DEMO REPORT

Respiratory

The network of organs and tissues that makes up your respiratory system is responsible for allowing you to breathe. This system aids in absorbing oxygen from the air, enabling your organs to function. It also removes waste gases from your blood, such as carbon dioxide. Allergies, illness, and infection are all common occurrences. The network of organs and tissues that makes it possible for you to breathe is known as the respiratory system. Airways, blood vessels, and the lungs are all a part of your respiratory system. In addition to the lungs, the respiratory system includes the muscles that move the lungs. These components operate in concert to transport oxygen around the body and remove waste gases like carbon dioxide from the system.

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

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16 entries

Full View

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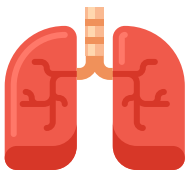
Sarcoidosis

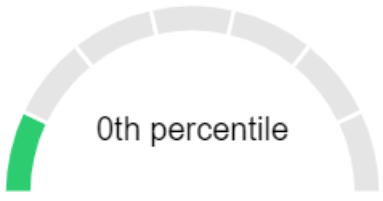


This result is based on **9 genetic variants** associated with **"Sarcoidosis"** analyzed in the scientific paper (09/15/2015 - Fischer A)

Respiratory

Your results

Very low genetic predisposition



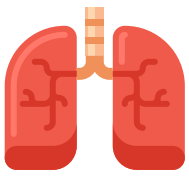
Your genetic predisposition is lower than the 100% average person from your genetic population

Study description

Genetic variation plays a significant role in the etiology of sarcoidosis. However, only a small fraction of its heritability has been explained so far.

Learn more

Asthma (adult onset)

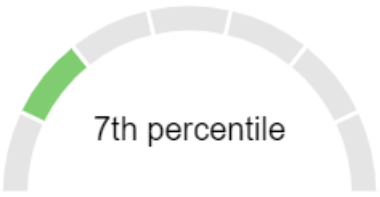


Respiratory

This result is based on **28 genetic variants** associated with **"Asthma (adult onset)"** analyzed in the scientific paper **(2019 Jun - Pividori M)**

Your results

Low genetic predisposition



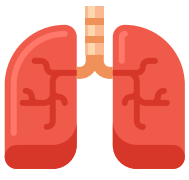
Your genetic predisposition is lower than the 93% average person from your genetic population

Study description

Childhood-onset and adult-onset asthma differ with respect to severity and comorbidities. Whether they also differ with respect to genetic risk factors has not been previously investigated in large samples. The goals of this study were to identify shared and distinct genetic risk loci for childhood-onset and adult-onset asthma, and to identify the genes that might mediate the effects of associated variation.

Learn more

Interstitial lung disease

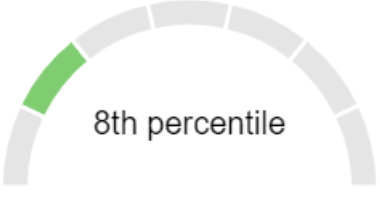


Respiratory

This result is based on **15 genetic variants** associated with **"Interstitial lung disease"** analyzed in the scientific paper **(2013 Jun - Fingerlin TE)**

Your results

Low genetic predisposition



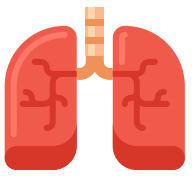
Your genetic predisposition is lower than the 92% average person from your genetic population

Study description

We performed a genome-wide association study of non-Hispanic, white individuals with fibrotic idiopathic interstitial pneumonias (IIPs; n = 1,616) and controls (n = 4,683), with follow-up replication analyses in 876 cases and 1,890 controls. We confirmed association with TERT at 5p15, MUC5B at 11p15 and the 3q26 region near TERC, and we identified seven newly associated loci (Pmeta = 2.4 × 10(-8) to 1.1 × 10(-19)), including FAM13A (4q22), DSP (6p24), OBFC1 (10q24), ATP11A (13q34), DPP9 (19p13) and chromosomal regions 7q22 and 15q14-15. Our results suggest that genes involved in host defense, cell-cell adhesion and DNA repair contribute to risk of fibrotic IIPs.

Learn more

Idiopathic pulmonary fibrosis

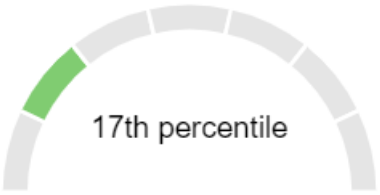


This result is based on **17 genetic variants** associated with **"Idiopathic pulmonary fibrosis"** analyzed in the scientific paper [(03/01/2020 - Allen RJ)]

Respiratory

Your results

Low genetic predisposition



Your genetic predisposition is lower than the 83% average person from your genetic population

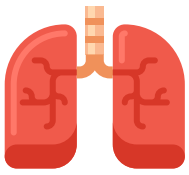
Study description

Rationale: Idiopathic pulmonary fibrosis (IPF) is a complex lung disease characterized by scarring of the lung that is believed to result from an atypical response to injury of the epithelium. Genome-wide association studies have reported signals of association implicating multiple pathways including host defense, telomere maintenance, signaling, and cell-cell adhesion.Objectives: To improve our understanding of factors that increase IPF susceptibility by identifying previously unreported genetic associations.Methods: We conducted genome-wide analyses across three independent studies and meta-analyzed these results to generate the largest genome-wide association study of IPF to date (2,668 IPF cases and 8,591 controls). We performed replication in two independent studies (1,456 IPF cases and 11,874 controls) and functional analyses (including statistical fine-mapping, investigations into gene expression, and testing for enrichment of IPF susceptibility signals in regulatory regions) to determine putatively causal genes. Polygenic risk scores were used to assess the collective effect of variants not reported as associated with IPF.Measurements and Main Results: We identified and replicated three new genome-wide significant (P < 5 × 10-8) signals of association with IPF susceptibility (associated with altered gene expression of KIF15, MAD1L1, and DEPTOR) and confirmed associations at 11 previously reported loci. Polygenic risk score analyses showed that the combined effect of many thousands of as yet unreported IPF susceptibility variants contribute to IPF susceptibility.Conclusions: The observation that decreased DEPTOR expression associates with increased susceptibility to IPF supports recent studies demonstrating the importance of mTOR signaling in lung fibrosis. New signals of association implicating KIF15 and MAD1L1 suggest a possible role of mitotic spindle-assembly genes in IPF susceptibility.

Learn more

Asthma (childhood onset)

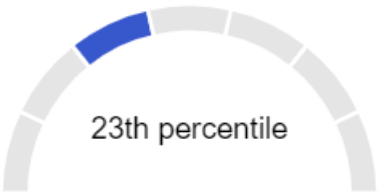
This result is based on **105 genetic variants** associated with **"Asthma (childhood onset)"** analyzed in the scientific paper [(04/04/2019 - Ferreira MAR)]



Respiratory

Your results

Below average genetic predisposition



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

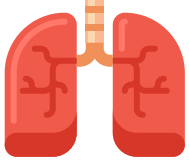
Study description

The extent to which genetic risk factors are shared between childhood-onset (COA) and adult-onset (AOA) asthma has not been estimated. On the basis of data from the UK Biobank study (n = 447,628), we found that the variance in disease liability explained by common variants is higher for COA (onset at ages between 0 and 19 years; h2g = 25.6%) than for AOA (onset at ages between 20 and 60 years; h2g = 10.6%). The genetic correlation (rg) between COA and AOA was 0.67. Variation in age of onset among COA-affected individuals had a low heritability (h2g = 5%), which we confirmed in independent studies and also among AOA-affected individuals. To identify subtype-specific genetic associations, we performed a genome-wide association study (GWAS) in the UK Biobank for COA (13,962 affected individuals) and a separate GWAS for AOA (26,582 affected individuals) by using a common set of 300,671 controls for both studies. We identified 123 independent associations for COA and 56 for AOA (37 overlapped); of these, 98 and 34, respectively, were reproducible in an independent study (n = 262,767). Collectively, 28 associations were not previously reported. For 96 COA-associated variants, including five variants that represent COA-specific risk factors, the risk allele was more common in COA- than in AOA-affected individuals. Conversely, we identified three variants that are stronger risk factors for AOA. Variants associated with obesity and smoking had a stronger contribution to the risk of AOA than to the risk of COA. Lastly, we identified 109 likely target genes of the associated variants, primarily on the basis of correlated expression quantitative trait loci (up to n = 31,684). GWAS informed by age of onset can identify subtype-specific risk variants, which can help us understand differences in pathophysiology between COA and AOA and so can be informative for drug development.

Learn more

Lung carcinoma (Familial squamous cell)

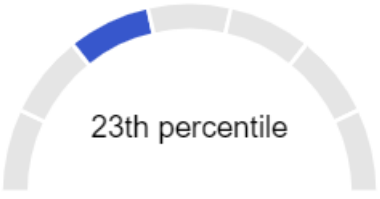
This result is based on **68 genetic variants** associated with **"Lung carcinoma (Familial squamous cell)"** analyzed in the scientific paper [(09/21/2018 - Byun J)]



Respiratory

Your results

Below average genetic predisposition



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

Study description

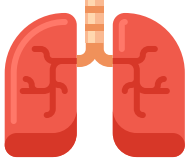
To identify genetic variation associated with lung cancer risk, we performed a genome-wide association analysis of 685 lung cancer cases that had a family history of two or more first or second degree relatives compared with 744 controls without lung cancer that were genotyped on an Illumina Human OmniExpressExome-8v1 array. To ensure robust results, we further evaluated these findings using data from six additional studies that were assembled through the

Transdisciplinary Research on Cancer of the Lung Consortium comprising 1993 familial cases and 33 690 controls. We performed a meta-analysis after imputation of all variants using the 1000 Genomes Project Phase 1 (version 3 release date September 2013). Analyses were conducted for 9 327 222 SNPs integrating data from the two sources. A novel variant on chromosome 4p15.31 near the LCORL gene and an imputed rare variant intergenic between CDKN2A and IFNA8 on chromosome 9p21.3 were identified at a genome-wide level of significance for squamous cell carcinomas. Additionally, associations of CHRNA3 and CHRNA5 on chromosome 15q25.1 in sporadic lung cancer were confirmed at a genome-wide level of significance in familial lung cancer. Previously identified variants in or near CHRNA2, BRCA2, CYP2A6 for overall lung cancer, TERT, SECISPB2L and RTEL1 for adenocarcinoma and RAD52 and MHC for squamous carcinoma were significantly associated with lung cancer.

Learn more

Nasal polyps

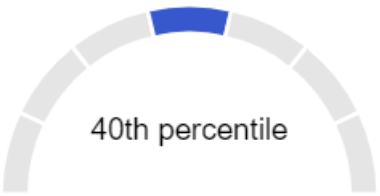
This result is based on **18 genetic variants** associated with **"Nasal polyps"** analyzed in the scientific paper **(2019 Feb - Kristjansson RP)**



Respiratory

Your results

Average genetic predisposition



Your genetic predisposition is lower than the 60% average person from your genetic population

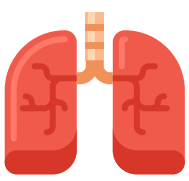
Study description

Nasal polyps (NP) are lesions on the nasal and paranasal sinus mucosa and are a risk factor for chronic rhinosinusitis (CRS). We performed genome-wide association studies on NP and CRS in Iceland and the UK (using UK Biobank data) with 4,366 NP cases, 5,608 CRS cases, and >700,000 controls. We found 10 markers associated with NP and 2 with CRS. We also tested 210 markers reported to associate with eosinophil count, yielding 17 additional NP associations. Of the 27 NP signals, 7 associate with CRS and 13 with asthma. Most notably, a missense variant in ALOX15 that causes a p.Thr560Met alteration in arachidonate 15-lipoxygenase (15-LO) confers large genome-wide significant protection against NP (P = 8.0 × 10-27, odds ratio = 0.32; 95% confidence interval = 0.26, 0.39) and CRS (P = 1.1 × 10-8, odds ratio = 0.64; 95% confidence interval = 0.55, 0.75). p.Thr560Met, carried by around 1 in 20 Europeans, was previously shown to cause near total loss of 15-LO enzymatic activity. Our findings identify 15-LO as a potential target for therapeutic intervention in NP and CRS.

Learn more

Small cell lung carcinoma

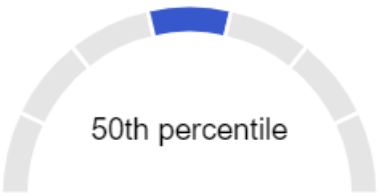
This result is based on **50 genetic variants** associated with **"Small cell lung carcinoma"** analyzed in the scientific paper **(2017 Jul - McKay JD)**



Respiratory

Your results

Average genetic predisposition



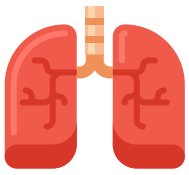
Your genetic predisposition is lower than the 50% average person from your genetic population

Study description

Although several lung cancer susceptibility loci have been identified, much of the heritability for lung cancer remains unexplained. Here 14,803 cases and 12,262 controls of European descent were genotyped on the OncoArray and combined with existing data for an aggregated genome-wide association study (GWAS) analysis of lung cancer in 29,266 cases and 56,450 controls. We identified 18 susceptibility loci achieving genome-wide significance, including 10 new loci. The new loci highlight the striking heterogeneity in genetic susceptibility across the histological subtypes of lung cancer, with four loci associated with lung cancer overall and six loci associated with lung adenocarcinoma. Gene expression quantitative trait locus (eQTL) analysis in 1,425 normal lung tissue samples highlights RNASET2, SECISBP2L and NRG1 as candidate genes. Other loci include genes such as a cholinergic nicotinic receptor, CHRNA2, and the telomere-related genes OFBC1 and RTEL1. Further exploration of the target genes will continue to provide new insights into the etiology of lung cancer.

Learn more

Lung cancer

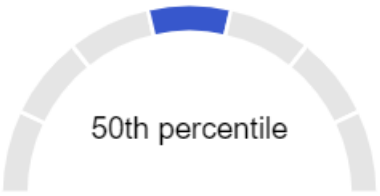


This result is based on **134 genetic variants** associated with **"Lung cancer"** analyzed in the scientific paper **(2017 Jul - McKay JD)**

Respiratory

Your results

Average genetic predisposition



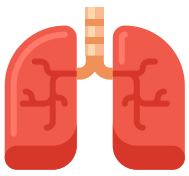
Your genetic predisposition is higher than the 50% average person from your genetic population

Study description

Although several lung cancer susceptibility loci have been identified, much of the heritability for lung cancer remains unexplained. Here 14,803 cases and 12,262 controls of European descent were genotyped on the OncoArray and combined with existing data for an aggregated genome-wide association study (GWAS) analysis of lung cancer in 29,266 cases and 56,450 controls. We identified 18 susceptibility loci achieving genome-wide significance, including 10 new loci. The new loci highlight the striking heterogeneity in genetic susceptibility across the histological subtypes of lung cancer, with four loci associated with lung cancer overall and six loci associated with lung adenocarcinoma. Gene expression quantitative trait locus (eQTL) analysis in 1,425 normal lung tissue samples highlights RNASET2, SECISBP2L and NRG1 as candidate genes. Other loci include genes such as a cholinergic nicotinic receptor, CHRNA2, and the telomere-related genes OFBC1 and RTEL1. Further exploration of the target genes will continue to provide new insights into the etiology of lung cancer.

Learn more

Diffusing capacity of carbon monoxide

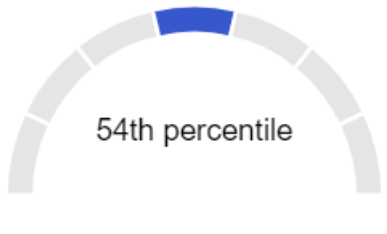


This result is based on **55 genetic variants** associated with **"Diffusing capacity of carbon monoxide"** analyzed in the scientific paper [(2019 May - Sakornsakolpat P)]

Respiratory

Your results

Average genetic predisposition



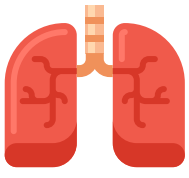
Your genetic predisposition is higher than the 54% average person from your genetic population

Study description

DlCO is a widely used pulmonary function test in clinical practice and a particularly useful measure for assessing patients with chronic obstructive pulmonary disease (COPD). We hypothesized that elucidating genetic determinants of DlCO could lead to better understanding of the genetic architecture of COPD. We estimated the heritability of DlCO using common genetic variants and performed genome-wide association analyses in four cohorts enriched for subjects with COPD (COPDGene [Genetic Epidemiology of COPD], NETT [National Emphysema Treatment Trial], GenKOLS [Genetics of Chronic Obstructive Lung Disease study], and TESRA [Treatment of Emphysema With a Gamma-Selective Retinoid Agonist study]) using a combined European ancestry white dataset and a COPDGene African American dataset. We assessed our genome-wide significant and suggestive associations for DlCO in previously reported genome-wide association studies of COPD and related traits. We also characterized associations of known COPD-associated variants and DlCO. We estimated the SNP-based heritability of DlCO in the European ancestry white population to be 22% (P = 0.0004). We identified three genome-wide significant associations with DlCO: variants near TGFB2, CHRNA3, and PDE11A loci (P < 5 × 10-8). In addition, 12 loci were suggestively associated with DlCO in European ancestry white (P < 1 × 10-5 in the combined analysis and P < 0.05 in both COPDGene and GenKOLS), including variants near NEGR1, CADM2, PCDH7, RETREG1, DACT2, NRG1, ANKRD18A, KRT86, NTN4, ARHGAP28, INSR, and PCBP3. Some DlCO-associated variants were also associated with COPD, emphysema, and/or spirometric values. Among 25 previously reported COPD loci, TGFB2, CHRNA3/CHRNA5, FAM13A, DSP, and CYP2A6 were associated with DlCO (P < 0.001). We identified several genetic loci that were significantly associated with DlCO and characterized effects of known COPD-associated loci on DlCO. These results could lead to better understanding of the heterogeneous nature of COPD.

Learn more

Squamous cell carcinoma

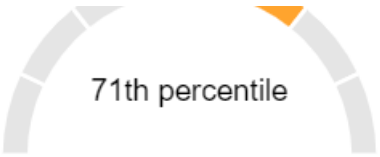


This result is based on **101 genetic variants** associated with **"Squamous cell carcinoma"** analyzed in the scientific paper [(2017 Jul - McKay JD)]

Respiratory

Your results

Above average genetic predisposition



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

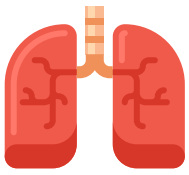
Study description

Although several lung cancer susceptibility loci have been identified, much of the heritability for lung cancer remains unexplained. Here 14,803 cases and 12,262 controls of European descent were genotyped on the OncoArray and combined with existing data for an aggregated genome-wide association study (GWAS) analysis of lung cancer in 29,266 cases and 56,450 controls. We identified 18 susceptibility loci achieving genome-wide significance, including 10 new loci. The new loci highlight the striking heterogeneity in genetic susceptibility across the histological subtypes of lung cancer, with four loci associated with lung cancer overall and six loci associated with lung adenocarcinoma. Gene expression quantitative trait locus (eQTL) analysis in 1,425 normal lung tissue samples highlights RNASET2, SECISBP2L and NRG1 as candidate genes. Other loci include genes such as a cholinergic nicotinic receptor, CHRNA2, and the telomere-related genes OFBC1 and RTEL1. Further exploration of the target genes will continue to provide new insights into the etiology of lung cancer.

Learn more

Lung adenocarcinoma

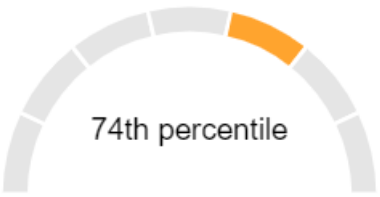
This result is based on **79 genetic variants** associated with **"Lung adenocarcinoma"** analyzed in the scientific paper [(2017 Jul - McKay JD)]



Respiratory

Your results

Above average genetic predisposition



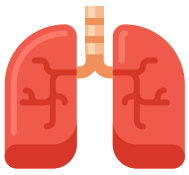
Your genetic predisposition is higher than the 74% average person from your genetic population

Study description

Although several lung cancer susceptibility loci have been identified, much of the heritability for lung cancer remains unexplained. Here 14,803 cases and 12,262 controls of European descent were genotyped on the OncoArray and combined with existing data for an aggregated genome-wide association study (GWAS) analysis of lung cancer in 29,266 cases and 56,450 controls. We identified 18 susceptibility loci achieving genome-wide significance, including 10 new loci. The new loci highlight the striking heterogeneity in genetic susceptibility across the histological subtypes of lung cancer, with four loci associated with lung cancer overall and six loci associated with lung adenocarcinoma. Gene expression quantitative trait locus (eQTL) analysis in 1,425 normal lung tissue samples highlights RNASET2, SECISBP2L and NRG1 as candidate genes. Other loci include genes such as a cholinergic nicotinic receptor, CHRNA2, and the telomere-related genes OFBC1 and RTEL1. Further exploration of the target genes will continue to provide new insights into the etiology of lung cancer.

Learn more

Airway responsiveness in chronic obstructive pulmonary disease

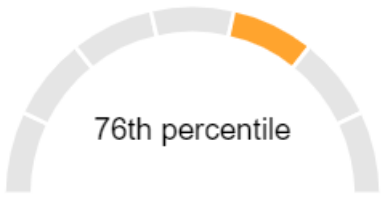


Respiratory

This result is based on **21 genetic variants** associated with **"Airway responsiveness in chronic obstructive pulmonary disease"** analyzed in the scientific paper (2015 Aug - Hansel NN)

Your results

Above average genetic predisposition



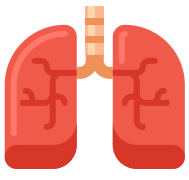
Your genetic predisposition is higher than the 76% average person from your genetic population

Study description

Increased airway responsiveness is linked to lung function decline and mortality in subjects with chronic obstructive pulmonary disease (COPD); however, the genetic contribution to airway responsiveness remains largely unknown. A genome-wide association study (GWAS) was performed using the Illumina (San Diego, CA) Human660W-Quad BeadChip on European Americans with COPD from the Lung Health Study. Linear regression models with correlated meta-analyses, including data from baseline (n = 2,814) and Year 5 (n = 2,657), were used to test for common genetic variants associated with airway responsiveness. Genotypic imputation was performed using reference 1000 Genomes Project data. Expression quantitative trait loci (eQTL) analyses in lung tissues were assessed for the top 10 markers identified, and immunohistochemistry assays assessed protein staining for SGCD and MYH15. Four genes were identified within the top 10 associations with airway responsiveness. Markers on chromosome 9p21.2 flanked by LINGO2 met a predetermined threshold of genome-wide significance ($P < 9.57 \times 10(-8)$). Markers on chromosomes 3q13.1 (flanked by MYH15), 5q33 (SGCD), and 6q21 (PDSS2) yielded suggestive evidence of association ($9.57 \times 10(-8) < P \leq 4.6 \times 10(-6)$). Gene expression studies in lung tissue showed single nucleotide polymorphisms on chromosomes 5 and 3 to act as eQTL for SGCD ($P = 2.57 \times 10(-9)$) and MYH15 ($P = 1.62 \times 10(-6)$), respectively. Immunohistochemistry confirmed localization of SGCD protein to airway smooth muscle and vessels and MYH15 to airway epithelium, vascular endothelium, and inflammatory cells. We identified novel loci associated with airway responsiveness in a GWAS among smokers with COPD. Risk alleles on chromosomes 5 and 3 acted as eQTLs for SGCD and MYH15 messenger RNA, and these proteins were expressed in lung cells relevant to the development of airway responsiveness.

Learn more

Chronic obstructive pulmonary disease

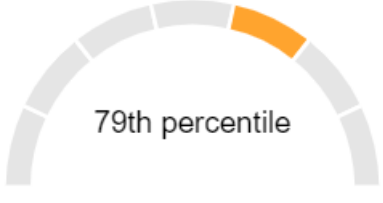


Respiratory

This result is based on **123 genetic variants** associated with **"Chronic obstructive pulmonary disease"** analyzed in the scientific paper (2019 Mar - Sakornsakolpat P)

Your results

Above average genetic predisposition



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

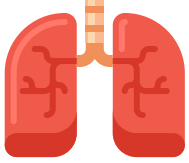
Study description

Chronic obstructive pulmonary disease (COPD) is the leading cause of respiratory mortality worldwide. Genetic risk loci provide new insights into disease pathogenesis. We performed a genome-wide association study in 35,735 cases and 222,076 controls from the UK Biobank and additional studies from the International COPD Genetics Consortium. We identified 82 loci associated with P < 5 × 10-8; 47 of these were previously described in association with either COPD or population-based measures of lung function. Of the remaining 35 new loci, 13 were associated with lung function in 79,055 individuals from the SpiroMeta consortium. Using gene expression and regulation data, we identified functional enrichment of COPD risk loci in lung tissue, smooth muscle, and several lung cell types. We found 14 COPD loci shared with either asthma or pulmonary fibrosis. COPD genetic risk loci clustered into groups based on associations with quantitative imaging features and comorbidities. Our analyses provide further support for the genetic susceptibility and heterogeneity of COPD.

Learn more

Non-small cell lung cancer

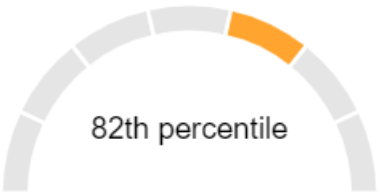
This result is based on **20 genetic variants** associated with **"Non-small cell lung cancer"** analyzed in the scientific paper (2019 Oct - Dai J)



Respiratory

Your results

Above average genetic predisposition



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

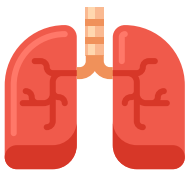
Study description

Genetic variation has an important role in the development of non-small-cell lung cancer (NSCLC). However, genetic factors for lung cancer have not been fully identified, especially in Chinese populations, which limits the use of existing polygenic risk scores (PRS) to identify subpopulations at high risk of lung cancer for prevention. We therefore aimed to identify novel loci associated with NSCLC risk, and generate a PRS and evaluate its utility and effectiveness in the prediction of lung cancer risk in Chinese populations.

Learn more

Post bronchodilator FEV1

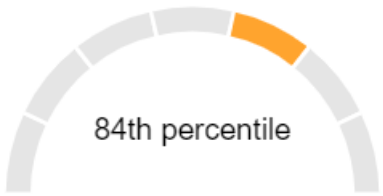
This result is based on **1175 genetic variants** associated with **"Post bronchodilator FEV1"** analyzed in the scientific paper (12/03/2015 - Lutz SM)



Respiratory

Your results

Above average genetic predisposition



Your genetic predisposition is higher than the 84% average person from your genetic population

Study description

Pulmonary function decline is a major contributor to morbidity and mortality among smokers. Post bronchodilator FEV1 and FEV1/FVC ratio are considered the standard assessment of airflow obstruction. We performed a genome-wide association study (GWAS) in 9919 current and former smokers in the COPDGene study (6659 non-Hispanic Whites [NHW] and 3260 African Americans [AA]) to identify associations with spirometric measures (post-bronchodilator FEV1 and FEV1/FVC). We also conducted meta-analysis of FEV1 and FEV1/FVC GWAS in the COPDGene, ECLIPSE, and GenKOLS cohorts (total n = 13,532).

Learn more