DEMO REPORT

Reproductive

The female reproductive organs are the vagina, uterus, fallopian tubes, and ovaries. To connect the uterus to the vagina, women use a muscular, hollow tube known as the vagina. Vasodilatation is possible because the vagina possesses muscular walls. For reproduction, the reproductive system performs four tasks: To produce sperm and egg cells. There are several reasons behind this. To care for and protect the offspring. The fallopian tubes, uterus, vagina, and similar structures in females, the penis, sperm channels (epididymis, ductus deferens, and ejaculatory ducts), and other related structures and glands in men, make up these tracts.

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

21 entries

Full View

Sort by risk | Sort by name

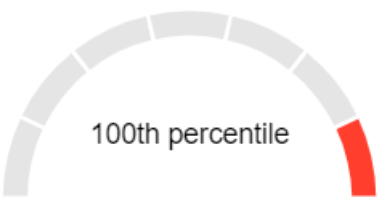
Epithelial ovarian cancer

This result is based on **23 genetic variants** associated with **"Epithelial ovarian cancer"** analyzed in the scientific paper (2017 May - Phelan CM)

Reproductive

Your results

Very high genetic predisposition



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

Study description

To identify common alleles associated with different histotypes of epithelial ovarian cancer (EOC), we pooled data from multiple genome-wide genotyping projects totaling 25,509 EOC cases and 40,941 controls. We identified nine new susceptibility loci for different EOC histotypes: six for serous EOC histotypes (3q28, 4q32.3, 8q21.11, 10q24.33, 18q11.2 and 22q12.1), two for mucinous EOC (3q22.3 and 9q31.1) and one for endometrioid EOC (5q12.3). We then performed meta-analysis on the results for high-grade serous ovarian cancer with the results from analysis of 31,448 BRCA1 and BRCA2 mutation carriers, including 3,887 mutation carriers with EOC. This identified three additional susceptibility loci at 2q13, 8q24.1 and 12q24.31. Integrated analyses of genes and regulatory biofeatures at each locus predicted candidate susceptibility genes, including OBFC1, a new candidate susceptibility gene for low-grade and borderline serous EOC.

Learn more

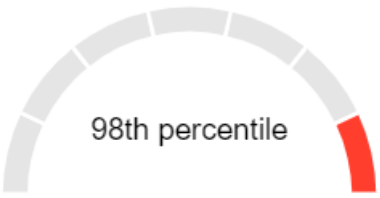
Invasive epithelial ovarian cancer

This result is based on **19 genetic variants** associated with **"Invasive epithelial ovarian cancer"** analyzed in the scientific paper [(2017 May - Phelan CM)]

Reproductive

Your results

Very high genetic predisposition



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

Study description

To identify common alleles associated with different histotypes of epithelial ovarian cancer (EOC), we pooled data from multiple genome-wide genotyping projects totaling 25,509 EOC cases and 40,941 controls. We identified nine new susceptibility loci for different EOC histotypes: six for serous EOC histotypes (3q28, 4q32.3, 8q21.11, 10q24.33, 18q11.2 and 22q12.1), two for mucinous EOC (3q22.3 and 9q31.1) and one for endometrioid EOC (5q12.3). We then performed meta-analysis on the results for high-grade serous ovarian cancer with the results from analysis of 31,448 BRCA1 and BRCA2 mutation carriers, including 3,887 mutation carriers with EOC. This identified three additional susceptibility loci at 2q13, 8q24.1 and 12q24.31. Integrated analyses of genes and regulatory biofeatures at each locus predicted candidate susceptibility genes, including OBFC1, a new candidate susceptibility gene for low-grade and borderline serous EOC.

Learn more

Testicular cancer

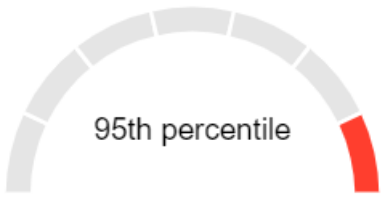
This result is based on **16 genetic variants** associated with **"Testicular cancer"** analyzed in the scientific paper [(09/04/2020 - Rashkin SR)]

Reproductive

Your results

Your results

Very high genetic predisposition



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

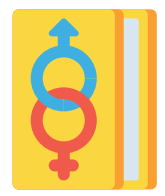
Study description

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.

Learn more

Testicular germ cell tumor

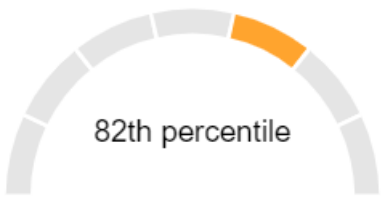
This result is based on **37 genetic variants** associated with **"Testicular germ cell tumor"** analyzed in the scientific paper **(2017 Jul - Wang Z)**



Reproductive

Your results

Above average genetic predisposition



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

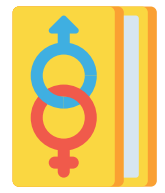
Study description

The international Testicular Cancer Consortium (TECAC) combined five published genome-wide association studies of testicular germ cell tumor (TGCT; 3,558 cases and 13,970 controls) to identify new susceptibility loci. We conducted a fixed-effects meta-analysis, including, to our knowledge, the first analysis of the X chromosome. Eight new loci mapping to 2q14.2, 3q26.2, 4q35.2, 7q36.3, 10q26.13, 15q21.3, 15q22.31, and Xq28 achieved genome-wide significance ($P < 5 \times 10-8$). Most loci harbor biologically plausible candidate genes. We refined previously reported associations at 9p24.3 and 19p12 by identifying one and three additional independent SNPs, respectively. In aggregate, the 39 independent markers identified to date explain 37% of father-to-son familial risk, 8% of which can be attributed to the 12 new signals reported here. Our findings substantially increase the number of known TGCT susceptibility alleles, move the field closer to a comprehensive understanding of the underlying genetic architecture of TGCT, and provide further clues to the etiology of TGCT.

Learn more

Uterine fibroids

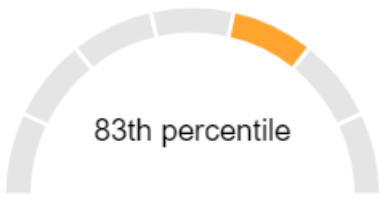
This result is based on **44 genetic variants** associated with **"Uterine fibroids"** analyzed in the scientific paper **(10/24/2019 - Gallagher CS)**



Reproductive

Your results

Above average genetic predisposition



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

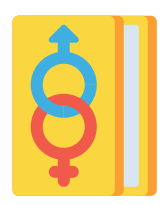
Study description

Uterine leiomyomata (UL) are the most common neoplasms of the female reproductive tract and primary cause for hysterectomy, leading to considerable morbidity and high economic burden. Here we conduct a GWAS meta-analysis in 35,474 cases and 267,505 female controls of European ancestry, identifying eight novel genome-wide significant (P < 5 × 10-8) loci, in addition to confirming 21 previously reported loci, including multiple independent signals at 10 loci. Phenotypic stratification of UL by heavy menstrual bleeding in 3409 cases and 199,171 female controls reveals genome-wide significant associations at three of the 29 UL loci: 5p15.33 (TERT), 5q35.2 (FGFR4) and 11q22.3 (ATM). Four loci identified in the meta-analysis are also associated with endometriosis risk; an epidemiological meta-analysis across 402,868 women suggests at least a doubling of risk for UL diagnosis among those with a history of endometriosis. These findings increase our understanding of genetic contribution and biology underlying UL development, and suggest overlapping genetic origins with endometriosis.

Learn more

Endometriosis

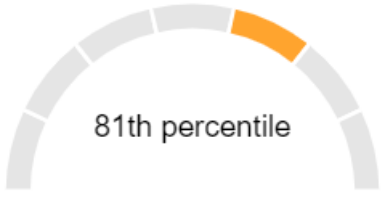
This result is based on **17 genetic variants** associated with **"Endometriosis"** analyzed in the scientific paper **(05/24/2017 - Sapkota Y)**



Reproductive

Your results

Above average genetic predisposition



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

Study description

Endometriosis is a heritable hormone-dependent gynecological disorder, associated with severe pelvic pain and reduced fertility; however, its molecular mechanisms remain largely unknown. Here we perform a meta-analysis of 11 genome-wide association case-control data sets, totalling 17,045 endometriosis cases and 191,596 controls. In addition to

replicating previously reported loci, we identify five novel loci significantly associated with endometriosis risk (P<5 × 10-8), implicating genes involved in sex steroid hormone pathways (FN1, CCDC170, ESR1, SYNE1 and FSHB). Conditional analysis identified five secondary association signals, including two at the ESR1 locus, resulting in 19 independent single nucleotide polymorphisms (SNPs) robustly associated with endometriosis, which together explain up to 5.19% of variance in endometriosis. These results highlight novel variants in or near specific genes with important roles in sex steroid hormone signalling and function, and offer unique opportunities for more targeted functional research efforts.

Learn more

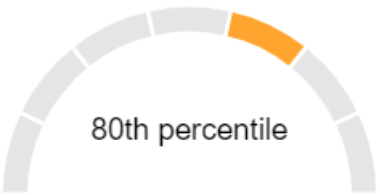
Placental abruption

This result is based on **9 genetic variants** associated with **"Placental abruption"** analyzed in the scientific paper **(2018 Jun - Workalemahu T)**

Reproductive

Your results

Above average genetic predisposition



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

Study description

Accumulating epidemiological evidence points to strong genetic susceptibility to placental abruption (PA). However, characterization of genes associated with PA remains incomplete. We conducted a genome-wide association study (GWAS) of PA and a meta-analysis of GWAS.

Learn more

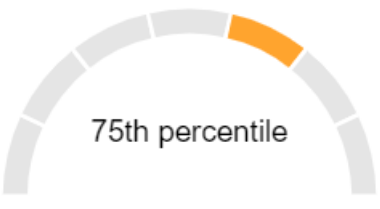
Menopause (age at onset) (earlier)

This result is based on **53 genetic variants** associated with **"Menopause (age at onset) (earlier)"** analyzed in the scientific paper **(2015 Nov - Day FR)**

Reproductive

Your results

Above average genetic predisposition



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

Study description

Menopause timing has a substantial impact on infertility and risk of disease, including breast cancer, but the underlying mechanisms are poorly understood. We report a dual strategy in ~70,000 women to identify common and low-frequency protein-coding variation associated with age at natural menopause (ANM). We identified 44 regions with common variants, including two regions harboring additional rare missense alleles of large effect. We found enrichment of signals in or near genes involved in delayed puberty, highlighting the first molecular links between the onset and end of reproductive lifespan. Pathway analyses identified major association with DNA damage response (DDR) genes, including the first common coding variant in BRCA1 associated with any complex trait. Mendelian randomization analyses supported a causal effect of later ANM on breast cancer risk (~6% increase in risk per year; P = 3 × 10(-14)), likely mediated by prolonged sex hormone exposure rather than DDR mechanisms.

Learn more

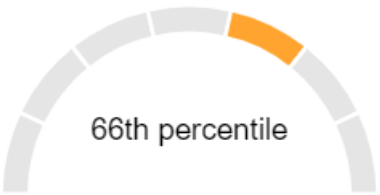
Offspring birth weight (higher)

This result is based on **131 genetic variants** associated with **"Offspring birth weight (higher)"** analyzed in the scientific paper [(2019 May - Warrington NM)]

Reproductive

Your results

Above average genetic predisposition



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

Study description

Birth weight variation is influenced by fetal and maternal genetic and non-genetic factors, and has been reproducibly associated with future cardio-metabolic health outcomes. In expanded genome-wide association analyses of own birth weight (n = 321,223) and offspring birth weight (n = 230,069 mothers), we identified 190 independent association signals (129 of which are novel). We used structural equation modeling to decompose the contributions of direct fetal and indirect maternal genetic effects, then applied Mendelian randomization to illuminate causal pathways. For example, both indirect maternal and direct fetal genetic effects drive the observational relationship between lower birth weight and higher later blood pressure: maternal blood pressure-raising alleles reduce offspring birth weight, but only direct fetal effects of these alleles, once inherited, increase later offspring blood pressure. Using maternal birth weight-lowering genotypes to proxy for an adverse intrauterine environment provided no evidence that it causally raises offspring blood pressure, indicating that the inverse birth weight-blood pressure association is attributable to genetic effects, and not to intrauterine programming.

Learn more

Polycystic ovary syndrome

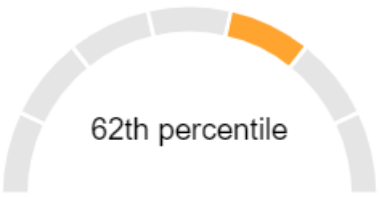
This result is based on **14 genetic variants** associated with **"Polycystic ovary syndrome"** analyzed in the

scientific paper [(12/19/2018 - Day F)]

Reproductive

Your results

Above average genetic predisposition



Your genetic predisposition is higher than the 62% average person from your genetic population

Study description

Polycystic ovary syndrome (PCOS) is a disorder characterized by hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology. Affected women frequently have metabolic disturbances including insulin resistance and dysregulation of glucose homeostasis. PCOS is diagnosed with two different sets of diagnostic criteria, resulting in a phenotypic spectrum of PCOS cases. The genetic similarities between cases diagnosed based on the two criteria have been largely unknown. Previous studies in Chinese and European subjects have identified 16 loci associated with risk of PCOS. We report a fixed-effect, inverse-weighted-variance meta-analysis from 10,074 PCOS cases and 103,164 controls of European ancestry and characterisation of PCOS related traits. We identified 3 novel loci (near PLGRKT, ZBTB16 and MAPRE1), and provide replication of 11 previously reported loci. Only one locus differed significantly in its association by diagnostic criteria; otherwise the genetic architecture was similar between PCOS diagnosed by self-report and PCOS diagnosed by NIH or non-NIH Rotterdam criteria across common variants at 13 loci. Identified variants were associated with hyperandrogenism, gonadotropin regulation and testosterone levels in affected women. Linkage disequilibrium score regression analysis revealed genetic correlations with obesity, fasting insulin, type 2 diabetes, lipid levels and coronary artery disease, indicating shared genetic architecture between metabolic traits and PCOS. Mendelian randomization analyses suggested variants associated with body mass index, fasting insulin, menopause timing, depression and male-pattern balding play a causal role in PCOS. The data thus demonstrate 3 novel loci associated with PCOS and similar genetic architecture for all diagnostic criteria. The data also provide the first genetic evidence for a male phenotype for PCOS and a causal link to depression, a previously hypothesized comorbid disease. Thus, the genetics provide a comprehensive view of PCOS that encompasses multiple diagnostic criteria, gender, reproductive potential and mental health.

Learn more

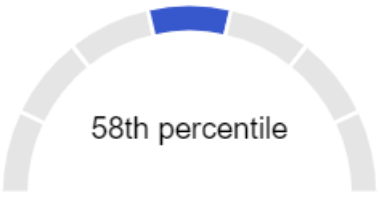
Infant length (higher)

This result is based on **15 genetic variants** associated with **"Infant length (higher)"** analyzed in the scientific paper [(02/15/2015 - van der Valk RJ)]

Reproductive

Your results

Average genetic predisposition



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

Study description

Common genetic variants have been identified for adult height, but not much is known about the genetics of skeletal growth in early life. To identify common genetic variants that influence fetal skeletal growth, we meta-analyzed 22 genome-wide association studies (Stage 1; N = 28 459). We identified seven independent top single nucleotide

polymorphisms (SNPs) (P < 1 × 10(-6)) for birth length, of which three were novel and four were in or near loci known to be associated with adult height (LCORL, PTCH1, GPR126 and HMGA2). The three novel SNPs were followed-up in nine replication studies (Stage 2; N = 11 995), with rs905938 in DC-STAMP domain containing 2 (DCST2) genome-wide significantly associated with birth length in a joint analysis (Stages 1 + 2; β = 0.046, SE = 0.008, P = 2.46 × 10(-8), explained variance = 0.05%). Rs905938 was also associated with infant length (N = 28 228; P = 5.54 × 10(-4)) and adult height (N = 127 513; P = 1.45 × 10(-5)). DCST2 is a DC-STAMP-like protein family member and DC-STAMP is an osteoclast cell-fusion regulator. Polygenic scores based on 180 SNPs previously associated with human adult stature explained 0.13% of variance in birth length. The same SNPs explained 2.95% of the variance of infant length. Of the 180 known adult height loci, 11 were genome-wide significantly associated with infant length (SF3B4, LCORL, SPAG17, C6orf173, PTCH1, GDF5, ZNFX1, HHIP, ACAN, HLA locus and HMGA2). This study highlights that common variation in DCST2 influences variation in early growth and adult height.

Learn more

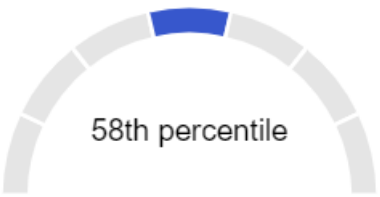
Endometrial cancer

This result is based on **27 genetic variants** associated with **"Endometrial cancer"** analyzed in the scientific paper [(08/09/2018 - O&#39)]

Reproductive

Your results

Average genetic predisposition



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

Study description

Endometrial cancer is the most commonly diagnosed cancer of the female reproductive tract in developed countries. Through genome-wide association studies (GWAS), we have previously identified eight risk loci for endometrial cancer. Here, we present an expanded meta-analysis of 12,906 endometrial cancer cases and 108,979 controls (including new genotype data for 5624 cases) and identify nine novel genome-wide significant loci, including a locus on 12q24.12 previously identified by meta-GWAS of endometrial and colorectal cancer. At five loci, expression quantitative trait locus (eQTL) analyses identify candidate causal genes; risk alleles at two of these loci associate with decreased expression of genes, which encode negative regulators of oncogenic signal transduction proteins (SH2B3 (12q24.12) and NF1 (17q11.2)). In summary, this study has doubled the number of known endometrial cancer risk loci and revealed candidate causal genes for future study.

Learn more

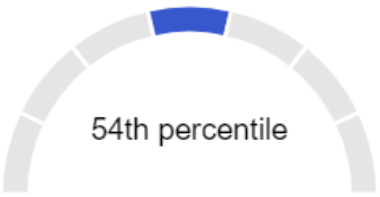
Dysmenorrheic pain

This result is based on **6 genetic variants** associated with **"Dysmenorrheic pain"** analyzed in the scientific paper [(2016 Nov - Jones AV)]

Reproductive

Your results

Average genetic predisposition



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

Study description

Dysmenorrhea is a common chronic pelvic pain syndrome affecting women of childbearing potential. Family studies suggest that genetic background influences the severity of dysmenorrhea, but genetic predisposition and molecular mechanisms underlying dysmenorrhea are not understood. In this study, we conduct the first genome-wide association study to identify genetic factors associated with dysmenorrhea pain severity. A cohort of females of European descent (n = 11,891) aged 18 to 45 years rated their average dysmenorrhea pain severity. We used a linear regression model adjusting for age and body mass index, identifying one genome-wide significant ($P < 5 \times 10$) association (rs7523086, $P = 4.1 \times 10$, effect size 0.1 [95% confidence interval, 0.074-0.126]). This single nucleotide polymorphism is colocalising with NGF, encoding nerve growth factor. The presence of one risk allele corresponds to a predicted 0.1-point increase in pain intensity on a 4-point ordinal pain scale. The putative effects on NGF function and/or expression remain unknown. However, genetic variation colocalises with active epigenetic marks in fat and ovary tissues, and expression levels in aorta tissue of a noncoding RNA flanking NGF correlate. Participants reporting extreme dysmenorrhea pain were more likely to report being positive for endometriosis, polycystic ovarian syndrome, depression, and other psychiatric disorders. Our results indicate that dysmenorrhea pain severity is partly genetically determined. NGF already has an established role in chronic pain disorders, and our findings suggest that NGF may be an important mediator for gynaecological/pelvic pain in the viscera.

Learn more

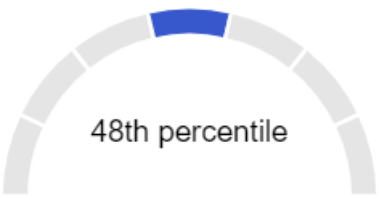
Birth weight (higher)

This result is based on **224 genetic variants** associated with **"Birth weight (higher)"** analyzed in the scientific paper **(2019 May - Warrington NM)**

Reproductive

Your results

Average genetic predisposition



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

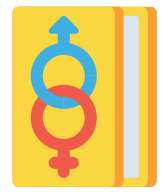
Study description

Birth weight variation is influenced by fetal and maternal genetic and non-genetic factors, and has been reproducibly associated with future cardio-metabolic health outcomes. In expanded genome-wide association analyses of own birth weight (n = 321,223) and offspring birth weight (n = 230,069 mothers), we identified 190 independent association signals (129 of which are novel). We used structural equation modeling to decompose the contributions of direct fetal and indirect maternal genetic effects, then applied Mendelian randomization to illuminate causal pathways. For example, both indirect maternal and direct fetal genetic effects drive the observational relationship between lower birth weight and higher later blood pressure: maternal blood pressure-raising alleles reduce offspring birth weight, but only direct fetal effects of these alleles, once inherited, increase later offspring blood pressure. Using maternal birth weight-lowering genotypes to proxy

for an adverse intrauterine environment provided no evidence that it causally raises offspring blood pressure, indicating that the inverse birth weight-blood pressure association is attributable to genetic effects, and not to intrauterine programming.

Learn more

Length of menstrual cycle

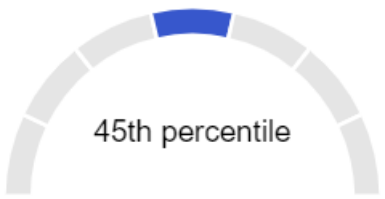


Reproductive

This result is based on **5 genetic variants** associated with **"Length of menstrual cycle"** analyzed in the scientific paper **(12/15/2018 - Laisk T)**

Your results

Average genetic predisposition



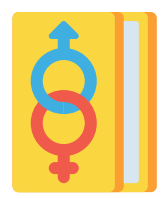
Your genetic predisposition is lower than the 55% average person from your genetic population

Study description

The normal menstrual cycle requires a delicate interplay between the hypothalamus, pituitary and ovary. Therefore, its length is an important indicator of female reproductive health. Menstrual cycle length has been shown to be partially controlled by genetic factors, especially in the follicle-stimulating hormone beta-subunit (FSHB) locus. A genome-wide association study meta-analysis of menstrual cycle length in 44 871 women of European ancestry confirmed the previously observed association with the FSHB locus and identified four additional novel signals in, or near, the GNRH1, PGR, NR5A2 and INS-IGF2 genes. These findings not only confirm the role of the hypothalamic-pituitary-gonadal axis in the genetic regulation of menstrual cycle length but also highlight potential novel local regulatory mechanisms, such as those mediated by IGF2.

Learn more

Mosaic loss of chromosome Y (Y chromosome dosage)

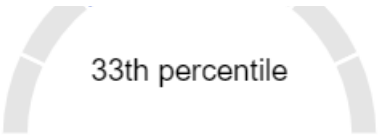


Reproductive

This result is based on **54 genetic variants** associated with **"Mosaic loss of chromosome Y (Y chromosome dosage)"** analyzed in the scientific paper **(10/17/2019 - Terao C)**

Your results

Below average genetic predisposition



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

Study description

Mosaic loss of chromosome Y (mLOY) is frequently observed in the leukocytes of ageing men. However, the genetic architecture and biological mechanisms underlying mLOY are not fully understood. In a cohort of 95,380 Japanese men, we identify 50 independent genetic markers in 46 loci associated with mLOY at a genome-wide significant level, 35 of which are unreported. Lead markers overlap enhancer marks in hematopoietic stem cells (HSCs, P ≤ 1.0 × 10-6). mLOY genome-wide association study signals exhibit polygenic architecture and demonstrate strong heritability enrichment in regions surrounding genes specifically expressed in multipotent progenitor (MPP) cells and HSCs (P ≤ 3.5 × 10-6). ChIP-seq data demonstrate that binding sites of FLI1, a fate-determining factor promoting HSC differentiation into platelets rather than red blood cells (RBCs), show a strong heritability enrichment (P = 1.5 × 10-6). Consistent with these findings, platelet and RBC counts are positively and negatively associated with mLOY, respectively. Collectively, our observations improve our understanding of the mechanisms underlying mLOY.

Learn more

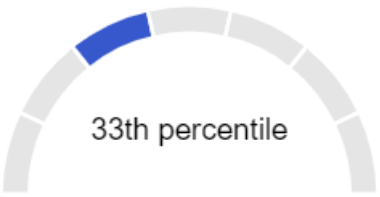
Woman Breast size (higher)

This result is based on **38 genetic variants** associated with **"Woman Breast size (higher)"** analyzed in the scientific paper **(06/30/2012 - Eriksson N)**

Reproductive

Your results

Below average genetic predisposition



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

Study description

While some factors of breast morphology, such as density, are directly implicated in breast cancer, the relationship between breast size and cancer is less clear. Breast size is moderately heritable, yet the genetic variants leading to differences in breast size have not been identified.

Learn more

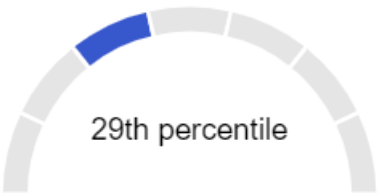
Menarche (age at onset) (earlier)

This result is based on **120 genetic variants** associated with **"Menarche (age at onset) (earlier)"** analyzed in the scientific paper **(10/02/2014 - Perry JR)**

Reproductive

Your results

Below average genetic predisposition



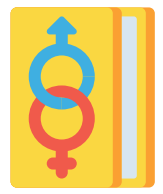
Your genetic predisposition is lower than the 71% average person from your genetic population

Study description

Age at menarche is a marker of timing of puberty in females. It varies widely between individuals, is a heritable trait and is associated with risks for obesity, type 2 diabetes, cardiovascular disease, breast cancer and all-cause mortality. Studies of rare human disorders of puberty and animal models point to a complex hypothalamic-pituitary-hormonal regulation, but the mechanisms that determine pubertal timing and underlie its links to disease risk remain unclear. Here, using genome-wide and custom-genotyping arrays in up to 182,416 women of European descent from 57 studies, we found robust evidence (P < 5 × 10(-8)) for 123 signals at 106 genomic loci associated with age at menarche. Many loci were associated with other pubertal traits in both sexes, and there was substantial overlap with genes implicated in body mass index and various diseases, including rare disorders of puberty. Menarche signals were enriched in imprinted regions, with three loci (DLK1-WDR25, MKRN3-MAGEL2 and KCNK9) demonstrating parent-of-origin-specific associations concordant with known parental expression patterns. Pathway analyses implicated nuclear hormone receptors, particularly retinoic acid and γ-aminobutyric acid-B2 receptor signalling, among novel mechanisms that regulate pubertal timing in humans. Our findings suggest a genetic architecture involving at least hundreds of common variants in the coordinated timing of the pubertal transition.

Learn more

Gestational age at birth (maternal effect)

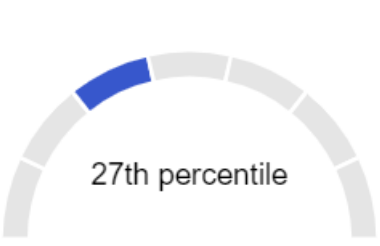


Reproductive

This result is based on **11 genetic variants** associated with **"Gestational age at birth (maternal effect)"** analyzed in the scientific paper **(09/21/2017 - Zhang G)**

Your results

Below average genetic predisposition



Your genetic predisposition is lower than the 73% average person from your genetic population

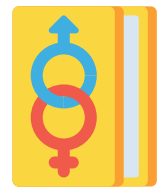
Study description

Despite evidence that genetic factors contribute to the duration of gestation and the risk of preterm birth, robust associations with genetic variants have not been identified. We used large data sets that included the gestational duration to determine possible genetic associations.

Learn more

Number of sexual partners

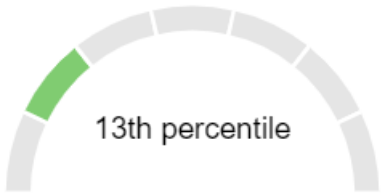
This result is based on **117 genetic variants** associated with **"Number of sexual partners"** analyzed in the scientific paper **(2019 Feb - Karlsson Linnér R)**



Reproductive

Your results

Low genetic predisposition



Your genetic predisposition is lower than the 87% average person from your genetic population

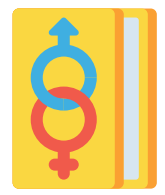
Study description

Humans vary substantially in their willingness to take risks. In a combined sample of over 1 million individuals, we conducted genome-wide association studies (GWAS) of general risk tolerance, adventurousness, and risky behaviors in the driving, drinking, smoking, and sexual domains. Across all GWAS, we identified hundreds of associated loci, including 99 loci associated with general risk tolerance. We report evidence of substantial shared genetic influences across risk tolerance and the risky behaviors: 46 of the 99 general risk tolerance loci contain a lead SNP for at least one of our other GWAS, and general risk tolerance is genetically correlated ([Formula: see text] ~ 0.25 to 0.50) with a range of risky behaviors. Bioinformatics analyses imply that genes near SNPs associated with general risk tolerance are highly expressed in brain tissues and point to a role for glutamatergic and GABAergic neurotransmission. We found no evidence of enrichment for genes previously hypothesized to relate to risk tolerance.

Learn more

Pubertal anthropometrics

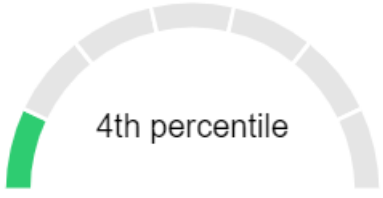
This result is based on **24 genetic variants** associated with **"Pubertal anthropometrics"** analyzed in the scientific paper **(07/01/2013 - Cousminer DL)**



Reproductive

Your results

Very low genetic predisposition



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

Study description

The pubertal height growth spurt is a distinctive feature of childhood growth reflecting both the central onset of puberty and local growth factors. Although little is known about the underlying genetics, growth variability during puberty correlates with adult risks for hormone-dependent cancer and adverse cardiometabolic health. The only gene so far associated with pubertal height growth, LIN28B, pleiotropically influences childhood growth, puberty and cancer progression, pointing to shared underlying mechanisms. To discover genetic loci influencing pubertal height and growth and to place them in context of overall growth and maturation, we performed genome-wide association meta-analyses in 18 737 European samples utilizing longitudinally collected height measurements. We found significant associations (P < 1.67 × 10(-8)) at 10 loci, including LIN28B. Five loci associated with pubertal timing, all impacting multiple aspects of growth. In particular, a novel variant correlated with expression of MAPK3, and associated both with increased

prepubertal growth and earlier menarche. Another variant near ADCY3-POMC associated with increased body mass index, reduced pubertal growth and earlier puberty. Whereas epidemiological correlations suggest that early puberty marks a pathway from rapid prepubertal growth to reduced final height and adult obesity, our study shows that individual loci associating with pubertal growth have variable longitudinal growth patterns that may differ from epidemiological observations. Overall, this study uncovers part of the complex genetic architecture linking pubertal height growth, the timing of puberty and childhood obesity and provides new information to pinpoint processes linking these traits.

Learn more