DEMO REPORT

Endocrine

Cells communicate with each other through hormones produced by the endocrine system. They're in charge of almost every cell, organ, and bodily function in your body. If the endocrine system is really not functioning correctly, you may have puberty, pregnancy, or stress management issues. Too much sugar in the blood prevents it from getting to your cells, where it is required for energy, so you may gain weight quickly, have weak bones, or have little energy. For example, the endocrine system produces hormones that affect your emotions and growth as well as your metabolism, organ function and reproduction.

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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Autoimmune thyroid disease

This result is based on **99 genetic variants** associated with **"Autoimmune thyroid disease"** analyzed in the scientific paper **(2020 Aug - Saevarsdottir S)**

Your results

Very high genetic predisposition

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

Study description

Autoimmune thyroid disease is the most common autoimmune disease and is highly heritable1. Here, by using a genome-wide association study of 30,234 cases and 725,172 controls from Iceland and the UK Biobank, we find 99 sequence variants at 93 loci, of which 84 variants are previously unreported2-7. A low-frequency (1.36%) intronic variant in FLT3 (rs76428106-C) has the largest effect on risk of autoimmune thyroid disease (odds ratio (OR) = 1.46, P = 2.37 × 10-24). rs76428106-C is also associated with systemic lupus erythematosus (OR = 1.90, P = 6.46 × 10-4), rheumatoid factor and/or anti-CCP-positive rheumatoid arthritis (OR = 1.41, P = 4.31 × 10-4) and coeliac disease (OR = 1.62, P = 1.20 × 10-4). FLT3 encodes fms-related tyrosine kinase 3, a receptor that regulates haematopoietic progenitor and dendritic cells. RNA sequencing revealed that rs76428106-C generates a cryptic splice site, which introduces a stop codon in 30% of transcripts that are predicted to encode a truncated protein, which lacks its tyrosine kinase domains. Each copy of rs76428106-C doubles the plasma levels of the FTL3 ligand. Activating somatic mutations in FLT3 are associated with acute myeloid leukaemia8 with a poor prognosis and rs76428106-C also predisposes individuals to acute myeloid leukaemia (OR = 1.90, P = 5.40 × 10-3). Thus, a predicted loss-of-function germline mutation in FLT3 causes a reduction in full-length FLT3, with a compensatory increase in the levels of its ligand and an increased disease risk, similar to that of a gain-of-function mutation.

Learn more

Breast cancer

This result is based on **798 genetic variants** associated with **"Breast cancer"** analyzed in the scientific paper **(11/02/2017 - Michailidou K)**

Your results

Low genetic predisposition

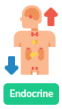
Your genetic predisposition is lower than the 88% average person from your genetic population

Study description

Breast cancer risk is influenced by rare coding variants in susceptibility genes, such as BRCA1, and many common, mostly non-coding variants. However, much of the genetic contribution to breast cancer risk remains unknown. Here we report the results of a genome-wide association study of breast cancer in 122,977 cases and 105,974 controls of European ancestry and 14,068 cases and 13,104 controls of East Asian ancestry. We identified 65 new loci that are associated with overall breast cancer risk at P < 5 × 10-8. The majority of credible risk single-nucleotide polymorphisms in these loci fall in distal regulatory elements, and by integrating in silico data to predict target genes in breast cells at each locus, we demonstrate a strong overlap between candidate target genes and somatic driver genes in breast tumours. We also find that heritability of breast cancer due to all single-nucleotide polymorphisms in regulatory features was 2-5-fold enriched relative to the genome-wide average, with strong enrichment for particular transcription factor binding sites. These results provide further insight into genetic susceptibility to breast cancer and will improve the use of genetic risk scores for individualized screening and prevention.

Learn more

Free thyroxine concentration



This result is based on **19 genetic variants** associated with **"Free thyroxine concentration"** analyzed in the scientific paper **(10/26/2018 - Teumer A)**

Your results

Above average genetic predisposition

Your genetic predisposition is higher than the 72% average person from your genetic population

Study description

Thyroid dysfunction is an important public health problem, which affects 10% of the general population and increases the risk of cardiovascular morbidity and mortality. Many aspects of thyroid hormone regulation have only partly been elucidated, including its transport, metabolism, and genetic determinants. Here we report a large meta-analysis of genome-wide association studies for thyroid function and dysfunction, testing 8 million genetic variants in up to 72,167 individuals. One-hundred-and-nine independent genetic variants are associated with these traits. A genetic risk score, calculated to assess their combined effects on clinical end points, shows significant associations with increased risk of both overt (Graves' disease) and subclinical thyroid disease, as well as clinical complications. By functional follow-up on selected signals, we identify a novel thyroid hormone transporter (SLC17A4) and a metabolizing enzyme (AADAT). Together, these results provide new knowledge about thyroid hormone physiology and disease, opening new possibilities for therapeutic targets.

Learn more

Hyperthyroidism

This result is based on **9 genetic variants** associated with **"Hyperthyroidism"** analyzed in the scientific paper **(10/26/2018 - Teumer A)**

Your results

High genetic predisposition

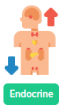
Your genetic predisposition is higher than the 95% average person from your genetic population

Study description

Thyroid dysfunction is an important public health problem, which affects 10% of the general population and increases the risk of cardiovascular morbidity and mortality. Many aspects of thyroid hormone regulation have only partly been elucidated, including its transport, metabolism, and genetic determinants. Here we report a large meta-analysis of genome-wide association studies for thyroid function and dysfunction, testing 8 million genetic variants in up to 72,167 individuals. One-hundred-and-nine independent genetic variants are associated with these traits. A genetic risk score, calculated to assess their combined effects on clinical end points, shows significant associations with increased risk of both overt (Graves' disease) and subclinical thyroid disease, as well as clinical complications. By functional follow-up on selected signals, we identify a novel thyroid hormone transporter (SLC17A4) and a metabolizing enzyme (AADAT). Together, these results provide new knowledge about thyroid hormone physiology and disease, opening new possibilities for therapeutic targets.

Learn more

Hypothyroidism



This result is based on **8 genetic variants** associated with **"Hypothyroidism"** analyzed in the scientific paper **(10/26/2018 - Teumer A)**

Your results

Very high genetic predisposition

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

Study description

Thyroid dysfunction is an important public health problem, which affects 10% of the general population and increases the risk of cardiovascular morbidity and mortality. Many aspects of thyroid hormone regulation have only partly been elucidated, including its transport, metabolism, and genetic determinants. Here we report a large meta-analysis of genome-wide association studies for thyroid function and dysfunction, testing 8 million genetic variants in up to 72,167 individuals. One-hundred-and-nine independent genetic variants are associated with these traits. A genetic risk score, calculated to assess their combined effects on clinical end points, shows significant associations with increased risk of both overt (Graves' disease) and subclinical thyroid disease, as well as clinical complications. By functional follow-up on selected signals, we identify a novel thyroid hormone transporter (SLC17A4) and a metabolizing enzyme (AADAT). Together, these results provide new knowledge about thyroid hormone physiology and disease, opening new possibilities for therapeutic targets.

Learn more

Pancreatic cancer

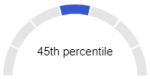
This result is based on **32 genetic variants** associated with **"Pancreatic cancer"** analyzed in the scientific paper

(02/08/2018 - Klein AP)

Endocrine

Your results

Average genetic predisposition



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

Study description

In 2020, 146,063 deaths due to pancreatic cancer are estimated to occur in Europe and the United States combined. To identify common susceptibility alleles, we performed the largest pancreatic cancer GWAS to date, including 9040 patients and 12,496 controls of European ancestry from the Pancreatic Cancer Cohort Consortium (PanScan) and the Pancreatic Cancer Case-Control Consortium (PanC4). Here, we find significant evidence of a novel association at rs78417682 (7p12/TNS3, P = 4.35 × 10-8). Replication of 10 promising signals in up to 2737 patients and 4752 controls from the PANcreatic Disease ReseArch (PANDoRA) consortium yields new genome-wide significant loci: rs13303010 at 1p36.33 (NOC2L, P = 8.36 × 10-14), rs2941471 at 8q21.11 (HNF4G, P = 6.60 × 10-10), rs4795218 at 17q12 (HNF1B, P = 1.32 × 10-8), and rs1517037 at 18q21.32 (GRP, P = 3.28 × 10-8). rs78417682 is not statistically significantly associated with pancreatic cancer in PANDoRA. Expression quantitative trait locus analysis in three independent pancreatic data sets provides molecular support of NOC2L as a pancreatic cancer susceptibility gene.

Learn more

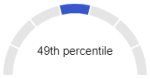
Thyroid cancer

This result is based on **13 genetic variants** associated with **"Thyroid cancer"** analyzed in the scientific paper
(09/04/2020 - Rashkin SR)

Endocrine

Your results

Average genetic predisposition



Your genetic predisposition is lower than the 51% average person from your genetic population

Study description

Deciphering the shared genetic basis of distinct cancers has the potential to elucidate carcinogenic mechanisms and inform broadly applicable risk assessment efforts. Here, we undertake genome-wide association studies (GWAS) and comprehensive evaluations of heritability and pleiotropy across 18 cancer types in two large, population-based cohorts: the UK Biobank (408,786 European ancestry individuals; 48,961 cancer cases) and the Kaiser Permanente Genetic Epidemiology Research on Adult Health and Aging cohorts (66,526 European ancestry individuals; 16,001 cancer cases). The GWAS detect 21 genome-wide significant associations independent of previously reported results. Investigations of pleiotropy identify 12 cancer pairs exhibiting either positive or negative genetic correlations; 25 pleiotropic loci; and 100 independent pleiotropic variants, many of which are regulatory elements and/or influence cross-tissue gene expression. Our findings demonstrate widespread pleiotropy and offer further insight into the complex genetic architecture of cross-cancer susceptibility.

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