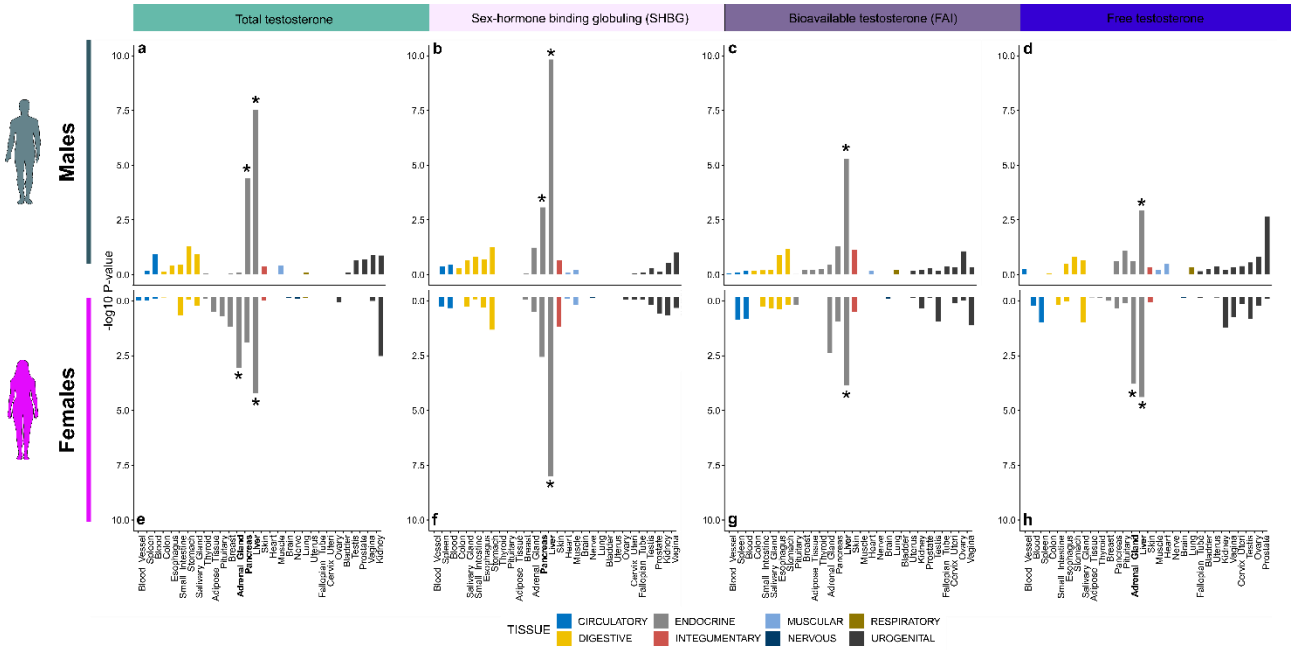
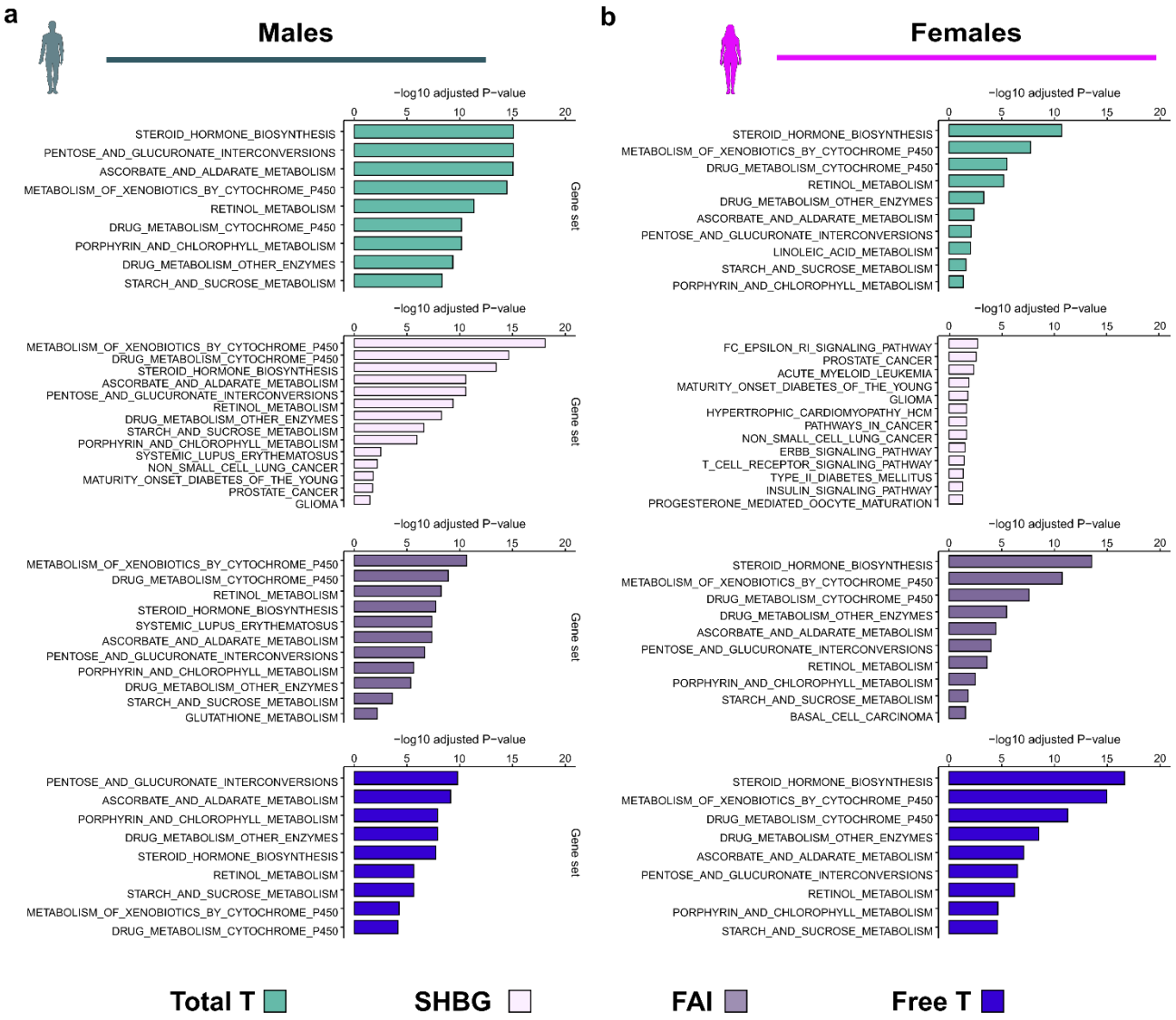
Supplementary Figures and Legends

Supplementary Figure 1. Results from tissue enrichment analyses from MAGMA implemented in FUMA



Panels **a-d** show tissue enrichment in males, and **e-h** in females. Green = total testosterone, white lilac = SHBG, grey = free androgen index (FAI), blue = free testosterone. The GTEx tissues shown in figure have been group based on tissue types, with code for tissue colors available in the figure. Based on the UK Biobank GWAS, a large proportion of the genes residing in the GWAS loci are preferably expressed in the liver. This applies to all studied traits. Interestingly, we simultaneously detected no evidence for the GWAS loci being enriched for genes expressed in the testis or the ovaries, the tissues responsible for the bulk of T production. Potentially, this can be a sign that at population level, individual differences in T metabolism may have larger effects on adult serum T levels than genetic differences in the tissues responsible for T production. Especially for males it seems thus possible that T production may not be the rate-limiting step for T levels. However, the situation may be slightly different in females, where adrenal gland is a major site for T production, and many genes in the loci associated with total and free T in females appeared preferably expressed in this tissue. Underscoring the close relationship between metabolism and T levels, pancreas also shows up as a tissue enriched for the GWAS signals for total T and SHBG in males.

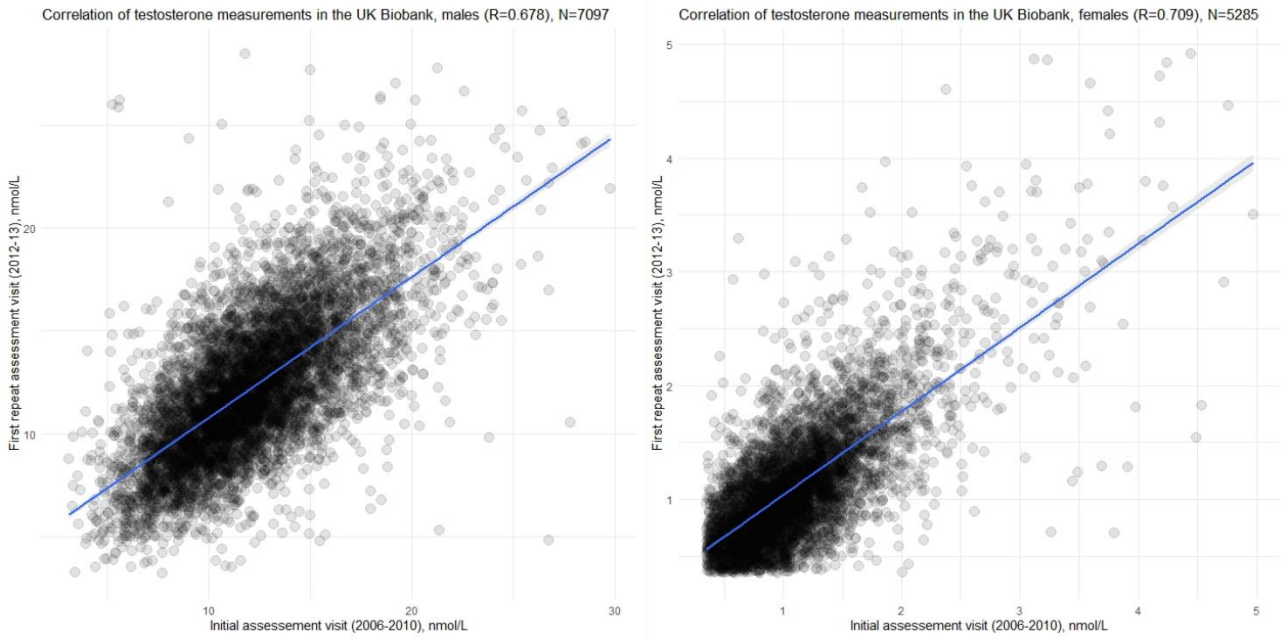
Supplementary Figure 2. Visualisation of results from KEGG pathway analyses in FUMA



The figure illustrates Kyoto Encyclopedia of Genes and Genomes (KEGG) pathways showing enrichment for the genes residing in the GWAS loci. Panel **a** shows the results for males, and **b** for females. Green = enriched pathways for total testosterone, white lilac = SHBG, grey = free androgen index (FAI), blue = free testosterone. These results are based on statistically significant enrichment on KEGG pathways, after adjusting for multiple testing in FUMA (Supplementary Table 10). The GWAS loci show clear enrichment for genes that affect steroid hormone biosynthesis, various metabolic pathways and metabolite excretion. Overall these results highlight that the genetic variation detected in the GWASs relates to many molecular pathways that are known to be crucial for T and SHBG processing and regulation. Importantly, these results also underscore that no single pathway dominates T regulation, but that genetic regulation of T levels in the human body likely results from simultaneous and combined action of many molecular processes. Notably, the results support the observations from the tissue enrichment analyses, emphasizing the significance of T metabolism (for which the major site of action is

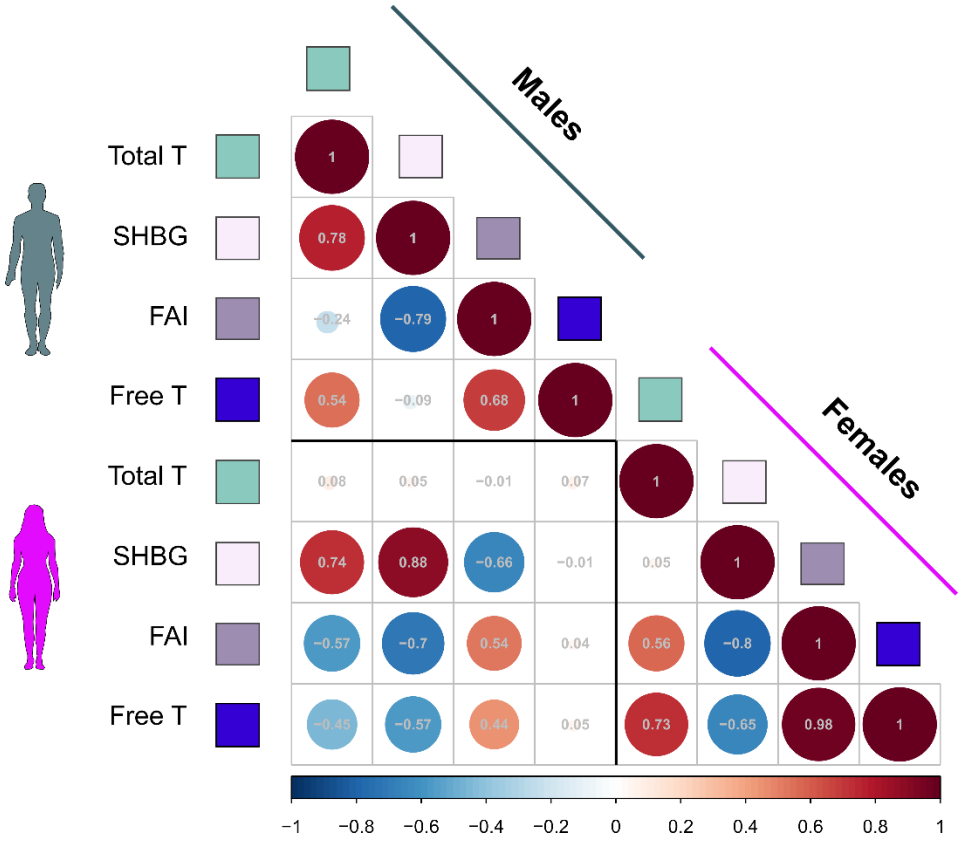
liver) to T levels, likely via specific and unspecific mechanisms. T=Testosterone, SHBG = Sex-hormone binding globulin, FAI = Free androgen index, Free T = calculated free testosterone.

Supplementary Figure 3. Correlation between testosterone levels ~5 years apart for a subset of UK Biobank participants



For both men (N=7,097) and women (N=5,285), testosterone (T) measurements from roughly five years apart are highly correlated (R=0.678 in men, R=0.709 in women) in the UK Biobank. The grey circles indicate individual measurements at time point 1 (original visit, x-axis) vs. time point 2 (repeat assessment, y-axis). The high correlations of T measurements between the two distinct time points provide strong evidence that the immunohistochemical method used reliably captures population-level variability in T levels. These correlations support also the concept that there exists a fairly stable individual-level baseline for T, known to be largely heritable in basis, reflecting an individual's life-long T exposure. Further supporting such a baseline for T levels, measurement at the first visit, several years prior the second, predicts T level at the second visit considerably better than for instance BMI from the current visit (Supplementary Figure 10). For visual purposes, the individuals with a likely medical condition leading to extremely low or high testosterone (<3 in men and >5 in women) were removed from the plots.

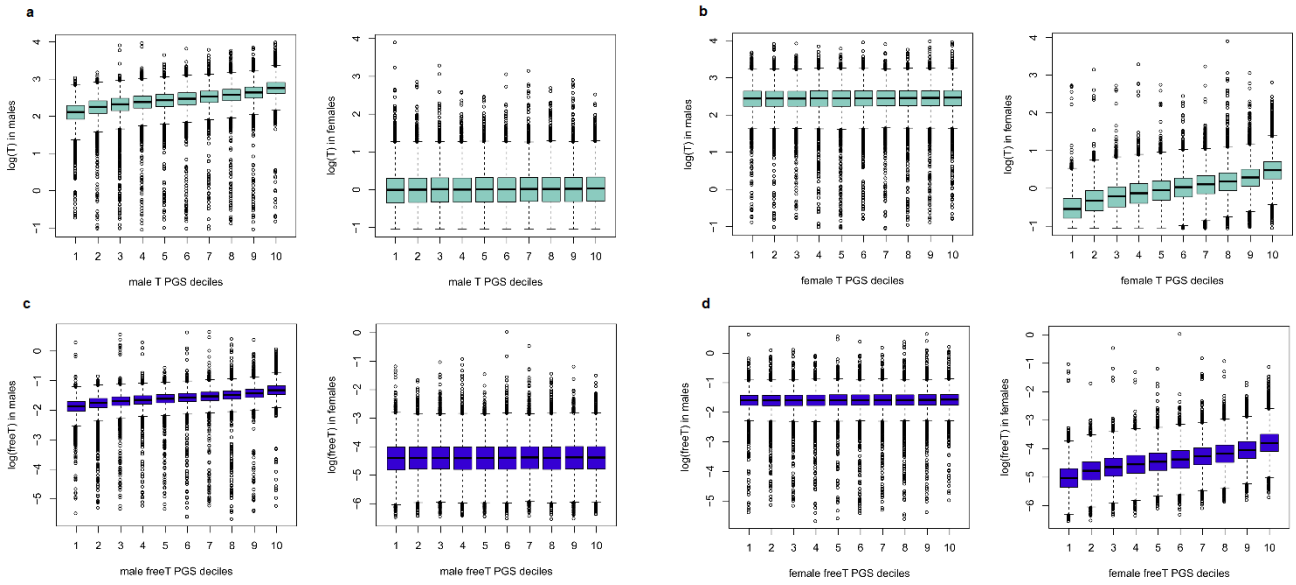
Supplementary Figure 4. Genetic correlation results between the studied testosterone traits



The figure shows genetic correlations based on LDSC analyses for males (area next to dark green symbol), females (pink) and over both sexes (bottom left corner surrounded by black lines in the correlation grid). Green = total testosterone, white lilac = SHBG, grey = free androgen index (FAI), blue = free testosterone. In line with recent findings, we observed only a very weak genetic correlation for total T levels between males and females (r_g=0.08, p=0.063). Similarly, free T showed low genetic correlation estimate between the sexes (r_g=0.05, p=0.102). This supports the concept that uniquely among complex traits, T levels are determined by distinct heritable factors in males and females, echoing results from earlier twin studies (20). In contrast, the genetic correlation for males and females SHBG was high (r_g=0.88, p=9.7e-197), and for FAI intermediate (r_g=0.57, p=5.8e-26), suggesting that similar genetic factors contribute to these traits in both sexes. Moreover, consistent with epidemiological observations that total T and SHBG levels are correlated in males, these traits showed a robust positive genetic correlation (r_g=0.78, p=1.0e-127). This observation supports the concept that under normal physiological conditions, based on homeostatic feedback SHBG levels increase when T increases and vice versa. At the same time, under the premise of unbound T being biologically the most potent form of T, this suggests that some genetic variants affecting total T in men do not necessarily reflect increased T activity. Yet, for females such connection between

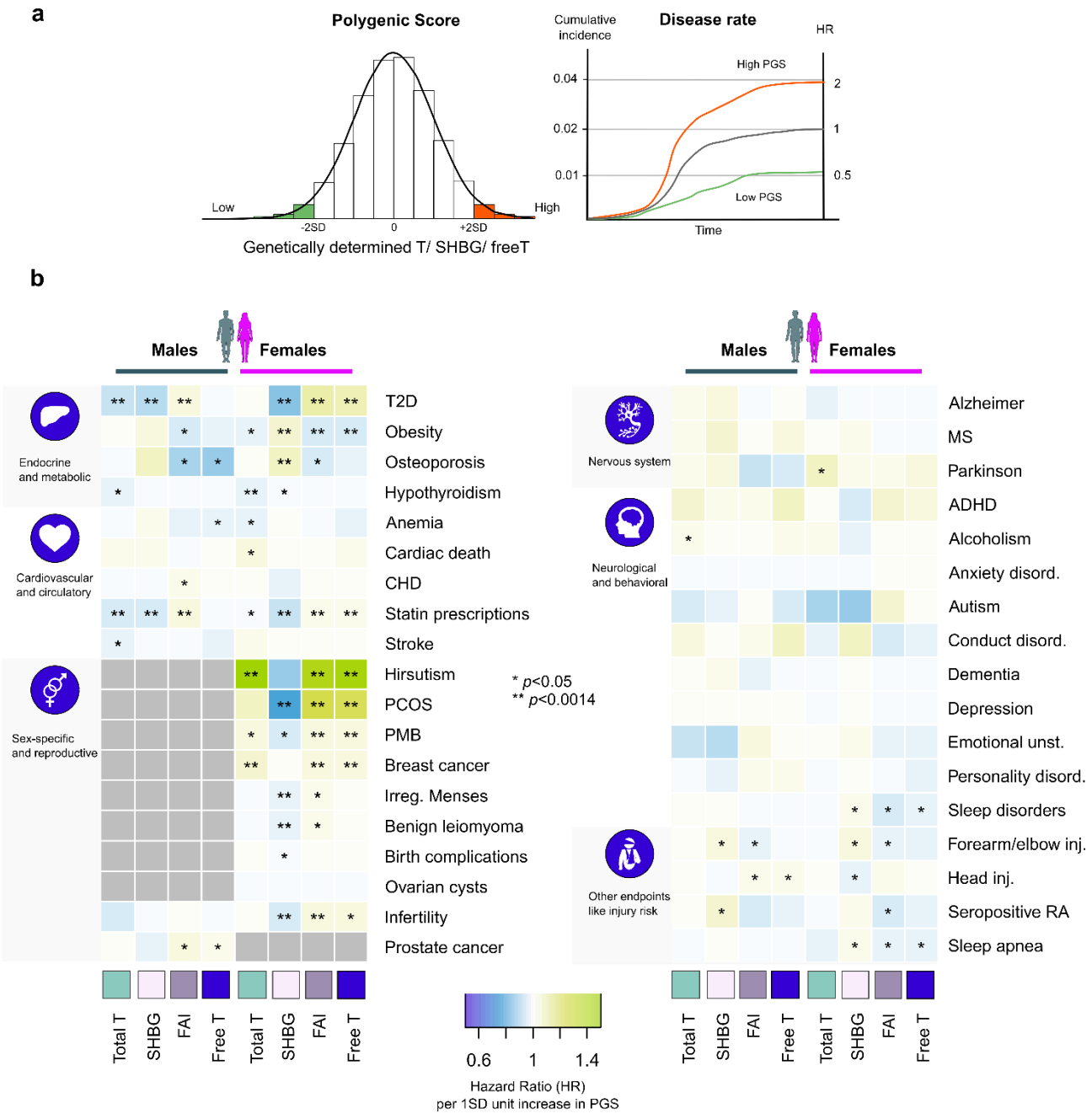
total T and SHBG did not exist (r_g=0.05, p=0.207), suggesting such feedback mechanism is not active in females, and that total T levels in females may be more closely related to T action than total T levels in males.

Supplementary Figure 5. Predictive ability of total T and free T PGSs for T levels in the UK Biobank



The figure shows how the sex-specific PGS correlates highly with T levels within the sex it was constructed, and has drastically limited predictive ability in the opposite sex. **a** Total T PGS vs T in men, **b** Total T PGS vs T in women, **c** Free T PGS in men, **d** Free T PGS in women. The box plots show median (black line), lower and upper quartiles (colored area of the box) for log T and free T per PGS decile (1=persons with T PGS in the lowest 10%, 10 = persons with T PGS higher than for 90% of the samples), and the error bars indicate 5% and 95% quantiles. Green boxplots = total testosterone values by PGS decile, blue = free testosterone values by PGS decile. Although the cross-sex comparisons in UK Biobank are performed using independent datasets (men vs. women, based on data from 159,110 unrelated men and 184,573 unrelated women with white British ancestry), please note that the training (PGS calculation) and the test (explained variance) datasets for the sex-specific PGSs include the same individuals from the UK Biobank, leading to inflated results.

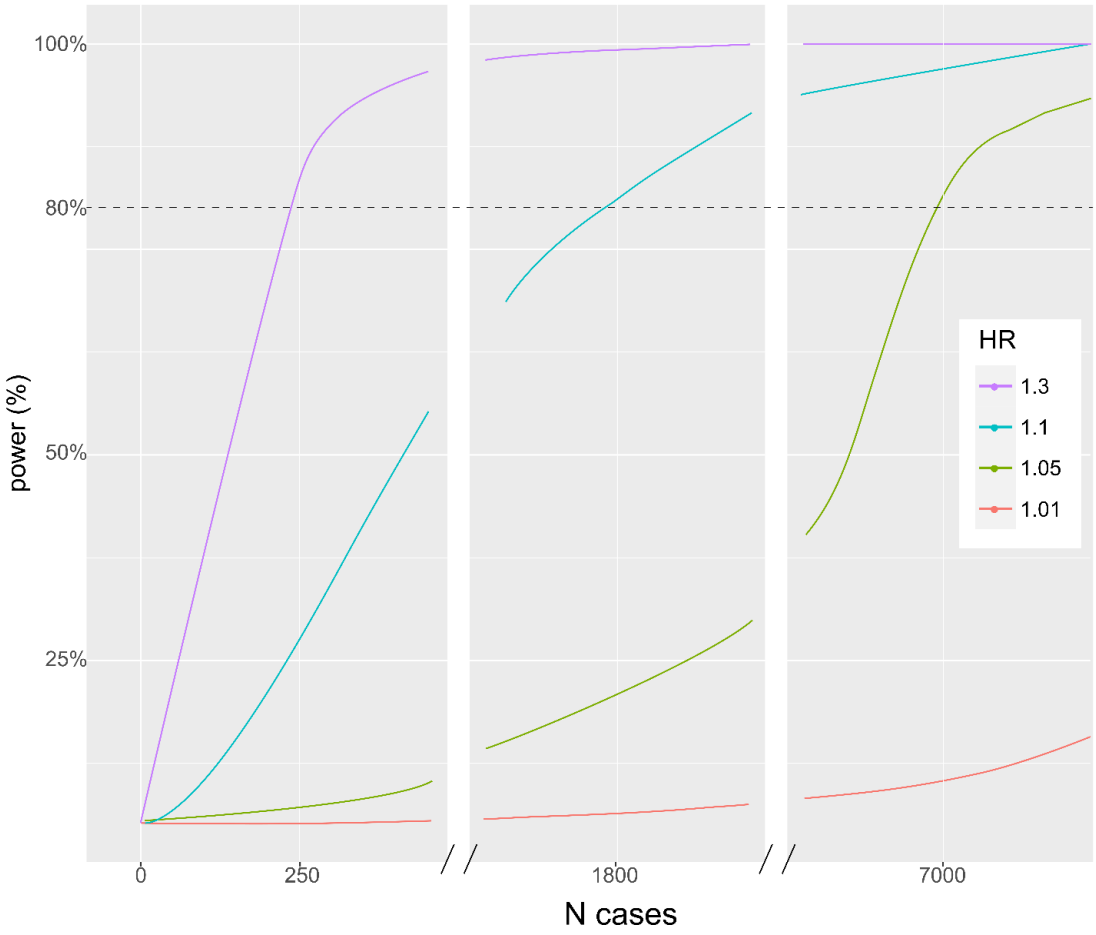
Supplementary Figure 6. PGS associations in the FinnGen



a Rationale of the PGS analyses. Calculation of the PGS in the FinnGen allows for ranking participants according to their genetic predisposition to higher T/ SHBG and free T levels. Then, the consequences of having either high/low genetically determined T can be estimated using the clinical information available from the FinnGen dataset. The figure on the right shows an example case where having a high PGS (2SD above population mean, orange) doubles the hazard ratio (HR) of getting diagnosed with a disease compared to those with an average PGS (grey line). The line green indicates those with low PGS (2SD below population mean) have only half of the risk of getting the diagnosis. **b** The heatmap illustrates hazard

ratios (HR) per 1SD increase in PGS for all studied traits in the sex-specific analyses. $p<0.0014$ corresponds to Bonferroni correction for 36 traits. Dark green symbol = males, pink = females. Green circles = total testosterone, white lilac = SHBG, grey = free androgen index, blue = free testosterone. Yellow shades = increased risk for endpoint, blue shades = decreased risk for endpoint. Grey box = endpoint not available for this sex. The data is based on 94,478 men and 122,986 women from FinnGen R5.

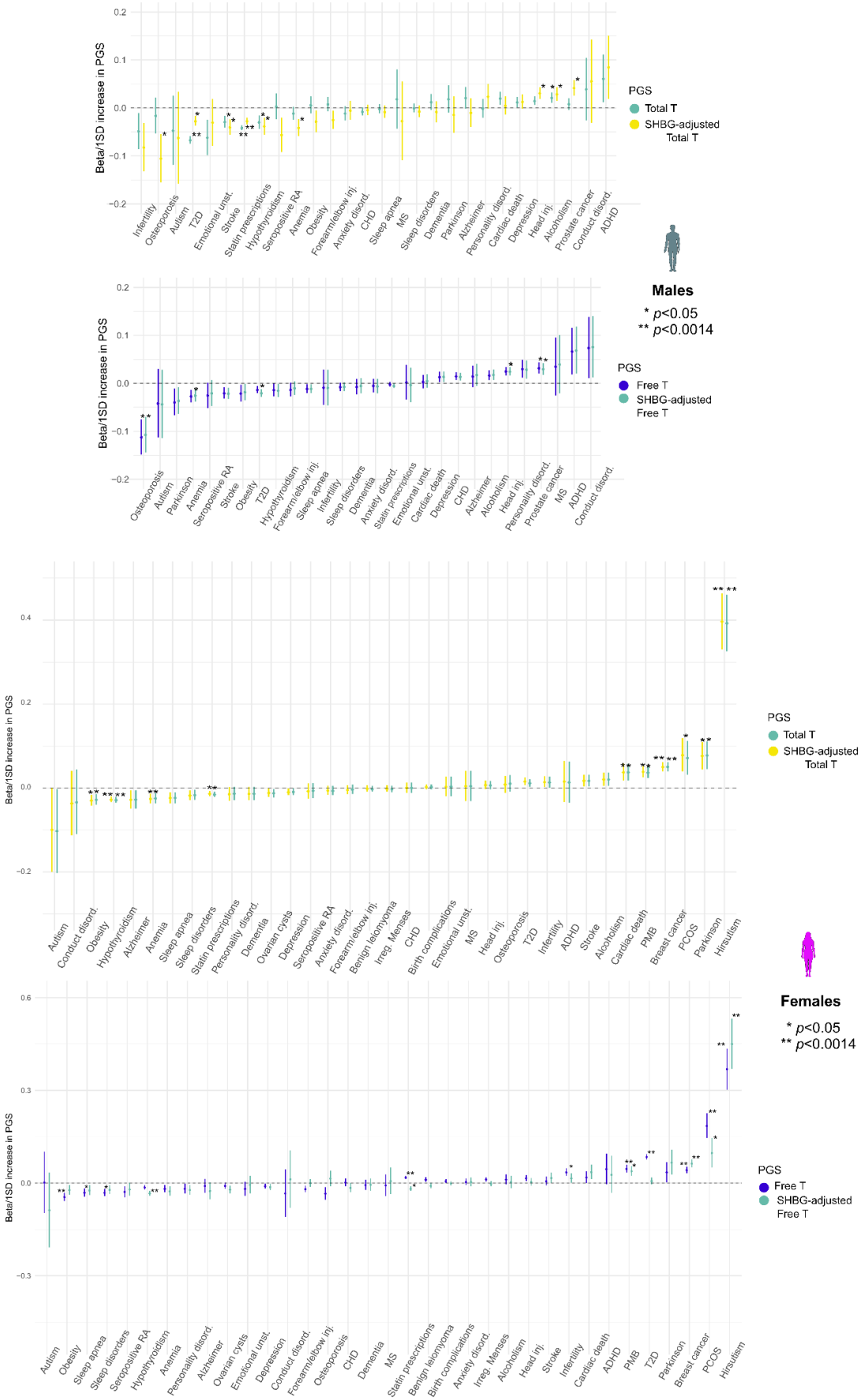
Supplementary Figure 7. Schematic illustration of the power analysis for FinnGen disease associations



We estimate that even for the rarest phenotypes studied, given the selected *p*-value threshold (0.0014), we had full power to detect any large effects (HR>1.3) for the PGSs, and that we could reliably detect also subtler effects for most phenotypes. To exemplify this in practical terms, for rare diagnoses such as hirsutism and conduct disorder we had adequate power (>80%) to detect large effects (HR>1.3, purple line). We could detect at least intermediate effects (HR>1.1, blue line) for the vast majority of the disease endpoints (over 1800 cases, 49/64 of the studied sex-specific endpoints (64 = the count of the studied disease endpoints when a sex-shared endpoint is considered as two independent endpoints, e.g. both male and female statin use are added to total sum). For the most common phenotypes like T2D and depression (more than 7000 cases, representing roughly a third of all included endpoints (20/64), we had the power to catch even smaller effects (HR>1.05, green line). We did not have the power to detect very small effects per 1SD change in PGS (HR<1.01, red line), yet such effects would be likely considered negligible in terms of medical relevance. The HR refers to risk per 1SD increase in the PGS for the studied T trait (total T, SHBG, FAI or free T) based on the PGS analysis in FinnGen. The curves are based on the selected *p*-value threshold of 0.0014, corresponding for multiple testing correction for 36 traits, and on power

calculation for Cox proportional Hazards Regression with powerSurvEpi package in R. Phenotypes and their N are listed in Supplementary Data 7 for both sexes separately. Please note that the X-axis marking case number is not continuous.

Supplementary Figure 8. Comparison of unadjusted and SHBG-adjusted T PGS associations in FinnGen.



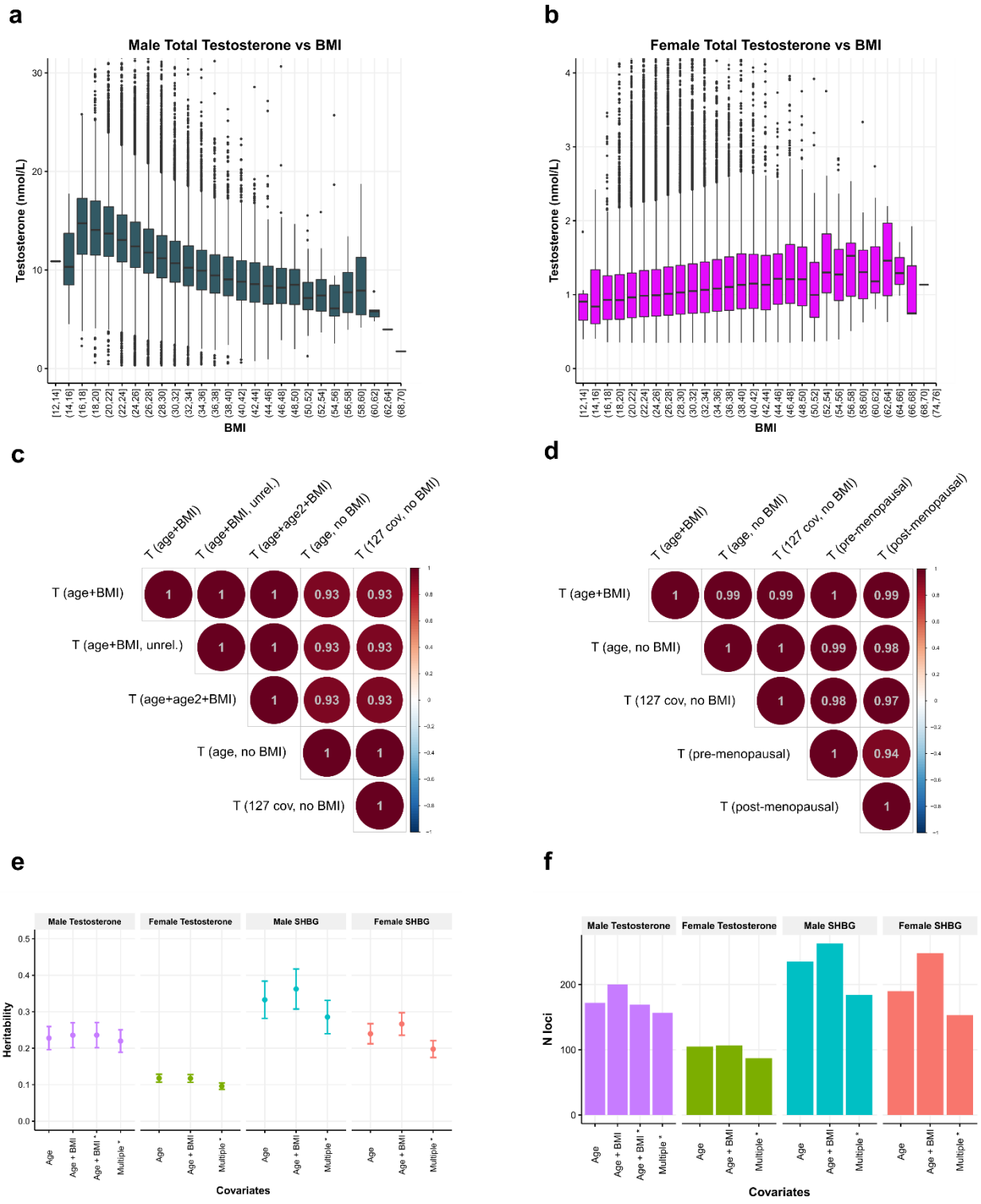
The figure shows disease risk per 1SD increase in PGS in a forest plot (beta and SE). Green = total T, yellow = SHBG-adjusted total T, blue = free T, light green = SHBG adjusted free T. The data is based on PGS association analyses comprising of 94,478 men and 122,986 women from FinnGen R5. For some endpoints, the effects between the unadjusted and the adjusted analysis appear different (e.g. only SHBG-adjusted total T showing nominally significant association to osteoporosis and prostate cancer in males). This suggests that the association of the unadjusted total T PGS to these endpoints suffers from the limitation that the PGS also reflects raised SHBG levels, which for these endpoints counteracts the effects of raised total T. This leads to at least two potential interpretations: first, this can be a sign of genetic pleiotropy, e.g. some genetic variants contributing to total T PGS affect both T and SHBG independently, or via another molecular pathway. Secondly, such a result may be a sign that a genetically set raise in SHBG leads to a biological compensation and raise in T levels (or vice versa), increased T binding by SHBG then counteracting the raise in T levels, and masking any associations that might result from direct (biologically available) T action.

Suggesting that both total T and SHBG in females likely contribute independently to PCOS, total T shows consistent association to PCOS both with and without SHBG adjustment, but free T association to PCOS is attenuated after SHBG adjustment. Notably, the statistically significant T associations to T2D disappear in both sexes (total T in men, free T in women) when taking into account the effect of SHBG in the analysis. For most other traits, the effects estimates remain highly similar between the unadjusted and adjusted analyses.

Supplementary Figure 9. Visualisation of genetic correlations and causality analyses between T, SHBG, FAI and free T and the studied disease endpoints in FinnGen

Heatmap colors indicate the direction and the strength of the genetic correlation (purple, negative correlation, green, positive correlation) based on LCV. Green = total testosterone, white lilac = SHBG, grey = free androgen index, blue = free testosterone. Positive genetic correlation means that same genetic variants that increase testosterone/SHBG increase also the risk for endpoint X. Asterisks indicate statistical support ($p<0.05$ and $p<0.0014$) for causality in either LCV or MR Egger analyses (Supplementary Data 8). Correlation without causation suggests that the genetic connection between the traits is likely mediated by genetic pleiotropy or that the causal effect is simply too weak to be detected. For both grids, the effects of male PGS on the studied trait is shown on left hand side (under green line), and the effect of the female PGS on the right (under pink line). Please note that this figure also illustrates the effects of male PGS for female traits, and vice versa. The data is based on PGS association analyses comprising of 94,478 men and 122,986 women from FinnGen R5.

Supplementary Figure 10. The effects of body mass index (BMI) and other covariates on testosterone (T) levels and GWAS results in the UK Biobank



Panels **a** and **b** show the relationship between serum total T levels and BMI (binned in two unit intervals) in the UK Biobank for males (**a,** green boxplots) and females (**b,** pink boxplots), based on 177,186 men and 175,435 women participants with

white British ancestry and testosterone measurements available. For men, a significant negative correlation exists between BMI and T levels (R=-0.295, p=0), whereas this relationship is reversed in women (R=0.081, p=2.9e-230). The box plots show median (black line) and lower and upper quartiles (colored area of the box) for serum total T, and the error bars indicate 5% and 95% quantiles. For visual purposes, the Y-axis is capped at 30nmol/l in men and 4nmol/l in women. Panels **c** and **d** show genetic correlation from LDSC between total T GWAS runs under varying covariate combinations in men (**c,** N=176,212) and women (**d,** N=174,850), implying BMI has the largest effects on results, and that for example menopause status has only negligible effects on the genetic findings. N pre-menopausal women included in study = 48,876; N post-menopausal = 136,236. Dark red indicates high genetic correlation. Adding BMI as a covariate increased both the estimated heritability (LDSC) and the number of loci found for both total T and SHBG. Panel **e** shows heritability estimates with SE**,** and **f** shows the number of significant GWAS hits under different models in a bar plot. Lilac = total testosterone in men, green = total testosterone in women, turquoise = SHBG in men, red = SHBG in women. In panels **e** and **f** * denotes a model with no relatives included in the analysis, and "multiple" refers to including 127 covariates from Sinnott-Armstrong et al. 2019. The results suggest that by using BMI as a covariate we can better capture any genetic variants affecting the studied traits in both sexes, and that the additional 127 covariates (for example, sample dilution factors) have negligible effects on the GWAS results.

Supplementary Information

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Neha Raghavan	Merck, Kenilworth, NJ, United States	Clinical Groups	Neurology Group
Martti Färkkilä	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Gastroenterology Group
Jukka Koskela	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Gastroenterology Group
Sampsa Pikkarainen	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Gastroenterology Group
Airi Jussila	Pirkanmaa Hospital District, Tampere, Finland	Clinical Groups	Gastroenterology Group
Katri Kaukinen	Pirkanmaa Hospital District, Tampere, Finland	Clinical Groups	Gastroenterology Group
Timo Blomster	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Gastroenterology Group
Mikko Kiviniemi	Northern Savo Hospital District, Kuopio, Finland	Clinical Groups	Gastroenterology Group
Markku Voutilainen	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Gastroenterology Group
Mark Daly	Institute for Molecular Medicine Finland (FIMM), HiLIFE, University of Helsinki, Helsinki; Broad Institute of MIT and Harvard; Massachusetts General Hospital, Boston MA, United States	Clinical Groups	Gastroenterology Group
Ali Abbasi	Abbvie, Chicago, IL, United States	Clinical Groups	Gastroenterology Group
Jeffrey Waring	Abbvie, Chicago, IL, United States	Clinical Groups	Gastroenterology Group
Nizar Smaoui	Abbvie, Chicago, IL, United States	Clinical Groups	Gastroenterology Group
Fedik Rahimov	Abbvie, Chicago, IL, United States	Clinical Groups	Gastroenterology Group
Anne Lehtonen	Abbvie, Chicago, IL, United States	Clinical Groups	Gastroenterology Group
Tim Lu	Genentech, San Francisco, CA, United States	Clinical Groups	Gastroenterology Group
Natalie Bowers	Genentech, San Francisco, CA, United States	Clinical Groups	Gastroenterology Group
Rion Pendergrass	Genentech, San Francisco, CA, United States	Clinical Groups	Gastroenterology Group
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Kirsi Kalpala	Pfizer, New York, NY, United States	Clinical Groups	Gastroenterology Group
Melissa Miller	Pfizer, New York, NY, United States	Clinical Groups	Gastroenterology Group
Xinli Hu	Pfizer, New York, NY, United States	Clinical Groups	Gastroenterology Group
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Pia Isomäki	Pirkanmaa Hospital District, Tampere, Finland	Clinical Groups	Rheumatology Group
Laura Pirilä	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Rheumatology Group
Oili Kaipiainen-Seppänen	Northern Savo Hospital District, Kuopio, Finland	Clinical Groups	Rheumatology Group
Johanna Huhtakangas	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Rheumatology Group
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Jeffrey Waring	Abbvie, Chicago, IL, United States	Clinical Groups	Rheumatology Group
Fedik Rahimov	Abbvie, Chicago, IL, United States	Clinical Groups	Rheumatology Group
Apinya Lertratanakul	Abbvie, Chicago, IL, United States	Clinical Groups	Rheumatology Group
Nizar Smaoui	Abbvie, Chicago, IL, United States	Clinical Groups	Rheumatology Group
Anne Lehtonen	Abbvie, Chicago, IL, United States	Clinical Groups	Rheumatology Group
Marla Hochfeld	Bristol Myers Squibb, New York, NY, United States	Clinical Groups	Rheumatology Group
Natalie Bowers	Genentech, San Francisco, CA, United States	Clinical Groups	Rheumatology Group
Rion Pendergrass	Genentech, San Francisco, CA, United States	Clinical Groups	Rheumatology Group
Jorge Esparza Gordillo	GlaxoSmithKline, Brentford, United Kingdom	Clinical Groups	Rheumatology Group
Kirsi Auro	GlaxoSmithKline, Espoo, Finland	Clinical Groups	Rheumatology Group
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Kirsi Kalpala	Pfizer, New York, NY, United States	Clinical Groups	Rheumatology Group
Nan Bing	Pfizer, New York, NY, United States	Clinical Groups	Rheumatology Group
Xinli Hu	Pfizer, New York, NY, United States	Clinical Groups	Rheumatology Group
Tarja Laitinen	Pirkanmaa Hospital District, Tampere, Finland	Clinical Groups	Pulmonology Group
Margit Pelkonen	Northern Savo Hospital District, Kuopio, Finland	Clinical Groups	Pulmonology Group
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Nizar Smaoui	Abbvie, Chicago, IL, United States	Clinical Groups	Pulmonology Group
Glenda Lassi	Astra Zeneca, Cambridge, United Kingdom	Clinical Groups	Pulmonology Group
Susan Eaton	Biogen, Cambridge, MA, United States	Clinical Groups	Pulmonology Group
Hubert Chen	Genentech, San Francisco, CA, United States	Clinical Groups	Pulmonology Group
Rion Pendergrass	Genentech, San Francisco, CA, United States	Clinical Groups	Pulmonology Group
Natalie Bowers	Genentech, San Francisco, CA, United States	Clinical Groups	Pulmonology Group
Joanna Betts	GlaxoSmithKline, Brentford, United Kingdom	Clinical Groups	Pulmonology Group
Kirsi Auro	GlaxoSmithKline, Espoo, Finland	Clinical Groups	Pulmonology Group
Rajashree Mishra	GlaxoSmithKline, Brentford, United Kingdom	Clinical Groups	Pulmonology Group
Majd Mouded	Novartis, Basel, Switzerland	Clinical Groups	Pulmonology Group

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Teemu Niiranen	Finnish Institute for Health and Welfare (THL), Helsinki, Finland	Clinical Groups	Cardiometabolic Diseases Group
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Jenni Aittokallio	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Cardiometabolic Diseases Group
Jussi Hernesniemi	Pirkanmaa Hospital District, Tampere, Finland	Clinical Groups	Cardiometabolic Diseases Group
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Benjamin Challis	Astra Zeneca, Cambridge, United Kingdom	Clinical Groups	Cardiometabolic Diseases Group
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Natalie Bowers	Genentech, San Francisco, CA, United States	Clinical Groups	Cardiometabolic Diseases Group
Rion Pendergrass	Genentech, San Francisco, CA, United States	Clinical Groups	Cardiometabolic Diseases Group
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Jaakko Parkkinen	Pfizer, New York, NY, United States	Clinical Groups	Cardiometabolic Diseases Group
Melissa Miller	Pfizer, New York, NY, United States	Clinical Groups	Cardiometabolic Diseases Group
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Heikki Joensuu	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Oncology Group
Olli Carpén	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Oncology Group
Johanna Mattson	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Oncology Group
Eveliina Salminen	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Oncology Group
Annika Auranen	Pirkanmaa Hospital District , Tampere, Finland	Clinical Groups	Oncology Group
Peeter Karihtala	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Oncology Group
Päivi Auvinen	Northern Savo Hospital District, Kuopio, Finland	Clinical Groups	Oncology Group
Klaus Elenius	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Oncology Group
Johanna Schleutker	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Oncology Group
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Jeffrey Waring	Abbvie, Chicago, IL, United States	Clinical Groups	Oncology Group
Bridget Riley-Gillis	Abbvie, Chicago, IL, United States	Clinical Groups	Oncology Group
Anne Lehtonen	Abbvie, Chicago, IL, United States	Clinical Groups	Oncology Group
Jennifer Schutzman	Genentech, San Francisco, CA, United States	Clinical Groups	Oncology Group

Natalie Bowers	Genentech, San Francisco, CA, United States	Clinical Groups	Oncology Group
Rion Pendergrass	Genentech, San Francisco, CA, United States	Clinical Groups	Oncology Group
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Heli Lehtonen	Pfizer, New York, NY, United States	Clinical Groups	Oncology Group
Stefan McDonough	Pfizer, New York, NY, United States	Clinical Groups	Oncology Group
Sauli Vuoti	Janssen-Cilag Oy, Espoo, Finland	Clinical Groups	Oncology Group
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Mengzhen Liu	Abbvie, Chicago, IL, United States	Clinical Groups	Opthalmology Group
Heiko Runz	Biogen, Cambridge, MA, United States	Clinical Groups	Opthalmology Group
Stephanie Loomis s	Biogen, Cambridge, MA, United State	Clinical Groups	Opthalmology Group
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Natalie Bowers	Genentech, San Francisco, CA, United States	Clinical Groups	Opthalmology Group
Hao Chen	Genentech, San Francisco, CA, United States	Clinical Groups	Opthalmology Group
Rion Pendergrass	Genentech, San Francisco, CA, United States	Clinical Groups	Opthalmology Group
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Sirkku Peltonen	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Dermatology Group
Leena Koulu	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Dermatology Group
Nizar Smaoui	Abbvie, Chicago, IL, United States	Clinical Groups	Dermatology Group
Fedik Rahimov	Abbvie, Chicago, IL, United States	Clinical Groups	Dermatology Group
Anne Lehtonen	Abbvie, Chicago, IL, United States	Clinical Groups	Dermatology Group
David Choy	Genentech, San Francisco, CA, United States	Clinical Groups	Dermatology Group
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Kirsi Kalpala	Pfizer, New York, NY, United States	Clinical Groups	Dermatology Group
Ying Wu	Pfizer, New York, NY, United States	Clinical Groups	Dermatology Group
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Tuula Salo	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Odontology Group
David Rice	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Odontology Group
Pekka Nieminen	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Odontology Group
Ulla Palotie	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Odontology Group
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Liisa Suominen	Northern Savo Hospital District, Kuopio, Finland	Clinical Groups	Odontology Group

Päivi Mäntylä	Northern Savo Hospital District, Kuopio, Finland	Clinical Groups	Odontology Group
Ulvi Gursoy	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Odontology Group
Vuokko Anttonen	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Odontology Group
Kirsi Sipilä	Research Unit of Oral Health Sciences Faculty of Medicine, University of Oulu, Oulu, Finland; Medical Research Center, Oulu, Oulu University Hospital and University of Oulu, Oulu, Finland	Clinical Groups	Odontology Group
Rion Pendergrass	Genentech, San Francisco, CA, United States	Clinical Groups	Odontology Group
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Laura Kotaniemi-Talonen	Pirkanmaa Hospital District, Tampere, Finland	Clinical Groups	Women's Health and Reproduction Group
Oskari Heikinheimo	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Women's Health and Reproduction Group
Ilkka Kalliala	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Women's Health and Reproduction Group
Lauri Aaltonen	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Women's Health and Reproduction Group
Varpu Jokimaa	Hospital District of Southwest Finland, Turku, Finland	Clinical Groups	Women's Health and Reproduction Group
Terhi Piltonen	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
Johannes Kettunen	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
Marja Vääräsmäki	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
Outi Uimari	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
Laure Morin-Papunen	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
Maarit Niinimäki	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
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Mary Pat Reeve	Institute for Molecular Medicine Finland (FIMM), HiLIFE, University of Helsinki, Helsinki, Finland	Clinical Groups	Women's Health and Reproduction Group
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Niko Välimäki	University of Helsinki, Helsinki, Finland	Clinical Groups	Women's Health and Reproduction Group
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Heidi Silven	University of Oulu, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
Eeva Sliz	University of Oulu, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
Riikka Arffman	University of Oulu, Oulu, Finland	Clinical Groups	Women's Health and Reproduction Group
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Mengzhen Liu	Abbvie, Chicago, IL, United States	Clinical Groups	Women's Health and Reproduction Group
Bridget Riley-Gillis	Abbvie, Chicago, IL, United States	Clinical Groups	Women's Health and Reproduction Group
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Chia-Yen Chen	Biogen, Cambridge, MA, United States	Clinical Groups	Depression group
Erkki Isometsä	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	Depression group
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Jaana Suvisaari	Finnish Institute for Health and Welfare (THL), Helsinki, Finland	Clinical Groups	Depression group
Thomas Damm Als	Aarhus University, Denmark	Clinical Groups	Depression group
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Antti Aarnisalo	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	ENT (ear, nose and throath) Group
Eveliina Salminen	Hospital District of Helsinki and Uusimaa, Helsinki, Finland	Clinical Groups	ENT (ear, nose and throath) Group
Elisa Rahikkala	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	ENT (ear, nose and throath) Group
Johannes Kettunen	Northern Ostrobothnia Hospital District, Oulu, Finland	Clinical Groups	ENT (ear, nose and throath) Group
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Wei Zhou	Broad Institute, Cambridge, MA, United States	FinnGen Analysis working group	FinnGen Analysis working group
Mutaamba Maasha	Broad Institute, Cambridge, MA, United States	FinnGen Analysis working group	FinnGen Analysis working group
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Taru Tukiainen	Institute for Molecular Medicine Finland (FIMM), HiLIFE, University of Helsinki, Helsinki, Finland	FinnGen Analysis working group	FinnGen Analysis working group
Tuula Palotie	University of Helsinki and Hospital District of Helsinki and Uusimaa, Helsinki, Finland	FinnGen Analysis working group	FinnGen Analysis working group
Kimmo Palin	University of Helsinki, Helsinki, Finland	FinnGen Analysis working group	FinnGen Analysis working group
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Harri Siirtola	University of Tampere, Tampere, Finland	FinnGen Analysis working group	FinnGen Analysis working group
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