Electronic Supplementary Material (ESI) for Metallomics.
This journal is © The Royal Society of Chemistry 2018

	Uniprot Id	Entry name	Gene names	Protein names	Predicted Pattern	Types of heme cofactors	Heme role	EC number	Subcellular location	Membrane associated	Involvement in disease	Gene ontology (biological process)
1	A0A024RAI7	A0A024RAI7_HUMAN	FLJ16008 hCG_42613	FLJ16008 protein, isoform CRA_a	Unknown	heme b	Catalytic		Unknown	No		
2	Q9NP58	ABCB6_HUMAN	ABCB6 MTABC3 PRP UMAT	ATP-binding cassette sub-family B member 6, mitochondrial (Mitochondrial ABC transporter 3) (Mt-ABC transporter 3) (P-glycoprotein-related protein) (Ubiquitously-expressed mammalian ABC half transporter)	Unknown	heme b	Substrate - transport		Endoplasmic reticulum, Golgi apparatus, Mitochondrion, Cell membrane, Endosome	Yes	DISEASE: Microphthalmia, isolated, with coloboma, 7 (MCOPCB7) [MIM:614497]: A disorder of eye formation, ranging from small size of a single eye to complete bilateral absence of ocular tissues. Ocular abnormalities like opacities of the cornea and lens, scaring of the retina and choroid, and other abnormalities may also be present. Ocular colobomas are a set of malformations resulting from abnormal morphogenesis of the optic cup and stalk, and the fusion of the fetal fissure (optic fissure). {ECO:0000269|PubMed:22226084}. Note=The disease is caused by mutations affecting the gene represented in this entry.; DISEASE: Dyschromatosis universalis hereditaria 3 (DUH3) [MIM:615402]: An autosomal dominant pigmentary genodermatosis characterized by a mixture of hyperpigmented and hypopigmented macules distributed randomly over the body, that appear in infancy or early childhood. The trunk and extremities are the dominant sites of abnormal pigmentation. Facial lesions can be seen in 50% of affected individuals, but involvement of palms and soles is unusual. Abnormalities of hair and nails have also been reported. Dyschromatosis universalis hereditaria may be associated with abnormalities of dermal connective tissue, nerve tissue, or other systemic complications. DISEASE: Pseudohyperkalemia, familial, 2, due to red cell leak (PSHK2) [MIM:609153]: A dominantly inherited condition characterized by increased serum potassium levels, measured in whole-blood specimens stored at or below room temperature. This condition is not accompanied by clinical symptoms or biological signs except for borderline abnormalities of red cell shape.	brain development [GO:0007420]; cellular iron ion homeostasis [GO:0006879]; heme transport [GO:0015886]; porphyrin-containing compound biosynthetic process [GO:0006779]; skin development [GO:0043588]; transmembrane transport [GO:0055085]; transport [GO:0006810]
3	O75027	ABCB7_HUMAN	ABCB7 ABC7	ATP-binding cassette sub-family B member 7, mitochondrial (ATP-binding cassette transporter 7) (ABC transporter 7 protein)	Unknown	heme b	Substrate - transport		Mitochondrion	Yes	DISEASE: Anemia, sideroblastic, spinocerebellar ataxia (ASAT) [MIM:301310]: A X-linked recessive disorder characterized by an infantile to early childhood onset of non-progressive cerebellar ataxia and mild anemia, with hypochromia and microcytosis. Note=The disease is caused by mutations affecting the gene represented in this entry.	cellular iron ion homeostasis [GO:0006879]; transmembrane transport [GO:0055085]; transport [GO:0006810]
4	Q9UNQ0	ABCG2_HUMAN	ABCG2 ABCP BCRP BCRP1 MXR	ATP-binding cassette sub-family G member 2 (Breast cancer resistance protein) (CDw338) (Mitoxantrone resistance-associated protein) (Placenta-specific ATP-binding cassette transporter) (Urate exporter) (CD antigen CD338)	Unknown	heme b	Substrate - transport		Mitochondrion, Cell membrane	Yes		cellular iron ion homeostasis [GO:0006879]; cholesterol efflux [GO:0033344]; response to drug [GO:0042493]; transport [GO:0006810]; urate metabolic process [GO:0046415]
5	Q8N7X0	ADGB_HUMAN	ADGB C6orf103 CAPN7L	Androglobin (Calpain-7-like protein)	Unknown	heme b	Oxygen storage/transport		Unknown	No		proteolysis [GO:0006508]
6	P43652	AFAM_HUMAN	AFM ALB2 ALBA	Afamin (Alpha-albumin) (Alpha-Alb)	Y377	heme b	Substrate - transport		Extracellular space	No		vitamin transport [GO:0051180]
7	P02768	ALBU_HUMAN	ALB GIG20 GIG42 PRO0903 PRO1708 PRO2044 PRO2619 PRO2675 UNQ696/PRO1341	Serum albumin	Y185	heme b	Substrate - transport		Extracellular space	No	DISEASE: Hyperthyroxinemia, familial dysalbuminemic (FDAH) [MIM:615999]: A disorder characterized by abnormally elevated levels of total serum thyroxine (T4) in euthyroid patients. It is due to abnormal serum albumin that binds T4 with enhanced affinity. Note=The disease is caused by mutations affecting the gene represented in this entry.; DISEASE: Analbuminemia (ANALBA) [MIM:616000]: A rare autosomal recessive disorder manifested by the presence of a very low amount of circulating serum albumin. Affected individuals manifest mild edema, hypotension, fatigue, and, occasionally, lower body lipodystrophy (mainly in adult females). The most common biochemical finding is hyperlipidemia, with a significant increase in the total and LDL cholesterol concentrations, but normal concentrations of HDL cholesterol and triglycerides. {ECO:0000269|PubMed:8134387}. Note=The disease is caused by mutations affecting the gene represented in this entry.	bile acid and bile salt transport [GO:0015721]; cellular protein metabolic process [GO:0044267]; cellular response to starvation [GO:0009267]; hemolysis by symbiont of host erythrocytes [GO:0019836]; high-density lipoprotein particle remodeling [GO:0034375]; maintenance of mitochondrion location [GO:0051659]; negative regulation of apoptotic process [GO:0043066]; negative regulation of programmed cell death [GO:0043069]; platelet degranulation [GO:0002576]; post-translational protein modification [GO:0043687]; receptor-mediated endocytosis [GO:0006898]; retina homeostasis [GO:0001895]; sodium-independent organic anion transport [GO:0043252]; transport [GO:0006810]
8	P02760	AMBP_HUMAN	AMBP HCP ITIL	Protein AMBP [Cleaved into: Alpha-1-microglobulin (Protein HC) (Alpha-1 microglycoprotein) (Complex-forming glycoprotein heterogeneous in charge); Inter-alpha-trypsin inhibitor light chain (ITI-LC) (Bikunin) (EDC1) (HI-30) (Uronic-acid-rich protein); Trypstatin]	Unknown	heme b	Substrate - degradation		Extracellular space	No		cell adhesion [GO:0007155]; female pregnancy [GO:0007565]; heme catabolic process [GO:0042167]; negative regulation of immune response [GO:0050777]; negative regulation of JNK cascade [GO:0046329]; protein catabolic process [GO:0030163]; protein-chromophore linkage [GO:0018298]; receptor-mediated endocytosis [GO:0006898]; viral process [GO:0016032]
9	O14867	BACH1_HUMAN	BACH1	Transcription regulator protein BACH1 (BTB and CNC homolog 1) (HA2303)	Unknown	heme b	Substrate - sensor		Nucleus	No		DNA repair [GO:0006281]; negative regulation of transcription from RNA polymerase II promoter [GO:0000122]; protein ubiquitination [GO:0016567]; regulation of transcription, DNA-templated [GO:0006355]; regulation of transcription from RNA polymerase II promoter in response to hypoxia [GO:0061418]; regulation of transcription involved in G1/S transition of mitotic cell cycle [GO:0000083]; regulation of transcription involved in G2/M transition of mitotic cell cycle [GO:0000117]

10	P15538	C11B1_HUMAN	CYP11B1 S11BH	Cytochrome P450 11B1, mitochondrial (CYPXIB1) (Cytochrome P-450c11) (Cytochrome P450C11) (Steroid 11-beta-hydroxylase) (EC 1.14.15.4)	C450	heme b	Catalytic	1.14.15.4	Mitochondrion	Yes	DISEASE: Adrenal hyperplasia 4 (AH4) [MIM:202010]: A form of congenital adrenal hyperplasia, a common recessive disease due to defective synthesis of cortisol. Congenital adrenal hyperplasia is characterized by androgen excess leading to ambiguous genitalia in affected females, rapid somatic growth during childhood in both sexes with premature closure of the epiphyses and short adult stature. Four clinical types: 'salt wasting' (SW, the most severe type), 'simple virilizing' (SV, less severely affected patients), with normal aldosterone biosynthesis, 'non-classic form' or late-onset (NC or LOAH) and 'cryptic' (asymptomatic). Note=The disease is caused by mutations affecting the gene represented in this entry.; DISEASE: Hyperaldosteronism, familial, 1 (HALD1) [MIM:103900]: A disorder characterized by hypertension, variable hyperaldosteronism, and abnormal adrenal steroid production, including 18-oxocortisol and 18-hydroxycortisol. There is significant phenotypic heterogeneity, and some individuals never develop hypertension. Note=The disease is caused by mutations affecting the gene represented in this entry. The molecular defect causing hyperaldosteronism familial 1 is an anti-Lepore-type fusion of the CYP11B1 and CYP11B2 genes. The hybrid gene has the promoting part of CYP11B1, ACTH-sensitive, and the coding part of CYP11B2.	aldosterone biosynthetic process [GO:0032342]; C21-steroid hormone biosynthetic process [GO:0006700]; cellular response to hormone stimulus [GO:0032870]; cellular response to potassium ion [GO:0035865]; cortisol biosynthetic process [GO:0034651]; glucocorticoid biosynthetic process [GO:0006704]; glucose homeostasis [GO:0042593]; immune response [GO:0006955]; regulation of blood pressure [GO:0008217]; sterol metabolic process [GO:0016125]
11	P19099	C11B2_HUMAN	CYP11B2	Cytochrome P450 11B2, mitochondrial (Aldosterone synthase) (ALDOS) (EC 1.14.15.4) (EC 1.14.15.5) (Aldosterone-synthesizing enzyme) (CYPXIB2) (Cytochrome P-450Aldo) (Cytochrome P-450C18) (Steroid 18-hydroxylase)	C450	heme b	Catalytic	1.14.15.4; 1.14.15.5	Mitochondrion	Yes	DISEASE: Corticosterone methyloxidase 1 deficiency (CMO-1 deficiency) [MIM:203400]: Autosomal recessive disorder of aldosterone biosynthesis. There are two biochemically different forms of selective aldosterone deficiency be termed corticosterone methyloxidase (CMO) deficiency type 1 and type 2. In CMO-1 deficiency, aldosterone is undetectable in plasma, while its immediate precursor, 18-hydroxycorticosterone, is low or normal. Note=The disease is caused by mutations affecting the gene represented in this entry.; DISEASE: Corticosterone methyloxidase 2 deficiency (CMO-2 deficiency) [MIM:610600]: Autosomal recessive disorder of aldosterone biosynthesis. In CMO-2 deficiency, aldosterone can be low or normal, but at the expense of increased secretion of 18-hydroxycorticosterone. Consequently, patients have a greatly increased ratio of 18-hydroxycorticosterone to aldosterone and a low ratio of corticosterone to 18-hydroxycorticosterone in serum. Note=The disease is caused by mutations affecting the gene represented in this entry.; DISEASE: Hyperaldosteronism, familial, 1 (HALD1) [MIM:103900]: A disorder characterized by hypertension, variable hyperaldosteronism, and abnormal adrenal steroid production, including 18-oxocortisol and 18-hydroxycortisol. There is significant phenotypic heterogeneity, and some individuals never develop hypertension. Note=The disease is caused by mutations affecting the gene represented in this entry. The molecular defect causing hyperaldosteronism familial 1 is an anti-Lepore-type fusion of the CYP11B1 and CYP11B2 genes. The hybrid gene has the promoting part of CYP11B1, ACTH-sensitive, and the coding part of CYP11B2.	aldosterone biosynthetic process [GO:0032342]; C21-steroid hormone biosynthetic process [GO:0006700]; cellular response to hormone stimulus [GO:0032870]; cellular response to potassium ion [GO:0035865]; cortisol biosynthetic process [GO:0034651]; mineralocorticoid biosynthetic process [GO:0006705]; potassium ion homeostasis [GO:0055075]; regulation of blood volume by renal aldosterone [GO:0002017]; renal water homeostasis [GO:0003091]; sodium ion homeostasis [GO:0055078]; sterol metabolic process [GO:0016125]
12	Q86VB7	C163A_HUMAN	CD163 M130	Scavenger receptor cysteine-rich type 1 protein M130 (Hemoglobin scavenger receptor) (CD antigen CD163) [Cleaved into: Soluble CD163 (sCD163)]	Unknown	heme b	Substrate - degradation		Extracellular space	No		acute-phase response [GO:0006953]; receptor-mediated endocytosis [GO:0006898]
13	Q4G0S4	C27C1_HUMAN	CYP27C1	Cytochrome P450 27C1 (EC 1.14.19.-) (All-trans retinol 3,4-desaturase)	C318	heme b	Catalytic	1.14.19.-	Unknown	Yes		retinal metabolic process [GO:0042574]; retinoic acid metabolic process [GO:0042573]; retinol metabolic process [GO:0042572]
14	Q6ZSU1	C2G1P_HUMAN	CYP2G1P CYP2GP1	Putative inactive cytochrome P450 2G1 (Cytochrome P450 2G1 pseudogene)	C91	heme b	Catalytic		Unknown	No		epoxygenase P450 pathway [GO:0019373]
15	Q99643	C560_HUMAN	SDHC CYB560 SDH3	Succinate dehydrogenase cytochrome b560 subunit, mitochondrial (Integral membrane protein CII-3) (QPs-1) (QPs1) (Succinate dehydrogenase complex subunit C) (Succinate-ubiquinone oxidoreductase cytochrome B large subunit) (CYBL)	H127	heme b	Electron transfer		Mitochondrion	Yes	DISEASE: Paragangliomas 3 (PGL3) [MIM:605373]: A neural crest tumor usually derived from the chromoreceptor tissue of a paraganglion. Paragangliomas can develop at various body sites, including the head, neck, thorax and abdomen. Most commonly, they are located in the head and neck region, specifically at the carotid bifurcation, the jugular foramen, the vagal nerve, and in the middle ear. {ECO:0000269|PubMed:11062460}. Note=The disease is caused by mutations affecting the gene represented in this entry.; DISEASE: Paraganglioma and gastric stromal sarcoma (PGGSS) [MIM:606864]: Gastrointestinal stromal tumors may be sporadic or inherited in an autosomal dominant manner, alone or as a component of a syndrome associated with other tumors, such as in the context of neurofibromatosis type 1 (NF1). Patients have both gastrointestinal stromal tumors and paragangliomas. Susceptibility to the tumors was inherited in an apparently autosomal dominant manner, with incomplete penetrance. {ECO:0000269|PubMed:17804857}. Note=The disease is caused by mutations affecting the gene represented in this entry.	aerobic respiration [GO:0009060]; mitochondrial electron transport, succinate to ubiquinone [GO:0006121]; oxidation-reduction process [GO:0055114]; tricarboxylic acid cycle [GO:0006099]
16	Q8N8Q1	C56D1_HUMAN	CYB561D1	Cytochrome b561 domain-containing protein 1	H55-H127; H93-H166	heme b	Electron transfer		Unknown	Yes		oxidation-reduction process [GO:0055114]
17	O14569	C56D2_HUMAN	CYB561D2 1O1F6 LUCA12.2	Cytochrome b561 domain-containing protein 2 (Putative tumor suppressor protein 101F6)	H48-H120;H86-H159	heme b	Electron transfer		Unknown	Yes		oxidation-reduction process [GO:0055114]