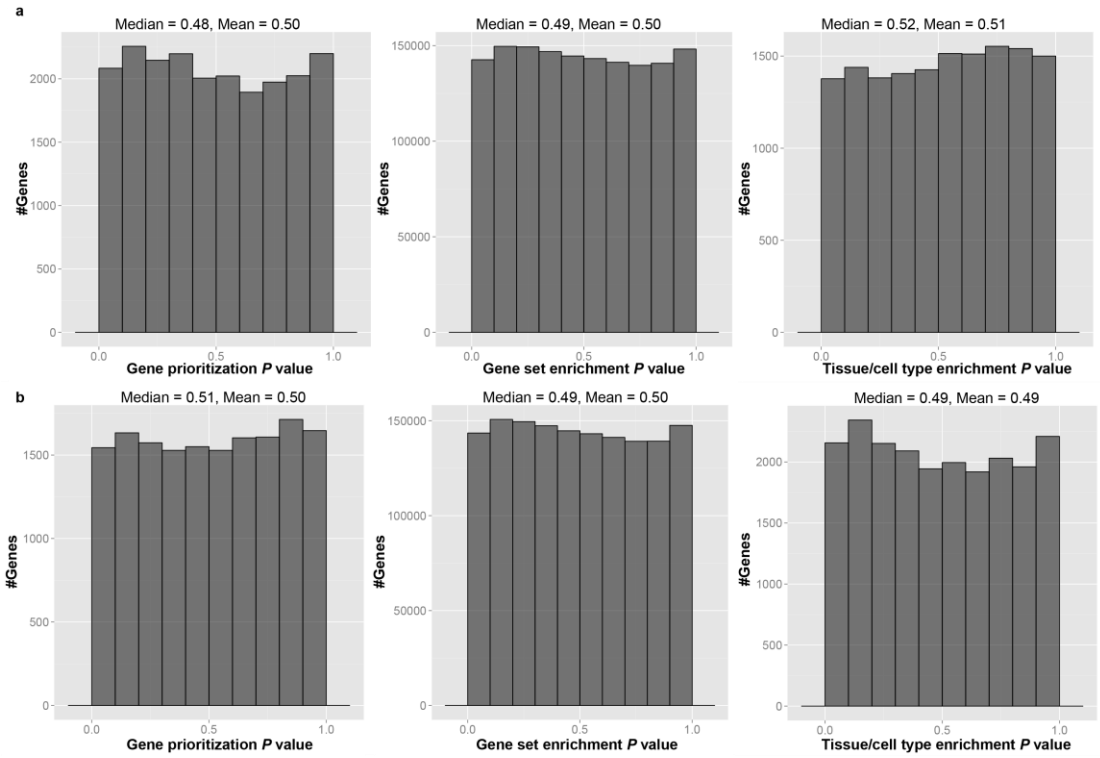
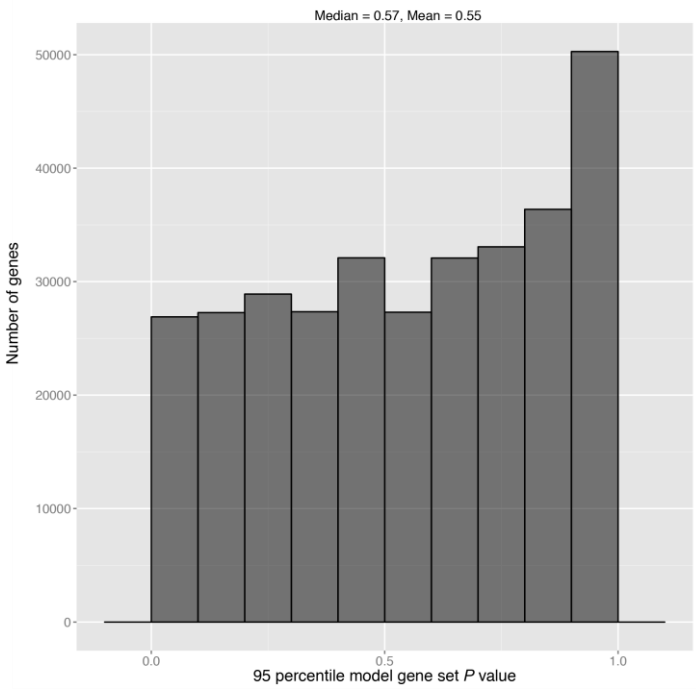
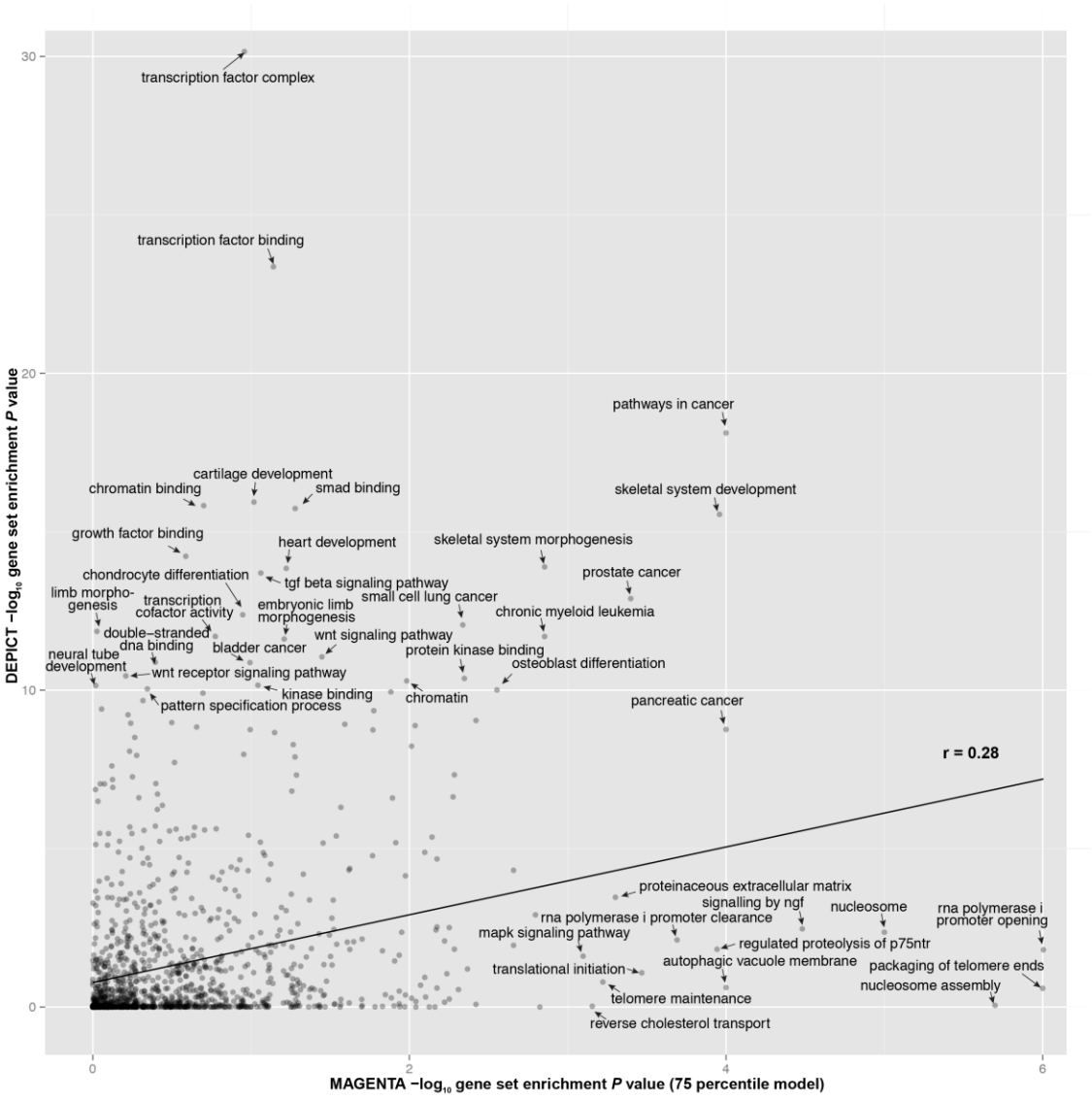


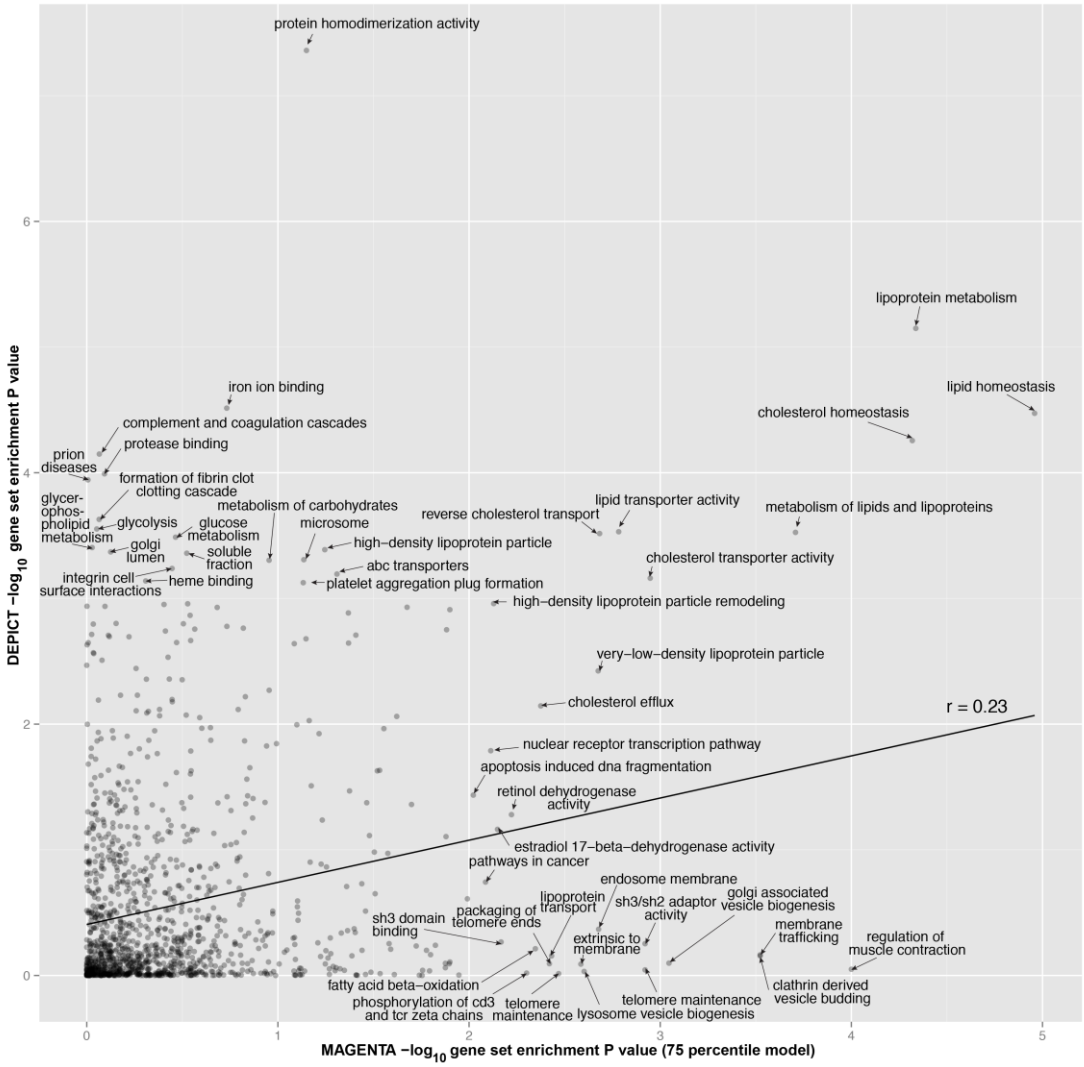
Supplementary Figure 1 – Type 1 error *P*-value histograms. Distribution of *P*-values from the gene prioritization analysis, the reconstituted gene set enrichment analysis and the tissue/cell type enrichment analysis. DEPICT was run using the top 100 independent loci from (a) 100x a simulated GWAS (i.e. using a Gaussian simulated phenotype) using Diabetes Genetics Initiative consortium genotype data[1] and (b) 100x a simulated GWAS using HAPGEN[2] simulated genotype data. The near uniform distributions of *P*-values from both shows that DEPICT has no pronounced type 1 error.



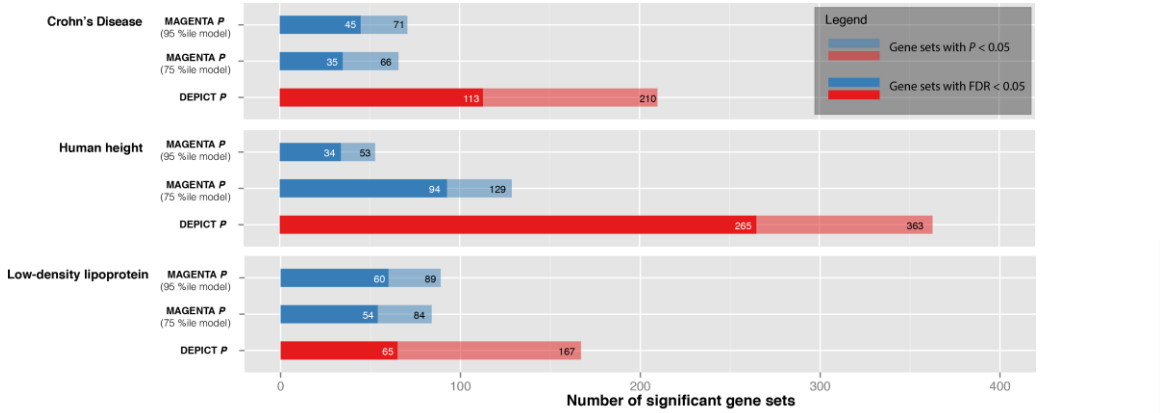
Supplementary Figure 2 – Distribution of MAGENTA gene set enrichment *P*-values. MAGENTA[3] was run 100 times using default settings on summary statistics from 100x a simulated GWAS (i.e. using a Gaussian simulated phenotype) using HapMap imputed Diabetes Genetics Initiative genotype data[1]. *P*-values from the 75 percentile model that was used are shown in the histogram.



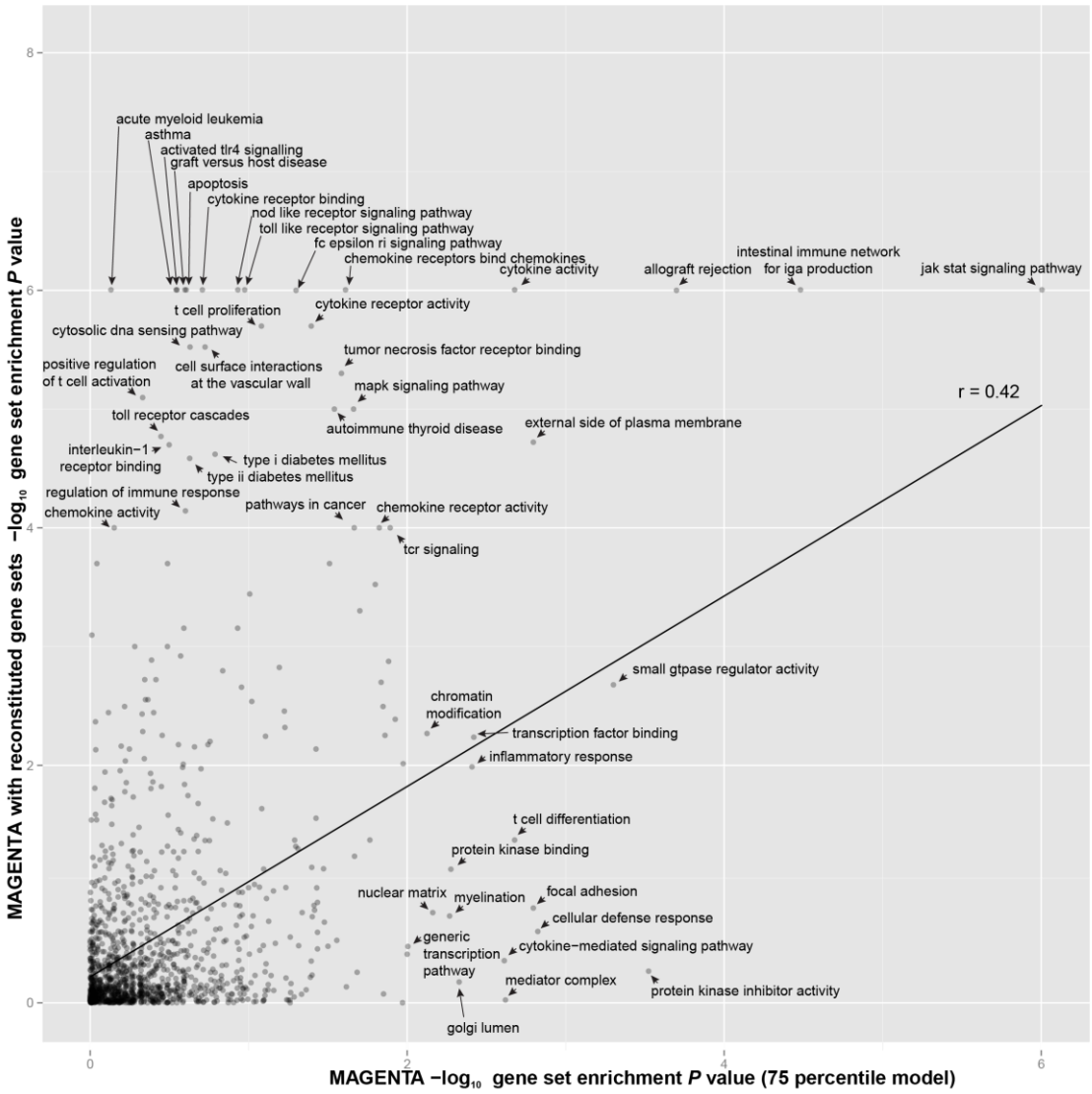
Supplementary Figure 3 – Comparison of DEPICT and MAGENTA for human height. Comparison of DEPICT, which was run with 697 genome-wide significant height SNPs as input, and MAGENTA, which was run using the complete list of human height GWAS summary statistics[4]. DEPICT was run using 1,280 reconstituted gene sets, whereas MAGENTA was run using the pre-defined versions of these 1,280 gene sets. Both methods were run with default settings and the 75 percentile model *P*-values are shown for MAGENTA.



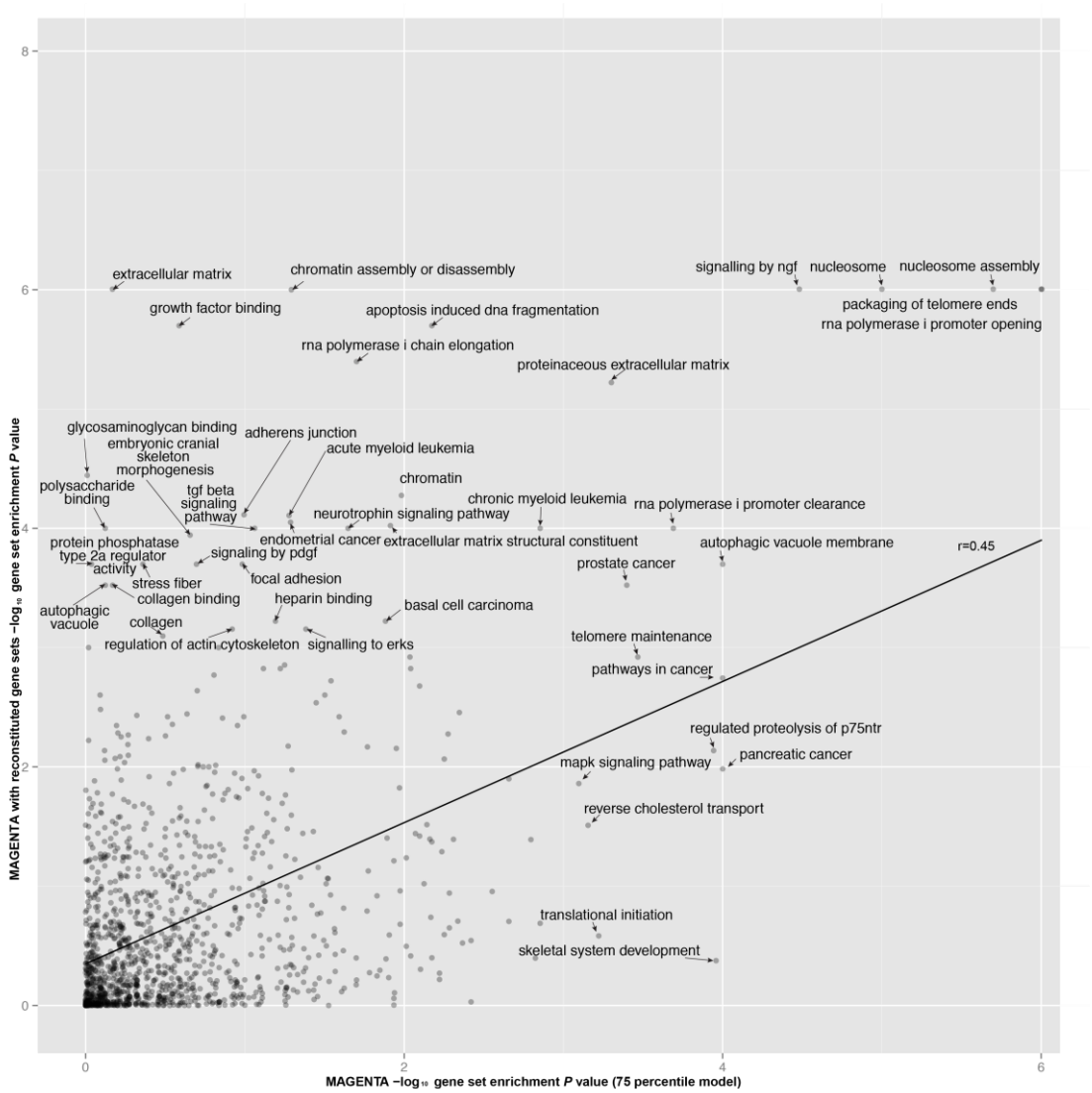
Supplementary Figure 4 – Comparison of DEPICT and MAGENTA for LDL.
Comparison of DEPICT, which was run with 67 genome-wide significant LDL SNPs as input, and MAGENTA, which was run using the complete list of LDL GWAS summary statistics[5]. DEPICT was run using 1,280 reconstituted gene sets, whereas MAGENTA was run using the pre-defined versions of these 1,280 gene sets. Both methods were run with default settings and the 75 percentile model *P*-values are shown for MAGENTA.



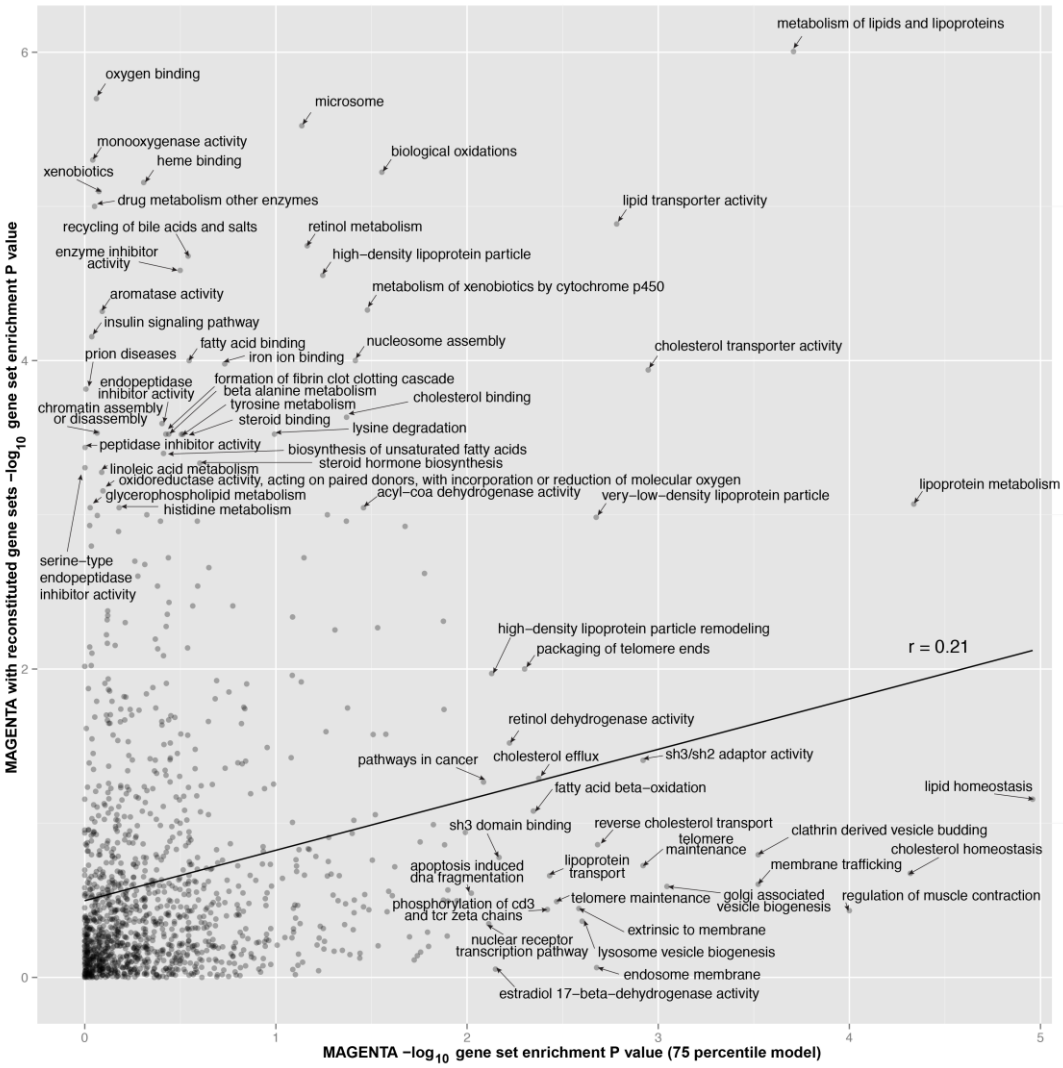
Supplementary Figure 5 – Overview of DEPICT and MAGENTA comparison. We compared DEPICT to MAGENTA[3] using GWAS data for Crohn's Disease from the Inflammatory Bowel Disease Consortium[6], for human height data from the GIANT Consortium[4], and data for low-density lipoprotein-cholesterol (LDL) from the Global Lipids Genetics Consortium[5]. We checked that DEPICT and MAGENTA exhibited correct type 1 error rates and ran each method on 1,280 gene sets having the same identifiers across both methods; DEPICT used reconstituted versions of the 1,280 gene sets and genome-wide significant SNPs only, while MAGENTA was run with its generic gene sets and considered the full set of association results, using both the 75th and 95th percentile cutoffs. For Crohn's Disease, DEPICT identified 2.5 times more significant gene sets (FDR < 0.05) than MAGENTA, 2.8x more significant gene sets for height, and 1.1 times more significant gene sets for LDL. A similar excess of gene sets was observed at nominal significance ($P < 0.05$).



Supplementary Figure 6 – MAGENTA with reconstituted gene sets for Crohn's Disease. Comparison of MAGENTA with predefined gene sets and reconstituted gene sets using Crohn's Disease summary statistics[6] (downloaded from www.ibdgenetics.org). MAGENTA was run using the pre-defined and reconstituted set 1,280 gene sets. Default settings were applied and the 75 percentile model *P*-values are shown.



Supplementary Figure 7 – MAGENTA with reconstituted gene sets for human height. Comparison of MAGENTA with predefined gene sets and reconstituted gene sets using genome-wide significant human height GWAS summary statistics[4]. MAGENTA was run using the pre-defined and reconstituted set 1,280 gene sets. Default settings were applied and the 75 percentile model *P*-values are shown.

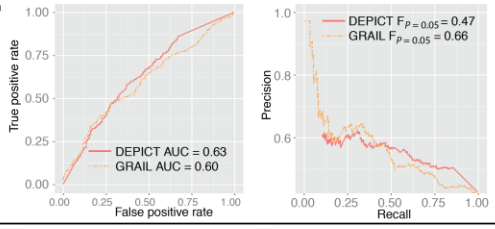


Supplementary Figure 8 – MAGENTA with reconstituted gene sets for LDL.

Comparison of MAGENTA with predefined gene sets and reconstituted gene sets using genome-wide significant low-density lipoprotein GWAS summary statistics[5]. MAGENTA was run using the pre-defined and reconstituted set 1,280 gene sets. Default settings were applied and the 75 percentile model *P*-values are shown.

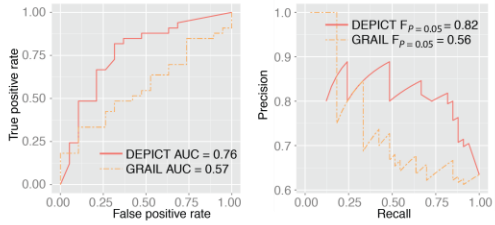
Priorization at loci with no Mendelian human skeletal growth genes (nearest gene benchmark)

Height



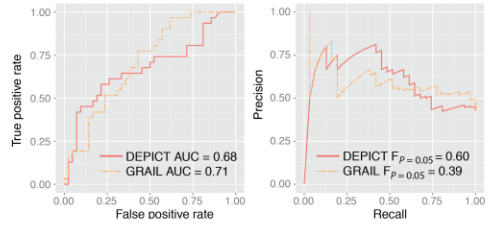
Priorization at loci with no Mendelian human skeletal growth genes (growth plate benchmark)

Height

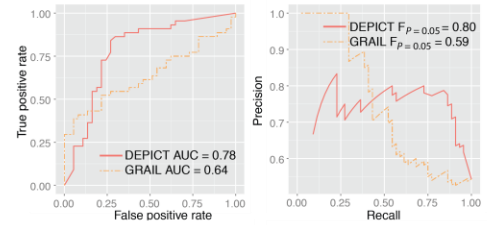


All loci, default method settings

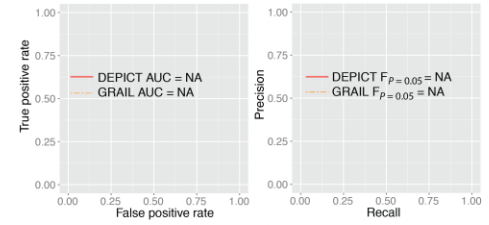
Crohn's Disease



Height

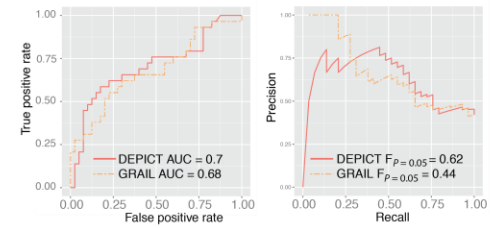


LDL

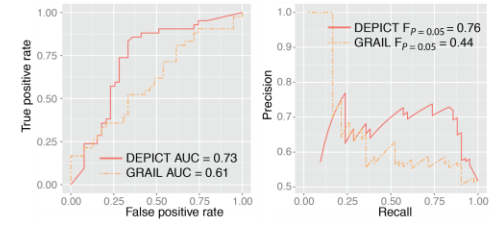


All loci, GRAIL with Gene Expression Atlas data

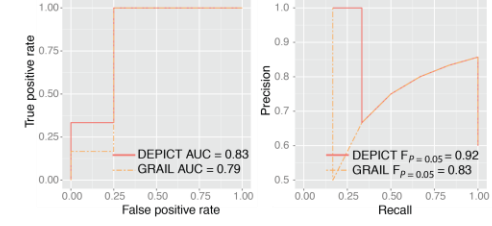
Crohn's Disease



Height

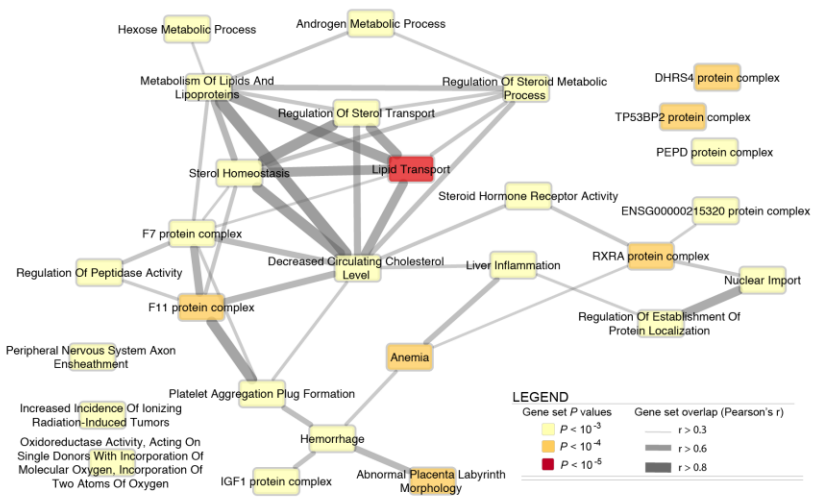


LDL



Supplementary Figure 9 – ROC and precision and recall estimates. Receiver operating characteristics (ROC) curves based on DEPICT and GRAIL[7] results for genome-wide significant SNPs for Crohn's Disease[6] (www.ibdgenetics.org) and human height[4]. The area under (AUC) the ROC curve estimate can be interpreted as the probability that a randomly chosen positive will rank higher than a randomly chosen negative. Precision measures the positive predicted value, whereas recall measures the sensitivity. A precision of 1 means that all positive genes were correctly classified as such and that there were no false positives. A recall of 1 means that all positive genes were classified as a positive gene.

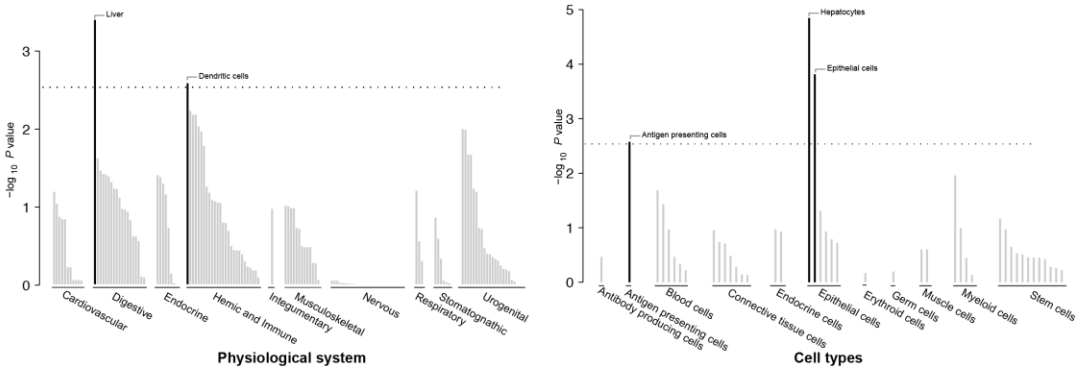
a



b

Locus	Chr.	Gene symbol	Gene *P* value	False discovery rate
rs1321195-rs2315065	6	*LPA*	3.12E-06	<0.006
rs1367117-rs520861-rs533617-rs6413458	2	*APOB*	3.39E-06	<0.006
rs1025447-rs4148191-rs4299376-rs6709904	2	*ABCG8*	2.38E-05	<0.006
rs17666993-rs2000999	16	*HP*	2.94E-05	<0.006
rs1025447-rs4148191-rs4299376-rs6709904	2	*ABCG5*	7.37E-05	<0.006
rs1308117	3	*DTX3L*	1.16E-04	0.008
rs1030431	8	*CYP7A1*	1.16E-04	0.007
rs4417316-rs964184	11	*APOA5*	1.81E-04	0.006
rs1564348	6	*SLC22A1*	2.21E-04	0.006
rs5930-rs6511720	19	*LDLR*	2.63E-04	0.010
rs2104417	20	*FER1L4*	3.56E-04	0.009
rs267733	1	*ANXA9*	4.89E-04	0.012
rs12735646	1	*NR0B2*	6.72E-04	0.012
rs1052639	2	*INSIG2*	6.77E-04	0.011
rs12916	5	*HMGCR*	0.001	0.010
rs174583	11	*FADS2*	0.001	0.011
rs3850634	1	*ANGPTL3*	0.001	0.009
rs1129555	10	*GPAM*	0.001	0.009
rs3852856-rs4420638	19	*APOC1*	0.002	0.013
rs11065987	12	*ALDH2*	0.002	0.015
rs364585	20	*SPTLC3*	0.003	0.017
rs3852856-rs4420638	19	*PVRL2*	0.003	0.016
rs12027135	1	*RHD*	0.003	0.015
rs4968845	17	*ABCA6*	0.003	0.015
rs1800978	9	*ABCA1*	0.004	0.017
rs2270690-rs4927206-rs2479409	1	*PCSK9*	0.004	0.018
rs1531517	19	*BCL3*	0.004	0.017
rs10401969	19	*TM6SF2*	0.005	0.021
rs2287622	2	*ABCB11*	0.005	0.021
rs13081171	3	*PARP9*	0.006	0.027
rs12048994	1	*SOAT1*	0.007	0.031
rs2807834	1	*MARC1*	0.007	0.027
rs11136341	8	*PARP10*	0.008	0.035
rs12027135	1	*RHCE*	0.008	0.036
rs11220462	11	*ST3GAL4*	0.010	0.040

c



Supplementary Figure 10 – DEPICT results LDL. The figure show significantly enriched gene sets (panel a), prioritized genes (panel b), and enriched tissue/cell types (panel c) for the DEPICT analysis of low-density lipoprotein cholesterol (LDL)[5]. The input to DEPICT were rsIDs from the 136 most associated SNPs ($P < 1 \times 10^{-5}$), which DEPICT converted to 87 fully independent loci (DEPICT locus definition). Unless otherwise stated, significance implies false discovery rate (FDR) below 5%. **a**, DEPICT identified 564 significant reconstituted gene sets. To group overlapping gene sets, we next computed the pairwise Pearson correlation of all significant gene sets and used Affinity Propagation method[8] on the Pearson distance matrix to cluster gene sets that were highly correlated. The Affinity Propagation method automatically defines the number of independent clusters and identifies an exemplar for each cluster. We used the exemplar gene sets to label cluster and referred to clusters as 'meta gene sets'. In the figure only exemplar gene sets are shown (there was no meta gene set that only contained a single gene set). Gene sets that had Pearson correlations above r = 0.3 were connected by edges, which were scaled according to degree of correlation (higher correlation is reflected by wider edges). Only gene sets with gene prioritization *P*-value $< 5.0 \times 10^{-4}$ are shown in the figure. Gene sets were color coded according to the DEPICT gene set enrichment *P*-value (lower *P*-values is reflected by darker colors). **b**, 35 genes were significantly prioritized (FDR < 0.05). Loci with labels containing multiple rsIDs denote DEPICT loci that resulted from overlapping input loci. **c**, DEPICT identified two significant tissue and three cell type annotations (marked in black).

Supplementary Note 1 – Locus definition calibration

In total we tested 21 locus definitions varying the genetic distance (measured by the LD r^2 parameter), physical distance and distance to nearest recombination hotspot (**Supplementary Table 2**). The nearest gene to a given best-associated SNP was always included, and loci that were overlapping in terms of the genes mapped to them were merged into a single locus. We calculated the fraction of the Mendelian skeletal growth genes that were significantly prioritized in associated height loci[4]. A locus delineation based solely on $r^2 < 0.5$ (corresponding to a mean and median locus size of 155.2 kb and 88.8 kb, respectively) optimized the capture of the positive control genes (40 of 54, or 74% of human skeletal growth genes at 53 loci were prioritized by DEPICT). We note that using a locus definition solely based on 10 kb flanks to the lead SNP resulted in a similar fraction of predicted Mendelian human skeletal growth genes, but at lower sensitivity (29 of 39, or 74%). We conducted a similar analysis for 67 genome-wide significant loci for low-density lipoprotein cholesterol (LDL) loci[5] using 14 Mendelian lipid genes reported in ref.[5] as positive genes. The r^2 cutoffs equal to 0.3 and 0.4 resulted in the highest fraction of prioritized Mendelian lipid genes with a better coverage than the other cutoffs at which all positive genes were correctly prioritized. Prioritized lipid genes at the $r^2 = 0.4$ cutoff had a slightly smaller mean gene prioritization P-value ($P = 1.2 \times 10^{-4}$) than prioritized lipid genes at the $r^2 = 0.3$ cutoff ($P = 4.4 \times 10^{-4}$).

Supplementary Note 2 – Varying the genotype data used by DEPICT

A possible concern with our approach is that the bias correction and false discovery rate estimation steps rely on the Diabetes Genetics Initiative (DGI) CEU genotype data[1] instead of the genotypes used to conduct the given primary GWAS. To explore how robust DEPICT is to changes in the genotype data used to compute null loci, we compared DEPICT results for 697 genome-wide significant SNPs for human height[4] from two different genotype data sets; DGI genotype data and data from a Dutch cohort[9] (imputed towards HapMap Project[10] release 2) comprising 1,240 unrelated individuals. The Dutch genotype data-based null GWAS was computed the same way as the DGI based null GWAS. We correlated the gene prioritization z-scores between the two runs as well as the gene set z-scores and observed that both analyses yielded highly correlated results ($r^2 = 0.995$, $P < 2.2 \times 10^{-16}$; $r^2 = 0.994$; $P < 2.2 \times 10^{-16}$), which strongly suggests that DEPICT results are not contingent on the genotypes used to compute null loci.

Supplementary Note 3 – Varying the number of null GWAS used by DEPICT

Having ensured that DEPICT is robust to changes in the underlying genotype data, we next assessed whether changing the number of permutated GWAS runs, used to generate null loci had any impact on results. We tested sets of background loci based on 200, 500 and 900 null GWAS runs and observed very high correlations between gene prioritization *P*-values, as well as between gene set enrichment *P*-values, and tissue/cell type enrichment analysis *P*-values (all Spearman r were between 0.991 and 0.99997) indicating that DEPICT is robust to changes in the number of null GWAS runs used to do bias adjustments**.**

Supplementary Note 4 – Names and affiliations of the members of the Genetic Investigation of ANthropometric Traits (GIANT) Consortium.

Andrew R Wood[1], Tonu Esko[2,3,4,5], Jian Yang[6,7], Sailaja Vedantam[3,4], Tune H Pers[3,4,5,8], Stefan Gustafsson[9,10], Audrey Y Chu[11], Karol Estrada[4,12,13], Jian'an Luan[14], Zoltán Kutalik[15,16,17], Najaf Amin[18], Martin L Buchkovich[19], Damien C Croteau-Chonka[19,20], Felix R Day[14], Yanan Duan[21], Tove Fall[9,10,22], Rudolf Fehrmann[23], Teresa Ferreira[24], Anne U Jackson[25], Juha Karjalainen[23], Ken Sin Lo[26], Adam E Locke[25], Reedik Mägi[2,24], Evelin Mihailov[2,27], Eleonora Porcu[28], Joshua C Randall[24,29], André Scherag[30,31], Anna AE Vinkhuyzen[6], Harm-Jan Westra[23], Thomas W Winkler[32], Tsegaselassie Workalemahu[33], Jing Hua Zhao[14], Devin Absher[34], Eva Albrecht[35], Denise Anderson[36], Jeffrey Baron[37], Marian Beekman[38,39], Ayse Demirkan[18,40], Georg B Ehret[41,42], Bjarke Feenstra[43], Mary F Feitosa[44], Krista Fischer[2], Ross M Fraser[45], Anuj Goel[24,46], Jian Gong[47], Anne E Justice[48], Stavroula Kanoni[49], Marcus E Kleber[50,51], Kati Kristiansson[52], Unhee Lim[53], Vaneet Lotay[54], Julian C Lui[37], Massimo Mangino[55], Irene Mateo Leach[56], Carolina Medina-Gomez[12,57,58], Michael A Nalls[59], Dale R Nyholt[60], Cameron D Palmer[3,4], Dorota Pasko[1], Sonali Pechlivanis[30], Inga Prokopenko[24,61,62], Janina S Ried[35], Stephan Ripke[13,63], Dmitry Shungin[64,65,66], Alena Stancáková[67], Rona J Strawbridge[68], Yun Ju Sung[69], Toshiko Tanaka[70], Alexander Teumer[71], Stella Trompet[72,73], Sander W van der Laan[74], Jessica van Setten[75], Jana V Van Vliet-Ostaptchouk[76], Zhaoming Wang[77,78,79,80], Loïc Yengo[81,82,83], Weihua Zhang[84,85], Uzma Afzal[84,85], Johan Ärnlöv[9,10,86], Gillian M Arscott[87], Stefania Bandinelli[88], Amy Barrett[61], Claire Bellis[89], Amanda J Bennett[61], Christian Berne[90], Matthias Blüher[91,92], Jennifer L Bolton[45], Yvonne Böttcher[91], Heather A Boyd[43], Marcel Bruinenberg[93], Brendan M Buckley[94], Steven Buyske[95,96], Ida H Caspersen[97], Peter S Chines[98], Robert Clarke[99], Simone Claudi-Boehm[100], Matthew Cooper[36], E Warwick Daw[44], Pim A De Jong[101], Joris Deelen[38,39], Graciela Delgado[50], Josh C Denny[102], Rosalie Dhonukshe-Rutten[103], Maria Dimitriou[104], Alex SF Doney[105], Marcus Dörr[77,106], Niina Eklund[52,107], Elodie Eury[81,82,83], Lasse Folkersen[68], Melissa E Garcia[108], Frank Geller[43], Vilmantas Giedraitis[109], Alan S Go[110], Harald Grallert[35,111,112], Tanja B Grammer[50], Jürgen Gräßler[113], Henrik Grönberg[22], Lisette C.P.G.M. de Groot[103], Christopher J Groves[61], Jeffrey Haessler[47], Per Hall[22], Toomas Haller[2], Goran Hallmans[114], Anke Hannemann[78], Catharina A Hartman[115], Maija Hassinen[116], Caroline Hayward[117], Nancy L Heard-Costa[118,119], Quinta Helmer[38,120,121], Gibran Hemani[6,7], Anjali K Henders[60], Hans L Hillege[56,122], Mark A Hlatky[123], Wolfgang Hoffmann[77,124], Per Hoffmann[125,126,127], Oddgeir Holmen[128], Jeanine J Houwing-Duistermaat[38,120], Thomas Illig[111,129], Aaron Isaacs[18,130], Alan L James[131,132], Janina Jeff[54], Berit Johansen[97], Åsa Johansson[133], Jennifer Jolley[134,135], Thorhildur Juliusdottir[24], Juhani Junttila[136], Abel N Kho[137], Leena Kinnunen[52], Norman Klopp[111,129], Thomas Kocher[138], Wolfgang Kratzer[139], Peter Lichtner[140], Lars Lind[141], Jaana Lindström[52], Stéphane Lobbens[81,82,83], Mattias Lorentzon[142], Yingchang Lu[54,143], Valeriya Lyssenko[144],

Patrik KE Magnusson[22], Anubha Mahajan[24], Marc Maillard[145], Wendy L McArdle[146], Colin A McKenzie[147], Stela McLachlan[45], Paul J McLaren[148,149], Cristina Menni[55], Sigrun Merger[100], Lili Milani[2], Alireza Moayyeri[55], Keri L Monda[48,150], Mario A Morken[98], Gabriele Müller[151], Martina Müller-Nurasyid[35,152,153,154], Arthur W Musk[155], Narisu Narisu[98], Matthias Nauck[77,78], Ilja M Nolte[122], Markus M Nöthen[126,127], Laticia Oozageer[84], Stefan Pilz[156,157], Nigel W Rayner[24,29,61], Frida Renstrom[64], Neil R Robertson[24,61], Lynda M Rose[11], Ronan Roussel[158,159,160], Serena Sanna[28], Hubert Scharnagl[161], Salome Scholtens[122], Fredrick R Schumacher[162], Heribert Schunkert[154,163], Robert A Scott[14], Joban Sehmi[84,85], Thomas Seufferlein[139], Jianxin Shi[164], Karri Silventoinen[165], Johannes H Smit[166,167], Albert Vernon Smith[168,169], Joanna Smolonska[23,122], Alice V Stanton[170], Kathleen Stirrups[29,49], David J Stott[171], Heather M Stringham[25], Johan Sundström[141], Morris A Swertz[23], Ann-Christine Syvänen[9,172], Bamidele O Tayo[173], Gudmar Thorleifsson[174], Jonathan P Tyrer[175], Suzanne van Dijk[12], Natasja M van Schoor[156], Nathalie van der Velde[12,176], Diana van Heemst[38,73], Floor VA van Oort[177], Sita H Vermeulen[178,179], Niek Verweij[56], Judith M Vonk[122], Lindsay L Waite[34], Melanie Waldenberger[111], Roman Wennauer[180], Lynne R Wilkens[53], Christina Willenborg[181,182], Tom Wilsgaard[183], Mary K Wojczynski[44], Andrew Wong[184], Alan F Wright[117], Qunyuan Zhang[44], Dominique Arveiler[185], Stephan JL Bakker[186], John Beilby[87,187], Richard N Bergman[188], Sven Bergmann[16,17], Reiner Biffar[189], John Blangero[89], Dorret I Boomsma[190], Stefan R Bornstein[113], Pascal Bovet[191,192], Paolo Brambilla[193], Morris J Brown[194], Harry Campbell[45], Mark J Caulfield[195], Aravinda Chakravarti[41], Rory Collins[99], Francis S Collins[98], Dana C Crawford[196,197], L Adrienne Cupples[118,198], John Danesh[199], Ulf de Faire[200], Hester M den Ruijter[74,201], Raimund Erbel[202], Jeanette Erdmann[181,182], Johan G Eriksson[52,203,204], Martin Farrall[24,46], Ele Ferrannini[205,206], Jean Ferrières[207], Ian Ford[208], Nita G Forouhi[14], Terrence Forrester[147], Ron T Gansevoort[186], Pablo V Gejman[209], Christian Gieger[35], Alain Golay[210], Omri Gottesman[54], Vilmundur Gudnason[168,169], Ulf Gyllensten[133], David W Haas[211], Alistair S Hall[212], Tamara B Harris[108], Andrew T Hattersley[213], Andrew C Heath[214], Christian Hengstenberg[154,163], Andrew A Hicks[215,216], Lucia A Hindorff[217], Aroon D Hingorani[218], Albert Hofman[57,58], G Kees Hovingh[219], Steve E Humphries[220], Steven C Hunt[221], Elina Hypponen[222,223,224], Kevin B Jacobs[79,80], Marjo-Riitta Jarvelin[85,225,226,227,228,229], Pekka Jousilahti[52], Antti M Jula[52], Jaakko Kaprio[52,107,230], John JP Kastelein[219], Manfred Kayser[57,231], Frank Kee[232], Sirkka M Keinanen-Kiukaanniemi[233,234], Lambertus A Kiemeney[178,235], Jaspal S Kooner[84,236,237], Charles Kooperberg[47], Seppo Koskinen[52], Peter Kovacs[91,92], Aldi T Kraja[44], Meena Kumari[238], Johanna Kuusisto[239], Timo A Lakka[116,240,241], Claudia Langenberg[14,238], Loic Le Marchand[53], Terho Lehtimäki[242], Sara Lupoli[243,244], Pamela AF Madden[214], Satu Männistö[52], Paolo Manunta[245,246], André Marette[247,248], Tara C Matise[96], Barbara McKnight[249], Thomas Meitinger[154], Frans L Moll[250], Grant W Montgomery[60], Andrew D Morris[105], Andrew P Morris[2,24,251], Jeffrey C Murray[252], Mari Nelis[2], Claes Ohlsson[142], Albertine J Oldehinkel[115], Ken K Ong[14,184], Willem H Ouwehand[134,135], Gerard Pasterkamp[74], Annette Peters[111,154,253], Peter P Pramstaller[215,216,254], Jackie F Price[45], Lu Qi[20,255], Olli T Raitakari[256,257], Tuomo Rankinen[258], DC Rao[44,69,214], Treva K Rice[69,214], Marylyn Ritchie[259], Igor Rudan[45,260], Veikko Salomaa[52], Nilesh J Samani[261,262], Jouko Saramies[263],, Mark A Sarzynski[258], Peter EH Schwarz[113,264], Sylvain Sebert[229], Peter Sever[265], Alan R Shuldiner[266,267], Juha Sinisalo[268], Valgerdur Steinthorsdottir[174], Ronald P Stolk[122], Jean-Claude Tardif[26,269], Anke Tönjes[91,92], Angelo Tremblay[270], Elena Tremoli[271], Jarmo Virtamo[52], Marie-Claude Vohl[248,272], The electronic medical records and genomics (eMERGE) consortium[273], The MIGen Consortium[274,275], The PAGE Consortium[275,276], The LifeLines Cohort Study[275,277], Philippe Amouyel[278], Folkert W Asselbergs[218,279,280], Themistocles L Assimes[123], Murielle Bochud[191,192], Bernhard O Boehm[100,281], Eric Boerwinkle[282], Erwin P Bottinger[54], Claude Bouchard[258], Stéphane Cauchi[81,82,83], John C Chambers[84,85,236], Stephen J Chanock[79], Richard S Cooper[173], Paul IW de Bakker[75,283,284], George Dedoussis[104], Luigi Ferrucci[70], Paul W Franks[64,65,255], Philippe Froguel[62,81,82,83], Leif C Groop[107,285], Christopher A Haiman[162], Anders Hamsten[68], M Geoffrey Hayes[137], Jennie Hui[87,187,222], David J. Hunter[20,255,286], Kristian Hveem[128], J Wouter Jukema[72,280,287], Robert C Kaplan[288], Mika Kivimaki[238], Diana Kuh[184], Markku Laakso[239], Yongmei Liu[289], Nicholas G Martin[60], Winfried März[50,161,290], Mads Melbye[43,123], Susanne Moebus[30], Patricia B Munroe[195],

Inger Njølstad[183], Ben A Oostra[18,130,291], Colin NA Palmer[105], Nancy L Pedersen[22], Markus Perola[2,52,107], Louis Pérusse[248,270], Ulrike Peters[47], Joseph E Powell[6,7], Chris Power[224], Thomas Quertermous[123], Rainer Rauramaa[116,241], Eva Reinmaa[2], Paul M Ridker[11,292], Fernando Rivadeneira[12,57,58], Jerome I Rotter[293], Timo E Saaristo[294,295], Danish Saleheen[199,296,297], David Schlessinger[298], P Eline Slagboom[38,39], Harold Snieder[122], Tim D Spector[55], Konstantin Strauch[35,153], Michael Stumvoll[91,92], Jaakko Tuomilehto[52,299,300,301], Matti Uusitupa[302,303], Pim van der Harst[23,56,280], Henry Völzke[77,124], Mark Walker[304], Nicholas J Wareham[14], Hugh Watkins[24,46], H-Erich Wichmann[305,306,307],, James F Wilson[45], Pieter Zanen[308], Panos Deloukas[29,49,309], Iris M Heid[32,35], Cecilia M Lindgren[4,24], Karen L Mohlke[19], Elizabeth K Speliotes[310], Unnur Thorsteinsdottir[174,311], Inês Barroso[29,312,313], Caroline S Fox[118], Kari E North[48,314], David P Strachan[315], Jacques S. Beckmann[16,17,316], Sonja I Berndt[79], Michael Boehnke[25], Ingrid B Borecki[44], Mark I McCarthy[24,61,317], Andres Metspalu[2,27], Kari Stefansson[174,311], André G Uitterlinden[12,57,58], Cornelia M van Duijn[18,57,58,130], Lude Franke[23], Cristen J Willer[318,319,320], Alkes L. Price[4,286,321], Guillaume Lettre[26,269], Ruth JF Loos[14,54,143,322], Michael N Weedon[1], Erik Ingelsson[9,10,24], Jeffrey R O'Connell[266], Goncalo R Abecasis[25], Daniel I Chasman[11,292‡], Michael E Goddard[323,324], Peter M Visscher[6,7], Joel N Hirschhorn[3,4,5], Timothy M Frayling[1]

Affiliations

1. Genetics of Complex Traits, University of Exeter Medical School, University of Exeter, Exeter EX1 2LU, UK
2. Estonian Genome Center, University of Tartu, Tartu 51010, Estonia
3. Division of Endocrinology, Genetics and Basic and Translational Obesity Research, Boston Children's Hospital, Boston, MA 02115, USA
4. Broad Institute of the Massachusetts Institute of Technology and Harvard University, Cambridge 02142, MA, USA
5. Department of Genetics, Harvard Medical School, Boston, MA 02115, USA
6. Queensland Brain Institute, The University of Queensland, Brisbane 4072, Australia
7. The University of Queensland Diamantina Institute, The Translation Research Institute, Brisbane 4012, Australia
8. Center for Biological Sequence Analysis, Department of Systems Biology, Technical University of Denmark, Lyngby 2800, Denmark
9. Science for Life Laboratory, Uppsala University, Uppsala 75185, Sweden
10. Department of Medical Sciences, Molecular Epidemiology, Uppsala University, Uppsala 75185, Sweden
11. Division of Preventive Medicine, Brigham and Women's Hospital, Boston, MA 02215, USA
12. Department of Internal Medicine, Erasmus Medical Center, 3015GE Rotterdam, The Netherlands
13. Analytic and Translational Genetics Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA
14. MRC Epidemiology Unit, University of Cambridge, Institute of Metabolic Science, Addenbrooke's Hospital, Hills Road, Cambridge, CB2 0QQ, UK
15. Institute of Social and Preventive Medicine (IUMSP), Centre Hospitalier Universitaire Vaudois (CHUV), Lausanne 1010, Switzerland
16. Swiss Institute of Bioinformatics, Lausanne 1015, Switzerland
17. Department of Medical Genetics, University of Lausanne, Lausanne 1005, Switzerland
18. Genetic Epidemiology Unit, Department of Epidemiology, Erasmus University Medical Center, 3015 GE Rotterdam, The Netherlands
19. Department of Genetics, University of North Carolina, Chapel Hill, NC 27599, USA
20. Channing Division of Network Medicine, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA 02115, USA

21. Division of Statistical Genomics, Department of Genetics Washington University School of Medicine, St. Louis, MO, USA
22. Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm 17177, Sweden
23. Department of Genetics, University Medical Center Groningen, University of Groningen, 9700 RB Groningen, The Netherlands
24. Wellcome Trust Centre for Human Genetics, University of Oxford, Oxford OX3 7BN, UK
25. Center for Statistical Genetics, Department of Biostatistics, University of Michigan, Ann Arbor, MI 48109, USA
26. Montreal Heart Institute, Montreal, Quebec H1T 1C8, Canada
27. Institute of Molecular and Cell Biology, University of Tartu, Tartu 51010, Estonia
28. Istituto di Ricerca Genetica e Biomedica (IRGB), Consiglio Nazionale delle Ricerche, Cagliari, Sardinia 09042, Italy
29. Wellcome Trust Sanger Institute, Hinxton, Cambridge CB10 1SA, UK
30. Institute for Medical Informatics, Biometry and Epidemiology (IMIBE), University Hospital Essen, Essen, Germany
31. Clinical Epidemiology, Integrated Research and Treatment Center, Center for Sepsis Control and Care (CSCC), Jena University Hospital, Jena, Germany
32. Department of Genetic Epidemiology, Institute of Epidemiology and Preventive Medicine, University of Regensburg, D-93053 Regensburg, Germany
33. Harvard School of Public Health, Department of Nutrition, Harvard University, Boston, MA 2115, USA
34. HudsonAlpha Institute for Biotechnology, Huntsville, AL 35806, USA
35. Institute of Genetic Epidemiology, Helmholtz Zentrum München - German Research Center for Environmental Health, D-85764 Neuherberg, Germany
36. Telethon Institute for Child Health Research, Centre for Child Health Research, The University of Western Australia, Western Australia 6008, Australia
37. Section on Growth and Development, Program in Developmental Endocrinology and Genetics, Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, MD 20892, USA
38. Netherlands Consortium for Healthy Aging (NCHA), Leiden University Medical Center, Leiden 2300 RC, The Netherlands
39. Department of Molecular Epidemiology, Leiden University Medical Center, 2300 RC Leiden, The Netherlands
40. Department of Human Genetics, Leiden University Medical Center, 2333 ZC Leiden, The Netherlands
41. Center for Complex Disease Genomics, McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA
42. Cardiology, Department of Specialties of Internal Medicine, Geneva University Hospital, Geneva 1211, Switzerland
43. Department of Epidemiology Research, Statens Serum Institut, Copenhagen DK-2300, Denmark
44. Department of Genetics, Washington University School of Medicine, St. Louis, MO 63110, USA
45. Centre for Population Health Sciences, University of Edinburgh, Teviot Place, Edinburgh, EH8 9AG, Scotland, UK
46. Division of Cardiovacular Medicine, Radcliffe Department of Medicine, University of Oxford, Oxford OX3 9DU, UK
47. Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA 98109, USA
48. Department of Epidemiology, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, USA
49. William Harvey Research Institute, Barts and The London School of Medicine and Dentistry, Queen Mary University of London, EC1M 6BQ UK

50. Vth Department of Medicine (Nephrology, Hypertensiology, Endocrinology, Diabetology, Rheumatology), Medical Faculty of Mannheim, University of Heidelberg, Germany
51. Department of Internal Medicine II, Ulm University Medical Centre, D-89081 Ulm, Germany
52. National Institute for Health and Welfare, FI-00271 Helsinki, Finland
53. Epidemiology Program, University of Hawaii Cancer Center, Honolulu, HI USA
54. The Charles Bronfman Institute for Personalized Medicine, Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA
55. Department of Twin Research and Genetic Epidemiology, King's College London, London SE1 7EH, UK
56. Department of Cardiology, University Medical Center Groningen, University of Groningen, 9700RB Groningen, The Netherlands
57. Netherlands Consortium for Healthy Aging (NCHA), 3015GE Rotterdam, The Netherlands
58. Department of Epidemiology, Erasmus Medical Center, 3015GE Rotterdam, The Netherlands
59. Laboratory of Neurogenetics, National Institute on Aging, National Institutes of Health, Bethesda, MD 20892, USA
60. QIMR Berghofer Medical Research Institute, Queensland 4006, Australia
61. Oxford Centre for Diabetes, Endocrinology and Metabolism, University of Oxford, Oxford OX3 7LJ, UK
62. Department of Genomics of Common Disease, School of Public Health, Imperial College London, Hammersmith Hospital, London, UK
63. Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard, Cambridge, MA 02142, USA
64. Department of Clinical Sciences, Genetic & Molecular Epidemiology Unit, Lund University Diabetes Center, Skåne University Hosptial, Malmö 205 02, Sweden
65. Department of Public Health and Clinical Medicine, Unit of Medicine, Umeå University, Umeå 901 87, Sweden
66. Department of Odontology, Umeå University, Umeå 901 85, Sweden
67. University of Eastern Finland, FI-70210 Kuopio, Finland
68. Atherosclerosis Research Unit, Center for Molecular Medicine, Department of Medicine, Karolinska Institutet, Stockholm 17176, Sweden
69. Division of Biostatistics, Washington University School of Medicine, St. Louis, MO 63110, USA
70. Translational Gerontology Branch, National institute on Aging, Baltimore MD 21225, USA
71. Interfaculty Institute for Genetics and Functional Genomics, University Medicine Greifswald, D-17475 Greifswald, Germany
72. Department of Cardiology, Leiden University Medical Center, 2300 RC Leiden, The Netherlands
73. Department of Gerontology and Geriatrics, Leiden University Medical Center, 2300 RC Leiden, The Netherlands
74. Experimental Cardiology Laboratory, Division Heart and Lungs, University Medical Center Utrecht, 3584 CX Utrecht, The Netherlands
75. Department of Medical Genetics, University Medical Center Utrecht, 3584 CX Utrecht, The Netherlands
76. Department of Endocrinology, University of Groningen, University Medical Center Groningen, Groningen, 9700 RB, The Netherlands
77. DZHK (Deutsches Zentrum für Herz-Kreislaufforschung – German Centre for Cardiovascular Research), partner site Greifswald, D-17475 Greifswald, Germany
78. Institute of Clinical Chemistry and Laboratory Medicine, University Medicine Greifswald, D-17475 Greifswald, Germany

79. Division of Cancer Epidemiology and Genetics, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, USA
80. Core Genotyping Facility, SAIC-Frederick, Inc., NCI-Frederick, Frederick, MD 21702, USA
81. CNRS UMR 8199, F-59019 Lille, France
82. European Genomic Institute for Diabetes, F-59000 Lille, France
83. Université de Lille 2, F-59000 Lille, France
84. Ealing Hospital NHS Trust, Middlesex UB1 3HW, UK
85. Department of Epidemiology and Biostatistics, Imperial College London, London W2 1PG, UK
86. School of Health and Social Studies, Dalarna University, Falun, Sweden
87. PathWest Laboratory Medicine of Western Australia, NEDLANDS, Western Australia 6009, Australia
88. Geriatric Unit, Azienda Sanitaria Firenze (ASF), Florence, Italy
89. Department of Genetics, Texas Biomedical Research Institute, San Antonio, TX, USA
90. Department of Medical Sciences, Endocrinology, Diabetes and Metabolism, Uppsala University, Uppsala 75185, Sweden
91. IFB Adiposity Diseases, University of Leipzig, D-04103 Leipzig, Germany
92. Department of Medicine, University of Leipzig, D-04103 Leipzig, Germany
93. LifeLines, University Medical Center Groningen, University of Groningen, 9700 RB Groningen, The Netherlands
94. Department of Pharmacology and Therapeutics, University College Cork, Cork, Ireland
95. Department of Statistics & Biostatistics, Rutgers University, Piscataway, N.J. USA
96. Department of Genetics, Rutgers University, Piscataway, N.J. USA.
97. Department of Biology, Norwegian University of Science and Technology, Trondheim, Norway
98. Genome Technology Branch, National Human Genome Research Institute, NIH, Bethesda, MD 20892, USA
99. Clinical Trial Service Unit, Epidemiological Studies Unit, Nuffield Department of Population Health, University of Oxford, Oxford OX3 7LF, UK
100. Division of Endocrinology, Diabetes and Metabolism, Ulm University Medical Centre, D-89081 Ulm, Germany
101. Department of Radiology, University Medical Center Utrecht, Utrecht, The Netherlands
102. Department of Biomedical Informatics, Vanderbilt University, Nashville, TN 37232, USA
103. Department of Human Nutrition, Wageningen University, Wageningen, The Netherlands
104. Department of Dietetics-Nutrition, Harokopio University, Athens, Greece
105. Medical Research Institute, University of Dundee, Ninewells Hospital and Medical School, Dundee DD1 9SY, UK
106. Department of Internal Medicine B, University Medicine Greifswald, D-17475 Greifswald, Germany
107. Institute for Molecular Medicine, University of Helsinki, FI-00014 Helsinki, Finland
108. Laboratory of Epidemiology and Population Sciences, National Institute on Aging, NIH, Bethesda, MD 20892, USA
109. Department of Public Health and Caring Sciences, Geriatrics, Uppsala University, Uppsala 75185, Sweden
110. Kaiser Permanente, Division of Research, Oakland, CA 94612, USA
111. Research Unit of Molecular Epidemiology, Helmholtz Zentrum München - German Research Center for Environmental Health, D-85764 Neuherberg, Germany
112. German Center for Diabetes Research (DZD), Neuherberg, Germany
113. Department of Medicine III, University Hospital Carl Gustav Carus, Technische Universität Dresden, D-01307 Dresden, Germany
114. Department of Public Health and Clinical Medicine, Unit of Nutritional Research, Umeå University , Umeå 90187, Sweden

115. Department of Psychiatry, University of Groningen, University Medical Center Groningen, Groningen, The Netherlands
116. Kuopio Research Institute of Exercise Medicine, Kuopio, Finland
117. MRC Human Genetics Unit, Institute of Genetics and Molecular Medicine, University of Edinburgh, Western General Hospital, Edinburgh, EH4 2XU, Scotland, UK
118. National Heart, Lung, and Blood Institute, the Framingham Heart Study, Framingham MA 01702, USA
119. Department of Neurology, Boston University School of Medicine, Boston, MA 02118, USA
120. Department of Medical Statistics and Bioinformatics, Leiden University Medical Center, 2300 RC Leiden, The Netherlands
121. Faculty of Psychology and Education, VU University Amsterdam, Amsterdam, The Netherlands
122. Department of Epidemiology, University Medical Center Groningen, University of Groningen, 9700 RB Groningen, The Netherlands
123. Department of Medicine, Stanford University School of Medicine, Stanford, CA 94305, USA
124. Institute for Community Medicine, University Medicine Greifswald, D-17475 Greifswald, Germany
125. Division of Medical Genetics, Department of Biomedicine, University of Basel, Basel, Switzerland
126. Department of Genomics, Life & Brain Center, University of Bonn, Bonn, Germany
127. Institute of Human Genetics, University of Bonn, Bonn, Germany
128. Department of Public Health and General Practice, Norwegian University of Science and Technology, Trondheim 7489, Norway
129. Hannover Unified Biobank, Hannover Medical School, Hannover, D-30625 Hannover, Germany
130. Center for Medical Sytems Biology, Leiden, The Netherlands
131. Department of Pulmonary Physiology and Sleep Medicine, NEDLANDS, Western Australia 6009, Australia
132. School of Medicine and Pharmacology, University of Western Australia, CRAWLEY 6009, Australia
133. Uppsala University, Department of Immunology, Genetics & Pathology, SciLifeLab, Rudbeck Laboratory, SE-751 85, Uppsala, Sweden
134. Department of Haematology, University of Cambridge, Cambridge CB2 0PT, UK
135. NHS Blood and Transplant, Cambridge CB2 0PT, UK
136. Department of Medicine, University of Oulo, Oulo, Finland
137. Department of Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL 60611, USA
138. Unit of Periodontology, Department of Restorative Dentistry, Periodontology and Endodontology, University Medicine Greifswald, D-17475 Greifswald, Germany
139. Department of Internal Medicine I, Ulm University Medical Centre, D-89081 Ulm, Germany
140. Institute of Human Genetics, Helmholtz Zentrum München - German Research Center for Environmental Health, D-85764 Neuherberg, Germany
141. Department of Medical Sciences, Cardiovascular Epidemiology, Uppsala University, Uppsala 75185, Sweden
142. Centre for Bone and Arthritis Research, Department of Internal Medicine and Clinical Nutrition, Institute of Medicine, Sahlgrenska Academy, University of Gothenburg, Gothenburg 413 45, Sweden
143. The Genetics of Obesity and Related Metabolic Traits Program, The Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA
144. Steno Diabetes Center A, S, Gentofte DK-2820, Denmark
145. Service of Nephrology, Department of Medicine, Lausanne University Hospital (CHUV), Lausanne 1005, Switzerland

146. School of Social and Community Medicine, University of Bristol, Bristol BS8 2BN, UK
147. Tropical Metabolism Research Unit, Tropical Medicine Research Institute, The University of the West Indies, Mona, Kingston 7, Jamaica
148. Global Health Institute, Department of Life Sciences, École Polytechnique Fédérale de Lausanne, Lausanne, Switzerland
149. Institute of Microbiology, University Hospital and University of Lausanne, Lausanne 1011, Switzerland
150. The Center for Observational Research, Amgen, Inc., Thousand Oaks, CA 91320, USA
151. Center for Evidence-based Healthcare, University Hospital Carl Gustav Carus, Technische Universität Dresden, D-01307 Dresden, Germany
152. Department of Medicine I, University Hospital Grosshadern, Ludwig-Maximilians-Universität, D-81377 Munich, Germany
153. Institute of Medical Informatics, Biometry and Epidemiology, Chair of Genetic Epidemiology, Ludwig-Maximilians-Universität, D-85764 Neuherberg, Germany
154. Deutsches Forschungszentrum für Herz-Kreislauferkrankungen (DZHK) (German Research Centre for Cardiovascular Research), Munich Heart Alliance, D-80636 Munich, Germany
155. Department of Respiratory Medicine, Sir Charles Gairdner Hospital, NEDLANDS, Western Australia 6009, Australia
156. Department of Epidemiology and Biostatistics, EMGO Institute for Health and Care Research, VU University Medical Center, Amsterdam, The Netherlands
157. Department of Internal Medicine, Division of Endocrinology and Metabolism, Medical University of Graz, 8036 Graz, Austria
158. Diabetology-Endocrinology-Nutrition, AP-HP, Bichat Hospital, F-75018 Paris, France
159. INSERM, U872, Centre de Recherche des Cordeliers, F-75006 Paris, France
160. Paris Diderot University, F-75018 Paris, France
161. Clinical Institute of Medical and Chemical Laboratory Diagnostics, Medical University of Graz, Graz 8036, Austria
162. Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA
163. Deutsches Herzzentrum München, Technische Universität München, D-80636 Munich, Germany
164. National Cancer Institute, Bethesda, MD, USA
165. Department of Sociology, University of Helsinki, Helsinki FI-00014, Finland
166. EMGO Institute for Health and Care Research, VU University, 1081BT Amsterdam, The Netherlands
167. Department of Psychiatry, Neuroscience Campus, VU University Amsterdam, Amsterdam, The Netherlands
168. Icelandic Heart Association, Kopavogur 201, Iceland
169. University of Iceland, Reykjavik 101, Iceland
170. Molecular & Cellular Therapeutics, Royal College of Surgeons in Ireland, 123 St Stephens Green, Dublin 2, Ireland
171. Institute of Cardiovascular and Medical Sciences, Faculty of Medicine, University of Glasgow, Glasgow G12 8TA, UK
172. Department of Medical Sciences, Molecular Medicine, Uppsala University, Uppsala 75144, Sweden
173. Department of Public Health Sciences, Stritch School of Medicine, Loyola University of Chicago, Maywood, IL 61053, USA
174. deCODE Genetics, Amgen inc., Reykjavik 101, Iceland
175. Department of Ocology, University of Cambridge, Cambridge CB2 0QQ, UK
176. Department of Internal Medicine section of Geriatrics, Academic Medical Center, Amsterdam, The Netherlands
177. Department of Child and Adolescent Psychiatry, Psychology, Erasmus University Medical Centre, 3000 CB Rotterdam, The Netherlands

178. Department for Health Evidence, Radboud University Medical Centre, 6500 HB Nijmegen, The Netherlands
179. Department of Genetics, Radboud University Medical Centre, 6500 HB Nijmegen, The Netherlands
180. Department of Clinical Chemistry, Ulm University Medical Centre, D-89081 Ulm, Germany
181. Deutsches Forschungszentrum für Herz-Kreislauferkrankungen (DZHK) (German Research Centre for Cardiovascular Research), partner site Hamburg/Lubeck/Kiel, Lubeck, Germany
182. Institut für Integrative und Experimentelle Genomik, Universität zu Lübeck, D-23562 Lübeck, Germany
183. Department of Community Medicine, Faculty of Health Sciences, UiT The Arctic University of Tromsø, Tromsø, Norway
184. MRC Unit for Lifelong Health and Ageing at UCL, London WC1B 5JU, UK
185. Department of Epidemiology and Public Health, EA3430, University of Strasbourg, Faculty of Medicine, Strasbourg, France
186. Department of Internal Medicine, University Medical Center Groningen, University of Groningen, 9700RB Groningen, The Netherlands
187. Pathology and Laboratory Medicine, The University of Western Australia, Western Australia 6009, Australia
188. Cedars-Sinai Diabetes and Obesity Research Institute, Los Angeles, CA, USA
189. Department of Prosthetic Dentistry, Gerostomatology and Dental Materials, University Medicine Greifswald, D-17475 Greifswald, Germany
190. Biological Psychology, VU University Amsterdam, 1081BT Amsterdam, The Netherlands
191. Institute of Social and Preventive Medicine (IUMSP), Centre Hospitalier Universitaire Vaudois and University of Lausanne, Lausanne, Switzerland
192. Ministry of Health, Victoria, Republic of Seychelles
193. Laboratory Medicine, Hospital of Desio, department of Health Sciences, University of Milano, Bicocca, Italy
194. Clinical Pharmacology Unit, University of Cambridge, Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ, UK
195. Clinical Pharmacology and Barts and The London Genome Centre, William Harvey Research Institute, Barts and The London School of Medicine and Dentistry, Queen Mary University of London, Charterhouse Square, London EC1M 6BQ, UK
196. Center for Human Genetics Research, Vanderbilt University Medical Center, Nashville TN 37203, USA
197. Department of Molecular Physiology and Biophysics, Vanderbilt University, Nashville, TN 37232, USA
198. Department of Biostatistics, Boston University School of Public Health, Boston, MA 02118, USA
199. Department of Public Health and Primary Care, University of Cambridge, Cambridge, UK
200. Division of Cardiovascular Epidemiology, Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden, Stockholm 17177, Sweden
201. Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, 3584 CX Utrecht, The Netherlands
202. Clinic of Cardiology, West-German Heart Centre, University Hospital Essen, Essen, Germany
203. Department of General Practice and Primary Health Care, University of Helsinki, FI-00290 Helsinki, Finland
204. Unit of General Practice, Helsinki University Central Hospital, Helsinki 00290, Finland
205. Department of Internal Medicine, University of Pisa, Pisa, Italy
206. CNR Institute of Clinical Physiology, University of Pisa, Pisa, Italy

207. Department of Cardiology, Toulouse University School of Medicine, Rangueil Hospital, Toulouse, France
208. Robertson Center for Biostatistics, University of Glasgow, Glasgow, UK
209. NorthShore University HealthSystem, Evanston, IL, University of Chicago, Chicago, IL, USA
210. Service of Therapeutic Education for Diabetes, Obesity and Chronic Diseases, Geneva University Hospital, Geneva CH-1211, Switzerland
211. Vanderbilt University School of Medicine, Department of Medicine, Pharmacology, Pathology, Microbiology and Immunology, Nashville, Tennessee, USA
212. Leeds MRC Medical Bioinformatics Centre, University of Leeds, UK
213. Institute of Biomedical & Clinical Science, University of Exeter, Barrack Road, Exeter, EX2 5DW
214. Department of Psychiatry, Washington University School of Medicine, St. Louis, MO 63110, USA
215. Center for Biomedicine, European Academy Bozen, Bolzano (EURAC), Bolzano 39100, Italy
216. Affiliated Institute of the University of Lübeck, D-23562 Lübeck, Germany
217. Division of Genomic Medicine, National Human Genome Research Institute, National Institutes of Health, Bethesda, MD, USA
218. Institute of Cardiovascular Science, University College London, WC1E 6BT, UK
219. Department of Vascular Medicine, Academic Medical Center, Amsterdam, The Netherlands
220. Centre for Cardiovascular Genetics, Institute Cardiovascular Sciences, University College London, London WC1E 6JJ, UK
221. Cardiovascular Genetics Division, Department of Internal Medicine, University of Utah, Salt Lake City, Utah 84108, USA
222. School of Population Health and Sansom Institute for Health Research, University of South Australia, Adelaide 5000, Australia
223. South Australian Health and Medical Research Institute, Adelaide, Australia
224. Centre for Paediatric Epidemiology and Biostatistics, UCL Institute of Child Health, London WC1N 1EH, UK
225. National Institute for Health and Welfare, FI-90101 Oulu, Finland
226. MRC Health Protection Agency (HPE) Centre for Environment and Health, School of Public Health, Imperial College London, UK
227. Unit of Primary Care, Oulu University Hospital, FI-90220 Oulu, Finland
228. Biocenter Oulu, University of Oulu, FI-90014 Oulu, Finland
229. Institute of Health Sciences, FI-90014 University of Oulu, Finland
230. Hjelt Institute Department of Public Health, University of Helsinki, FI-00014 Helsinki, Finland
231. Department of Forensic Molecular Biology, Erasmus MC, 3015GE Rotterdam, The Netherlands
232. UKCRC Centre of Excellence for Public Health (NI), Queens University of Belfast, Northern Ireland
233. Faculty of Medicine, Institute of Health Sciences, University of Oulu, Oulu, Finland
234. Unit of General Practice, Oulu University Hospital, Oulu, Finland
235. Department of Urology, Radboud University Medical Centre, 6500 HB Nijmegen, The Netherlands
236. Imperial College Healthcare NHS Trust, London W12 0HS, UK
237. National Heart and Lung Institute, Imperial College, London W12 0NN, UK
238. Department of Epidemiology and Public Health, UCL London, WC1E 6BT, UK
239. Department of Medicine, Kuopio University Hospital and University of Eastern Finland, FI-70210 Kuopio, Finland
240. Department of Physiology, Institute of Biomedicine, University of Eastern Finland, Kuopio Campus, Kuopio, Finland

241. Department of Clinical Physiology and Nuclear Medicine, Kuopio University Hospital and University of Eastern Finland, Kuopio, Finland
242. Department of Clinical Chemistry, Fimlab Laboratories and School of Medicine University of Tampere, FI-33520 Tampere, Finland
243. Department of Health Sciences, University of Milano, I 20142, Italy
244. Fondazione Filarete, Milano I 20139, Italy
245. Division of Nephrology and Dialysis, San Raffaele Scientific Institute, Milano I 20132, Italy
246. Università Vita-Salute San Raffaele, Milano I 20132, Italy
247. Institut Universitaire de Cardiologie et de Pneumologie de Québec, Faculty of Medicine, Laval University, Quebec, QC G1V 0A6, Canada
248. Institute of Nutrition and Functional Foods, Laval University, Quebec, QC G1V 0A6, Canada
249. Department of Biostatistics, University of Washington, Seattle, WA 98195, USA
250. Department of Surgery, University Medical Center Utrecht, 3584 CX Utrecht, The Netherlands
251. Department of Biostatistics, University of Liverpool, Liverpool L69 3GA, UK
252. Department of Pediatrics, University of Iowa, Iowa City, Iowa IA 52242, USA
253. Institute of Epidemiology II, Helmholtz Zentrum München - German Research Center for Environmental Health, Neuherberg, Germany, D-85764 Neuherberg, Germany
254. Department of Neurology, General Central Hospital, Bolzano 39100, Italy
255. Department of Nutrition, Harvard School of Public Health, Boston, Massachusetts, USA
256. Department of Clinical Physiology and Nuclear Medicine, Turku University Hospital, FI-20521 Turku, Finland
257. Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, FI-20521 Turku, Finland
258. Human Genomics Laboratory, Pennington Biomedical Research Center, Baton Rouge, LA 70808, USA
259. Center for Systems Genomics, The Pennsylvania State University, University Park, PA 16802, USA
260. Croatian Centre for Global Health, Faculty of Medicine, University of Split, 21000 Split, Croatia
261. Department of Cardiovascular Sciences, University of Leicester, Glenfield Hospital, Leicester LE3 9QP, UK
262. National Institute for Health Research (NIHR) Leicester Cardiovascular Biomedical Research Unit, Glenfield Hospital, Leicester, LE3 9QP, UK
263. South Carelia Central Hospital. 53130 Lappeenranta. Finland
264. Paul Langerhans Institute Dresden, German Center for Diabetes Research (DZD), Dresden, Germany
265. International Centre for Circulatory Health, Imperial College London, London W2 1PG, UK
266. Program for Personalized and Genomic Medicine, and Division of Endocrinology, Diabetes and Nutrition, University of Maryland School of Medicine, Baltimore, MD 21201, USA
267. Geriatric Research and Education Clinical Center, Vetrans Administration Medical Center, Baltimore, MD 21201, USA
268. HUCH Heart and Lungcenter, Department of Medicine, Helsinki University Central Hospital, FI-00290 Helsinki, Finland
269. Université de Montréal, Montreal, Quebec H1T 1C8, Canada
270. Department of Kinesiology, Laval University, Quebec, QC G1V 0A6, Canada
271. Dipartimento di Scienze Farmacologiche e Biomolecolari, Università di Milano & Centro Cardiologico Monzino, IRCCS, Milan 20133, italy
272. Department of Food Science and Nutrition, Laval University, Quebec, QC G1V 0A6, Canada

273. The electronic medical records and genomics (eMERGE) consortium
274. Myocardial Infarction Genetics (MIGen) Consortium
275. Membership to this consortium is provided below.
276. Population Architecture using Genomics and Epidemiology Consortium
277. The LifeLines Cohort Study, University of Groningen, University Medical Center Groningen, Groningen, The Netherlands
278. Institut Pasteur de Lille; INSERM, U744; Université de Lille 2; F-59000 Lille, France
279. Department of Cardiology, Division Heart and Lungs, University Medical Center Utrecht, 3584 CX Utrecht, The Netherlands
280. Durrer Center for Cardiogenetic Research, Interuniversity Cardiology Institute Netherlands-Netherlands Heart Institute, 3501 DG Utrecht, The Netherlands
281. Lee Kong Chian School of Medicine, Imperial College London and Nanyang Technological University, Singapore, 637553 Singapore, Singapore
282. Health Science Center at Houston, University of Texas, Houston, TX, USA
283. Department of Medicine, Division of Genetics, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA
284. Department of Epidemiology, University Medical Center Utrecht, Utrecht, The Netherlands
285. Lund University Diabetes Centre and Department of Clinical Science, Diabetes & Endocrinology Unit, Lund University, Malmö 221 00, Sweden
286. Harvard School of Public Health, Department of Epidemiology, Harvard University, Boston, MA 2115, USA
287. Interuniversity Cardiology Institute of the Netherlands (ICIN), Utrecht, the Netherlands
288. Albert Einstein College of Medicine. Department of epidemiology and population health, Belfer 1306, NY 10461, USA
289. Center for Human Genetics, Division of Public Health Sciences, Wake Forest School of Medicine, Winston-Salem, NC 27157, USA
290. Synlab Academy, Synlab Services GmbH, Mannheim, Germany
291. Department of Clinical Genetics, Erasmus University Medical Center, Rotterdam, The Netherlands
292. Harvard Medical School, Boston, MA 02115, USA
293. Institute for Translational Genomics and Population Sciences, Los Angeles BioMedical Research Institute at Harbor-UCLA Medical Center, Torrance, CA, USA
294. Finnish Diabetes Association, Kirjoniementie 15, FI-33680 Tampere, Finland
295. Pirkanmaa Hospital District, Tampere, Finland
296. Center for Non-Communicable Diseases, Karatchi, Pakistan
297. Department of Medicine, University of Pennsylvania, Philadelphia, USA
298. Laboratory of Genetics, National Institute on Aging, Baltimore, MD 21224, USA
299. Instituto de Investigacion Sanitaria del Hospital Universario LaPaz (IdiPAZ), Madrid, Spain
300. Diabetes Research Group, King Abdulaziz University, Jeddah, Saudi Arabia
301. Centre for Vascular Prevention, Danube-University Krems, 3500 Krems, Austria
302. Department of Public Health and Clinical Nutrition, University of Eastern Finland, Finland
303. Research Unit, Kuopio University Hospital, Kuopio, Finland
304. Institute of Cellular Medicine, Newcastle University, Newcastle NE1 7RU, UK
305. Institute of Medical Informatics, Biometry and Epidemiology, Chair of Epidemiology, Ludwig-Maximilians-Universität, D-85764 Munich, Germany
306. Klinikum Grosshadern, D-81377 Munich, Germany
307. Institute of Epidemiology I, Helmholtz Zentrum München - German Research Center for Environmental Health, Neuherberg, Germany, D-85764 Neuherberg, Germany
308. Department of Pulmonology, University Medical Center Utrecht, Utrecht, The Netherlands
309. King Abdulaziz University, Jeddah 21589, Saudi Arabia

310. Department of Internal Medicine, Division of Gastroenterology, and Department of Computational Medicine and Bioinformatics, University of Michigan, Ann Arbor, MI 48109
311. Faculty of Medicine, University of Iceland, Reykjavik 101, Iceland
312. University of Cambridge Metabolic Research Laboratories, Institute of Metabolic Science, Addenbrooke's Hospital, Cambridge CB2 OQQ, UK
313. NIHR Cambridge Biomedical Research Centre, Institute of Metabolic Science, Addenbrooke's Hospital, Cambridge CB2 OQQ, UK
314. Carolina Center for Genome Sciences, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599, USA
315. Division of Population Health Sciences & Education, St George's, University of London, London SW17 0RE, UK
316. Service of Medical Genetics, CHUV University Hospital, Lausanne, Switzerland
317. Oxford NIHR Biomedical Research Centre, Oxford University Hospitals NHS Trust, Oxford, OX3 7LJ, UK
318. Department of Internal Medicine, Division of Cardiovascular Medicine, University of Michigan, Ann Arbor, MI, USA
319. Department of Computational Medicine and Bioinformatics, University of Michigan, Ann Arbor, MI, USA
320. Department of Human Genetics, University of Michigan, Ann Arbor, MI, USA
321. Harvard School of Public Health, Department of Biostatistics, Boston, MA 02115, USA
322. The Mindich Child Health and Development Institute, Icahn School of Medicine at Mount Sinai, New York, NY 10029, USA
323. Biosciences Research Division, Department of Primary Industries, Victoria 3083, Australia
324. Department of Food and Agricultural Systems, University of Melbourne, Victoria 3010, Australia

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