**S2 Note. Summary of biological description for novel BP loci**

Some information of the nearest genes for blood pressure novel loci

**rs73884351** (***LOC105374235-KCNMB2-IT1***, 3q26.32): Two genes reside within ± 500 kb of the lead SNV. The rs73884351 is located within an uncharacterized ***LOC105374235*** and 248 kb downstream of ***KCNMB2-IT1*** (***KCNMB2*** antisense RNA 1, transcript variant 1, long non-coding RNA). ***KCNMB2*** (Potassium Calcium-Activated Channel Subfamily M Regulatory Beta Subunit 2) protein is a regulatory subunit of the calcium activated potassium ***KCNMA1*** (maxiK) channels, which are large conductance, voltage and calcium-sensitive potassium channels that are fundamental to the control of smooth muscle tone and neuronal excitability[1]. The ***KCNMB2*** protein decreases the activation time of MaxiK alpha subunit currents and may participate in ***KCNMA1*** inactivation in chromaffin cells of the adrenal gland or in hippocampal neurons[2, 3]. Studies of ***KCNMB2*** knockout mice have demonstrated a link of hypertension to deficient potassium (K) secretion and aldosteronism[4, 5]. The ***KNCMB2***(-/-) mice had reduced ability to excrete K(+) into the urine but achieved K(+) balance through an aldosterone-mediated, β2-independent mechanism. The ***KNCMB2***(-/-) mice did not display salt-sensitive hypertension and were able to decrease plasma aldosterone on a high-Na(+) diet, although plasma aldosterone remained elevated[5]. GWA studies have found suggestive associations of near or within ***KCNMB2-IT1*** variants with cognitive performance[6], plasma clusterin levels[7], hippocampal sclerosis[8], bipolar disorder and schizophrenia[9], amyotrophic lateral sclerosis[10], and asthma childhood onset[11].

**rs145429126** (***GABRB1***, 4p12): Five genes reside within ± 500 kb of the lead SNV. The rs145429126 is located within an intron of ***GABRB1*** (gamma-aminobutyric acid type A receptor alpha4 subunit). The gamma-aminobutyric acid (GABA) A receptor is a multisubunit chloride channel that acts as inhibitory neurotransmitters in the central nervous system[12]. Most of the genes encoding GABAA receptors are placed in chromosomal clusters, and the GABAA cluster on chromosome 4p12 includes ***GABRB1***, ***GABRA4***, ***GABRA2*** and ***GABRG1***[13]. Alcohol abuse has been associated with facilitation of neurotransmission mediated by the brain’s major inhibitory transmitter, GABA, acting via GABAA receptors[14-16]. Mutations in ***GABRB1*** have been shown to promote alcohol consumption in mice, causing high ethanol consumption accompanied by spontaneous GABA ion channel opening and increased accumbal tonic current[16]. In addition, studies in humans have shown significant allelic association between the risk of alcohol dependence and ***GABRB1*** polymorphisms[14, 15, 17, 18]. ***GABRB1*** has also been associated with bipolar disorder[13], schizophrenia[19], autism spectrum disorder[20], and thalamus volume and their interactive effects on intelligence[21]. GWA studies have shown evidence of association of variants within or near to ***GABRB1*** with age of onset of Alzheimer’s disease[22], or suggestive associations with type 2 diabetes[23], migraine clinic-based[24], and post bronchodilator FEV1[25].

**rs80158983** (***EYS***, 6q12): Two genes reside within ± 500 kb of the lead SNV. The rs80158983 is located within an intron of ***EYS*** (eyes shut homolog, Drosophila). ***EYS*** protein is expressed in the photoreceptor layer of the retina, and the gene is mutated in autosomal recessive retinitis pigmentosa. Analysis of ***EYS*** cDNA has demonstrated that ***EYS*** gene products are expressed with relative abundance in the spinal cord[26]. The structural similarities of these ***EYS*** products to members of the Notch signaling pathway and to agrin suggest a possible functional role in the maintenance and regeneration of the structural integrity of skeletal muscle[26]. Evidence of association of ***PTP4A1-PHF3****-****EYS*** of rare[27] and common[26] variants were reported for alcohol dependence in a multi-racial population study[28]. A GWA study found a suggestive association of ***EYS*** variant with heart failure related metabolomic profile (dihydroxy docosatrienoic acid) in African Ancestry[29] and in European ancestry (serotonin)[30] In addition, GWA studies have reported suggestive associations of variants near or within ***EYS*** with type 2 diabetes[31], statin-induced myopathy[26], and glycosylation of immunoglobulin G[32].

**rs76987554** (***TARID***, 6q23.2): Six genes reside within ± 500 kb of the lead SNV. The rs76987554 is within an intron of ***TARID*** (transcription factor 21). ***TARID*** is a member of the basic helix-loop-helix (bHLH) transcription factor (TF) family and is essential for the development of diverse cell types during embryogenesis of the heart, lung, kidney, and spleen[33-36]. Transcriptional studies in human coronary artery smooth muscle cells demonstrated that ***JUN*** family members and other AP-1 (atypical activator protein 1) -related TFs regulate ***TARID*** transcription, and disruption of ***TARID*** transcription pathway may account in part for coronary artery disease susceptibility[37, 38]. GWA studies have identified variants of ***TARID*** associated with coronary artery disease[39, 40] and coronary heart disease[33, 41]. The lead SNV rs12190287 for these diseases was also identified as an expression quantitative trait locus (eQTL) associated with increased ***TARID*** gene expression in both liver and adipose tissue[34, 41]. The rs12190287 is located within the 3’ untranslated region (3’UTR) of ***TARID***[37]; however rs12190287 and our lead SNV rs76987554 are in low LD (r2 = 0.01). In addition, GWA studies have found suggestive associations (1.0x10-5<*P*>5.0x10-8) near or within variants of ***TARID*** with antihypertensive response to angiotensin II receptor blocker therapy [42], post bronchodilator FEV1[25], schizophrenia[43], age of onset for Alzheimer’s disease[22], developmental language disorder[44], and visceral fat[45]. Another biologically potential candidate gene is ***SGK1***, which encodes serine/threonine-protein kinase that participates in the regulation of renal Na(+) retention, renal K(+) elimination, salt appetite, gastric acid secretion, intestinal Na(+)/H(+) exchange and nutrient transport, insulin-dependent salt sensitivity of blood pressure, salt sensitivity of peripheral glucose uptake, cardiac repolarization and neuroexcitability[46]. Insulin and growth factors, via ***PI3K*** (phosphatidylinositol-3-kinase), ***PDK1*** (3-phosphoinositide-dependent kinase 1), and mTOR (mammalian target of rapamycin), activate ***SGK1***[47] which regulates ***SMCT1/SLC5A8*** (Na+/monocarboxylate transporter 1)[48, 49], ***SGLT1/ SLC5A1*** (Na+-glucose transporter 1)[50], and ***SMIT/SLC5A3*** (myo-inositol cotransporter)[49, 50]. Our lead SNV rs76987554 is located 410 kb downstream of ***SGK1***, which has been associated with increased blood pressure[51-55], obesity and prevalence of type 2 diabetes[47], and stroke[56]. In addition, ***SGK1*** may contribute to the mechanisms underlying behavioral responses to chronic ethanol exposure. A study demonstrated that the hypothalamic pituitary adrenal axis and glucocorticoid receptor signaling mediate acute ethanol induction of Sgk1 transcription in mouse prefrontal cortex[57].

**rs6995407** (***LOC105379224*** (ncRNA)-***SGK223***, 8p23.1): The rs6995407 is located 145 kb upstream of ***SGK223***, which encodes a human pseudokinase and functions as an oncogenic scaffold recruiting a distinct repertoire of signaling proteins[58]. A similar ***SGK223*** protein in rat (Pragmin) binds to Rho family GTPase 2 (Rnd2) and regulates neurite outgrowth via activation of Ras homolog gene family, member A (RhoA)[59]. GWA studies have identified variants near to ***SGK223*** associated with schizophrenia[60] and neuroticism[61].

**rs453301** (***LOC102724880*** (ncRNA)-***PPP1R3B***, 8p23.1): The rs453301 is located 24 kb upstream of ***PPP1R3B***, which encodes the regulatory subunit 3b of protein phosphatase-1 and is expressed in skeletal muscle and liver. ***PPP1R3B*** promotes glycogen synthesis and inhibits glycogen breakdown to glucose 1-phosphate that can be converted to glucose 6-phosphate by phosphoglucomutase[62, 63]. ***PPP1R3B*** regulates protein phosphatase-1 (PP1) catalytic subunit and increases PP1 dephosphorylation of glycogen synthase and phosphorylase kinase[63]. The glycogen synthase is activated by dephosphorylation and the phosphorylase kinase is inactivated by dephosphorylation[62]. ***PPP1R3B*** has been reported to associate with liver enzyme levels (alkaline phosphatase)[64], fasting insulin and glucose interaction with BMI[45], LDL cholesterol, HDL cholesterol and total cholesterol[65-69], C-reactive protein levels[70], glycemic traits (pregnancy)[71], metabolite levels[72], Alzheimer's disease[73], and systemic lupus erythematosus[74].

**rs11774915** (***LOC157273*** (ncRNA), 8p23.1): The rs11774915 is located 29 kb upstream of ***LOC157273***. A GWA study have identified association between ***LOC157273*** with fibrinogen levels[75]. Evidence of association has been observed between ***LOC157273*** with plasma fibrinogen level and incident hypertension among men, but not among women[76, 77]. In addition, moderate drinking was shown to decrease the levels of fibrinogen[78]. and both factors have been suggested to contribute to the protective effect on cardiovascular disease.

**rs55868514** (***TNKS***, 8p23.1): The rs55868514 is located 41 kb upstream of ***TNKS***, which encodes a tankyrase (Tnks). Tnks proteins belong to the superfamily of poly(ADP-ribose) polymerases (PARPs) that catalyze the addition of poly(ADP-ribose) onto substrates, which influence the activity and stability of the modified proteins[79, 80]. ***TNKS*** also regulates the centrosome function[81]. *TNKS* proteins are expressed in a large number of tissues and control a broad range of cellular processes that include DNA damage repair, Wnt signaling, and telomere length maintenance[79, 82]. Deletion of ***TNKS*** gene results in embryonic lethality[83]. Tnks protein binds directly to axin, a negative regulator of the canonical Wnt/β-catenin signaling pathway, forming a destruction complex with glycogen synthase kinase 3β (GSK-3β) and adenomatous polyposis coli (APC) to degrade β-catenin[84]. *TNKS* is also involved in the regulation of ***GLUT4*** (glucose transporter type 4) trafficking in 3T3-L1 adipocytes[85]. Variants of ***TNKS*** are associated with type 2 diabetes[86] and cancers, including gastric cancer[87], breast cancer[88], colon cancer[88], and lung cancer[89]. GWA studies have identified associations between ***TNKS/MSRA*** with obesity limited to children and adolescents[85], neuroticism[61], and osteoarthritis[90].

**rs483916** (***MIR124-1***, 8p23.1): The rs483916 is located 33 kb upstream of ***MIR124-1***, a microRNA 124-1 gene. miRNAs are short non‑coding RNAs that play a key role as post‑transcriptional modulators of gene expression by targeting mRNAs for translational repression or destabilization. miRNAs participate in various biological events and pathological processes[91]. A regulatory network study demonstrated that ***miR-124*** and ***miR-135a*** are potential regulators of the mineralocorticoid receptor gene (***NR3C2***) expression, and could participate in the regulation of renin-angiotensin-aldosterone system and thereby might be involved in BP regulation[92].

**rs11786677** (***MSRA***, 8p23.1): The rs11786677 is located within an intron of ***MSRA***, which encodes a ubiquitous and highly conserved protein, the methionine sulfoxide reductase A. ***MSRA*** catalyzes the enzymatic reduction of methionine sulfoxide to methionine and is implicated in oxidative stress protection, reducing methionine sulfoxide residues in proteins back to methionine[93], thereby repairing and protecting proteins from oxidation. ***MSRA*** is mainly expressed in kidney, liver, brain, and adipose tissue. Chronic excess of ROS leads to mitochondrial dysfunction in liver and skeletal muscle which contribute to insulin resistance[94]. Deletion of *Msra* show high-fat-diet-induced insulin resistance in mice, most likely due to increased oxidative stress[95], while overexpression reduces insulin resistance in old mice[96]. ***MSRA*** may also play a neuroprotective role in Alzheimer’s disease[97, 98]; however the role of methionine sulfoxide reductase in the neurodegenerative diseases is yet to be determined. GWA studies have identified associations of ***MSRA*** with obesity limited to children and adolescents[85], neuroticism[61], schizophrenia[60], chronotype[99], and suggestive association with hypertension[100].

**rs4841409** (***RP1L1***, 8p23.1): The rs4841409 is located 3 kb upstream of ***RP1L1***, which encodes a retinitis pigmentosa 1-like 1 protein that belongs to a member of the doublecortin family. Mutations in the***RP1L1*** gene cause autosomal dominant occult macular dystrophy [101].The expression of ***RP1L1*** protein is limited to the retina, and it appears to be specific to photoreceptors [102].

**rs7814795** (***MIR4286***, 8p23.1): The rs7814795 is located 5 kb of ***MIR4286*** (Homo sapiens microRNA 4286). A study of microRNA expression revealed that ***MIR4286*** mediates proliferation and apoptosis in melanoma cells[103]. However, the regulatory function of ***MIR4286*** remains unknown.

**rs7814757** (***PINX1***, 8p23.1): The rs7814757 is located within an intron of ***PINX1*** (PIN2/TRF1-interacting telomerase inhibitor 1). ***PINX1*** is involved in preventing telomere degradation and facilitating telomerase-based telomere elongation[104]. Overexpression of ***PINX1*** inhibits telomerase activity, shortens telomeres, and induces crisis, whereas reduction of ***PINX1*** of endogenous PinX1 results in an increase in telomerase activity and elongation of telomeres[105]. ***PINX1*** inhibits cell proliferation and may be a putative tumor suppressor[106]. Studies have suggested that ***PINX1*** participates in the development of cancers[105, 106]. including hepatocellular carcinomas. Overexpression of ***PINX1*** were associated with alcohol-related cirrhosis and fibrosis[107]. ***PINX1*** was reported genome-wide associated with carotid intima-media thickness[108], triglycerides[66], and substance related to lung cancer (3-hydroxy-1-methylpropylmercapturic acid levels) in smokers[109].

**rs4841465** (***XKR6***, 8p23.1): The rs4841465 is located within an intron of ***XKR6*** (XK, Kell blood group complex subunit-related family, member 6). GWA studies have shown evidence of association between ***XKR6*** with triglycerides[110] and eosinophilic esophagitis[66], and suggestive associations with asthma and hay fever[111], systemic lupus erythematosus[112, 113], retinal vascular caliber[114], and response to antipsychotic therapy[115].

XKR6 was found to be

associated with SLE in the SLEGEN GWAS (13). In a further

European case study, evidence of XKR6 susceptibility loci

associated with certain sub-phenotypes of SLE was found,

such as between SNV rs4240671 in XKR6 and lupus nephritis

(P = 0.0006) (14). TMEM39A (rs1132200) was identied as

a susceptibility locus in multiple sclerosis in a comprehen-

sive follow-up of the rst GWAS, which was validated in a

replication study in Spain (15, 16).

**rs9969423** (***FAM167A-AS1***, 8p23.1): The rs9969423 is located within an intron of ***FAM167A-AS1*** (FAM167A antisense RNA 1). A suggestive associations of ***FAM167A-AS1*** with cognitive performance was described in a GWA study[116].

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**rs6983727** (***BLK***, 8p23.1): The rs6983727 is located 130 kb downstream of ***BLK***, which encodes a non-receptor tyrosine-kinase of the SRC family of proto-oncogenes that are typically involved in cell proliferation and differentiation. The ***BLK*** protein is present in many tissues, but its expression is highly restricted to the B-cell lineage and is dependent on developmental stage[117]. The ***BLK*** protein stimulates insulin synthesis and secretion in response to glucose and enhances the expression of several pancreatic beta-cell transcription factors. Mutations at the ***BLK*** locus are associated with maturity onset diabetes of the young and β-cell dysfunction[118]. A GWA study identified association of ***BLK-LINC00208*** variants with Barrett's oesophagus or oesophageal adenocarcinoma[119], and the strongest associated SNV rs10108511 (*P* = 2.12 x 10-9) is in high LD with our lead SNV rs4841564 for alcohol-BP interaction (r2 = 0.91). Evidence of association of alcohol use disorders with an increased risk of both squamous cell carcinoma and adenocarcinoma of the esophagus has been reported in a large population-based study in Sweden[120]. In addition, a suggestive genome-wide association was observed between a variant near to ***BLK-LINC00208*** with alcohol dependence[121]. GWA studies have also shown association between ***BLK*** or near genes with systemic lupus erythematosus[117, 122-124], rheumatoid arthritis[125, 126], and Kawasaki disease[127, 128].

**rs13280442** (***LINC00208***, 8p23.1): The rs13280442 is located 106 kb upstream of ***LINC00208*** (long intergenic non-protein coding RNA 208).

**rs36038176** (***GATA4***, 8p23.1): Twenty genes reside within ± 500 kb of the lead SNV. The rs36038176 is located within intron of ***GATA4***, which encodes a binding protein 4 of zinc-finger transcription factor (TF)[129]. ***GATA4*** regulates genes involved in embryogenesis and myocardial differentiation and function[130] .***GATA4*** also regulates negatively astrocyte cell proliferation and positively apoptosis[131], and is a TF of atrial natriuretic peptide (ANP) associated with the pathophysiology of alcohol dependence[132]. ***GATA4*** rs13273672 has been associated with alcohol dependence in several studies[133-136]. A functional magnetic resonance imaging study in alcohol-dependent patients observed a stronger alcohol-specific amygdala response, which predicted a lowered risk for relapse to heavy drinking in the ***GATA4*** rs13273672 -AA-homozygotes as compared with -G allele carriers[137]. In addition, studies have reported association between ***GATA4*** with idiopathic atrial fibrillation[138] and neurological and psychological disorders[131].

**rs79505281** (***UNC5D***, 8p12): ***UNC5D*** is the only gene that resides within ± 500 kb of the lead SNV. The rs79505281 is located 47 kb downstream of ***UNC5D***, which is a netrin-1 receptor UNC5H family member. ***UNC5D*** is induced during DNA damage-mediated apoptosis and transcriptional target of tumor suppressor p53[139]. Experimental model suggested that Unc5D regulates p53-dependent apoptosis in neuroblastoma cells[140] and in renal cell carcinoma[141]. A GWA study found a suggestive association between variant near ***UNC5D*** with post bronchodilator FEV1/FVC ratio[25].

**rs115888294** (***CDH17***, 8q22.1): Nine genes reside within ± 500 kb of the lead SNV. The rs115888294 is 22 kb downstream of ***CDH17*** (cadherin 17). This gene is a member of the cadherin superfamily, genes encoding calcium-dependent, membrane-associated glycoproteins. ***CDH17*** protein catalyzes the initial reaction in O-linked oligosaccharide biosynthesis, the transfer of an N-acetyl-D-galactosamine residue to a serine or threonine residue on the protein receptor. The protein is a component of the gastrointestinal tract and pancreatic ducts, acting as an intestinal proton-dependent peptide transporter in the first step in oral absorption of many medically important peptide-based drugs[142], and may be involved in the morphological organization of liver and intestine[143]. Association of ***CDH17*** with hypertension-related traits was suggested in a correlated meta-analysis of the African ancestry of the Continental Origin and Genetic Epidemiology Network (COGENT). The cross-phenotype association method was demonstrated to improve statistical power with summary statistics in the combined effects of hypertension, SBP and DBP traits over a single-trait analysis[144]. A GWA study showed evidence of association of ***CDH17*** with diisocyanate-induced asthma[145].

**rs61494734** (***LINGO2***, 9p21.1): Three genes reside within ± 500 kb of the lead SNV. The rs61494734 is located within an intron of ***LINGO2*** (leucine rich repeat and Ig domain containing 2), which has been implicated in essential tremor and Parkinson disease[146, 147], and neurological pathways[148]. A positron emission tomography (PET) study indicated that alcohol-induced suppression of essential tremor patients, which is mediated via a reduction of cerebellar synaptic overactivity, resulted in increased afferent input to the inferior olivary nuclei[149]. Evidence of association between ***LINGO2***-rs12348435 with age at onset of alcohol dependence has been reported in a genome-wide survival analysis in large high-risk families from the Collaborative Study on the Genetics of Alcoholism (COGA)[150]. Also, a suggestive association between ***LINGO2***-rs10968576 with body mass has been described in GIANT Consortium[151] and in a cohort of elderly Swedes[152]. In addition, GWA studies reported significant association of ***LINGO2*** with motion sickness[148], and suggestive associations between variants near or within ***LINGO2*** with pharmacokinetics of olanzapine in severe mental disorder[153], schizophrenia[60], cannabis dependence[154], type 2 diabetes[155], post bronchodilator FEV1/FVC ratio[25], airway responsiveness in COPD[156], and lupus nephritis in systemic lupus erythematosus[157].

**rs73655199** (***CORO2A***, 9q22.33): Eleven genes reside within ± 500 kb of the lead SNV. The rs73655199 is located within an intron of ***CORO2A*** (coronin 2A). Coronins are highly conserved F-actin binding proteins that are important for cell motility, actin dynamics, cell cycle progression, signal transduction, apoptosis, and gene regulation[158]. ***CORO2A*** has been implicated in the regulation of the focal adhesion turnover rate[158], and identified as a component of the nuclear receptor co-repressor (NCoR) complex with a function as an NCoR exchange factor[159]. ***CORO2A*** mediates toll-like receptors (TLRs) -induced NCoR turnover by a mechanism involving interaction with oligomeric nuclear actin[160, 161]. The interaction of ***CORO2A*** via a SIM-motif (small ubiquitin-like modifier (SUMO) 2/3 interacting motif) located in its coiled coil region with SUMOylated liver X receptors (LXRs) prevents NCoR clearance from target gene promoters[160, 161]. ***CORO2A*** expression is associated with colorectal adenoma-adenocarcinoma sequence and oncogenic signaling[160], and may mediate actin-dependent de-repression of inflammatory response genes[161]. Our lead SNV rs73655199 is located 143 kb downstream of ***GABBR2***, which encodes gamma-aminobutyric acid type B receptor subunit 2 that belongs to the G-protein coupled receptor 3 family and GABA-B receptor subfamily. The GABA-B receptors inhibit neuronal activity through G protein-coupled second-messenger systems, which regulate the release of neurotransmitters, and the activity of ion channels and adenylyl cyclase. GABA-B receptors are expressed in human aortic smooth muscle cells and regulate the intracellular Ca(2+) concentration[162]. Evidence of association of ***GABBR2*** variants were described for alcohol dependence[163] and nicotine dependence[164, 165]. In addition, significant reductions were noticed in protein levels of ***GABBR2*** in lateral cerebella from subjects with schizophrenia, bipolar disorder, and major depression when compared with controls[166]. GWA studies have reported significant associations of variants near or within ***CORO2A*** for serum thyroid-stimulating hormone levels[167-169], hypothyroidism[170, 171], thyroid cancer[168, 172-174], severe influenza A (H1N1)[175], and plasma homocysteine[176]. Suggestive genome-wide associations were also detected for coronary heart disease[177], Alzheimer’s disease age of onset[22], schizophrenia[60], and posttraumatic stress disorder[178].

**rs4253197** (***ERCC6***, 10q11.23): Fourteen genes reside within ± 500 kb of the lead SNV. The rs4253197 is located within intron of ***ERCC6*** (excision-repair cross-complementing rodent repair deficiency, complementation group 6), which encodes the Cockayne Syndrome Group B (CSB) protein that participates in DNA repair and gene expression. CSB belongs to the SWI2/SNF2 ATP-dependent chromatin remodeler family, which is conserved from yeast to human[179]. It was indicated that CSB and CTCF (11-zinc finger protein) can regulate each other's chromatin association, and thus modulating chromatin structure and coordinating gene expression in response to oxidative stress [179]. Mutations in ***ERCC6*** are associated with growth failure, intellectual disability, neurological dysfunction and decline[180]. In addition, the pro-carries of ***ERCC6*** 1230Pro allele showed a decreased risk for laryngeal cancer with stronger association in high alcohol consumers, which suggest that ***ERCC6*** may modulate an individual’s ability to repair the effect of alcohol consumption to the risk for laryngeal cancer[181]. Other potential gene influencing alcohol consumption is ***CHAT*** (choline O-acetyltransferase), which encodes an enzyme which catalyzes the biosynthesis of the neurotransmitter acetylcholine. Our lead SNV rs4253197 is located 136 kb upstream of ***CHAT***. A study suggested that adolescent binge ethanol decreases adult ChAT expression, possibly through neuroimmune mechanisms, which might impact adult cognition, arousal, or reward sensitivity[182]. In addition, ***CHAT*** may be involved in the acetylcholine neuronal activity that modulates brain-derived neurotrophic factor production and inflammation in the brain, and in the development of Alzheimer’s disease[183].

**rs201383951** (***GRK5***, 10q26.11): Fourteen genes reside within ± 500 kb of the lead SNV. The rs201383951 is located 13 kb downstream of ***GRK5***, which encodes a member of the guanine nucleotide-binding protein (G protein)-coupled receptor membrane-associated serine/threonine protein kinase. The protein phosphorylates the activated forms of G protein-coupled receptors (GPCRs), which initiates beta-arrestin-mediated receptor desensitization, internalization, and signaling events leading to their down-regulation[184]. Desensitization of GPCRs regulates the number of polymorphonuclear leukocytes (PMNs), which are critical effector cells of the innate immune system and GPCR desensitization is mediated by GRKs[185]. The chemokine macrophage inflammatory protein-2 (***MIP2***) induces ***GRK2*** and ***GRK5*** expression in PMNs through phosphoinositide-3-kinase (PI3K)-gamma signaling. The lipopolysaccharide (LPS)-activated signaling through the Toll-like receptor 4 (***TLR4***) pathway transcriptionally downregulates the expression of ***GRK2*** and ***GRK5*** in response to ***MIP2***. The reduced expression of GRKs lowers chemokine receptor desensitization and augments the PMN migratory response[185]. In addition, it was demonstrated that ***GRK5*** is localized in the centrosome and regulates microtubule nucleation and normal cell cycle progression[186]. Knockdown of ***Grk5*** expression in HeLa cells induced G2/M arrest or delay, which appeared to be due to increased expression of p53, reduced activity of aurora A kinase and a subsequent delay in the activation of polo-like kinase 1[186]. GRKs 2 and 5 are highly expressed in the heart and known to be upregulated in heart failure (HF)[187]. Variant of ***GRK5*** (rs17098707 A>T; Gln41Leu) has revealed association with differential survival in African Americans HF patients[188, 189], but the role of ***GRK5*** in cardiac pathophysiology remains unclear. ***GRK5*** may also have a role in regulating blood pressure through the smooth muscle cells (SMC), which control vascular tone[187, 190]. A study of SMC-specific ***Grk5*** overexpression in mice demonstrated hypertension in a Gi-dependent manner[191]. However, studies in human have failed to find association of ***GRK5*** with BP variation levels[189, 192]. GWA studies have reported significant association of ***GRK5*** with type 2 diabetes in East Asians[193] and suggestive association with coronary artery aneurysm in Kawasaki disease[194]. Other prominent gene on 10q26.11 for vascular homeostasis is ***BAG3*** (B-cell lymphoma 2-associated athanogene 3). Our lead SNV is located 183 kb upstream of ***BAG3***, which is a member of a conserved family of cyto-protective co-chaperone proteins containing a conserved domain able to interact with heat shock HSC70/HSP70 and sHSPs proteins. Mouse experiment has suggested that ***BAG3*** exerts a vasorelaxing effect through the activation of the PI3K/Akt/eNOS signaling pathway, and may influence blood pressure regulation[195]. A GWA study identified significant association of ***BAG3*** with dilated cardiomyopathy[196], and suggestive association with alcohol dependence[133]. ***BAG3*** was also suggested to contribute to alcohol-induced neurodegeneration[197, 198]. The BAG proteins modulate the switch between autophagy and endoplasmic reticulum (ER)-associated degradation through competing for binding of the adapter proteins, p62 and NBR (neighbor of BRC1). Chronic alcohol intake affects both p62 and ***BAG3***[199-201], which could break the molecular switch and disrupt the balance between the ER and lysosomes for protein degradation[198]. ***BAG3*** is also involved in numerous activities including macro-autophagic protein degradation in aging cells[202]. ***BAG3*** is abundantly expressed in the heart and in striated muscle. Mutations in ***BAG3*** causes severe dominant childhood muscular dystrophy with cardiomyopathy[203]. Our lead SNV is located 258 kb upstream of ***INPP5F***, which encodes an inositol 1,4,5-trisphosphate (InsP3) 5-phosphatase and contains a Sac domain. The activity of this protein is specific for phosphatidylinositol 4,5-bisphosphate and phosphatidylinositol 3,4,5-trisphosphate. A GWA study observed association between ***INPP5F*** (inositol polyphosphate-5-phosphatase F) with Parkinson's disease[155]. Our lead SNV is located 424 kb upstream of ***SEC23IP*** (SEC23 interacting protein), which encodes a member of the phosphatidic acid preferring-phospholipase A1 family that degrades phospholipids and is involved in membrane trafficking[204]. ***SEC23IP*** protein has a role in the organization of ER exit sites and the Golgi apparatus, and in ER Golgi transport[205]. A GWA study found association of ***SEC23IP*** with menarche age at onset[206]. ***SEC23IP*** has been reported to associate with adult attention deficit hyperactivity disorder in exome chip analyses[207] and with neurodevelopmental disorders using exome sequencing in consanguineous families[208].

**rs11200509** (***TACC2***, 10q26.13): Ten genes reside within ± 500 kb of the lead SNV. The rs11200509 is located 2.4 kb downstream of ***TACC2*** (transforming acidic coiled-coil containing protein 2), which encodes a protein that concentrates at centrosomes throughout the cell cycle. TCCA are centrosomes/microtubules interaction-associated proteins containing a highly conserved C-terminal coiled-coil “TACC domain”. ***TACC2*** may promote androgen-mediated growth in the prostate cancer[209] and be involved in the cell proliferation of breast carcinoma[210]. Our lead SNV rs11200509 is located 198 kb and 205 Kb upstream of ***ARMS2*** and ***HTRA1***, respectively. GWA studies have identified several SNVs of ***ARMS2*** and ***HTRA1*** genes associated with age-related macular degeneration[211-218]. Suggestive associations within 1 Mb of the lead SNV were also detected with height[219], type 2 diabetes[220], smoking cessation[221, 222], schizophrenia[60], bipolar disorder[223], late-onset Alzheimer’s disease[224], and breast cancer[225].

**rs10741534** (***GALNT18***, 11p15.4): Seven genes reside within ± 500 kb of the lead SNV. The rs10741534 is 37.5 kb downstream of ***GALNT18*** (polypeptide N-acetylgalactosaminyltransferase 18). ***GALNT18*** protein catalyzes the initial reaction in O-linked oligosaccharide biosynthesis, the transfer of an N-acetyl-D-galactosamine residue to a serine or threonine residue on the protein receptor. GWA studies have suggested associations between variants near or within ***GALNT18*** withtype 2 diabetes in the African-American population , post bronchodilator FEV1 [25], psychotic symptoms in prion disease[226], Alzheimer’s disease[22], diisocyanate-induced asthma[145], and response to tocilizumab for the treatment of rheumatoid arthritis[227].

**rs139077481** (***ELMOD1***, 11q22.3): Eight genes reside within ± 500 kb of the lead SNV. The rs139077481 is located within an intron of ***ELMOD1*** (cell engulfment and motility domain containing 1). Its protein acts as a GTPase-activating protein (GAP) toward guanine nucleotide exchange factors like ARL2, ARL3, ARF1 and ARF6, but not for GTPases outside the Arf family[228]. The non-opioid sigma-1 receptor (S1R) is an effector of GAP activity of ELMOD1–3 proteins as its direct binding to either ***ELMOD1*** or ***ELMOD2***[228]. GWA studies have described suggestive associations (6.0 x 10-8 ≤ *P* ≤ 9.0 x 10-6) within the 1 Mb region of rs139077481 for large artery stroke[40], parental extreme longevity (95 years and older)[229], autism spectrum disorder, attention deficit-hyperactivity disorder, bipolar disorder, major depressive disorder, and schizophrenia (combined)[230], optimism[231], and IgG glycosylation[32].

**rs186331780** (***LOC105369793*** -***FAM19A2***, 12q14.1): One gene reside within ± 500 kb of the lead SNV. The rs186331780 is located 391 kb downstream of ***FAM19A2*** (family with sequence similarity 19 member A2, C-C motif chemokine like). ***FAM19A2*** is a member of the TAFA family of five homologous genes that encode small secreted proteins. TAFA proteins appear distantly related to MIP-1alpha, a member of the CC-chemokine family. TAFA mRNAs are highly expressed in specific brain regions, with little expression in colon, heart, lung, spleen, kidney, and thymus[232]. The biological functions of TAFA family members continues unclear; however, there are some indications that TAFAs may modulate immune responses in the CNS, may represent a novel class of neurokines that acts as regulators of immune nervous cells[233], and may control axonal sprouting following brain injury. A GWA study has identified a significant association of ***FAM19A2*** for modified stumvoll insulin sensitivity index[234]. Other GWA studies have found suggestive associations of ***FAM19A2*** with IgG glycosylation[32], schizoaffective disorder[235], oppositional defiant disorder dimensions in attention-deficit hyperactivity disorder[236], hippocampal volume[237], post bronchodilator FEV1/FVC ratio[25], chronic obstructive pulmonary disease[238], pulmonary function decline[239], and asthma and hay fever[111]. In addition, a genome-wide admixture analysis showed a suggestive association between ***FAM19A2*** (rs348644) with FEV1/FVC among African Americans in the COPDGene Study[240].

**rs187888844** (***LOC105370250*** -***PCDH9***, 13q21.32): One gene resides within ± 500 kb of the lead SNV. The rs187888844 is located 476 kb upstream of ***PCDH9*** (protocadherin 9). ***PCDH9*** protein belongs to a calcium-dependent cell–cell adhesion molecule of the cadherin superfamily. ***PCDH9*** predominantly is expressed in the brain but also in other tissues, and the expression patterns appear to be developmentally regulated[241]. A study of tissue microarrays and immunohistochemistry suggested that ***PCDH9*** might function as a tumor suppressor during cancer development and progression. The ***PCDH9*** expression was decreased in human cerebral glial tumors, and the loss correlated significantly with higher histological grade[242]. GWA studies have found suggestive associations of variants within or near ***PCDH9*** with obesity[243], post bronchodilator FEV1/FVC ratio[25], pulmonary function decline[239], 3-hydroxy-1-methylpropylmercapturic acid levels in smokers[109], symmetrical dimethylarginine levels[244], response to platinum-based chemotherapy[245], sense of smell[246], and schizophrenia[60].

**rs116464496** (***LINC00343***, 13q33.2): Two genes reside within ± 500 kb of the lead SNV. The rs116464496 is located 228 kb upstream of ***LINC00343*** (long intergenic non-protein coding RNA 343) and 444 kb downstream of ***DAOA*** (D-amino acid oxidase activator). ***DAOA*** protein activates d-amino acid oxidase in the brain, which oxidizes d-serine, an important co-agonist for the N-methyl-d-Aspartate receptor[247]. Studies have shown evidence that DAOA may be involved in the pathophysiology of psychotic disorders[248]. GWA studies have described suggestive associations of variants within or near ***DAOA*** with Alzheimer’s disease and age of onset[22], bipolar disorder and schizophrenia[249], left superior temporal gyrus thickness (schizophrenia interaction)[250], subcutaneous adipose tissue[251], visceral fat[45], obesity-related traits[252], smoking initiation[253], adverse response to chemotherapy (neutropenia/leucopenia)[254], and immune response to smallpox (secreted IL-2)[255].

**rs7185735 (*FTO***, 16q12.2): Six genes reside within ± 500 kb of the lead SNV. The rs7185735 is located within an intron in ***FTO*** (alpha-ketoglutarate dependent dioxygenase). The fat-mass and obesity-associated gene, ***FTO***, demethylates nuclear RNA and newly synthesized mRNAs[256]. An epitranscriptomic function study demonstrated that ***FTO*** preferentially demethylates *N*6,2′-*O*-dimethyladenosine (m6Am) rather than *N*6-methyladenosine (m6A) and reduces the stability of m6Am mRNAs. Demethylation of cytoplasmic m6Am mRNAs may be induced by stimuli that induce cytosolic translocation of ***FTO***[256]. ***FTO*** has been implicated with enhanced food intake and reduced satiety, and in the regulation of the global metabolic rate, energy expenditure and energy homeostasis, which contribute to the regulation of body size and body fat accumulation[257-262]. ***Fto*** mouse deficiency induces browning of white adipose tissue including enhanced uncoupling protein 1 (***Ucp-1***) expression and mitochondrial uncoupling in adipocytes[258]. The regulatory importance of the ***FTO*** locus in the early adipocyte differentiation with a causal role of a risk-conferring ***FTO*** on adiposity was proposed in a study using epigenomic data, observations from patients and mice, and CRISPR–Cas9 genome editing[261]. This study found that the ***FTO***-intronic rs1421085 disrupts a conserved motif for the ***ARID5B*** repressor leading to derepression of a potent preadipocyte enhancer and a doubling of ***IRX3*** and ***IRX5*** expression during early adipocyte differentiation[261, 262]. These findings suggest a pathway for adipocyte thermogenesis regulation involving ***FTO*** and these other genes in the developmental swing between energy-dissipating beige (brite) fat cells and energy-accumulating white adipocytes, which reduces the thermogenesis and subsequently increases lipid deposition. Variants of ***FTO*** have been associated in diverse ancestries with obesity-related traits factors[260]. T2D susceptibility after adjustment for BMI[257, 263]. long-term incidence of cardiovascular disease and related death independent of traditional risk factors[259], depression and mood[264, 265], Alzheimer’s disease[266], alcohol consumption and alcohol dependency[27, 267, 268]. In addition, frequency of alcohol consumption was suggested to modify the effect of FTO variants on BMI[269]. Inconsistent associations have been reported for ***FTO*** with BP traits. Significant association for ***FTO*** with higher systolic and diastolic blood pressures was found after considering adiposity as a covariate in Mexican children[270], but not in Chinese children[271]. GWA studies have identified SNVs near or within ***FTO*** to be associated with BMI[151, 272-282], weight[282], obesity[282-284], body fat percentage[285], waist circumference[286], adiposity[287], circulating leptin levels[288], type 2 diabetes[31, 220, 289-291], dietary macronutrient intake[292], menarche age at onset[206, 293], triglycerides and HDL-cholesterol[66], vitiligo (non-segmental)[294], breast cancer[295, 296], and melanoma[297].

**rs140520944** (***LOC105372045***-***MIR302F***, 18q12.1): No genes reside within ± 500 kb of the lead SNV. The rs140520944 is located 790 kb upstream of ***MIR302F*** (microRNA 302f). microRNAs (miRNAs) are short (20-24 nt) non-coding RNAs that participate in the gene regulatory networks of several multicellular organisms by regulating a variety of biological processes, such as, cell growth, differentiation, apoptosis and metastasis[298-300]. Evidence suggests that miRNAs are transcribed by RNA polymerase II, the transcripts are capped and polyadenylated, which can be either protein-coding or non-coding[301]. miRNAs can cause mRNA degradation or inhibit protein translation through binding to complementary sequences in the 3′-untranslated region (3′-UTR) of target genes[300, 301]. Reports have suggested that dysregulation of miRNA expression may play a role in a series of human cancers, such as breast cancer, colon cancer, osteosarcoma, lung cancer, melanoma and hepatocellular carcinoma[299, 301, 302]. GWA studies have found suggestive associations between intergenic variants within 1Mb of our lead SNV rs140520944 with LDL peak particle diameter- total fat intake interaction[303], post bronchodilator FEV1[25], cognitive performance[116], and bipolar disorder[304].

**rs142673685** (***LOC105372361***-***THEG5***, 19q12): Five genes reside within ± 500 kb of the lead SNV. The rs142673685 is located 76 kb downstream of ***THEG5*** (testis highly expressed protein 5), and 321 upstream of ***TSHZ3*** (teashirt zinc finger homeobox 3), which encodes a zinc-finger transcription factor involved in developmental processes. ***TSHZ3*** in conjunction with ***APBB1*** (amyloid beta precursor protein binding family B member 1), ***SET*** (SET nuclear oncogene) and histone deacetylase (HDAC1 and DHAC2) factors act as transcriptional repressors, which inhibit the expression of ***CASP4*** (Caspase 4)[305]. ***TSHZ3***-mediated transcription repression involves the recruitment of histone deacetylases HDAC1 and HDAC2. FE65 (the ***APBB1*** protein), simultaneously recruits SET (a component of the inhibitor of acetyl transferase), and that in turn recruits histone deacetylases to produce a powerful gene-silencing complex[306]. ***TSHZ3*** regulates the development of neurons involved in respiratory rhythm and airflow control[306], and is expressed in smooth muscle cell precursors that form the wall of the forming mammalian ureter[307]. GWA studies have described suggestive associations between variants within 1Mb of our lead SNV rs140520944 with post bronchodilator FEV1 in COPD[32], cognitive performance[6], narcolepsy with cataplexy[308], and estradiol levels[309].

Some BP-known genes from European ancestry cohorts[310, 311] extended as significant to other ancestry groups, e.g. the *LSP1-TNNT3* in Asians. The novel findings have been also associated with other traits, from different GWAS.

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