which will enable elucidation of the genetics of thrombotic diseases, and serves as an example for the genetic study of other diseases.

Learn more

Venous thromboembolism

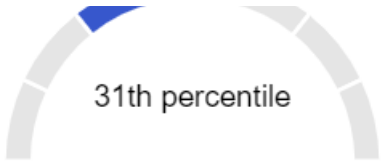


Circulatory

This result is based on **132 genetic variants** associated with **"Venous thromboembolism"** analyzed in the scientific paper **(11/07/2019 - Lindström S)**

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 69% average person from your genetic population

Study description

Venous thromboembolism (VTE) is a significant contributor to morbidity and mortality. To advance our understanding of the biology contributing to VTE, we conducted a genome-wide association study (GWAS) of VTE and a transcriptome-wide association study (TWAS) based on imputed gene expression from whole blood and liver. We meta-analyzed GWAS data from 18 studies for 30 234 VTE cases and 172 122 controls and assessed the association between 12 923 718 genetic variants and VTE. We generated variant prediction scores of gene expression from whole blood and liver tissue and assessed them for association with VTE. Mendelian randomization analyses were conducted for traits genetically associated with novel VTE loci. We identified 34 independent genetic signals for VTE risk from GWAS meta-analysis, of which 14 are newly reported associations. This included 11 newly associated genetic loci (C1orf198, PLEK, OSMR-AS1, NUGGC/SCARA5, GRK5, MPHOSPH9, ARID4A, PLCG2, SMG6, EIF5A, and STX10) of which 6 replicated, and 3 new independent signals in 3 known genes. Further, TWAS identified 5 additional genetic loci with imputed gene expression levels differing between cases and controls in whole blood (SH2B3, SPSB1, RP11-747H7.3, RP4-737E23.2) and in liver (ERAP1). At some GWAS loci, we found suggestive evidence that the VTE association signal for novel and previously known regions colocalized with expression quantitative trait locus signals. Mendelian randomization analyses suggested that blood traits may contribute to the underlying risk of VTE. To conclude, we identified 16 novel susceptibility loci for VTE; for some loci, the association signals are likely mediated through gene expression of nearby genes.

Learn more