DEMO REPORT

Nervous

A structured set of cells in the nervous system dedicated to transmitting electrical inputs from sensory receptors to the region where a reaction occurs through a network. Diffuse and centralized nervous systems are the two main kinds. Lower invertebrates have diffuse systems, which do not have brains and have their neurons dispersed throughout the body in a netlike way. A part of the nervous system has a prominent role in organizing information and steering responses in higher invertebrate and vertebrate central nervous systems. All of your body's functions are controlled by your neurological system. Your brain governs your actions, thoughts and reactions to the environment around you automatically. Additionally, it regulates the body's digestive, respiratory, and sexual systems (puberty).

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

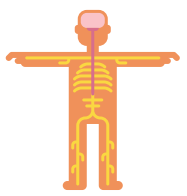
18 entries

Full View

Sort by risk | Sort by name

Anorexia nervosa

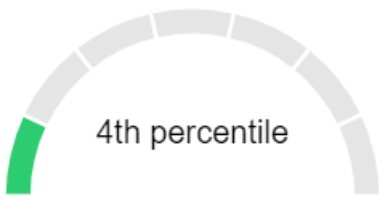
This result is based on **6 genetic variants** associated with **"Anorexia nervosa"** analyzed in the scientific paper **(09/01/2017 - Duncan L)**



Nervous

Your results

Very low genetic predisposition



Your genetic predisposition is lower than the 96% average person from your genetic population

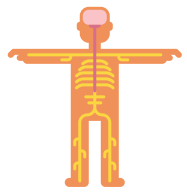
Study description

The authors conducted a genome-wide association study of anorexia nervosa and calculated genetic correlations with a series of psychiatric, educational, and metabolic phenotypes.

Learn more

Epilepsy

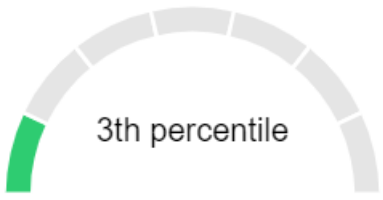
This result is based on **9 genetic variants** associated with **"Epilepsy"** analyzed in the scientific paper (2014 Sep -)



Nervous

Your results

Very low genetic predisposition



Your genetic predisposition is lower than the 97% average person from your genetic population

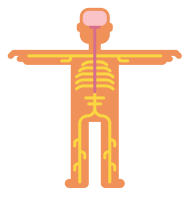
Study description

The epilepsies are a clinically heterogeneous group of neurological disorders. Despite strong evidence for heritability, genome-wide association studies have had little success in identification of risk loci associated with epilepsy, probably because of relatively small sample sizes and insufficient power. We aimed to identify risk loci through meta-analyses of genome-wide association studies for all epilepsy and the two largest clinical subtypes (genetic generalised epilepsy and focal epilepsy).

Learn more

Parkinson's disease

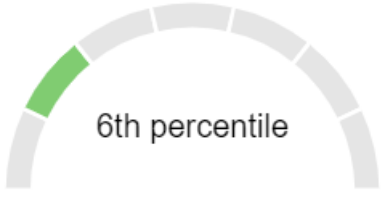
This result is based on **55 genetic variants** associated with **"Parkinson's disease"** analyzed in the scientific paper (2017 Oct - Chang D)



Nervous

Your results

Low genetic predisposition



Your genetic predisposition is lower than the 94% average person from your genetic population

Study description

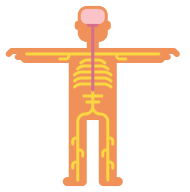
Study description

Common variant genome-wide association studies (GWASs) have, to date, identified >24 risk loci for Parkinson's disease (PD). To discover additional loci, we carried out a GWAS comparing 6,476 PD cases with 302,042 controls, followed by a meta-analysis with a recent study of over 13,000 PD cases and 95,000 controls at 9,830 overlapping variants. We then tested 35 loci (P < 1 × 10-6) in a replication cohort of 5,851 cases and 5,866 controls. We identified 17 novel risk loci (P < 5 × 10-8) in a joint analysis of 26,035 cases and 403,190 controls. We used a neurocentric strategy to assign candidate risk genes to the loci. We identified protein-altering or cis-expression quantitative trait locus (cis-eQTL) variants in linkage disequilibrium with the index variant in 29 of the 41 PD loci. These results indicate a key role for autophagy and lysosomal biology in PD risk, and suggest potential new drug targets for PD.

Learn more

Amyotrophic lateral sclerosis

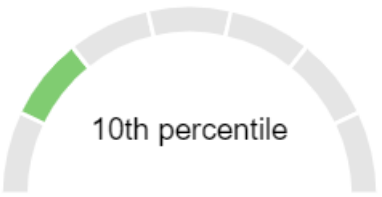
This result is based on **15 genetic variants** associated with **"Amyotrophic lateral sclerosis"** analyzed in the scientific paper [(2013 Jan - Ahmeti KB)]



Nervous

Your results

Low genetic predisposition



Your genetic predisposition is lower than the 90% average person from your genetic population

Study description

Amyotrophic lateral sclerosis (ALS) is the third most common adult-onset neurodegenerative disease. Individuals with ALS rapidly progress to paralysis and die from respiratory failure within 3 to 5 years after symptom onset. Epidemiological factors explain only a modest amount of the risk for ALS. However, there is growing evidence of a strong genetic component to both familial and sporadic ALS risk. The International Consortium on Amyotrophic Lateral Sclerosis Genetics was established to bring together existing genome-wide association cohorts and identify sporadic ALS susceptibility and age at symptom onset loci. Here, we report the results of a meta-analysis of the International Consortium on Amyotrophic Lateral Sclerosis Genetics genome-wide association samples, consisting of 4243 ALS cases and 5112 controls from 13 European ancestry cohorts from across the United States and Europe. Eight genomic regions provided evidence of association with ALS, including 9p21.2 (rs3849942, odds ratio [OR] = 1.21; p = 4.41 × 10(-7)), 17p11.2 (rs7477, OR = 1.30; p = 2.89 × 10(-7)), and 19p13 (rs12608932, OR = 1.37, p = 1.29 × 10(-7)). Six genomic regions were associated with age at onset of ALS. The strongest evidence for an age of onset locus was observed at 1p34.1, with comparable evidence at rs3011225 (R(2)(partial) = 0.0061; p = 6.59 × 10(-8)) and rs803675 (R(2)(partial) = 0.0060; p = 6.96 × 10(-8)). These associations were consistent across all 13 cohorts. For rs3011225, individuals with at least 1 copy of the minor allele had an earlier average age of onset of over 2 years. Identifying the underlying pathways influencing susceptibility to and age at onset of ALS may provide insight into the pathogenic mechanisms and motivate new pharmacologic targets for this fatal neurodegenerative disease.

Learn more

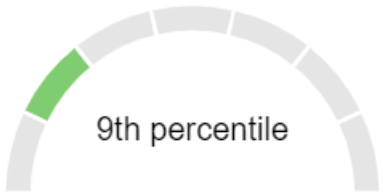
Migraine

Nervous

This result is based on **44 genetic variants** associated with **"Migraine"** analyzed in the scientific paper [(2016 Aug - Gormley P)]

Your results

Low genetic predisposition



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

Study description

Migraine is a debilitating neurological disorder affecting around one in seven people worldwide, but its molecular mechanisms remain poorly understood. There is some debate about whether migraine is a disease of vascular dysfunction or a result of neuronal dysfunction with secondary vascular changes. Genome-wide association (GWA) studies have thus far identified 13 independent loci associated with migraine. To identify new susceptibility loci, we carried out a genetic study of migraine on 59,674 affected subjects and 316,078 controls from 22 GWA studies. We identified 44 independent single-nucleotide polymorphisms (SNPs) significantly associated with migraine risk ($P < 5 \times 10(-8)$) that mapped to 38 distinct genomic loci, including 28 loci not previously reported and a locus that to our knowledge is the first to be identified on chromosome X. In subsequent computational analyses, the identified loci showed enrichment for genes expressed in vascular and smooth muscle tissues, consistent with a predominant theory of migraine that highlights vascular etiologies.

Learn more

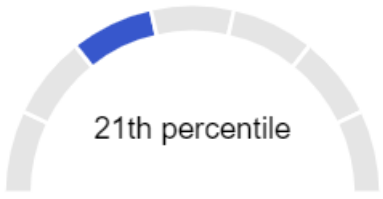
Headache

Nervous

This result is based on **28 genetic variants** associated with **"Headache"** analyzed in the scientific paper [(2018 Feb - Meng W)]

Your results

Below average genetic predisposition



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

Study description

Headache is the most common neurological symptom and a leading cause of years lived with disability. We sought to identify the genetic variants associated with a broadly-defined headache phenotype in 223,773 subjects from the UK Biobank cohort.

Learn more

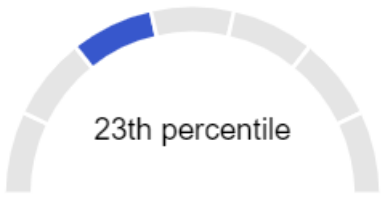
Heschl's gyrus morphology

Nervous

This result is based on **32 genetic variants** associated with **"Heschl's gyrus morphology"** analyzed in the scientific paper [(2014 Sep - Cai DC)]

Your results

Below average genetic predisposition



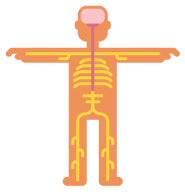
Your genetic predisposition is lower than the 77% average person from your genetic population

Study description

Heschl's gyrus (HG) is a core region of the auditory cortex whose morphology is highly variable across individuals. This variability has been linked to sound perception ability in both speech and music domains. Previous studies show that variations in morphological features of HG, such as cortical surface area and thickness, are heritable. To identify genetic variants that affect HG morphology, we conducted a genome-wide association scan (GWAS) meta-analysis in 3054 healthy individuals using HG surface area and thickness as quantitative traits. None of the single nucleotide polymorphisms (SNPs) showed association P values that would survive correction for multiple testing over the genome. The most significant association was found between right HG area and SNP rs72932726 close to gene DCBLD2 (3q12.1; P=2.77 × 10(-7)). This SNP was also associated with other regions involved in speech processing. The SNP rs333332 within gene KALRN (3q21.2; P=2.27 × 10(-6)) and rs143000161 near gene COBLL1 (2q24.3; P=2.40 × 10(-6)) were associated with the area and thickness of left HG, respectively. Both genes are involved in the development of the nervous system. The SNP rs7062395 close to the X-linked deafness gene POU3F4 was associated with right HG thickness (Xq21.1; P=2.38 × 10(-6)). This is the first molecular genetic analysis of variability in HG morphology.

Learn more

Neurofibrillary tangles

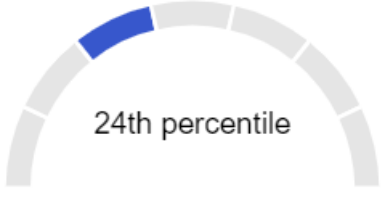


Nervous

This result is based on **31 genetic variants** associated with **"Neurofibrillary tangles"** analyzed in the scientific paper [(09/01/2019 - Dumitrescu L)]

Your results

Below average genetic predisposition



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

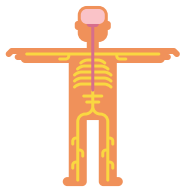
Study description

Autopsy measures of Alzheimer's disease neuropathology have been leveraged as endophenotypes in previous genome-wide association studies (GWAS). However, despite evidence of sex differences in Alzheimer's disease risk, sex-stratified models have not been incorporated into previous GWAS analyses. We looked for sex-specific genetic associations with

Alzheimer's disease endophenotypes from six brain bank data repositories. The pooled dataset included 2701 males and 3275 females, the majority of whom were diagnosed with Alzheimer's disease at autopsy (70%). Sex-stratified GWAS were performed within each dataset and then meta-analysed. Loci that reached genome-wide significance ($P < 5 \times 10\text{-}8$) in stratified models were further assessed for sex interactions. Additional analyses were performed in independent datasets leveraging cognitive, neuroimaging and CSF endophenotypes, along with age-at-onset data. Outside of the APOE region, one locus on chromosome 7 (rs34331204) showed a sex-specific association with neurofibrillary tangles among males ($P = 2.5 \times 10\text{-}8$) but not females ($P = 0.85$, sex-interaction $P = 2.9 \times 10\text{-}4$). In follow-up analyses, rs34331204 was also associated with hippocampal volume, executive function, and age-at-onset only among males. These results implicate a novel locus that confers male-specific protection from tau pathology and highlight the value of assessing genetic associations in a sex-specific manner.

Learn more

White matter lesion progression

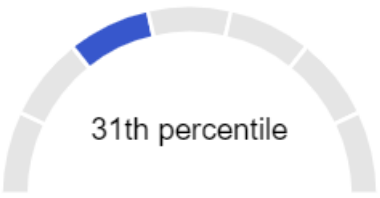


Nervous

This result is based on **7 genetic variants** associated with **"White matter lesion progression"** analyzed in the scientific paper [(2015 Nov - Hofer E)]

Your results

Below average genetic predisposition



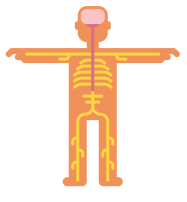
Your genetic predisposition is lower than the 69% average person from your genetic population

Study description

Background and White matter lesion (WML) progression on magnetic resonance imaging is related to cognitive decline and stroke, but its determinants besides baseline WML burden are largely unknown. Here, we estimated heritability of WML progression, and sought common genetic variants associated with WML progression in elderly participants from the Cohorts for Heart and Aging Research in Genomic Epidemiology (CHARGE) consortium.

Learn more

Deep white matter hyperintensities

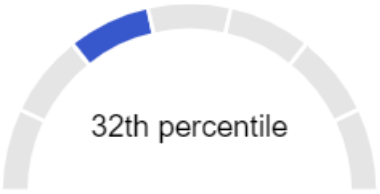


Nervous

This result is based on **45 genetic variants** associated with **"Deep white matter hyperintensities"** analyzed in the scientific paper [(2020 Jul - Armstrong NJ)]

Your results

Below average genetic predisposition



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

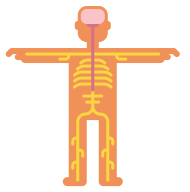
Study description

Background and Periventricular white matter hyperintensities (WMH; PVWMH) and deep WMH (DWMH) are regional classifications of WMH and reflect proposed differences in cause. In the first study, to date, we undertook genome-wide association analyses of DWMH and PVWMH to show that these phenotypes have different genetic underpinnings.

Learn more

Hearing function

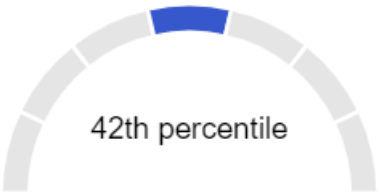
This result is based on **5 genetic variants** associated with **"Hearing function"** analyzed in the scientific paper **(12/01/2014 - Wolber LE)**



Nervous

Your results

Average genetic predisposition



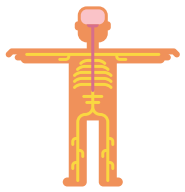
Your genetic predisposition is lower than the 58% average person from your genetic population

Study description

Hearing function is known to be heritable, but few significant and reproducible associations of genetic variants have been identified to date in the adult population. In this study, genome-wide association results of hearing function from the G-EAR consortium and TwinsUK were used for meta-analysis. Hearing ability in eight population samples of Northern and Southern European ancestry (n = 4591) and the Silk Road (n = 348) was measured using pure-tone audiometry and summarized using principal component (PC) analysis. Genome-wide association analyses for PC1-3 were conducted separately in each sample assuming an additive model adjusted for age, sex and relatedness of subjects. Meta-analysis was performed using 2.3 million single-nucleotide polymorphisms (SNPs) tested against each of the three PCs of hearing ability in 4939 individuals. A single SNP lying in intron 6 of the salt-inducible kinase 3 (SIK3) gene was found to be associated with hearing PC2 (P = 3.7×10(-8)) and further supported by whole-genome sequence in a subset. To determine the relevance of this gene in the ear, expression of the Sik3 protein was studied in mouse cochlea of different ages. Sik3 was expressed in murine hair cells during early development and in cells of the spiral ganglion during early development and adulthood. Our results suggest a developmental role of Sik3 in hearing and may be required for the maintenance of adult auditory function.

Learn more

Neuritic plaque

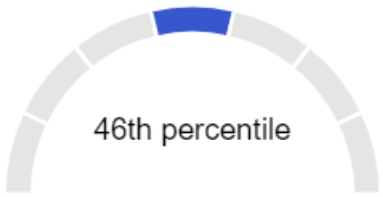


Nervous

This result is based on **30 genetic variants** associated with **"Neuritic plaque"** analyzed in the scientific paper **(09/04/2014 - Beecham GW)**

Your results

Average genetic predisposition



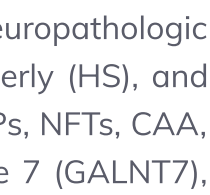
Your genetic predisposition is lower than the 54% average person from your genetic population

Study description

Alzheimer's disease (AD) and related dementias are a major public health challenge and present a therapeutic imperative for which we need additional insight into molecular pathogenesis. We performed a genome-wide association study and analysis of known genetic risk loci for AD dementia using neuropathologic data from 4,914 brain autopsies. Neuropathologic data were used to define clinico-pathologic AD dementia or controls, assess core neuropathologic features of AD (neuritic plaques, NPs; neurofibrillary tangles, NFTs), and evaluate commonly co-morbid neuropathologic changes: cerebral amyloid angiopathy (CAA), Lewy body disease (LBD), hippocampal sclerosis of the elderly (HS), and vascular brain injury (VBI). Genome-wide significance was observed for clinico-pathologic AD dementia, NPs, NFTs, CAA, and LBD with a number of variants in and around the apolipoprotein E gene (APOE). GalNAc transferase 7 (GALNT7), ATP-Binding Cassette, Sub-Family G (WHITE), Member 1 (ABCG1), and an intergenic region on chromosome 9 were associated with NP score; and Potassium Large Conductance Calcium-Activated Channel, Subfamily M, Beta Member 2 (KCNMB2) was strongly associated with HS. Twelve of the 21 non-APOE genetic risk loci for clinically-defined AD dementia were confirmed in our clinico-pathologic sample: CR1, BIN1, CLU, MS4A6A, PICALM, ABCA7, CD33, PTK2B, SORL1, MEF2C, ZCWPW1, and CASS4 with 9 of these 12 loci showing larger odds ratio in the clinico-pathologic sample. Correlation of effect sizes for risk of AD dementia with effect size for NFTs or NPs showed positive correlation, while those for risk of VBI showed a moderate negative correlation. The other co-morbid neuropathologic features showed only nominal association with the known AD loci. Our results discovered new genetic associations with specific neuropathologic features and aligned known genetic risk for AD dementia with specific neuropathologic changes in the largest brain autopsy study of AD and related dementias.

Learn more

Diabetic retinopathy

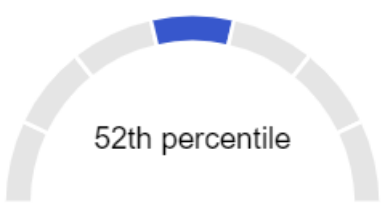


Nervous

This result is based on **19 genetic variants** associated with **"Diabetic retinopathy"** analyzed in the scientific paper **(06/15/2011 - Grassi MA)**

Your results

Average genetic predisposition



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

Study description

Diabetic retinopathy is a leading cause of blindness. The purpose of this study is to identify novel genetic loci associated with the sight threatening complications of diabetic retinopathy. We performed a meta-analysis of genome-wide association data for severe diabetic retinopathy as defined by diabetic macular edema or proliferative diabetic retinopathy in unrelated cases ascertained from two large, type I diabetic cohorts: the Genetics of Kidney in Diabetes (GoKinD) and the Epidemiology of Diabetes Intervention and Control Trial (EDIC) studies. Controls were other diabetic subjects in the cohort. A combined total of 2829 subjects (973 cases, 1856 controls) were studied on 2 543 887 single nucleotide polymorphisms (SNPs). Subjects with nephropathy were excluded in a sub-analysis of 281 severe retinopathy cases. We also performed an association analysis of 1390 copy number variations (CNVs) using tag SNPs. No associations were significant at a genome-wide level after correcting for multiple measures. The meta-analysis did identify several associations that can be pursued in future replication studies, including an intergenic SNP, rs476141, on chromosome 1 (P-value 1.2 × 10(-7)). The most interesting signal from the CNV analysis came from the sub-group analysis without nephropathy subjects and is rs10521145 (P-value 3.4 × 10(-6)) in the intron of CCDC101, a histone acetyltransferase. This SNP tags the copy number region CNVR6685.1 on chromosome 16 at 28.5 Mb, a gain/loss site. In summary, this study nominates several novel genetic loci associated with the sight-threatening complications of diabetic retinopathy and anticipates future large-scale consortium-based validation studies.

Learn more

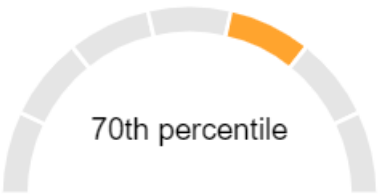
Essential tremor

Nervous

This result is based on **12 genetic variants** associated with **"Essential tremor"** analyzed in the scientific paper (2016 Dec - Müller SH)

Your results

Above average genetic predisposition



Your genetic predisposition is higher than the 70% average person from your genetic population

Study description

We conducted a genome-wide association study of essential tremor, a common movement disorder characterized mainly by a postural and kinetic tremor of the upper extremities. Twin and family history studies show a high heritability for essential tremor. The molecular genetic determinants of essential tremor are unknown. We included 2807 patients and 6441 controls of European descent in our two-stage genome-wide association study. The 59 most significantly disease-associated markers of the discovery stage were genotyped in the replication stage. After Bonferroni correction two markers, one (rs10937625) located in the serine/threonine kinase STK32B and one (rs17590046) in the transcriptional coactivator PPARGC1A were associated with essential tremor. Three markers (rs12764057, rs10822974, rs7903491) in the cell-adhesion molecule CTNNA3 were significant in the combined analysis of both stages. The expression of STK32B was increased in the cerebellar cortex of patients and expression quantitative trait loci database mining showed association between the protective minor allele of rs10937625 and reduced expression in cerebellar cortex. We found no expression differences related to disease status or marker genotype for the other two genes. Replication of two lead single nucleotide polymorphisms of previous small genome-wide association studies (rs3794087 in SLC1A2, rs9652490 in LINGO1) did not confirm the association with essential tremor.

Learn more

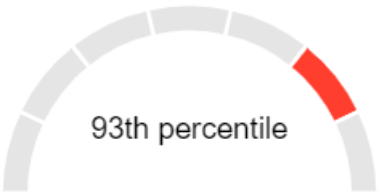
Glioblastoma

Nervous

This result is based on **9 genetic variants** associated with **"Glioblastoma"** analyzed in the scientific paper **(05/09/2018 - Ostrom QT)**

Your results

High genetic predisposition



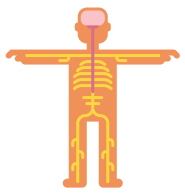
Your genetic predisposition is higher than the 93% average person from your genetic population

Study description

Incidence of glioma is approximately 50% higher in males. Previous analyses have examined exposures related to sex hormones in women as potential protective factors for these tumors, with inconsistent results. Previous glioma genome-wide association studies (GWAS) have not stratified by sex. Potential sex-specific genetic effects were assessed in autosomal SNPs and sex chromosome variants for all glioma, GBM and non-GBM patients using data from four previous glioma GWAS. Datasets were analyzed using sex-stratified logistic regression models and combined using meta-analysis. There were 4,831 male cases, 5,216 male controls, 3,206 female cases and 5,470 female controls. A significant association was detected at rs11979158 (7p11.2) in males only. Association at rs55705857 (8q24.21) was stronger in females than in males. A large region on 3p21.31 was identified with significant association in females only. The identified differences in effect of risk variants do not fully explain the observed incidence difference in glioma by sex.

Learn more

Non-glioblastoma glioma

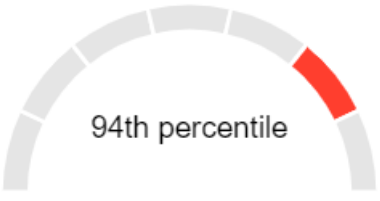


Nervous

This result is based on **7 genetic variants** associated with **"Non-glioblastoma glioma"** analyzed in the scientific paper **(05/09/2018 - Ostrom QT)**

Your results

High genetic predisposition



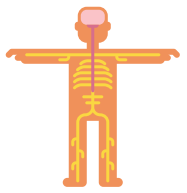
Your genetic predisposition is higher than the 94% average person from your genetic population

Study description

Incidence of glioma is approximately 50% higher in males. Previous analyses have examined exposures related to sex hormones in women as potential protective factors for these tumors, with inconsistent results. Previous glioma genome-wide association studies (GWAS) have not stratified by sex. Potential sex-specific genetic effects were assessed in autosomal SNPs and sex chromosome variants for all glioma, GBM and non-GBM patients using data from four previous glioma GWAS. Datasets were analyzed using sex-stratified logistic regression models and combined using meta-analysis. There were 4,831 male cases, 5,216 male controls, 3,206 female cases and 5,470 female controls. A significant association was detected at rs11979158 (7p11.2) in males only. Association at rs55705857 (8q24.21) was stronger in females than in males. A large region on 3p21.31 was identified with significant association in females only. The identified differences in effect of risk variants do not fully explain the observed incidence difference in glioma by sex.

Learn more

Glioma

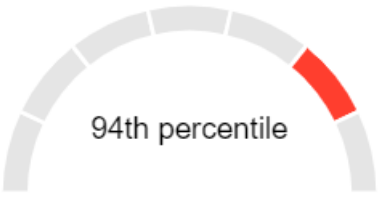


Nervous

This result is based on **18 genetic variants** associated with **"Glioma"** analyzed in the scientific paper [(2017 May - Melin BS)]

Your results

High genetic predisposition



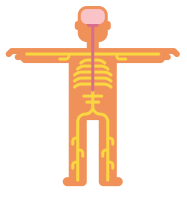
Your genetic predisposition is higher than the 94% average person from your genetic population

Study description

Genome-wide association studies (GWAS) have transformed our understanding of glioma susceptibility, but individual studies have had limited power to identify risk loci. We performed a meta-analysis of existing GWAS and two new GWAS, which totaled 12,496 cases and 18,190 controls. We identified five new loci for glioblastoma (GBM) at 1p31.3 (rs12752552; P = 2.04 × 10-9, odds ratio (OR) = 1.22), 11q14.1 (rs11233250; P = 9.95 × 10-10, OR = 1.24), 16p13.3 (rs2562152; P = 1.93 × 10-8, OR = 1.21), 16q12.1 (rs10852606; P = 1.29 × 10-11, OR = 1.18) and 22q13.1 (rs2235573; P = 1.76 × 10-10, OR = 1.15), as well as eight loci for non-GBM tumors at 1q32.1 (rs4252707; P = 3.34 × 10-9, OR = 1.19), 1q44 (rs12076373; P = 2.63 × 10-10, OR = 1.23), 2q33.3 (rs7572263; P = 2.18 × 10-10, OR = 1.20), 3p14.1 (rs11706832; P = 7.66 × 10-9, OR = 1.15), 10q24.33 (rs11598018; P = 3.39 × 10-8, OR = 1.14), 11q21 (rs7107785; P = 3.87 × 10-10, OR = 1.16), 14q12 (rs10131032; P = 5.07 × 10-11, OR = 1.33) and 16p13.3 (rs3751667; P = 2.61 × 10-9, OR = 1.18). These data substantiate that genetic susceptibility to GBM and non-GBM tumors are highly distinct, which likely reflects different etiology.

Learn more

Subcortical brain region volumes

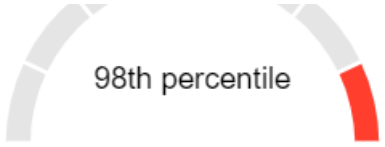


Nervous

This result is based on **11 genetic variants** associated with **"Subcortical brain region volumes"** analyzed in the scientific paper [(04/09/2015 - Hibar DP)]

Your results

Very high genetic predisposition



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

Study description

The highly complex structure of the human brain is strongly shaped by genetic influences. Subcortical brain regions form circuits with cortical areas to coordinate movement, learning, memory and motivation, and altered circuits can lead to abnormal behaviour and disease. To investigate how common genetic variants affect the structure of these brain regions, here we conduct genome-wide association studies of the volumes of seven subcortical regions and the intracranial volume derived from magnetic resonance images of 30,717 individuals from 50 cohorts. We identify five novel genetic variants influencing the volumes of the putamen and caudate nucleus. We also find stronger evidence for three loci with previously established influences on hippocampal volume and intracranial volume. These variants show specific volumetric effects on brain structures rather than global effects across structures. The strongest effects were found for the putamen, where a novel intergenic locus with replicable influence on volume (rs945270; P = 1.08 × 10(-33); 0.52% variance explained) showed evidence of altering the expression of the KTN1 gene in both brain and blood tissue. Variants influencing putamen volume clustered near developmental genes that regulate apoptosis, axon guidance and vesicle transport. Identification of these genetic variants provides insight into the causes of variability in human brain development, and may help to determine mechanisms of neuropsychiatric dysfunction.

Learn more