DEMO REPORT

Metabolism

Metabolism, the sum of the chemical reactions that take place within each cell of a living organism and that provide energy for vital processes and for synthesizing new organic material.

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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5 entries

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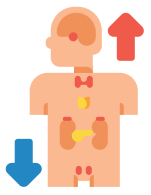
Metabolic Response

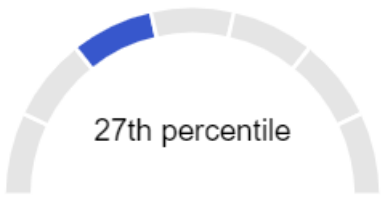


Metabolism

This result is based on **1 genetic variants** associated with **"Metabolic Response"** analyzed in the scientific paper (01/07/2020 - Oh SW)

Your results

Below average genetic predisposition



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

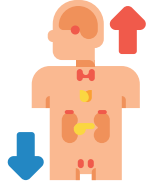
Study description

Metabolic syndrome (MetS) which is caused by obesity and insulin resistance, is well known for its predictive capability for the risk of type 2 diabetes mellitus and cardiovascular disease. The development of MetS is associated with multiple genetic factors, environmental factors and lifestyle. We performed a genome-wide association study to identify single-nucleotide polymorphism (SNP) related to MetS in large Korean population based samples of 1,362 subjects with MetS and 6,061 controls using the Axiom® Korean Biobank Array 1.0. We replicated the data in another sample including 502 subjects with MetS and 1,751 controls. After adjusting for age and sex, rs662799 located in the APOA5 gene were significantly associated with MetS. 15 SNPs in GCKR, C2orf16, APOA5, ZPR1, and BUD13 were associated with high triglyceride (TG). 14 SNPs in APOA5, ALDH1A2, LIPC, HERPUD1, and CETP, and 2 SNPs in MTNR1B were associated with low high density lipoprotein cholesterol (HDL-C) and high fasting blood glucose respectively. Among these SNPs, 6 TG SNPs: rs1260326, rs1260333, rs1919127, rs964184, rs2075295 and rs1558861 and 11 HDL-C SNPs: rs4775041, rs10468017, rs1800588, rs72786786, rs173539, rs247616, rs247617, rs3764261, rs4783961, rs708272, and rs7499892 were first discovered in Koreans. Additional research is needed to confirm these 17 novel SNPs in Korean population.

Learn more

Energy expenditure

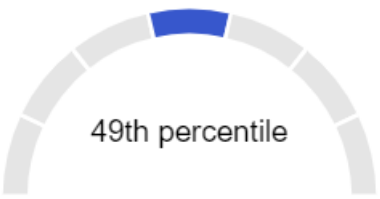
This result is based on **8 genetic variants** associated with **"Energy expenditure"** analyzed in the scientific paper (08/02/2018 - Jiang L)



Metabolism

Your results

Average genetic predisposition



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

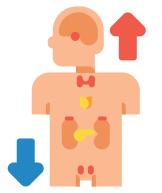
Study description

Excessive energy intake or insufficient energy expenditure, which result in energy imbalance, contribute to the development of obesity. Obesity-related genes, such as FTO, are associated with energy traits. No genome-wide association studies (GWAS) have been conducted to detect the genetic associations with energy-related traits, including energy intake and energy expenditure, among European-ancestry populations. In this study, we conducted a genome-wide study using pooled GWAS including 12,030 European-ancestry women and 6,743 European-ancestry men to identify genetic variants associated with these two energy traits. We observed a statistically significant genome-wide SNP heritability for energy intake of 6.05% (95%CI = (1.76, 10.34), P = 0.006); the SNP heritability for expenditure was not statistically significantly greater than zero. We discovered three SNPs on chromosome 12q13 near gene ANKRD33 that were genome-wide significantly associated with increased total energy intake among all men. We also identified signals on region 2q22 that were associated with energy expenditure among lean people. Body mass index related SNPs were found to be significantly associated with energy intake and expenditure through SNP set analyses. Larger GWAS studies of total energy traits are warranted to explore the genetic basis of energy intake, including possible differences between men and women, and the association between total energy intake and other downstream phenotypes, such as diabetes and chronic diseases.

Learn more

Weight

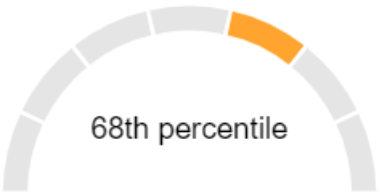
This result is based on **198 genetic variants** associated with **"Weight"** analyzed in the scientific paper [(06/01/2017 - Tachmazidou I)]



Metabolism

Your results

Above average genetic predisposition



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

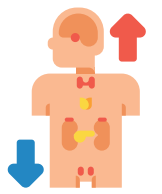
Study description

Deep sequence-based imputation can enhance the discovery power of genome-wide association studies by assessing previously unexplored variation across the common- and low-frequency spectra. We applied a hybrid whole-genome sequencing (WGS) and deep imputation approach to examine the broader allelic architecture of 12 anthropometric traits associated with height, body mass, and fat distribution in up to 267,616 individuals. We report 106 genome-wide significant signals that have not been previously identified, including 9 low-frequency variants pointing to functional candidates. Of the 106 signals, 6 are in genomic regions that have not been implicated with related traits before, 28 are independent signals at previously reported regions, and 72 represent previously reported signals for a different anthropometric trait. 71% of signals reside within genes and fine mapping resolves 23 signals to one or two likely causal variants. We confirm genetic overlap between human monogenic and polygenic anthropometric traits and find signal enrichment in cis expression QTLs in relevant tissues. Our results highlight the potential of WGS strategies to enhance biologically relevant discoveries across the frequency spectrum.

Learn more

Lean body mass

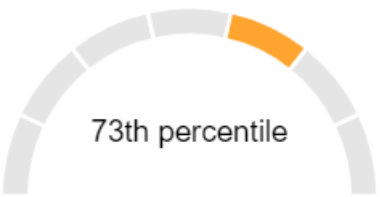
This result is based on **76 genetic variants** associated with **"Lean body mass"** analyzed in the scientific paper [(06/01/2017 - Tachmazidou I)]



Metabolism

Your results

Above average genetic predisposition



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

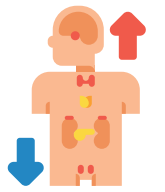
Study description

Deep sequence-based imputation can enhance the discovery power of genome-wide association studies by assessing previously unexplored variation across the common- and low-frequency spectra. We applied a hybrid whole-genome sequencing (WGS) and deep imputation approach to examine the broader allelic architecture of 12 anthropometric traits associated with height, body mass, and fat distribution in up to 267,616 individuals. We report 106 genome-wide significant signals that have not been previously identified, including 9 low-frequency variants pointing to functional candidates. Of the 106 signals, 6 are in genomic regions that have not been implicated with related traits before, 28 are independent signals at previously reported regions, and 72 represent previously reported signals for a different anthropometric trait. 71% of signals reside within genes and fine mapping resolves 23 signals to one or two likely causal

variants. We confirm genetic overlap between human monogenic and polygenic anthropometric traits and find signal enrichment in cis expression QTLs in relevant tissues. Our results highlight the potential of WGS strategies to enhance biologically relevant discoveries across the frequency spectrum.

Learn more

Childhood obesity

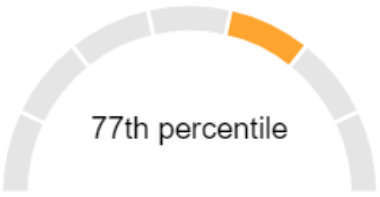


Metabolism

This result is based on **57 genetic variants** associated with **"Childhood obesity"** analyzed in the scientific paper **(10/01/2019 - Bradfield JP)**

Your results

Above average genetic predisposition



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

Study description

Although hundreds of genome-wide association studies-implicated loci have been reported for adult obesity-related traits, less is known about the genetics specific for early-onset obesity and with only a few studies conducted in non-European populations to date. Searching for additional genetic variants associated with childhood obesity, we performed a trans-ancestral meta-analysis of 30 studies consisting of up to 13 005 cases (≥95th percentile of body mass index (BMI) achieved 2-18 years old) and 15 599 controls (consistently <50th percentile of BMI) of European, African, North/South American and East Asian ancestry. Suggestive loci were taken forward for replication in a sample of 1888 cases and 4689 controls from seven cohorts of European and North/South American ancestry. In addition to observing 18 previously implicated BMI or obesity loci, for both early and late onset, we uncovered one completely novel locus in this trans-ancestral analysis (nearest gene, METTL15). The variant was nominally associated with only the European subgroup analysis but had a consistent direction of effect in other ethnicities. We then utilized trans-ancestral Bayesian analysis to narrow down the location of the probable causal variant at each genome-wide significant signal. Of all the fine-mapped loci, we were able to narrow down the causative variant at four known loci to fewer than 10 single nucleotide polymorphisms (SNPs) (FAIM2, GNPDA2, MC4R and SEC16B loci). In conclusion, an ethnically diverse setting has enabled us to both identify an additional pediatric obesity locus and further fine-map existing loci.

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