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**SNPs ARRAY KARYOTYPING REVEALS
RECURRENT LESIONS IN PRIMARY
MYELOFIBROSIS**

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SUMMARY

The molecular pathogenesis of primary myelofibrosis (PMF) is still largely unknown. Recently, single-nucleotide polymorphism arrays (SNP-A) were shown to allow for genome-wide profiling of copy-number alterations and copy-neutral runs of homozygosity (aUPD) at high resolution.

In this study we performed SNP-A analysis of primary myelofibrosis, aiming to identify novel recurrent genomic abnormalities,

We analyzed DNA of 20 PMF patients using the Affymetrix Genome-Wide Human SNP Array 6.0 and conventional bioinformatic tools. Validation was carried on by TaqMan Copy Number Assay and Immunohistochemistry. We observed a highly complex karyotype in all cases, detecting all the previously reported alterations (including del 20q, del13q, aUPD of 9p24, aUPD on chromosome 11 and abnormalities on chromosome 1). In addition, we identified several novel cryptic lesions.

Remarkable, in overall 95% of cases, we found the 20p13 cytoband affected by lesions. Among them we defined a minimal affected region (MAR), a recurrent lesion occurring in 55% of patients. In particular, the MAR is a genomic amplification extending for 9,911 bps and overlapping the *SIPB1* gene locus.

These results were confirmed by real-time PCR and validated *in silico* in an independent series of myeloproliferative diseases.

Furthermore, by immunohistochemistry assay, we assessed a consistent deregulation of the SIRPB1 protein which was high up-regulated in PMF patients carrying the amplification.

In conclusion, we found a novel highly recurrent genomic lesion in PMF patients responsible of the aberrant proliferation of the encoded protein.

1. INTRODUCTION

1.1 Myeloproliferative Neoplasm

The concept of myeloproliferative disease was first proposed in 1951 by the hematologist William Dameshek, who introduced the term "myeloproliferative disorders (MPD)" to encompass polycythemia vera (PV), essential thrombocythemia (ET), primary myelofibrosis (PMF), chronic myelogenous leukemia (CML), and erythroleukemia. His suggestion was based on the clinical phenotype overlapping of these disorders and on the hypothesis that a generalized proliferation of bone marrow cells, due to some unknown "myelostimulatory factor", was the underlying cause [Dameshek 1951].

In the 2001, the World Health Organization committee for the classification of hematologic malignancies undertook the first attempt to classify MPD and MPD-like clinicopathologic entities; CML, PV, ET, and PMF were included under the category of "chronic myeloproliferative diseases" (CMPD) including also other "nonclassic" MPD-like disorders such as chronic neutrophilic leukemia (CNL), chronic eosinophilic leukemia/hypereosinophilic syndrome (CEL/HES), and "unclassified CMPD" (the erythroleukemia was instead recognized as a variant of acute myeloid leukaemia) [1].

However, seven years later, in the 2008, the WHO decided to rename all this group of diseases from "myeloproliferative diseases" to "myeloproliferative neoplasms" stressing the accent on the clonal genetic changes intended as a salient feature of this group of disease. [1]

The MPNs are in fact clonal hematopoietic stem cell disorders characterized by abnormal, clonal excess proliferation with complete maturation of one or more of the myeloid lineages (i.e granulocytic, erythroid, megacaryocytic and mast cell).

MPNs are rare, with an aggressive incidence of approximately 6-10 cases per 100,000 people annually [2, 3]. They are primarily neoplasms of adults that peak in frequency in the 5th to 7th decade, but some subtypes, particularly CML and ET are reported in children as well.

Initially MPNs are characterized by hypercellularity of the bone marrow with effective haematopoietic maturation and increased number of granulocytes,

red blood cells and or platelets in the peripheral blood. Further, splenomegaly and hepatomegaly are common and caused by sequestration of excess blood cells proliferation and abnormal haematopoietic cells. Despite an often insidious onset, each MPN entity has the potential to undergo to bone marrow failure due to myelofibrosis, ineffective hematopoiesis, transformation to an overt blast phase or any combination of these events [1].

1.2 Myeloproliferative Neoplasms Ph-negative

The four "classic" MPNs (ie, CML, PV, ET, and PMF) should be distinguished from the other "nonclassic" MPNs (CNL, CELNOS, SM, MPN U) by the presence of t(9;22)(q34;q11) [4].

The result of this alteration is the fusion gene *BCR-ABL* created by juxtapositioning the *ABL* gene on chromosome 9 (region q34) to a part of the *BCR* ("breakpoint cluster region") gene on chromosome 22 (region q11). This is a reciprocal translocation, with an elongated chromosome 9 and a truncated chromosome 22: the Philadelphia chromosome [5].

In the 1960 the association of the Philadelphia (Ph1)-chromosome with CML [6] allowed to distinguish the other three "classic" disorders as Ph1-negative MPD [7].

Nevertheless, until recently, no specific genetic alterations inside the *BCR-ABL*-negative classic MPNs were identified and the distinction between clonal from reactive myeloproliferation and between one type of MPN from another one was allowed by the integration of a variety of features like the bone marrow histology and the few clinical and laboratory findings at disposal [8].

Fortunately, in the last 4 years have been done many fundamental advances in understanding the molecular pathogenesis of classic *BCR-ABL*-negative MPNs. In particular was assessed the central role of altered tyrosine kynase activity with the discovery of specific molecular abnormalities associated with PV, ET, and PMF [9].

1.3 Primary Myelofibrosis

1.3.1 Definition

Primary myelofibrosis is a new name, updating previous labels such as *chronic idiopathic myelofibrosis*, *agnogenic myeloid metaplasia*, and *myelofibrosis with myeloid metaplasia*, among others. It is a clonal myeloproliferative disorder that is characterised by megakaryocytic and granulocytic proliferation with intact maturation, progressive bone marrow fibrosis, splenomegaly and extramedullary hematopoiesis. Nevertheless the etiology of PMF is unknown, nearly 25% of PMF patients are actually with post-essential thrombocythemia myelofibrosis and post-polycythemia vera myelofibrosis [10].

Primary myelofibrosis is the rarest of the classic myeloproliferative neoplasms, with an incidence rate of approximately 0,5-1,5 cases per 100,000 persons per year. PMF patients tend to be older than those with essential thrombocytosis and polycythemia vera because the median age at diagnosis is often in the seventh decade without sex predominance [10].

PMF is associated with poorer prognosis compared with other classic BCR/ABL-negative chronic myeloproliferative neoplasms, the clinical course of the disease is extremely variable, with a median survival from diagnosis less than five years.

Currently, drug therapy has not altered the natural history of the disease [11]. At present, allogeneic stem cell transplantation (ASCT) is the only means of altering the natural history of patients with PMF and provides the only hope for cure even if the majority of affected patients are not suitable candidates for the particular treatment modality [12]

To this regard, the greater understanding of the cellular and molecular events that lead to the development of PMF, could open the possibility for more targeted and effective therapies.

1.3.2 Morphology

Primary myelofibrosis (PMF) is a clonal MPN characterized by a proliferation of predominantly megakaryocytes and granulocytes in the bone marrow that in fully developed disease is associated with reactive deposition of fibrous tissue and with extramedullary haematopoiesis (EMH). PMF is classically subdivided in pre-fibrotic or fibrotic phase.

The pre-fibrotic phase is characterized by hypercellular BM with an increased number of neutrophils and atypical megakaryocytes while the marrow fibrosis is yet minimal or absent. The recognition key of this stage is the morphology and histotopography of megakaryocytes which are marked abnormal and often organized in clusters adjacent to BM vascular sinuses and the bone trabeculae.

Instead, in fibrotic phase the clear-cut reticulin and or collagen fibrosis become evident often with osteosclerosis. The megakaryocytes are atypical and often occur in large clusters or sheets within vascular sinuses. Furthermore, the increased number of CD34⁺ with cluster formation indicate an accelerated phase of disease.

The WHO in the 2008 has recognized a group of major and minor criteria for a diagnosis of primary myelofibrosis.

The major criteria encompass the presence of atypical megakaryocytes in the setting of collagen or reticulin fibrosis, the exclusion of other myeloid neoplasms such as chronic myelogenous leukemia or polycythemia vera, the detection of a clonal marker such as *JAK2V617F*, and the exclusion of secondary causes of marrow fibrosis.

In addition, the minor criteria for primary myelofibrosis require high level of lactate dehydrogenase, leukoerythroblastosis, anemia, and splenomegaly [1, 10].

1.3.3 Molecular Biology

Beginning in early 2005, a number of novel mutations involving Janus kinase 2 (*JAK2*), Myeloproliferative Leukemia Virus (*MPL*), *TET* oncogene family member 2 (*TET2*), Additional Sex Combs-Like 1 (*ASXL1*), Casitas B-lineage lymphoma proto-oncogene (*CBL*), Isocitrate dehydrogenase (*IDH*) and *IKAROS* family zinc finger 1 (*IKZF1*) have been described in BCR-ABL-negative MPNs [13]. In PMF these alterations are present with a frequency ranging from 0% to 65%. However, none of these mutations were PMF specific, being recorded in different MPNs. In fact, they were not found to be mutually exclusive nor could be traced back to a common ancestral clone, at the contrary they highlight a certain degree of heterogeneity in MPNs genetics and postulate the existence of multiple abnormal clones.

Of note, sixty five percent of patients with primary myelofibrosis present an acquired recurrent mutation located in exon 14 of *JAK2* (*JAK2V617F*) [14-17] while an additional 8% to 10% harbors a mutation at codon 515 in *MPL*, ie, *W515L/K/A* [18-20]. These mutations represent a major criterion for diagnosis according to the 2008 World Health Organization classification of myeloid neoplasms [21]

Discovery of *JAK2* mutation has revolutioned the diagnostic approach to MPNs Ph-negative.

However these mutations has been reported not only in MPN but in myelodysplastic/myeloproliferative neoplasm (MDS/MPN), myelodysplastic syndrome (MDS) and acute myeloid leukaemia (AML).

JAK2 gene is located on chromosome 9p24, includes 25 exons and its protein is 1132 amino acids. *JAK2* protein is one of the four Janus family non-receptor protein tyrosine kinases, responsible of cytokine signalling and signal transduction trough their association to the type I cytokine receptor lacking of intrinsic kinase activity.

In particular, *JAK2* protein associates with erythropoietin receptor (EPOR), thrombopoietin (TPO) receptor (*MPL*) and granulocyte colony-

stimulating factor receptor (GCSFR). These receptors after binding activation to their specific ligand, activate in turn the JAK2 kinase. The activated JAK2 kinase complex is involved in the recruitment and phosphorylation of signal transducers and activators of the STAT signalling accountable for a wide spectrum of cellular processes, including proliferation, survival or normal functioning of hematopoietic, immune, cardiac and other cells [22, 23].

The acquired somatic mutation *JAK2V617F* is a guanine-to-thymidine substitution resulting in a substitution of valine for phenylalanine at position 617 in JAK2 protein and in a constitutive activation of JAK2 kinase [14-17]. Thereof, the abnormal association of JAK2 to different growth factors and cytokine receptor, particularly MPL, EPOR and GCSFR, become responsible for the proliferation of multiple lineages.

The oncogenic potential of *JAK2V617F* has been well documented, being able to reproduce an MPN phenotype in murine bone marrow transplantation assays [24]. Additionally, different clinical studies have demonstrated an effect of mutant allele burden on phenotype. Mutant allele burden, in fact, directly correlates with hemoglobin value, leukocyte count, and, inversely, with platelet count. [25, 26]

1.3.4 Cytogenetic Characterization

According to metaphase cytogenetic (MC) studies the prevalence of abnormal karyotype in PMF at diagnosis is about 34% [27-29]. However, no specific defect for PMF has been reported. The most frequent abnormalities are del 20q del 13q and abnormalities of chromosome 1. Additional lesions observed included +8, +9, and, with less frequency, alterations on chromosomes 3, -5/del(5q), -7/del7(q), del(12p) and +21. Chromosomal Breakpoints regions involved with del(20q) included q11.2-13.1, for del(13q) the q12-22 and for del(12p) the bands p11-13. There is no presence of Philadelphia chromosome or *BCR-ABL* fusion gene [28-33].

1.4 Human genome variability

In 2001, the International Human Genome Sequencing Consortium, led in the United States by the National Human Genome Research Institute (NHGRI) and the Department of Energy (DOE), shared the results of the first draft of the human genome sequence [34] and would later, in 2004, announce the successful completion of a more accurate version [35].

The final form of the human genome contained 2,85 billion nucleotides, with a predicted error rate of 1 event per 100,000 bases sequenced and only 341 gaps; regions like centromeres and telomeres and a few dozen gaps scattered around the genome, some of them rather large, cannot be considered finished due to the presence of highly repetitive DNA sequences that are difficult to sequence using the current clone-based sequencing technology. In summary: the best estimates of total genome size indicate that about 92.3% of the genome has been completed [35].

Although the earlier draft publications in 2001 had predicted as many as 40,000 protein-encoding genes, the finishing genome sequence version reduced this estimate to approximately 20,000-25,000 protein-encoding genes. In fact, only about 1.5% of the genome codes for proteins, while the rest consists of non-coding RNA genes, regulatory sequences, introns, and noncoding DNA. The daunting task now remains to analyze the billions of base pairs that have been sequenced in order to uncover the biological knowledge within the raw sequence information.

One particularly striking finding of the Human Genome Project research is that the human nucleotide sequence is nearly identical (99,9%) between any two individuals but only a single nucleotide change can be responsible for causing human disease.

We have to consider that the human genome is composed of approximately 3 billion base pairs arranged in a double helix where each single base can exist as one of four DNA molecules: adenine (A), guanine (G), cytosine (C) or thymine (T) [36]. After comparing two human individuals, of these 3

billion bases, approximately 99% are the same in any two given positions [36]. However, 1% of 3 billion still leaves room for millions of bases of variation [36]. One way to begin to understand the genome is to study the different kinds of genetic variation between individuals and how they contribute to various traits or diseases. If a group of individuals with a disease share a specific kind of variation, studying this alteration could be useful for understanding how the disease functions.

However, the versions of the human genome available until 2007, provided by the Human Genome Sequencing Consortium [35] were assembled from haploid genome of donors and, almost exclusively, reported DNA variation in the form of single nucleotide polymorphisms (SNPs). Nevertheless, smaller-scale (<100 bp) insertion/deletion sequences (indels) or large-scale variants also contributed to human biology and disease and warranted an extensive survey.

Finally, in 2007, the first sequencing of an individual diploid human genome shed light on the contribution of these structural variations to the genome variability. After a more accurate estimation of variations, including for the first time the copy number variation (CNVs), was finally estimated that, two human genomes shared a similarity of 99,5% with a genetic variation accounting for 0,5% [37].

At present, variations are classified as either a single nucleotide or a structural variant [38].

1.4.1 Single nucleotide variations

Single nucleotide variations are single base pair changes in the genome between individuals in a given population [39]. They are more commonly referred to as single nucleotide polymorphisms, or SNPs, if the minor allele of the variant is at least 1% [39]. The SNPs make up about 90% of all human genetic variations occurring every 100 to 300 bases at specific genomic sites although not at uniform density.

Each individual has a unique SNPs pattern that is made up of many different single nucleotide variations. However, because only about 3-5 percent of human DNA codes for proteins, SNPs are generally found outside of coding sequences without affecting protein function.

Most SNPs are not responsible for a disease state but indeed serve as biological markers for pinpointing a disease on the human genome map, because they are usually located near a gene found to be associated with a certain disease.

Furthermore, SNPs may be considered the ultimate genetic markers as they represent the finest resolution of a DNA sequence (a single nucleotide), occur frequently throughout the genome and are genetically stable in populations with a low mutation rate. In order to use SNPs as markers, many scientists and researchers are carrying out studies to discover SNPs in genome sequences. It is estimated that a total of about 10 million SNPs exist in the human population, so far [International HapMap Project 2010].

1.4.2 Structural variants

Structural variants are genomic alterations involving segments of DNA larger in size than 1 kb. These alterations can be microscopic or sub microscopic with no specified frequency or clear association with disease or phenotype.

There are different types of structural variant:

Inversion: A segment of DNA that is reversed in orientation with respect to the rest of the chromosome. In more detail, while pericentric inversions include the centromere, the paracentric ones do not.

Translocation: After a chromosome break a portion of chromosome reattaches to a different chromosome (inter-chromosomal translocations) or to an other part of the same chromosome (intra-chromosomal translocations)

Segmental uniparental disomy: Uniparental disomy occur when a pair of homologous chromosomes in a diploid individual is inherited from a single parent.

Specifically, in the acquired uniparental disomy, only a portion of the chromosome pair is involved and this alteration is not inherited but is a somatic acquisition

Copy number variation (CNV): A segment of DNA that is 1 kb or larger and is present at a variable copy number in comparison with a reference genome. The term of CNVs includes insertions, deletions and duplications. When they occur in more than 1% of the population could be referred as copy-number polymorphism (CNP).

1.4.3 Copy number variations

The copy number variations, ranging in size from one kilobase to several megabases, account for roughly 12% of human genomic DNA and can be either inherited or can arise de novo during development [40]. However, the extent to which CNVs effectively contribute to human genetic diversity, and may or may not convey phenotypes, is still object of study.

A CNV can encompass part of a gene, an entire gene or several genes and can confer different clinical phenotype according to the genes, to the genomic region involved and to the distinct mechanism triggered like gene dosage effects, gene disruption, position effects or unmasking of a recessive allele [41, 42].

Thus, as well as causing disease, human-specific CNVs may be responsible for the emergence of advantageous human-specific traits, such as cognition [43, 44]. They therefore are likely to be subject to evolutionary pressures such as selection as well as genetic drift [45].

A lot of studies have tried to estimate the presence of CNVs in healthy individuals and to eventually clarify their role. For this purpose, in fact, in 2001, the International HapMap Consortium launched the International HapMap Project to catalogue genetic similarities and differences in human beings. The genetic data were collected from healthy individuals coming from Nigeria, China, Japan, and U.S.A, constituting the so-called HapMap samples. In the initial phase, the project accounts for 270 HapMap samples while in the 2007 they increased up to 1184.

The Hap Map consortium analyzed the genomic variations of the all above samples to create the haplotype map of the human genome describing the common patterns of human DNA sequence variation in terms of SNPs and CNVs. The haplotype map data are published on line at disposal of researchers to find genetic variants affecting health, disease, responses to drugs and environmental factors [46, 47].

In 2004, two studies conducted by Sebat et al and Iafrate et al., and using two different methods of investigation, reported for the first time the presence of many CNVs affecting large DNA genomic segments in normal human individuals. In particular, Sebat and colleagues used a technology called ROMA (representational oligonucleotide microarray analysis) with 85,000 interrogating probes and an average spacing of 35 kilobase (kb) to study the large-scale CNV(>100-kb). They identified between 22 healthy individuals a total of 221 copy number changes at 76 CNV loci [48]

Instead, Iafrate et al employed a BAC (bacterial artificial chromosome) CGH array with resolution of 1 Mb (megabases), and investigated large-scale CNVs in 55 unrelated healthy individuals. They successful identified 255 clones with copy number gain or loss [49].

About half of the observed copy number changes were identified in more than one individual, thus representing not rare but potential polymorphic variations.

Furthermore, analyzing in detail the sequence of such CNVs, they revealed to include many functional genes involved in regulation of cell growth and metabolism, implicating an important potential role of CNVs in human traits, disease, and evolution [48, 49].

Of note, in 2006, Redon et al. [50] constructed a first-generation CNVs map of the human genome. They employed both SNPs genotyping arrays (Affymetrix GeneChip Human Mapping 500K, 47,4642 SNPs) for comparative analysis of hybridization intensities and Whole Genome TilePath BAC arrays (WGTP, 26,574 large insert BAC clones) for CGH in a cohort of 270 HapMap samples [46]. A total of 1,447 CNV regions were identified and approximately half of these were detected in multiple individuals [50]

However, in some initial studies, CNVs size was often overestimated because of the relative low resolution of some platforms (e.g., BAC arrays) used in CNV analysis.

Mills et al. in 2006 analysed genomic DNA of 36 individuals, generated by shotgun sequencing. They identified 415,436 polymorphic CNVs, ranging in size from 1 to 9,989 bp. Of them, over 148,000 CNVs (35,7%) were placed within known genes and 5,542 CNVs were in correspondence to promoters or exons of genes, where gene function would be expected to be influenced the greatest [51].

McCarroll et al. in 2008 contributed to a more accurate description of CNV map by using on the hybrid SNP-CNV genotyping arrays at higher resolution to study 270 HapMap samples. They documented that large-scale (>100-kb) CNV affects far less of the human genome than reported in precedence, mainly because most CNVs are far smaller than reported CNV regions [52]. However this estimate is still challenged by resolution methods and the results are based on a reference genome that is limited in scope. In addition to the array-based platforms (SNPs or CGH arrays), CNVs can also be investigated by DNA sequencing.

In subsequent years, many additional studies using a multitude of different high-resolution genome analysis platforms may advance our knowledge of CNVs in healthy individuals, and a comprehensive CNV map is beginning to emerge.

1.4.4 Possible genesis of CNV

To explain the origin of copy number variation (CNV) have been proposed four major causative mechanisms which probably account for the majority of CNV: non allelic homologous recombination (NAHR), non homologous end-joining (NHEJ), microhomology-mediated break-induced replication (MMBIR) and long interspersed element-1 (L1) -mediated retrotransposition.

NAHR is the rearrangement between two non allelic DNA sequence repeats sharing high similarity to each other. The crossover generally leads to duplication and/or deletion if the repeats are on the same chromosome and in direct orientation; inversion if the repeats are inverted; chromosomal translocation if the sequence repeats are on different chromosomes. Substrates for NAHR are

low copy repeats (LCRs) or segmental duplication (SDs) of >10 kb in length with 95%–97% similarity [53, 54]

NHEJ is a DNA repair mechanism of double strand breaks (DSBs) caused by ionizing radiation or reactive oxygen species (NHEJ is also responsible for physiological V(D)J recombination. [55, 56] and unlike NAHR, it occurs in absence of extended DNA sequence similarity.

MMBIR occurs during DNA replication; the active replication fork can stall and switch templates using complementary template microhomology to anneal and prime DNA replication. The sequence of homology are represented by the L-1 elements which cover 89% of human genomic DNA and are the only currently active autonomous transposons in the human genome [57, 58].

1.4.5 CNV in disease

More than 1,000 genes have now been mapped within or close to regions that are affected by structural variants, and a number of disorders associated with CNVs have been described [39, 59, 60].

CNV have been related with sporadic and Mendelian genetic diseases in humans, and recently have also been shown to be associated with human complex traits such as susceptibility to HIV infection, Chron disease, autism, and schizophrenia; CNVs like other types of genetic variation, could be interrelated with susceptibility or resistance to disease. Furthermore gene copy number can be elevated in cancer cells. For instance, the EGFR copy number can be higher than normal in non-small cell lung cancer [61] and the number of CAG repeats on the fourth chromosome is a determining genetic factor for Huntington's disease, a trinucleotide repeat disorder [62].

A lot of study have been conducting to elucidate the role of CNVs in haematological malignancies; Breunis et al demonstrated that a CNV affecting FCGR2C is responsible to predispose to idiopathic thrombocytopenic purpura [63], two studies on acute myeloid leukemia [64, 65] revealed the presence of a region significantly affected by recurrent CNVs; Thoenissen et in MPN Ph1-negative demonstrated that the number of CNV increases with the progression of

disease and identified commonly altered regions involved in development disease including not only established targets (*ETV6*, *TP53*, and *RUNXI*) but also new candidate genes on 7q, 16q, 19p, and 21q [66]; Gondek et al. showed how patients affected by myeloid disorders are affected by CNVs and detected additional important CNVs in MDS/MPD-U patients [67].

However, no one study has analyzed CNVs in patients exclusively affected by PMF, so far.

1.5 Databases of CNV

In parallel to the growing of knowledge, several databases are created to catalogue this newly appreciated form of human genetic variation. Of note, the Database of Genomic Variants (DGV) and DatabasE of Chromosomal Imbalance and Phenotype in Humans using Ensembl Resources (DECIPHER).

The DGV (<http://projects.tcag.ca/variation/>) documents structural variants of 1 kb or more that were detected in apparently healthy individuals (<http://projects.tcag.ca/variation>). This database is still growing and researchers can use it to exclude from their analysis the CNVs identified as physiologic. However, it should be cautious when using DGV as this database is not always reliable; are reported CNVs even if reported in a single individual, different platforms have been used in determining the CNVs and finally, data submitted to this database are not subject to an editorial screening.

Instead, DECIPHER (<https://decipher.sanger.ac.uk>) collects clinical information about patients with microdeletions, microduplications, insertions, translocations and inversions, in order to elucidate the pathogenetic role of a single CNV for a specific disease.

1.6 Acquired Uniparental Disomy (aUPD)

Copy number changes aren't the only genomic change with important implications for disease. Loss of heterozygosity (LOH) is also an important genomic aberration. Regions with LOH can extend several megabases (Mb), encompassing multiple genetic loci or even whole chromosome arms.

LOH refers to the loss of a state of heterozygosity which was previously present in the normal cell. Often, in tumour genome, the loss of one allele (LOH) implies the duplication of the remaining one leading to the creation of a new region of homozygosity, also reported as acquired Uniparental Disomy (aUPD)

The aUPD in somatic cells might arise during mitosis from non-disjunction chromosomal segregation error or from non allelic homologous recombination (NAHR) in presence of low-copy repeats [68]; both errors result in a homozygous chromosome.

In summary, knowledge gathered over the past 5 years has strongly supported the notion that aUPD might be a far more common, non random contributing factor to the development of many diseases, including cancer. aUPD regions might harbor homozygous mutated, deleted or promoter-hypermethylated genes or miRNAs or imprinted genes.

The distribution of aUPD regions appears non-random, and homozygous gene mutations have been discovered in aUPD regions in various cancers. For example, associations have been found between aUPD and homozygous mutations: in *c-CBL* in acute myeloid leukemia and atypical chronic myeloid leukemia [69, 70]; in *JAK2* and myeloproliferative disorders [71]; in *NF1* and juvenile myelomonocytic leukemia [72]; in *A20* and B-cell lymphoma [73]; in *TET* and myelodysplastic syndrome [74] and in *MPL* and refractory anemia with ringed sideroblasts and thrombocytosis [75].

1.7 New Challenge: Single Nucleotide Polymorphism (SNP) array

Deep-sequencing approaches are particularly powerful at identifying copy number changes but the cost of this technique remains very high. Nevertheless, it is most probable that most CNV discovery over the next few years will be achieved using DNA microarray technology of continually increasing resolution and cost effectiveness as the comparative genomic hybridization (CGH) array and Single nucleotide polymorphism (SNP) array.

Importantly, the advantage of employing SNP-A respect to CGH array is that allows the identification of copy-number and uniparental disomy too, in the same experiment using analytical tools. The aUPD is extremely important in both cancer samples and general cytogenetic cases, but unfortunately cannot be easily detected neither by traditional cytogenetic methods such as FISH nor by more recent technique as CGH array.

At present the most updated SNP oligonucleotide genomic microarray is the Genome-wide SNP array 6.0 provided by Affymetrix.

The Affymetrix method is a single colour assay, composed of multiple overlapping allele specific hybridization probes that are complementary to SNP regions present in the genome processed in the assay.

The first SNP array (10K) produced by Affymetrix contained 10,000 SNP probes, followed by the 100K (100,000 SNP probes), 500K (250,000 SNP probes), the 5.0. array (500,000 SNP probes) up to the newest array the SNP A 6.0 with a total of 1,8 million probes on a single slide.

1.8 Genome-wide SNP array 6.0

Genome-Wide Human SNP Array 6.0 provides the coverage and sensitivity needed to identify large and small chromosomal abnormalities.

In particular, this array is an oligonucleotide array that allows the detection of single SNP genotype as well as the presence of CNV in terms of deletions and amplifications, regions of Loss of Heterozygosity and segments of Uniparental Disomy.

The Affymetrix Genome-Wide Human SNP Array 6.0 consists of a single array that enables genotyping of approximately 906,600 single nucleotide polymorphisms (SNPs) and assaying of approximately 945,826 non-polymorphic probes for detection of copy number variation.

SNPs were chosen and represented on arrays based upon reproducibility, call rate, accuracy and concordance with genotypes from the International HapMap Project. These SNPs have been validated across a minimum of 500 individuals from multiple populations, including Caucasian, Asian and African.

Each array has 6,8 million 5 x 5 μm features. A feature consists of more than 1 million copies of a 25-basepair-oligonucleotide probe of defined sequence. Each polymorphic SNP is interrogated by six to eight oligonucleotide probes (three or four probes for the single SNP allele). Approximately 482,000 SNP probes derive from the previous-generation Mapping 500K and SNP 5.0 arrays while the novel additional 424,000 SNP probes encompass tag SNP markers derived from the International HapMap Project, SNPs on chromosomes X and Y, mitochondrial SNPs, SNPs in recombination hotspots, and new SNPs added to the dbSNP database after completion of the GeneChip Human Mapping 500K Array Set.

Moreover this array contains a total of 946,000 non-polymorphic copy number probes. About 202,000 probes targeted 5,677 known copy number changes reported in the Toronto Database of Genomic Variants (DGV), additional 744,000 probes are evenly spaced along the genome, enabling to detect copy number changes more precisely and assuring a more complete coverage of CNP than other SNP/CN platforms.

1.9 SNP array: Paired Unpaired

The SNPs array data can be studied by paired or unpaired analysis. In the latter, the SNPs genotyping and the CNV pattern identified in the tumour sample are compared to the SNPs genotyping and the CNV pattern of a healthy genome reference line provided by HapMap Consortium and available *in silico*. This genome reference line is the virtual addition of all CNVs and SNPs calls found in the HapMap samples.

In the unpaired analysis, CNVs detected in the tumour sample but also present in the genome reference line, are considered to be polymorphic alterations, while CNVs identified only in the tumour samples but not present in the healthy HapMap samples, are lesions potentially associated with the disease.

Indeed, in the paired analysis the SNPs calls and CNVs found in the tumour samples are compared to that ones detected in the match normal sample of the same individual. In this case, the CNVs identified both in the tumour sample and in its matched control sample are alterations belonging to the physiological germline variations of the individual inherited by the tumour as other cell type. On the contrary, the CNVs found exclusively in the tumour sample are alteration most likely acquired during the development disease.

The last approach is highly preferred to the other one thanks to the significant reduction of false discovery rates.

2. AIMS

The biology of PMF is not yet well known and no consistent cytogenetic abnormalities were detected; therefore, it is not clear what role acquired abnormalities plays in the pathogenesis of disease. Nevertheless, all the studies of karyotyping conducted by now, have analysed cohorts of patients affected by different MPNs but no study has ever specifically focused on PMF patients.

With the resolution of genomic arrays growing exponentially, they are becoming increasingly powerful tools for describing the genetic events underlying cancer development.

In this study using the powerful Genome-Wide Human SNP Array 6.0 we performed a paired analysis on patients exclusively affected by primary myelofibrosis, in order to identify novel cryptic genomic aberrations.

3. MATERIALS AND METHODS

3.1 Patients

We studied 20 PMF patients diagnosed according to WHO criteria [1]. To this regard, the bone marrow biopsies of all patients were reviewed by at least two experienced hematopathologists. The cases were further equally divided into a training set (14 PMF) and a test set (14 PMF). The main clinical features were homogeneously represented in the two groups (Table1). In particular, the patients of the training test and test set reported a median age of 68 and 74 years, a female/male ratio of 50% and 40%, a median follow up of 28,5 and 38,5 months, a ratio $JAK2^{wt} / JAK2^{mut}$ of 60% and 40% and a leukemic transformation of 0% and 2%, respectively.

In both training and test sets the genomic DNA was isolated from peripheral blood (PB) myeloid cells while additionally in the training set we also studied non neoplastic T-lymphocytes isolated from peripheral blood (see below). In addition, we collected genomic DNA from 10 healthy individuals, which served as controls.

The local ethical committee approved all the study and written permission and informed consent have been obtained from all patients before sample collection.

Table 1. Patients' characteristics

	Training set	Test set
Patients' number	10	10
Median age, years (range)	68	74
Patients aged over 65	5 (50%)	8 (80%)
Female/male ratio	5/5 (50%)	4/6 (40%)
JAK2 ^{wt} /JAK2 ^{mut}	6/4 (60%)	4/6 (40%)
Median follow up, months	24,5	38,5
Leucemic transformation	0 (0%)	2 (20%)

3.2 Purification of myeloid and CD3⁺ cells from peripheral blood

Peripheral blood mononuclear cells (PBMC) and granulocytes were obtained from 20 patients by Ficoll-Paque by density gradient separation (GE Healthcare Wisconsin, USA). Subsequently, in 10 cases (training set), we collected granulocytes, representing the clonal population, on one hand, and the PBMC on the other. The latter were then processed in order to isolate *bona fide* non neoplastic T-lymphocytes. CD3⁺ cells isolation was performed by immunomagnetic labelling of CD3⁺ cells followed by a separation process using the MACS device (Miltenyi Biotech, Bergisch Gladbach, Germany). In brief, up to 2x10⁸ PBMNC were washed with PBS and 1% human albumin to reduce the labelling volume to 10 ml. The cells were then incubated with a clinical grade anti-CD 3 monoclonal antibody (MoAb) conjugated to microbeads (MACS CD3 reagent, Miltenyi Biotech) for 20 minutes. After washing, CD3⁺ cells were purified on a MS column (Miltenyi Biotech). After selection, cell present in the

positive (CD3) and negative (PBMNC) fractions were counted and submitted to flow cytometry analysis. Assessment of CD3⁺ cells before and after the separation process was carried out by labelling of the target cells with anti-CD3-phycoerythrin (PE) MoAb (Becton Dickinson, San Jose, CA, USA), followed by cytometric reanalysis of purified cells performed on a gated population set on scatter properties by FACScan equipment (Becton Dickinson). A minimum of 10,000 events was collected in list mode on FACScan software.

In the other 10 cases, conversely, only granulocytes were collected and used for the subsequent analyses (test set).

Notably, to ensure the collection of adequate neoplastic components, a morphological evaluation of the granulocytic fractions was performed on smears obtained after cytopspin enrichment. Only samples for which at least 80% of the cellular population was constituted by myeloid elements were selected for the following analysis.

3.3 JAK2 617V>F genotyping

JAK2 617V>F allele burden was measured by a quantitative real-time polymerase chain reaction assay (qPCR) as previously described [76]. In particular, 20 ng of genomic DNA were isolated and purified from peripheral blood granulocytes. Polymerase chain reaction amplification and detection were performed on ABI Prism 7300 analyzer (Applied Biosystems, Foster City, CA) according to the following cycling conditions: 10 minutes at 95°C followed by 40 cycles of 15 seconds at 95°C and 60 seconds at 60°C. Primers flanking the mutant region (forward primer 5'-AAGCTTTCTCACAAGCATTGGTTT-3'; reverse primer 5'-AGAAAGGCATTAGAAAGCCTGTAGTT-3') were employed together with TaqMan probes (Applied Biosystems, Milano, Italy) which were specific for either wild type (VIC-5'-TCTCCACAGACACATAC-3'MGB) or mutant *JAK2* allele (FAM-5'-TCCACAGAAACATAC-3'-MGB). All samples were analyzed in triplicate and the amount of *JAK2*617V>F allele was calculated by comparison with serial dilutions of mutant DNA, obtained from a

polycythemia vera patient harbouring 100% mutant allele, and wild-type DNA from healthy subjects. The mean of triplicate Δ CT determinations (CTJAK2617V>F – CTJAK2WT) was used to calculate the percentage of mutant allele. Positive and negative controls were included in each assay; inter- and intra-assay variation was 3% and 5%, respectively.

3.4 SNP array procedure

Array: Genome-wide SNP array 6.0

This array contains 906,600 Single Nucleotide Polymorphism (SNP) probes and 945,826 copy number variant (CNV) probes, providing marker spacing in the range of as low as 700 bases (Affymetrix, Santa Clara, CA, USA).

General DNA Requirements

Genomic DNA was extracted using QIAamp DNA Mini Kit (QIAGEN Inc., Valencia, CA, U.S.A.). Concentration and quality samples were quantified by Nanodrop ND-1000 spectrophotometer running software version 3.0.1 (NanoDrop Technologies, Inc., Rockland DE). Genomic DNA was assessed to be double strand, free of inhibitors and not highly degraded. When A₂₆₀/A₂₈₀ ratio and A₂₆₀/A₂₃₀ were lower than 1.9 and 2 respectively, we proceeded with DNA clean up, according to manufacturer's instructions (Affymetrix, Santa Clara, CA, USA).

SNP array process

During the SNPs array experiments is highly recommended to use two separate rooms. This device can greatly reduce the risk of sample contamination due to previously amplified PCR products. These rooms are referred to as the:

- Pre-PCR Clean Room
- Post-PCR Room

Pre-PCR Clean Room

Briefly, a total of 500 ng of highly purified genomic DNA from each patient was processed in each step by a provided Affymetrix® Genome-Wide Human SNP Nsp/Sty Assay Kit 5.0/6. and other prescribed kits. For all samples, 250 ng of DNA was digested with *NspI* and 250 ng with Sty enzymes (*Nsp I* Enzyme, Sty I Enzyme by New England Biolabs) followed by adaptor ligation (T4 DNA Ligase by New England Biolabs). A generic primer that recognizes the adaptor sequence was used to amplify adaptor-ligated DNA fragments (Clontech TITANIUM DNA Amplification Kit by CELBIO). PCR conditions have been optimized to preferentially amplify fragments in the 200 to 1,100 bp size range.

Post-PCR Clean Room

In post PCR room, PCR products were pooled, purified (Magnetic Beads by Agencourt), and fragmented. Fragmented PCR products were then labeled with a fluorochrome, denatured, and hybridized onto a Genome-Wide Human SNP Array 6.0. Arrays were then washed by the use of Affymetrix fluidics stations and after washing, they were incubated with a streptavidin-phycoerythrin conjugate to enable detection of hybridized fragments on the array. The fragment binding (which reflects the genotype) was then measured by a scanner picking up the fluorescent signals on the array feature scanned (GeneChip® 3000 Scanner with 7G upgrade Affymetrix, Santa Clara, CA, USA).

3.5 Data Analysis

Data were analysed using Affymetrix Genotyping Console (GTC) 4.0 and Partek Genomic Suite 6.5. First we loaded on GTC 4.0 all sample attribute (ARR) and intensity data (CEL) files. We thus performed the intensity Quality Control (QC) for determining the basic data quality. All the arrays passed the recommended QC threshold (≥ 0.4) according to manual instruction (GTC 4.0 manual, Affymetrix, Santa Clara, CA, USA). Thus, we generated CHP files by performing genotyping analysis, based on Birdseed v.2 algorithm and using default parameters (confidence threshold 0.1 and block size 0).

Subsequently, all CEL and CHP files were uploaded on Partek Genomic suite 6.5. We updated all the arrays to the new human genome build Genome Reference Consortium GRCh37 (ucsc hg19), corrected the GC waviness and used only the latest builds of databases (Refseq, Database of Genomic Variants).

We first analyzed the 10 PMF samples constituting the training set for which matched normal DNA was available. Starting from CEL files we performed a paired copy number analysis comparing directly normal CNV pattern and tumour CNV pattern of each patient. In particular, we detected amplifications and deletions using genomic segmentation algorithm with 10 minimum genomic markers and a p-value threshold $\leq 0,0001$.

After that, starting from training set CHP files we performed LOH analysis with the following parameters: max probability equal to 0,99; genomic equal to 0, and genotype equal to 0,1.

Finally we overlapped the Copy Number and LOH analysis results obtaining a complete tumour DNA characterization in terms of Amplification with/without LOH, Deletion with/without LOH and Copy Neutral LOH.

As paired analysis is the gold standard for SNP-A analysis (Heinrichs 2010) we decided to re-study the training set by an unpaired analysis too as to find the differences between the two approaches (paired *versus* unpaired analysis) and most importantly to find the best parameters to reduce them.

In the unpaired analysis, Partek directly provided you two baselines for tumour DNA normalization: LOH baseline and CN baseline, both generated from

Hap Map samples (individuals from Nigeria, China, Japan and Western Europe)[46]

Additionally we built two homemade baselines (one for CN analysis and the other one for LOH analysis) made up by DNA from 10 healthy individuals.

In practise, we conducted an unpaired analysis of the training set both with Partek and with our homemade baselines. We detected amplifications and deletions using genomic segmentation algorithm and tested different parameters for each analysis.

The homemade baseline was preferred to the Partek one, basing on its consistency with the paired training test results. The best parameters found were 20 minimum genomic markers, a p-value threshold $< 0,0001$ for the CN analysis and max probability of 0,999999, a genomic decay of 0 and a genotype error of 0,1 for the LOH analysis.

According to these findings we proceeded with unpaired analysis of the test set. We thus analyzed the test set constituting of 10 PMF samples for which matched normal DNA was not available. Starting from CEL and CHP files we performed an unpaired copy number and LOH analysis comparing the tumour DNA of each patient to the homemade baselines with the parameters previously found.

In all analyses we excluded from the study the chromosomes x, y, the mitochondrion and finally the CNV segment <1 Kb and UPDs regions < 1 Mb. Furthermore we excluded all the regions with low density markers, where the insufficient probe coverage didn't assure the right CN attribution.

After that, in order to evaluate the prevalence of CNVs within each chromosome we normalized the number of alterations on chromosome length (in Mb).

3.6 TaqMan Copy Number assay

To validate target genomic copy number amplification found by SNP-A analysis, we used a TaqMan Copy Number assay (Applied Biosystems; Foster

City, CA). In particular, the target copy number was assessed with a specific TaqMan Copy Number Assay (Hs04039030_cn; Applied Biosystems; Foster City, CA) and TaqMan Copy Number Reference Assay *RNase P* (Applied Biosystems). For each sample 4 repeats of 20 ng of DNA were run in a duplex real-time Polymerase Chain Reaction with TaqMan Universal Genotyping Master Mix, a FAM® dye-labeled TaqMan® Copy Number Assay and a VIC® dye-labeled TaqMan® Copy Number Reference Assay (Applied Biosystems; Foster City, CA). The *RNaseP* reference genes are known to be present in two copies in a diploid genome, regardless of the copy number of the target of interest, and are used to normalize sample input and minimize the variation between the target of the test and reference assay.

The reactions were performed on the genomic DNA from the original tumour samples and, according to the manufacturer's protocol, were run on an ABI 7900HT real-time PCR machine (Applied Biosystems; Foster City, CA) and primarily analyzed by SDS Software 2.3.

The relative quantitation analysis, without calibrator sample, was then performed by CopyCaller™ Software v1.0

3.7 *In silico* validation

To confirm our results in a validation set, we downloaded two data sets of Affymetrix CEL files publicly accessible in the GEO database (<http://www.ncbi.nlm.nih.gov/geo/>). As were not present series only constituted by PMF we included in the validation set all the available MPN cases.

In particular, we used the GEO accession number GSE19647 (<http://www.ncbi.nlm.nih.gov/gds/?term=GSE19647>) and downloaded from this data set 7 CEL files of patients in PMF blast phase studied by GeneChip® Human Mapping 250K Nsp and 39 CEL files of MPN patients analysed by Affymetrix Human Mapping 50K Array Xba 240 [66].

Moreover, we downloaded from another data set, with accession number GSE21991 (<http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE21991>), 14

patients affected by MDS/MPN and analysed by the our same array, Genome-Wide SNP array 6.0 [77].

(All cel files number are reported in Appendix A)

3.8 Immunohistochemical validation

In order to evaluate the expression of SIRPB1, we studied by immunohistochemistry selected cases carrying or not the amplification within the 20p13 cytoband (containing the *SIRPB1* gene). Immunohistochemistry was performed as previously reported (Tripodo et al. Haematologica 2009). Briefly, Four-micrometers-thick sections of bone marrow biopsies were deparaffinized and rehydrated to water, subsequently the slides were microwave treated in Tris-HCl/EDTA buffer pH 9.0 (DakoCytomation) for a total of 20 minutes prior to PBS washing. After neutralization of the endogenous peroxidase with H₂O₂ for 10 minutes, the sections were first incubated with protein block (Novocastra, UK) for 10 minutes. Sections were then incubated with the primary polyclonal antibody rabbit anti-human SIRPB1 (Proteintech Group, USA; dilution 1:50). Incubation time was overnight at 4°C. Normal rabbit serum was used instead of primary antibodies as negative control. Binding of the primary antibody was revealed by a polymer detection system (Novolink max polymer detection system, Novocastra,UK) using DAB (3,3'-Diaminobenzidine, Novocastra,UK) substrate-chromogen. After counterstaining with hematoxylin (Novocastra,UK), the sections were analysed under a Leica DM2000 optical microscope (Leica, Germany) and captions were collected using a Leica DFC320 digital camera (Leica).

4. RESULTS

4.1 Identification of CNV, LOH and aUPD

First, we aimed to detect unbalanced chromosomal defects as well as aUPD. To address this issue we analysed cases included in the training set (10 cases), and performed a paired analysis for copy number determination by genome wide SNP array 6.0. Specifically, we directly compared normal (T lymphocytes) and neoplastic (myeloid cells) matched samples.

We identified a total of 2,765 CNVs, ranging in size from 1 kb to 23 Mb (Appendix B). As shown in Fig.1, all the CNVs are displayed in a whole-genome karyoview: along each chromosome the CNVs, depicted in different colours (amplifications in blue, deletions in violet and aUPD in green), are distributed in virtual vertical columns, each one representing a single patients.

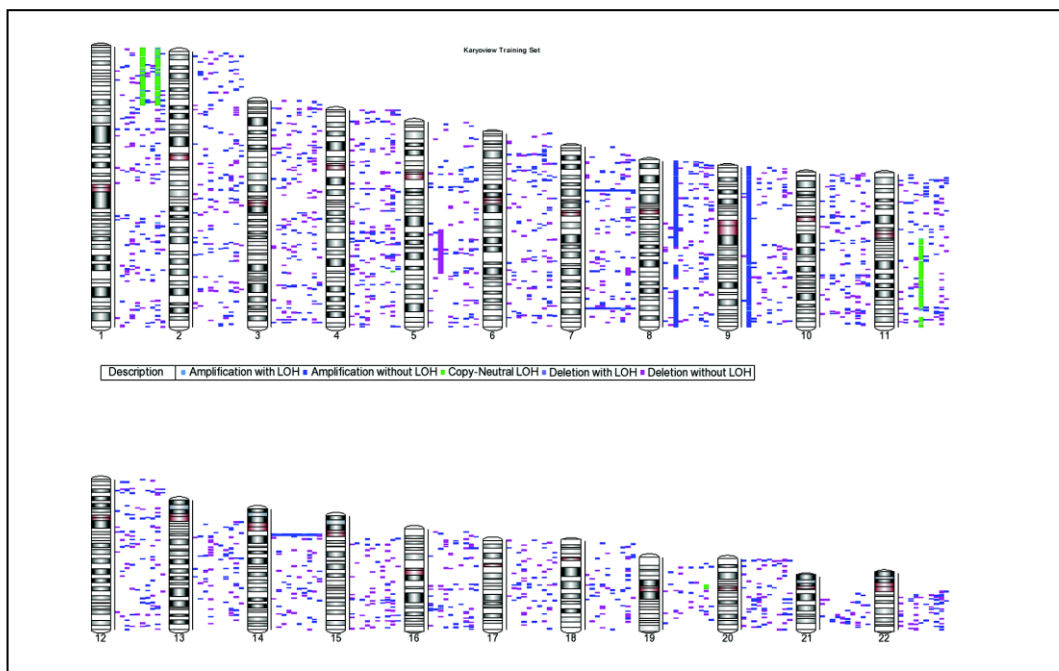


Figure 1. *Karyoview of training set.* Amplification with LOH (Light blue), Amplification without LOH (Dark blue), Copy Neutral LOH (aUPD, Green), Deletion with LOH (Violet), and Deletion without LOH (Cyclamen) are represented. All the 2,765 lesions recorded in the training set are depicted.

Globally, the CNVs were constituted by either amplifications (N=1,584, median size 14,5 Kb - range of 1Kb-2,3 Mb), deletions (N= 1,132, median size

16,7 Kb - range of 1 kb- 9 Mb) and aUPD (N=49, median size 1,9 Mb - range of 1 Mb-7,2 Mb) affecting the PMF genome in variable proportions (Figure 2).

Based on the most update Genomic Toronto Database version (<http://projects.tcag.ca/variation/>), 1,773 CNVs (64%) of detected CNVs were then regarded as known, while 992 CNVs (36%) were considered as novel.

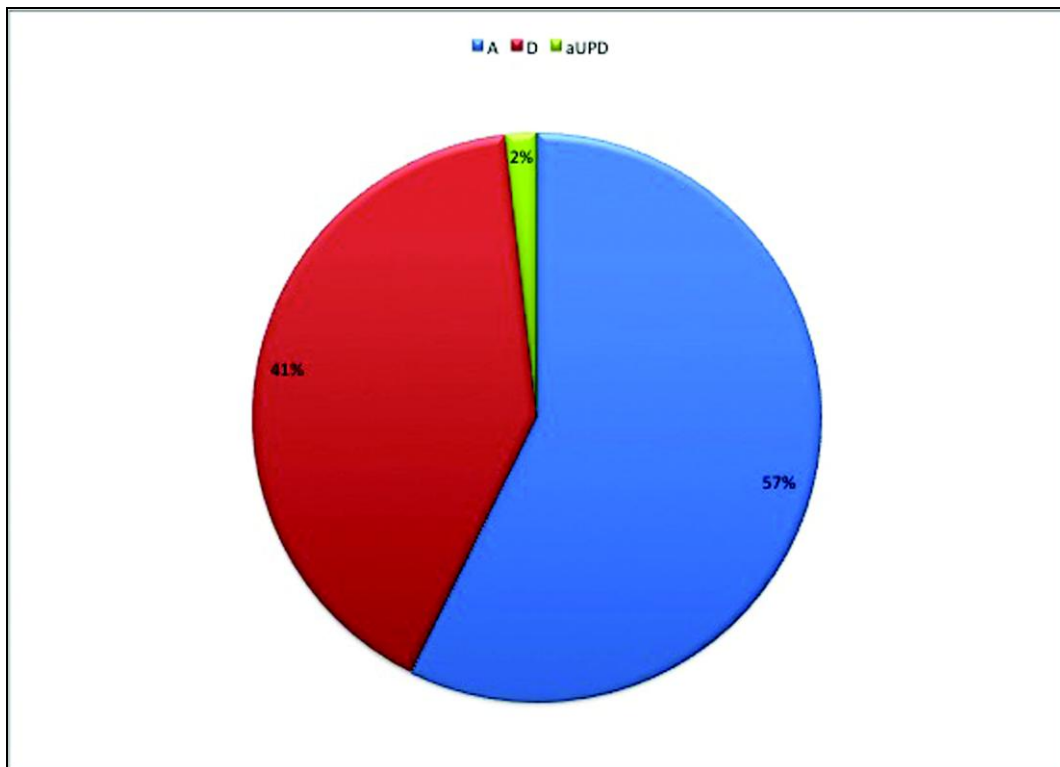


Figure 2. *Global distribution of CNVs across genomes*

We then looked at the CNVs distribution across the chromosomes in order to assess whether certain chromosomes were predominantly affected. After opportune normalization on chromosome size, we observed that the number of CNVs was over the median value in the chromosomes 4, 7, 8, 9, 10, 11, 14, 16, 20 and 22, while it was below in the remaining ones. However the difference was not significant (χ^2 , p value= 0,99) (Figure 3).

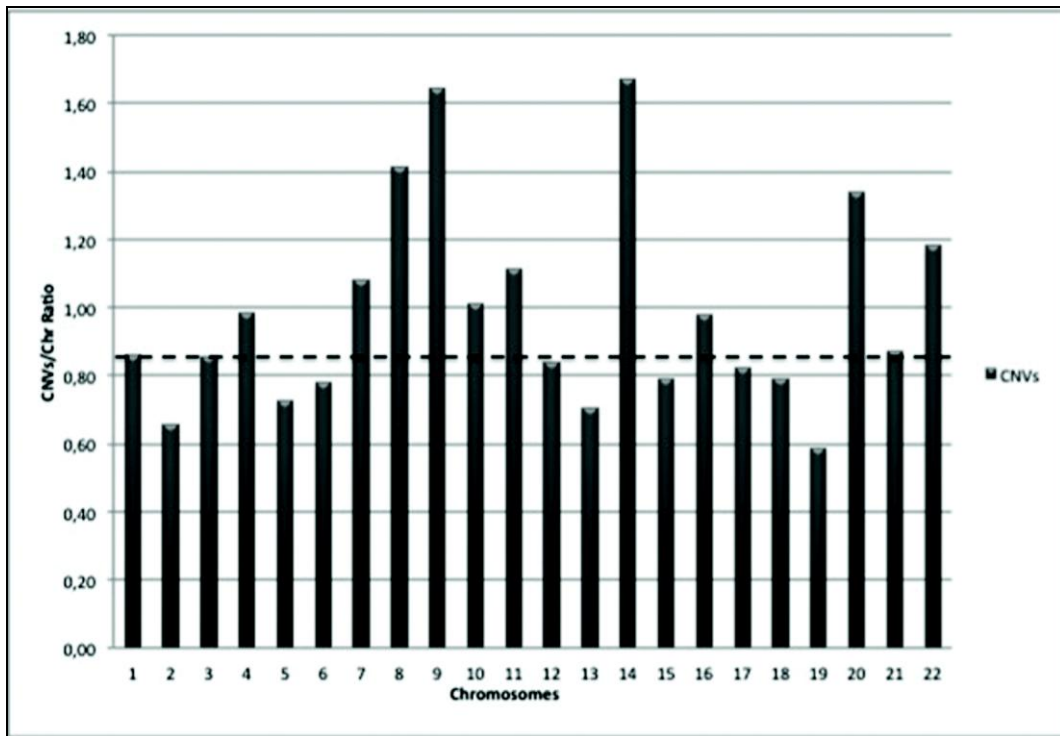


Figure 3. *CNVs distribution across chromosomes.* On the x-axis, the chromosomes (excluded x, y and mitochondrial chromosomes) are presented. On the y-axis, the number of CNVs affecting each chromosome is offered.

As far as the single cases were concerned, we identified a mean of 343 CNVs/patient (range 188-602), with a mean size of 168,623 kb and a median size of 15,912 kb. Amplifications were confirmed to be the commonest lesions in all patients, though all the types of abnormalities were detected in most instances (Figure 4).

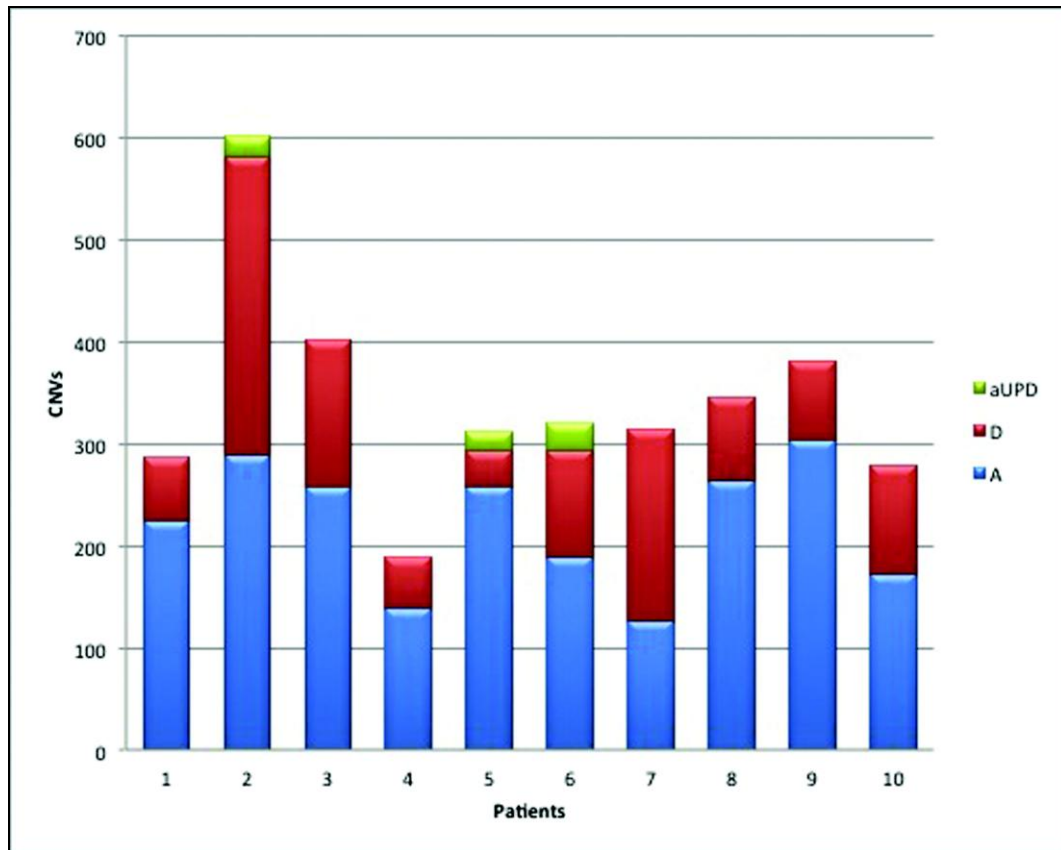


Figure 4. *Recurrence of CNVs across PMF patients.* On the x-axis, the number of patients (evaluated in the training set) is presented. On the y-axis, the number of CNVs recurring in a certain number of patients is offered.

Subsequently, we investigated whether the above CNVs were present in the test set (N=10 cases) as well. First, we visualized by karyoview the lesions found in test set patients confirming the presence of a complex karyotype in this set too. Then we examine whether the CNVs of test set were affecting the same genomic regions reported in the training set. To do this, we compared the 744 cytobands found to be altered in the training set with the 419 ones found in the test set. Indeed, a significant overlap was recorded between the two groups; specifically, in the test set we confirmed 327/744 lesions (44%)($p= 0.0053$) (Figure 5 A-B). In this way we could appreciate a consistent degree of homogeneity between the two groups, sharing a high level of comparability not only for the clinical features but for the genomic characteristics too.

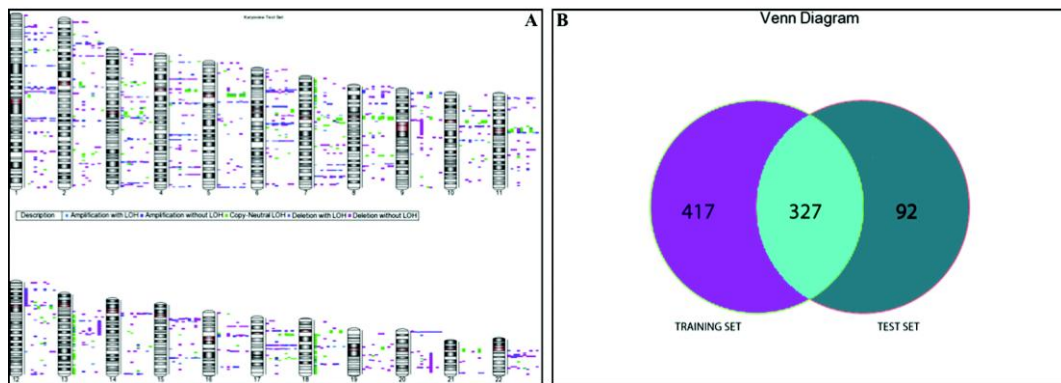


Figure 5. CNVs recorded in the test set. A) Karyoview of test set: Amplification with LOH (Light blue), Amplification without LOH (Dark blue), Copy Neutral LOH (aUPD, Green), Deletion with LOH (Violet), and Deletion without LOH (Cyclamen) are represented. All the 2,096 lesions recorded in the test set are depicted. B) Venn-Diagram representing the significant overlap between the cytobands recognised as affected by any imbalance in the training (violet) and test set (green), respectively ($p=0.0053$).

In the training set, as internal control validation, we confirmed the presence of the two abnormalities previously detected by MC: a trisomy 8 and a 5q deletion (Figure 6 A-B)

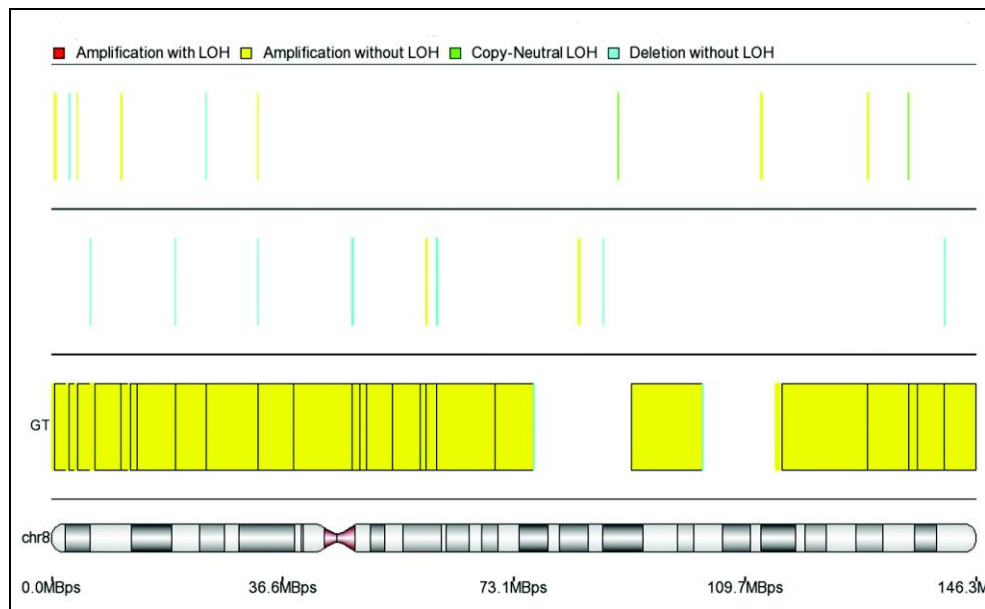


Figure 6 A. Correspondence between metaphase cytogenetic and SNPs-A karyotyping. A) Trisomy of chromosome 8 identified in one patient (yellow bar) known to carry the same abnormality basing on metaphase cytogenetic results. Please note two different cases randomly selected and presented as negative controls (arrows).

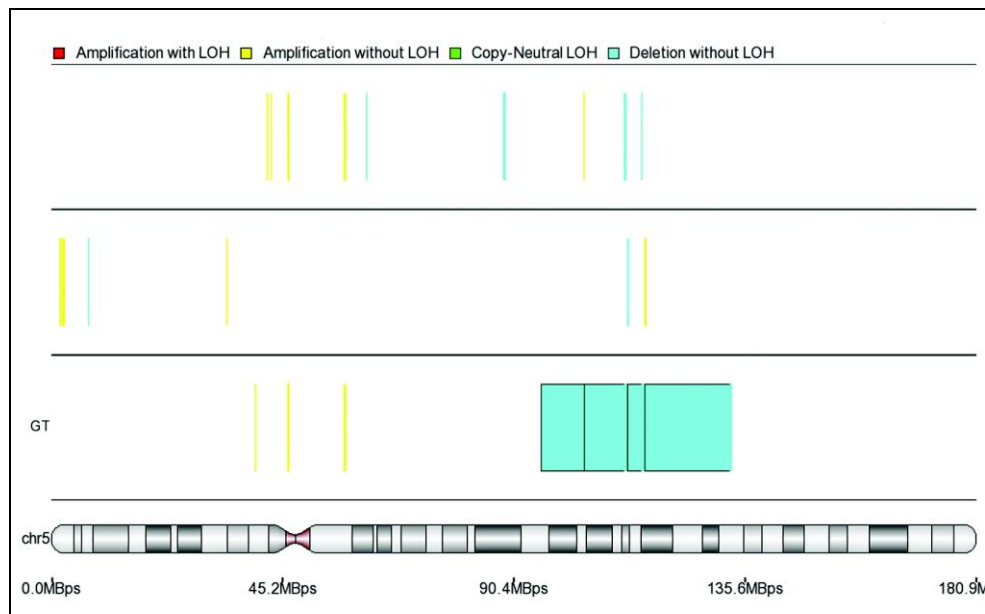


Figure 6 B. Correspondence between metaphase cytogenetic and SNPs-A karyotyping. B) Deletion of chromosome 5q identified in one patient (light blue bar) known to carry the same abnormality basing on metaphase cytogenetic results. Please note two different cases randomly selected and presented as negative controls (arrows).

We then compared our findings with the ones derived from the previously published SNP-A profiling studies, though they were more often performed at a lower resolution. We confirmed the presence of different regions of aUPD on chr9 at *JAK2* locus [78], on chromosome 13q, on chromosome 1 and on chromosome 11 [70, 79]. More specifically, concerning the *JAK2* locus, we found 5 amplifications, 1 deletion and 1 aUPD in cases carrying *JAK2* somatic mutations (as detected by PCR and direct sequencing), while 1 amplification, 1 deletion and 1 aUPD were recorded in unmutated cases. In addition, we detected in 2/20 patients and in 3/20 patients respectively, two large region of aUPDs in correspondence to the *MPL* and *CBL* genes, which are commonly mutated in PMF. Furthermore, we identified in 2/20 patients the deletion and in 3/20 patients the aUPD of *RBI*, a gene frequently deleted in MF. Such results are consistent with the prevalence reported in literature for such lesions [10, 80, 81].

Unfortunately, further validation by direct sequencing was not possible due to the lack of residual material (Figure 7 A-B-C).

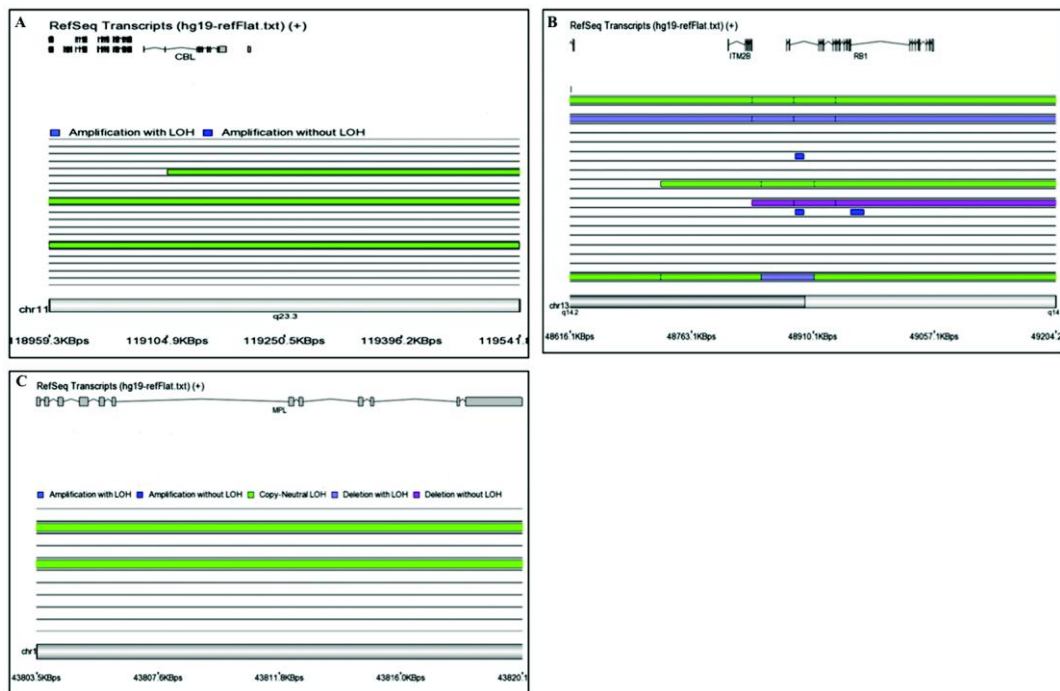


Figure 7. Acquired uniparental disomy in PMF patients A) aUPD detected in 3 patients on chromosome 11 in correspondence to *MPL* gene (green bars). B) aUPD detected in 3 patients on chromosome 13. In particular, in correspondence of *RBI* gene 3 patients presented aUPD (green bars) and two deletions (violet and cyclamen bars). C) aUPD detected in 2 patients on chromosome 1 in correspondence to *MPL* gene. Please note for each figure, different cases randomly selected and presented as negative controls (empty bars).

Taken together, our results confirmed the presence of a complex and heterogeneous karyotype in MF patients, basically consistent with previous studies [50] and proposed novel CNVs for further characterisation.

4.2 PMF presents with recurrent abnormalities on 20p13

We then looked for CNVs consistently recurrent in our dataset. First the analysis was carried out in the training set (10 cases). As the matched control was represented by T-lymphocytes, we excluded CNVs on chromosomes 7p34,

7p14.1, 14q11.2 (recurrent in all instances) because of possible artefacts due to the physiological alteration secondary to T-cell receptor editing process.

No lesion was found to be present in up to 6/10 cases. Conversely, we found 1 CNV in 5/10 samples, 5 in 4/10 samples, 47 in 3/10 and 285 in at least 2/10 (Figure 8).

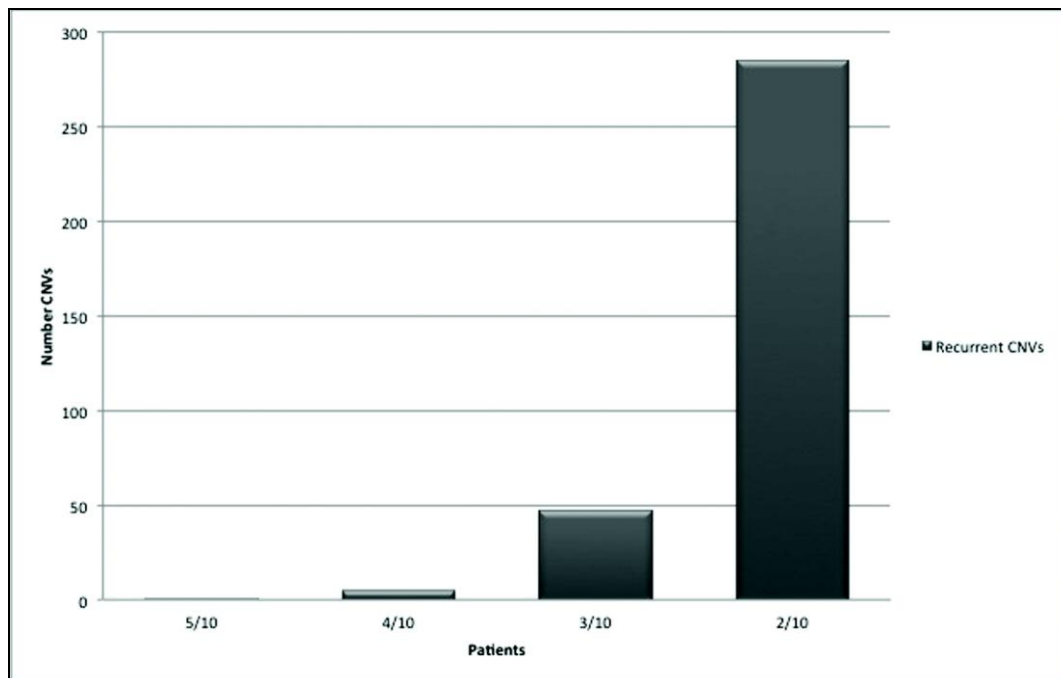


Figure 8. Recurrence of CNVs across PMF patients. On the x-axis, the number of patients (evaluated in the training set) is presented. On the y-axis, the number of CNVs recurring in a certain number of patients is offered.

Specifically, the commonest recurrent lesion was an amplification occurring in 1p31.1 cytoband. However, as this was an already known lesion [82] we focused on CNVs occurring in 4/10 cases and evaluated them in the test set. These genomic imbalances, in particular, involved the cytobands 1p31.1, 10q23.31, 4q25, 4q31.3 and 20p13. By looking for these lesions in the test set, we found the 20p13 abnormalities to be the most frequently occurring one (7/10 cases). Thus, such lesion, representing the Minimally Affected Region (MAR) within the 20p13 cytoband, was then overall recorded in 11/20 patients (55%). Specifically, the identified MAR was a segment of DNA amplified 4 times (media

copy number = 4,1) on chromosome 20 from 1572019 to 1581930 nucleotides, extending for 9,911 bps and overlapping the *SIRPB1* gene (Figure 5).

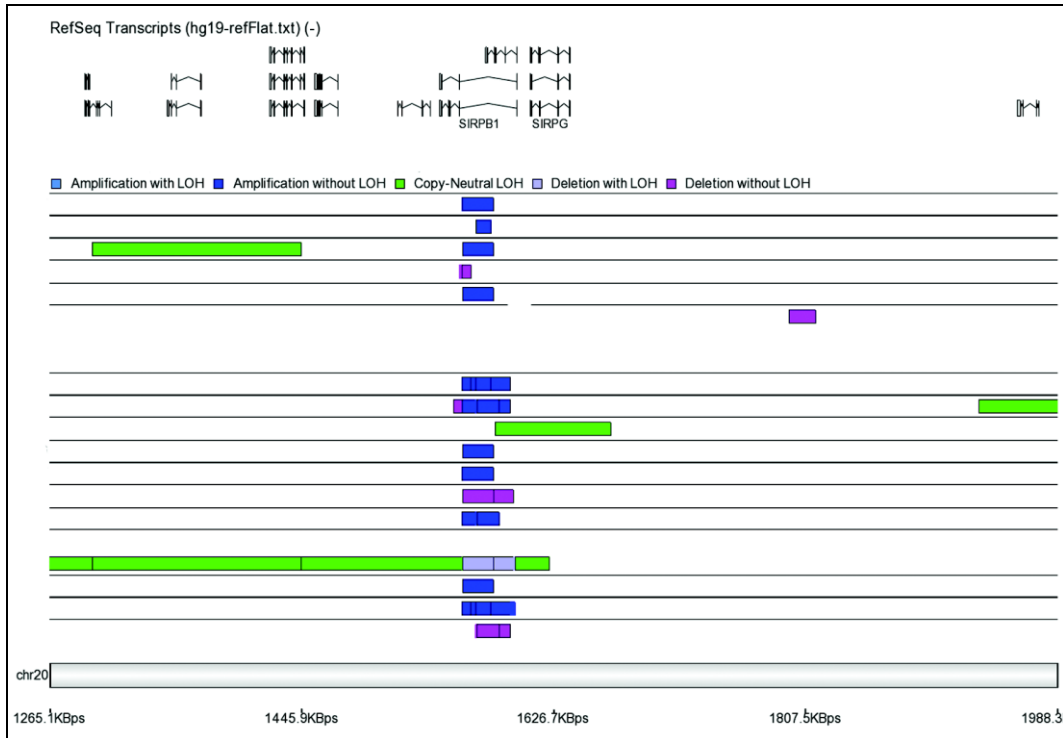


Figure 9. *Minimally affected region (MAR) definition.* Genomes from all patients are represented as single lines (named P1-P20). MAR (black arrow) was defined as the minimal DNA fragment affected by any imbalance in the majority of cases. Correspondence to *SIRPB1* gene was found (red arrow). Please note the occurrence of other abnormalities rather than amplification within the MAR or in the adjacent area in some cases.

Subsequently, due to the high prevalence of the lesion and the known role of chromosome 20 in myeloid malignancies [83, 84], we further extended our attention to the context of areas adjacent to the MAR and considered the entire 20p13 cytoband. In particular, 9/10 patients in the training set showed different alterations affecting the cytoband 20p13. We then tested the presence of 20p13 abnormalities in the test set too, and we found it in 10/10 cases. Thus, overall, 19/20 patients showed abnormalities on chromosome 20p13.

Finally, in order to assess the prevalence of 20p13 abnormalities in an independent panel of cases, we tested an *in silico* validation set, including 14 cases of MDS/MPN, 7 cases of MF in blast phase and 39 cases of MPN

previously reported and for which high SNP karyotyping CEL files were available (<http://www.ncbi.nlm.nih.gov/projects/geo/>) [83, 85].

Notably, we could confirm the presence of lesions affecting 20p13 cytoband in 7/14 MDS/MPN patients, 5/7 MF blast phase patients, and in 1/39 MPN patient, though the majority of analyzed cases were originally studied with less sensitive arrays (Figure 10).

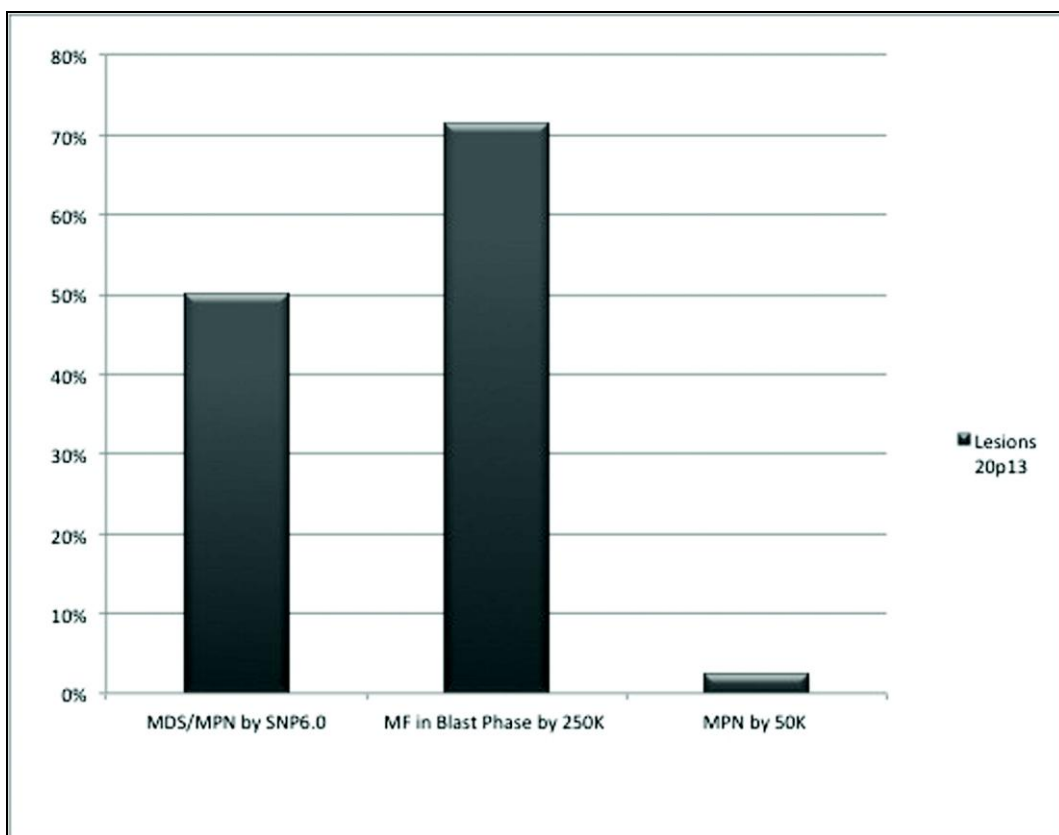


Figure 10. *Recurrence of 20p13 in a panel of myeloproliferative neoplasms.* Basing on *in silico* analysis, 20p13 abnormalities were recorded in a significant proportion of MPN. On the x-axis, three different datasets are reported, based on different microarray technology (Affymetrix SNPs-array 6.0, Affymetrix Human Mapping 250K Nsp, and Affymetrix Human Mapping 50K Array Xba 240, respectively).

4.3 TaqMan Copy Number assay of MAR

In order to validate the results obtained with the SNP-A, we used the TaqMan Copy Number Assay using a specific probe overlapping under the CNV of interest on 20p13 cytoband (Hs04039030_cn; Applied Biosystems; Foster City, CA). Specifically, we studied 10/11 tumor samples, in which the MAR appeared to be involved, and for which residual DNA was available. Furthermore, we analyzed 6 DNA control samples, including DNA from 1 healthy donor as well as DNA from matched non neoplastic T lymphocytes of 5 patients. Additionally we tested the target copy number amplification on a human genomic DNA control (Reference Genomic DNA, 103, Affymetrix, Santa Clara, CA, USA). The analysis was performed in quadruplicate and three times in three different days for further control.

High inter-assay and intra-assay reproducibility was recorded. In particular, analogue results were obtained in the replicates and consistent results were found within each repetition with a $\sigma\Delta Ct$ below 0,15. Overall, the assay confirmed the SNP-A results, with a consistency of 70%. Specifically, we detected the target copy number to be amplified in 7/10 patients (Figure 11).

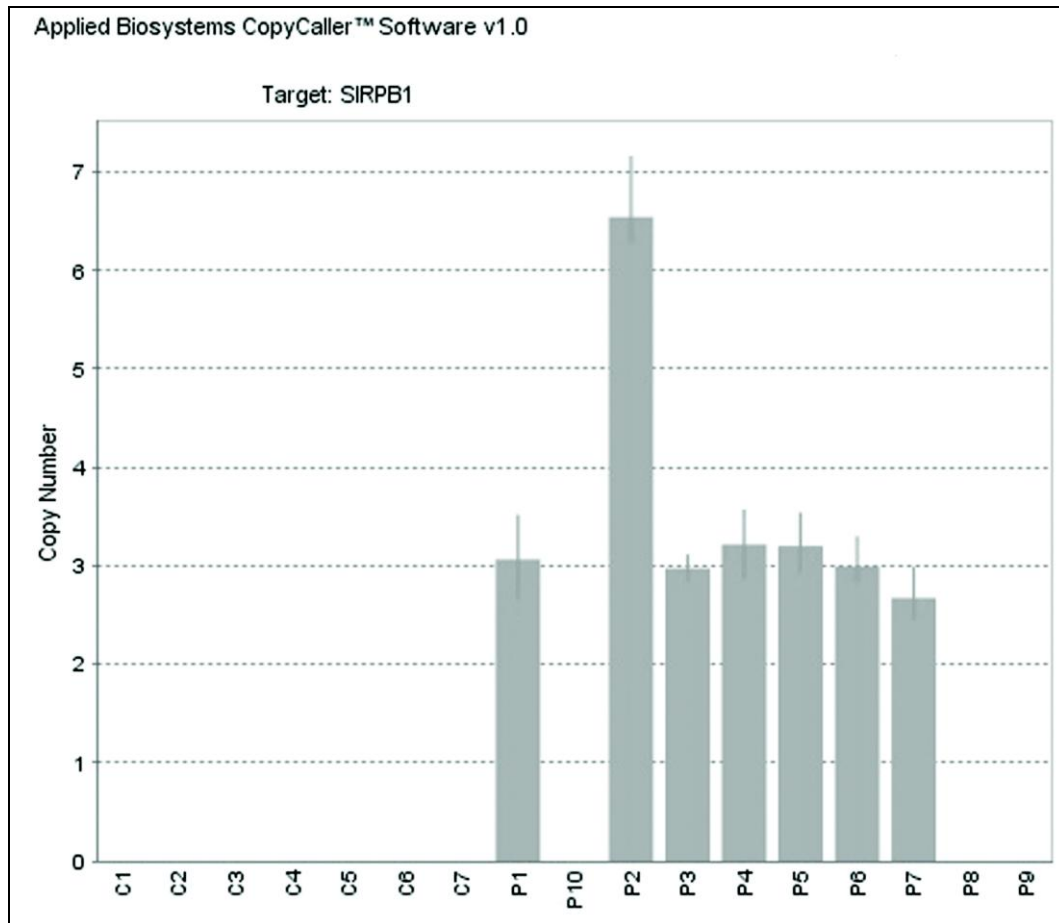


Figure 11. Validation of MAR abnormalities TaqMan Copy Number Assay. Each column represents a single genome (P=patient; C= control). Bars correspond to standard deviations. On the y axis, detected copy number values are presented. C1= gDNA from Affymetrix; C2=healthy donor; C3-C7=patients matched DNA from non neoplastic cells; P1-P10=patients.

4.4 SIRPB1 protein detection

Following the detection of a 20p13 amplification overlapping the *SIRPB1* gene, we tested the expression of SIRPB1 protein by immunohistochemistry on 5 PMF cases characterized by 20p13 amplification and on 5 PMF cases not showing the molecular event. Interestingly, we found a different expression of SIRPB1 between the two groups of PMF cases (Figure 12), as the protein proved to be diffusely expressed in myeloid cells, megakaryocytes, and macrophages/dendritic cells in cases with *SIRPB1* amplification, while it was almost exclusively

expressed by macrophages/dendritic cells and on the surface of scattered myeloid cells in PMF cases not showing 20p13 abnormalities (Figure 12). In PMF cases characterized by 20p13 amplification, SIRPB1 expression in myeloid and megakaryocytic cells showed a combined cytoplasmatic and membrane pattern (Figure 12 A-B).

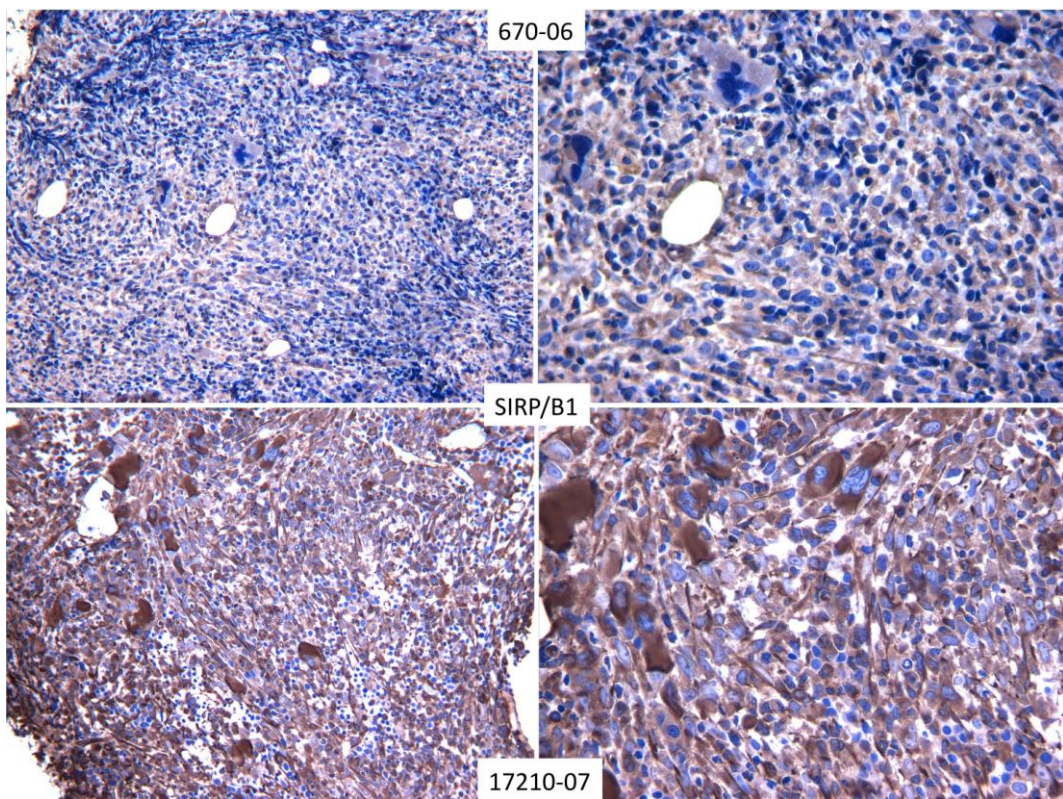


Figure 12 A. SIRPB1 expression in PMF. Patient numbered 670-06 without 20p13 amplification shows a basal SIRPB1 expression. On the contrary, patient numbered 17210-07 with 20p13 amplification presents a consistent SIRPB1 expression.

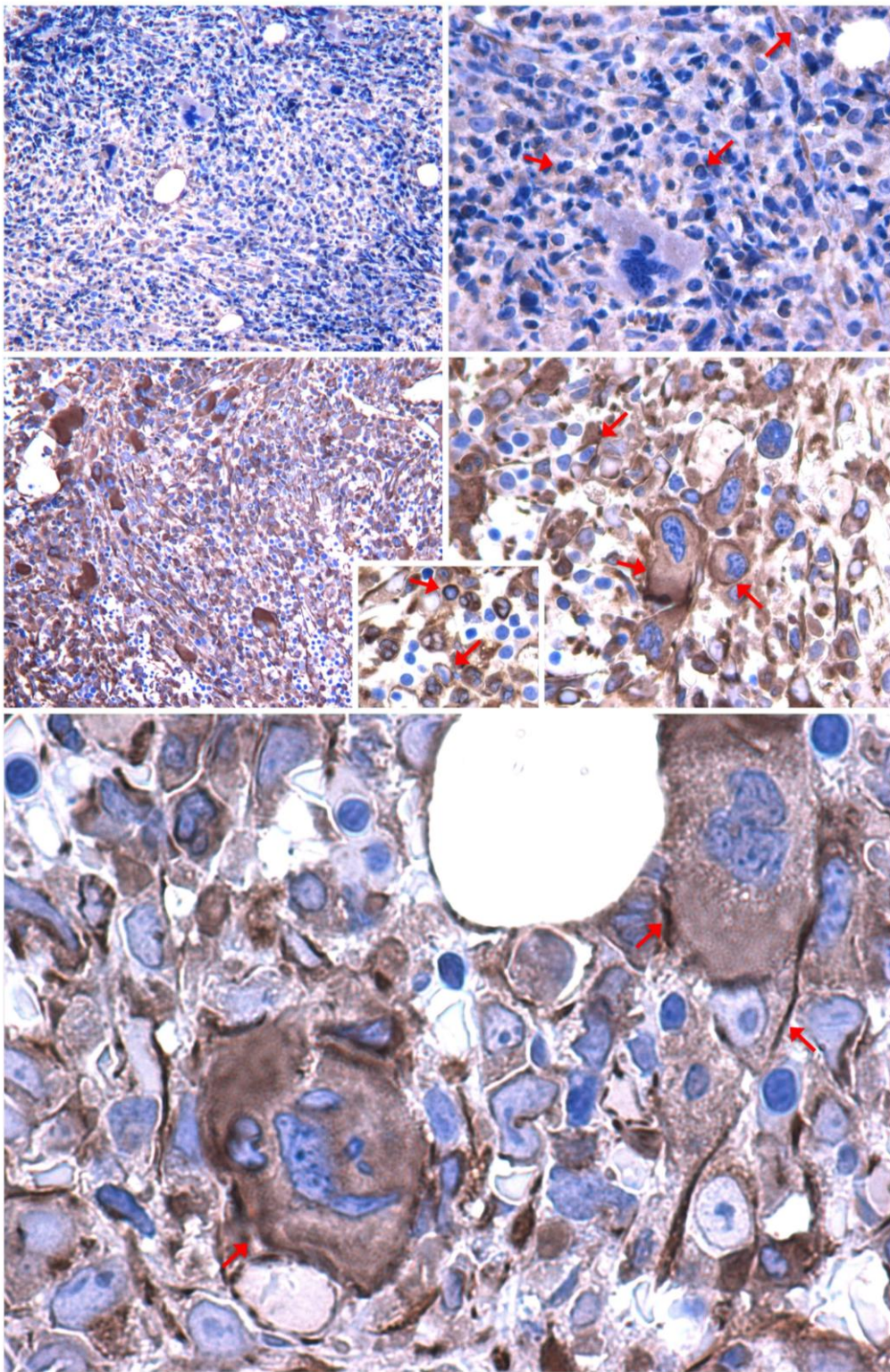


Figure 12 B. Images at higher resolution of patients with 20p13 amplification and a strong SIRPB1 expression as shown by red arrows.

5. DISCUSSION

In this study, we aimed to identify novel cytogenetic abnormalities in PMF patients by using high-density SNP-A. Previous studies showed the ability of such technique in detecting novel genetic lesions in both myeloid and lymphoid disorders [82, 86]. In particular, SNP-A profiling retains several advantages if compared to metaphase cytogenetics (MC), including the ability to detect lesions even when cells are not actively dividing, and definitely higher sensitivity and capacity to detect aUPD. Noteworthy, the latter two features also distinguish SNP-A analysis from CGH. On the other hand, a major limitation of SNP-A, in comparison to MC, is its inability to recognize chromosomal translocations [84].

Indeed, with this approach we were able to detect a remarkable number of lesions (2,765 CNVs) including aUPD, deletion and amplification, many of which (992/2,765; 36%) turned out to be previously uncovered. Notably, according to our study design, all copy number changes detected by SNP-A paired analysis in the training set, were then validated in a test set. Specifically, the correspondence between the two groups was significant ($p=0.0053$), with 44% of lesions confirmed in both (Figure 5 B).

Concerning the lesions previously associated with MPN, we identified del(13q), del(20q), and abnormalities on chromosome 1, +9 +8 and del(5q); moreover, we observed regions of aUPD on chromosome 9 at *JAK2* locus [67, 70], on chr13q, and at loci corresponding to *MPL*, *CBL* and *RBI* genes [13, 87]. Importantly, as internal validation, we also recognized two lesions previously detected by MC in our cohort (namely del(5q) and trisomy 8). Thus, SPN-A karyotyping was confirmed to be an effective tool for the identification of both cryptic and larger genomic abnormalities.

As far as newly identified aberrations were concerned, we focused on the lesions that recurred at higher frequency in our series, thus being candidate to retain a potential pathogenic role. Interestingly, as indicated by other studies using less sensitive techniques [27], PMF was confirmed to have heterogeneous lesions at cytogenetic level. Actually, all patients presented with a complex karyotype, with a minimum number of CNV/patient of 188. Remarkably, however, when a paired analysis was performed in the training set, no lesion was consistently

present in all cases, nor in up to 60% of them. Among 50% of patients, was found a lesion already reported in literature and for this reason no further investigated.

On the other hand, a few new alteration were found in 40% of patients. Among them, we focused on an amplification affecting the cytoband 20p13, as the most frequent in the test set. The overall prevalence in patients of such CNV was of 55%.

To better understand the context of this lesion we primarily extend our attention to the adjacent areas, in the 20p13 cytoband.

Interestingly, we found that all the patients expected one, (19/20) have at least one alteration (amplification, deletion or aUPD) occurring in the 20p13 cytoband.

Subsequently, we assessed the prevalence of 20p13 abnormalities in an independent validation data set. Specifically, we studied a group of 60 myeloid tumours, basing on previously published data which were available online. Remarkably, we confirmed the presence of lesions affecting 20p13 cytoband in 7/14 MDS/MPN patients, 5/7 MF patients and 1/39 MPN patient. Of note, this finding was then consistent with the concept that different myeloid malignancies often share common genetic abnormalities [88, 89].

Specifically, 20p13 gains were recently described in MDS [80], though occurring at very low frequency. Nevertheless, in general, other loci on chromosome 20 have been found to be involved in myeloid malignancies, though the implicated genes and the exact consequences are poorly known [79, 81].

Finally, after exploring the lesions along the 20p13 cytoband, the above mentioned CNV occurring in the 55% of patients, was specifically, recognized as a minimally affected region (MAR).

The MAR was an amplification of DNA extending for 9,911 bps, in correspondence to the *SIRPB1* gene.

Thus, first of all we validated the detection of such lesion by a specific copy number real time PCR assay. We successfully confirmed its presence but unfortunately, we couldn't proceed with a further characterization at genomic level, due to the lack of residual DNA.

Subsequently we test if the copy number amplification within *SIRPB1* gene could be eventually reflected at protein level, by SIRPB1 protein deregulation. Interestingly, according to this hypothesis, we found the SIRPB1 protein remarkably more expressed in the PMF patients presenting the copy number amplification than in patients without it.

The encoded protein is a member of the signal-regulatory-protein (SIRP) family, and also belongs to the immunoglobulin superfamily. SIRP family members are receptor-type transmembrane glycoproteins known to be involved in the regulation of receptor tyrosine kinase-coupled signaling processes.

Specifically, SIRPB1 protein is expressed in monocytes and myeloid dendritic cells and associates with the signal adaptor DAP12 on the cell surface. Several studies conducted *in vitro*, have demonstrated that the complex SIRPB1/DAP12 is involved in the recruitment of the protein tyrosine kinase Syk, in a marked activation of MAPK and MEK kinases and in promotion of cell proliferation [90, 91]. Additionally this complex has been found to promote the phagocytosis by macrophages and the migration of neutrophils [91, 92].

Thus, SIRPB1 is a new DAP12-associated receptor involved in the activation and proliferation of myeloid cells[93, 94].

A deregulated SIRPB1 expression and/or function might eventually be implicated in aberrant myeloid cell proliferation and survival in PMF cases with MAR. This hypothesis is also suggested by the detection of a diffuse SIRPB1 expression on myeloid cells of PMF cases characterized by 20p13 amplification. In this setting, the combined cytoplasmatic and membrane expression pattern of SIRPB1 might be suggestive of the *SIRPB1* synthesis exceeding its membrane exposure possibly owing to the limiting DAP12 expression. Overall, our results identify an amplification in correspondence of SIRPB1 as potentially involved in PMF and represent a starting point for further studies aimed to identify the actual role of these events in the PMF pathogenesis.

In conclusion, we detected, for the first time to the best of our knowledge, a recurrent lesion involving 20p13 and *SIRPB1* in PMF patients. Further studies are definitely warranted in order to 1) deeply characterize the genomic lesion; 2) assess the possible pathogenetic role of 20p13 amplification and *SIRPB1* over-expression; 3) assess the prognostic value of such lesion.

APPENDIX A

Validation *in silico*

For target validation *in silico* we downloaded two data sets of Affymetrix CEL files publicly available in the GEO database (<http://www.ncbi.nlm.nih.gov/geo/>). We used the GEO accession number GSE19647 (<http://www.ncbi.nlm.nih.gov/gds/?term=GSE19647>) and downloaded from this data set a total of 46 CEL file of patients affected by MF, 7 of them were studied by Affymetrix 250k SNP-A with the following sample number: GSM489831, GSM489832, GSM489834, GSM489835, GSM489836, GSM489837, GSM489838 while the other 39 by Affymetrix 50K SNP-A: SM489870, GSM489871, GSM489873, GSM489874, GSM489875, GSM489876, GSM489877 GSM489882, GSM489883, GSM489884 GSM489885 GSM489886 GSM489887 GSM489889 GSM489890 GSM489891 GSM489892 GSM489893 GSM489894 GSM489895 GSM489896 GSM489897 GSM489898 GSM489899 GSM489901 GSM489902 GSM489903 GSM489904 GSM489905 GSM489906 GSM489907 GSM489908 GSM489909 GSM489910 GSM489911 GSM489912 GSM489913 GSM489914 GSM489915 [66].

Moreover we downloaded from another data set, with accession number GSE21991 (<http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE21991>), 14 patients affected by MDS/MPD analysed by the our same array, Genome-Wide SNP array 6.0: GSM546489, GSM546867, GSM546868, GSM546869, GSM546870, GSM546871, GSM546872, GSM546873. GSM546874, GSM546875, GSM546886, GSM546887, GSM546888, GSM546890 [77].

APPENDIX B

	<i>bands</i>	<i>length</i>	<i>CNV</i>	<i>Gene</i>	<i>Chr</i>	<i>bands</i>	<i>length</i>	<i>CNV</i>	<i>Gene</i>
1	1p12	9437	D	ZNF697 (-)	9	9p13.3	31274	D	SMU1 (-)
1	1p13.1	11209	A	CASQ2 (-)	9	9p13.3 - 9p13.2	2989672	A	DNAJB5 (+)
1	1p13.2	3859	A	KCND3 (-)	9	9p21.1 - 9p13.3	1282508	A	TAFIL (-)
1	1p13.2	24646	A	TRIM33 (-)	9	9p21.2	10397	A	C9orf82 (-)
1	1p13.2	13150	D	CTTNBP2NL (+)	9	9p21.2	29099	A	TUSC1 (-)
1	1p13.3	5588	A	GSTM5 (+)	9	9p21.2	121904	A	TUSC1 (-)
1	1p13.3	11662	A	GSTM1 (+)	9	9p21.2	218225	A	TUSC1 (-)
1	1p13.3	117663	A	KCNC4 (+)	9	9p21.2	220910	A	TUSC1 (-)
1	1p13.3	14998	D	FAM102B (+)	9	9p21.2	10397	D	C9orf82 (-)
1	1p13.3	24808	D	WDR47 (-)	9	9p21.2	29099	D	TUSC1 (-)
1	1p13.3	80496	D	NBPF6 (+)	9	9p21.2 - 9p21.1	5372157	A	C9orf72 (-)
1	1p21.1	29110	A	AMY2A (+)	9	9p21.3	4333	A	IFNA5 (-)
1	1p21.1	12159	D	PRMT6 (+)	9	9p21.3	5510	A	CDKN2B (-)
1	1p21.1	12497	D	COL11A1 (-)	9	9p21.3	12285	A	TUSC1 (-)
1	1p22.2	4202	D	LMO4 (+)	9	9p21.3	12751	A	ELAVL2 (-)
1	1p22.3	1898	A	LMO4 (+)	9	9p21.3	108273	A	IFNA14 (-)
1	1p22.3	23320	D	SYDE2 (-)	9	9p21.3	213835	A	ELAVL2 (-)
1	1p31.1	1114	A	NEGR1 (-)	9	9p21.3	441880	A	MIR548H2 (+)
1	1p31.1	1704	A	NEGR1 (-)	9	9p21.3	704424	A	MIR31 (-)
1	1p31.1	3033	A	NEGR1 (-)	9	9p21.3	1128303	A	CDKN2B (-)
1	1p31.1	10469	A	NEGR1 (-)	9	9p21.3	1610451	A	ELAVL2 (-)
1	1p31.1	27965	A	NEGR1 (-)	9	9p21.3	5510	D	CDKN2B (-)
1	1p31.1	10469	D	NEGR1 (-)	9	9p21.3	12285	D	TUSC1 (-)
1	1p31.3	6425	A	INADL (+)	9	9p21.3 - 9p21.2	752779	A	TUSC1 (-)
1	1p31.3	13006	A	IL23R (+)	9	9p22.1	13245	A	SLC24A2 (-)
1	1p31.3	20892	D	INADL (+)	9	9p22.1	15175	A	FAM154A (-)
1	1p32.1	20300	D	MYSM1 (-)	9	9p22.1	727327	A	SCARNA8 (-)
1	1p32.3	19870	A	FAM151A (-)	9	9p22.1 - 9p21.3	1062244	A	SLC24A2 (-)
1	1p32.3	43917	A	DHCR24 (-)	9	9p22.2	3276	A	SH3GL2 (+)
1	1p32.3	6071	D	USP24 (-)	9	9p22.2	11126	A	ADAMTSL1 (+)
1	1p33	1293434	aUPD	CMPK1 (+)	9	9p22.2	407132	A	SH3GL2 (+)
1	1p33 - 1p32.3	3344446	aUPD	ELAVL4 (+)	9	9p22.2	902009	A	CNTLN (+)
1	1p34.1	33320	A	ERI3 (-)	9	9p22.2 - 9p22.1	681282	A	ADAMTSL1 (+)
1	1p34.1 - 1p33	2105884	aUPD	C1orf223 (+)	9	9p22.3 - 9p22.2	1394307	A	C9orf93 (+)
1	1p34.2	1297679	aUPD	EDN2 (-)	9	9p23	29382	A	TYRP1 (+)
1	1p34.2	41020	D	FOXJ3 (-)	9	9p23	2982	A	PTPRD (-)
1	1p34.2 - 1p34.1	1935643	aUPD	KDM4A (+)	9	9p23	14215	A	PTPRD (-)
1	1p34.3	12216	A	GJB5 (+)	9	9p23	21254	A	PTPRD (-)
1	1p34.3	41601	A	POU3F1 (-)	9	9p23	244292	A	PTPRD (-)
1	1p34.3	4802	A	RRAGC (-)	9	9p23	380898	A	PTPRD (-)
1	1p34.3	20406	A	RRAGC (-)	9	9p23	828198	A	TYRP1 (+)
1	1p34.3	3494858	aUPD	NCDN (+)	9	9p23	2606882	A	C9orf150 (+)
1	1p34.3 - 1p34.2	2156873	aUPD	MYCBP (-)	9	9p23	2982	D	PTPRD (-)
1	1p35.1 - 1p34.3	1190377	aUPD	HMGB4 (+)	9	9p23	14215	D	PTPRD (-)
1	1p35.2	35891	D	TINAGL1 (+)	9	9p23	21254	D	PTPRD (-)

1	1p35.2 - 1p35.1	1686364	aUPD	LOC100128071 (-)	9	9p23	25003	D	PTPRD (-)
1	1p35.3	57092	A	EPB41 (+)	9	9p23 - 9p22.3	1430422	A	LOC389705 (+)
1	1p35.3	38526	D	AHDC1 (-)	9	9p24.1	3459079	A	INSL4 (+)
1	1p35.3 - 1p35.2	2642787	aUPD	EPB41 (+)	9	9p24.1 - 9p23	1360180	A	PTPRD (-)
1	1p36.11	6175	A	RHD (+)	9	9p24.2	2646	A	KIAA0020 (-)
1	1p36.11	20137	A	RHD (+)	9	9p24.2	4388	A	C9orf70 (+)
1	1p36.11	6175	D	RHD (+)	9	9p24.2	4505	A	C9orf70 (+)
1	1p36.11	13682	D	RHD (+)	9	9p24.2	274843	A	RFX3 (-)
1	1p36.11	20137	D	RHD (+)	9	9p24.2	733879	A	C9orf70 (+)
1	1p36.11	34537	D	RHD (+)	9	9p24.2	4388	D	C9orf70 (+)
1	1p36.11	43513	D	CLIC4 (+)	9	9p24.2 - 9p24.1	1042317	A	AK3 (-)
1	1p36.11 - 1p35.3	3682935	aUPD	FAM76A (+)	9	9p24.3	2926	A	KANK1 (+)
1	1p36.12	13159	A	RAP1GAP (-)	9	9p24.3	19476	A	DOCK8 (+)
1	1p36.12	44992	A	RAP1GAP (-)	9	9p24.3	25795	A	SMARCA2 (+)
1	1p36.12	1833	A	RAP1GAP (-)	9	9p24.3	275384	A	C9orf66 (-)
1	1p36.12	13159	A	RAP1GAP (-)	9	9p24.3	315090	A	KANK1 (+)
1	1p36.12	1024767	aUPD	VWA5B1 (+)	9	9p24.3	941117	A	DMRT3 (+)
1	1p36.12	28889	D	EIF4G3 (-)	9	9p24.3	19476	D	DOCK8 (+)
1	1p36.12 - 1p36.11	2711784	aUPD	MYOM3 (-)	9	9p24.3	25795	D	SMARCA2 (+)
1	1p36.13	6485	A	UQCRHL (-)	9	9p24.3 - 9p24.2	1367788	A	SMARCA2 (+)
1	1p36.13	73304	A	NBPF1 (-)	9	9q12	3087	A	PGM5P2 (-)
1	1p36.13	1037424	aUPD	ATP13A2 (-)	9	9q12	6373	A	PGM5P2 (-)
1	1p36.13	73403	D	NBPF1 (-)	9	9q12	7628	A	FOXD4L5 (-)
1	1p36.13 - 1p36.12	1316741	aUPD	MRT04 (+)	9	9q12	8148	A	FOXD4L5 (-)
1	1p36.22 - 1p36.13	7246036	aUPD	SLC25A33 (+)	9	9q12	11633	A	PGM5P2 (-)
1	1p36.23	79986	A	CAMTA1 (+)	9	9q12	1192673	A	CBWD6 (-)
1	1p36.23	2786	D	ENO1 (-)	9	9q12	7628	D	FOXD4L5 (-)
1	1p36.31 - 1p36.23	2504913	aUPD	PHF13 (+)	9	9q12	8148	D	FOXD4L5 (-)
1	1p36.32	8753	A	PANK4 (-)	9	9q12 - 9q21.11	1066555	A	FOXD4L3 (+)
1	1p36.32	1908846	aUPD	MEGF6 (-)	9	9q21.11	6625	A	PRKACG (-)
1	1p36.32	19344	D	PANK4 (-)	9	9q21.11	16505	A	FAM189A2 (+)
1	1p36.32 - 1p36.31	1999667	aUPD	HES3 (+)	9	9q21.11	72744	A	APBA1 (-)
1	1p36.33 - 1p36.32	1459536	aUPD	GABRD (+)	9	9q21.11	179896	A	APBA1 (-)
1	1q21.1	49387	A	NOTCH2NL (+)	9	9q21.11	487326	A	TJP2 (+)
1	1q21.2	23979	A	LOC388692 (+)	9	9q21.11	490646	A	FAM122A (+)
1	1q21.2	99154	A	NBPF16 (+)	9	9q21.11	6625	D	PRKACG (-)
1	1q21.2	31658	D	FCGR1C (+)	9	9q21.11	16505	D	FAM189A2 (+)
1	1q21.3	5617	A	LCE1D (+)	9	9q21.11 - 9q21.12	599535	A	PTAR1 (-)
1	1q21.3	33473	A	LCE3C (+)	9	9q21.12 - 9q21.13	3754789	A	C9orf57 (-)
1	1q21.3	22773	D	POGZ (-)	9	9q21.13	1780283	A	C9orf40 (-)
1	1q23.1	25873	A	MIR555 (-)	9	9q21.13	2695	D	GCNT1 (+)
1	1q23.1	29491	D	LOC645676 (+)	9	9q21.13 - 9q21.2	807555	A	RPSAP9 (+)
1	1q23.2	8617	D	OR6Y1 (-)	9	9q21.2	15032	A	LOC100286938 (-)
1	1q24.2	34249	A	MIR921 (-)	9	9q21.2	444589	A	FOXB2 (+)
1	1q24.2	18396	D	FMO9P (+)	9	9q21.2	15032	D	LOC100286938 (-)
1	1q24.3	37955	A	NME7 (-)	9	9q21.2 - 9q21.31	1296206	A	VPS13A (+)

1	1q25.1	10244	D	ZBTB37 (+)	9	9q21.31 - 9q21.32	3404889	A	TLE4 (+)
1	1q25.1	20035	D	BAT2L2 (+)	9	9q21.32	16211	A	FLJ43950 (+)
1	1q25.2	6294	A	RABGAP1L (+)	9	9q21.32	1406912	A	FLJ46321 (+)
1	1q25.2	16036	A	GPR52 (+)	9	9q21.32 - 9q21.33	1798681	A	RMI1 (+)
1	1q25.3	24385	A	LHX4 (+)	9	9q21.33	10127	A	AGTPBP1 (-)
1	1q25.3	4875	D	RALGPS2 (+)	9	9q21.33	16047	A	AGTPBP1 (-)
1	1q25.3	40623	D	DHX9 (+)	9	9q21.33	43486	A	AGTPBP1 (-)
1	1q31.1	18341	A	FAM5C (-)	9	9q21.33	550948	A	AGTPBP1 (-)
1	1q31.3	8611	A	CFHR1 (+)	9	9q21.33	53612	D	AGTPBP1 (-)
1	1q31.3	10691	A	CFHR3 (+)	9	9q21.33 - 9q22.2	3213991	A	CTSL1 (+)
1	1q31.3	11484	A	KCNT2 (-)	9	9q22.2	2446	A	C9orf47 (+)
									LOC100129066
1	1q31.3	25086	A	ASPM (-)	9	9q22.2	6216	A	(+)
1	1q31.3	26720	A	B3GALT2 (-)	9	9q22.2	705733	A	SEMA4D (-)
									LOC100129066
1	1q31.3	31625	A	CFH (+)	9	9q22.2 - 9q22.31	840310	A	(+)
1	1q31.3	37552	A	CFHR3 (+)	9	9q22.31	26549	A	SYK (+)
1	1q31.3	1236	D	KCNT2 (-)	9	9q22.31	182570	A	SYK (+)
1	1q31.3	13178	D	KCNT2 (-)	9	9q22.31 - 9q22.32	3112029	A	C9orf129 (-)
1	1q32.1	20638	D	ATP6V1G3 (-)	9	9q22.32	49455	D	HIATL1 (+)
1	1q32.1	21167	D	KIF14 (-)	9	9q22.32 - 9q22.33	1384214	A	MIR24-1 (+)
1	1q32.2	30888	A	CDK18 (+)	9	9q22.33	1414823	A	CDC14B (-)
1	1q42.12	15745	A	CNIH4 (+)	9	9q22.33	66926	D	HIATL2 (-)
1	1q42.12	15990	D	DEGS1 (+)	9	9q22.33 - 9q31.1	1976348	A	XPA (-)
1	1q42.12	87271	D	TP53BP2 (-)	9	9q31.1	32489	A	CYL2 (+)
1	1q42.13	51151	A	RNF187 (+)	9	9q31.1	4053765	A	TGFBR1 (+)
1	1q42.13	51916	A	WNT9A (-)	9	9q31.1	32489	D	CYL2 (+)
1	1q42.2	11374	A	TSNAX-DISC1 (+)	9	9q31.1 - 9q31.2	2437110	A	OR13D1 (+)
1	1q42.2	3570	D	URB2 (+)	9	9q31.2 - 9q31.3	2117944	A	FKTN (+)
1	1q42.2	10664	D	TSNAX-DISC1 (+)	9	9q31.3	1636349	A	C9orf6 (+)
1	1q42.2	33889	D	GALNT2 (+)	9	9q31.3 - 9q32	2927525	A	AKAP2 (+)
1	1q43	5768	A	LOC339535 (-)	9	9q32	156146	A	ROD1 (-)
1	1q43	7696	D	ACTN2 (+)	9	9q32 - 9q33.1	1597100	A	CDC26 (-)
1	1q44	1918	A	LOC731275 (-)	9	9q33.1	92373	A	COL27A1 (+)
1	1q44	2015	A	SCCPDH (+)	9	9q33.1	268458	A	DBC1 (-)
1	1q44	79355	A	LOC731275 (-)	9	9q33.1	465792	A	SNORA70C (+)
1	1q44	89730	A	LOC731275 (-)	9	9q33.1	1002177	A	COL27A1 (+)
1	1q44	19734	D	AHCTF1 (-)	9	9q33.1	1536549	A	TLR4 (+)
1	1q44	23653	D	EXO1 (+)	9	9q33.1	1537552	A	TRIM32 (+)
1	1q44	34202	D	KIF26B (+)	9	9q33.1	21402	D	TLR4 (+)
1	1q44	43592	D	TFB2M (-)	9	9q33.1 - 9q33.2	802984	A	DBC1 (-)
1		28350	A	OR2T10 (-)	9	9q33.2 - 9q33.3	3495662	A	RBM18 (-)
1		442531	A	SH3BP5L (-)	9	9q33.3	139740	A	NEK6 (+)
1	1q23.2	1459	D	OR6Y1 (-)	9	9q33.3	6592	A	CRB2 (+)
1	1p36.31	25352	A	ACOT7 (-)	9	9q33.3	21997	A	FAM125B (+)
1	1p13.3	13521	D	PRMT6 (+)	9	9q33.3	863477	A	DENND1A (-)
1	1p22.3	15154	A	LMO4 (+)	9	9q33.3	1794220	A	SCAI (-)
1	1q23.2	16944	A	OR6Y1 (-)	9	9q33.3 - 9q34.11	3142322	A	ST6GALNAC4

1	1p21.1	29739	D	COL11A1 (-)	9	9q34.11	87816	A	C9orf106 (+)
1	1q23.3	1778	A	C1orf110 (-)	9	9q34.11 - 9q34.13	1377116	A	TOR1B (+)
1	1q32.1	28175	A	IGFN1 (+)	9	9q34.13	39373	D	ABL1 (+)
1	1p21.3	11156	A	RWDD3 (+)	9	9q34.13 - 9q34.2	2190109	A	UCK1 (-)
1	1q25.1	23422	D	SLC9A11 (-)	9	9q34.2	1047	A	SURF6 (-)
1	1p35.2	48030	D	PRO0611 (+)	9	9q34.2	3803	A	SURF6 (-)
1	1p36.12	69481	D	PLA2G2A (-)	9	9q34.2	4779	A	SURF6 (-)
1	1p32.3	29393	A	OSBPL9 (+)	9	9q34.2	390273	A	GTF3C5 (+)
1	1q32.2	14832	A	CDK18 (+)	9	9q34.2	393125	A	C9orf96 (+)
1	1p32.3	49596	A	LRP8 (-)	9	9q34.2	1047	D	SURF6 (-)
1	1q25.1	35240	D	DNM3 (+)	9	9q34.2	4514	D	SURF6 (-)
1	1q21.3	19526	A	POGZ (-)	9	9q34.2 - 9q34.3	34410	A	SARDH (-)
1	1p34.2	16987	D	GUCA2B (+)	9	9q34.3	21881	A	KIAA0649 (+)
1	1p32.2	9227	A	MIR548D2 (-)	9	9q34.3	1567401	A	BRD3 (-)
1	1p33	44647	D	SPATA6 (-)	9	9q34.3 -	2881392	A	LCN6 (-)
1	1p36.11	51568	A	RUNX3 (-)	9	9p21.3	14066	A	ELAVL2 (-)
1	1q21.3	21867	A	THEM5 (-)	9	9p21.3	14066	D	ELAVL2 (-)
1	1p12	15530	D	PHGDH (+)	9	9q33.3	5174	A	CRB2 (+)
1	1p31.1	35938	D	SLC44A5 (-)	9	9q31.2	26508	A	FSD1L (+)
1	1p36.32	1798	A	LOC284661 (+)	9	9q31.2	26508	D	FSD1L (+)
1	1q25.3	26608	D	GLUL (-)	9	9q33.1	12764	A	SNORA70C (+)
1	1p34.2	25849	A	KCNQ4 (+)	9	9q32	10609	A	HSDL2 (+)
1	1q32.2	17736	D	DYRK3 (+)	9	9q32	10609	D	HSDL2 (+)
1	1q44	7199	A	AKT3 (-)	9	9q33.3	25809	A	CRB2 (+)
1	1p36.31	9660	D	NPHP4 (-)	9	9q22.32	68163	A	PTPDC1 (+)
1	1q22	38768	A	KCNN3 (-)	9	9q21.12	20627	A	SMC5 (+)
1	1q24.2	7605	A	LOC400794 (-)	9	9q21.12	20627	D	SMC5 (+)
1	1q31.3	5479	D	B3GALT2 (-)	9	9q31.3	7573	A	KLF4 (-)
1	1q23.2	12015	A	OR6N2 (-)	9	9q31.3	7573	D	KLF4 (-)
1	1q42.13	21957	D	SRP9 (+)	9	9q33.1	3397	A	TRIM32 (+)
1	1q32.1	21333	D	DENND1B (-)	9	9q34.2	2386	A	TSC1 (-)
1	1q32.2	10218	D	IKBKE (+)	9	9p23	13769	A	NFIB (-)
1	1q23.2	36577	D	CD5L (-)	9	9p13.3	40321	A	SMU1 (-)
1	1p31.3	15532	A	ATG4C (+)	9	9p21.3	4342	A	MIR491 (+)
1	1p36.12	25629	D	HNRNPR (-)	9	9q21.13	29928	A	RORB (+)
1	1q42.2	19948	A	URB2 (+)	9	9q21.13	29928	D	RORB (+)
1	1p31.1	16492	D	MGC27382 (+)	9	9q21.31	12832	A	PSAT1 (+)
1	1q25.3	14071	A	GLUL (-)	9	9q21.31	12832	D	PSAT1 (+)
1	1p31.3	20391	A	LEPROT (+)	9	9p21.2	2134	A	TUSC1 (-)
1	1p31.3	11076	D	SERBP1 (-)	9	9p21.2	2134	D	TUSC1 (-)
1	1p22.1	6955	A	CDC7 (+)	9	9q34.13	2462	A	PRDM12 (+)
1	1p36.13	65144	A	RCC2 (-)	9	9p23	10437	A	NFIB (-)
1	1p32.1	14902	A	TACSTD2 (-)	9	9q34.13	20755	A	PRDM12 (+)
1	1p33	29560	D	CYP4X1 (+)	9	9q21.13	14266	D	PCSK5 (+)
1	1q43	27197	D	GREM2 (-)	9	9p24.1	3637	A	JAK2 (+)
1	1q25.1	23951	D	DNM3 (+)	9	9p24.1	3637	D	JAK2 (+)
1	1p36.13	21758	A	ARHGEF10L (+)	9	9p24.1	7407	A	JAK2 (+)

1	1p32.2	13274	A	MIR548D2 (-)	9	9p24.1	7407	D	JAK2 (+)
1	1p31.1	31864	D	AK5 (+)	9	9q21.2	2503	A	PRUNE2 (-)
1	1q43	9273	A	RYR2 (+)	9	9q32	37663	A	ROD1 (-)
1	1p31.3	14965	D	ATG4C (+)	9	9q21.2	3458	A	PRUNE2 (-)
1	1p13.1	23391	A	ATP1A1 (+)	9	9p24.1	1954	A	JAK2 (+)
1	1q42.2	7731	A	SIPAIL2 (-)	9	9p23	1372	A	NFIB (-)
1	1p31.3	16174	A	PDE4B (+)	9	9q21.2	22613	A	PRUNE2 (-)
1	1q25.1	11579	A	C1orf105 (+)	9	9q22.32	11993	D	FBP1 (-)
1	1q25.3	3861	D	ABL2 (-)	9	9p13.3	21944	A	UBE2R2 (+)
1	1p36.11	14280	D	MAN1C1 (+)	9	9q33.1	9236	D	SNORA70C (+)
1	1p33	9445	A	AGBL4 (-)	9	9q33.2	1866	A	MIR147 (-)
1	1p33	27072	A	CYP4A11 (-)	9	9p21.2	13281	A	TUSC1 (-)
1	1p36.11	9590	D	GRHL3 (+)	9	9p21.2	13281	D	TUSC1 (-)
1	1p12	22376	D	VTCN1 (-)	9	9p24.1	30182	A	PTPRD (-)
1	1q32.1	21613	A	PTPRV (+)	9	9q22.31	13421	A	DIRAS2 (-)
1	1p22.3	7350	A	MCOLN2 (-)	9	9q22.33	6072	A	PTCH1 (-)
1	1p21.3	25623	D	DPYD (-)	9	9q22.33	2978	D	PTCH1 (-)
1	1p22.3	35598	D	HS2ST1 (+)	9	9p22.3	10779	A	PSIP1 (-)
1	1p36.13	18745	A	PAX7 (+)	9	9q22.33	3095	D	PTCH1 (-)
1	1q24.3	42800	D	KIFAP3 (-)	9	9q22.33	49298	A	PTCH1 (-)
1	1q43	9643	D	CHRM3 (+)	9	9q33.1	25375	A	DEC1 (+)
1	1q23.3	13390	A	VSIG8 (-)	9	9q22.31	291570	A	DIRAS2 (-)
1	1q43	15288	D	CHRM3 (+)	9	9q31.3	46284	A	EPB41L4B (-)
1	1p31.3	14772	D	JAK1 (-)	9	9q34.11	17291	D	DNM1 (+)
1	1q32.1	17855	D	NR5A2 (+)	9	9p21.1	24130	A	ACO1 (+)
1	1p22.2	18162	A	LRRC8D (+)	9	9q22.33	7202	A	PTCH1 (-)
1	1p34.1	12675	A	PTCH2 (-)	9	9q22.33	7202	D	PTCH1 (-)
1	1p21.3	26050	D	SNX7 (+)	9	9q21.11	22817	A	APBA1 (-)
2	2p11.1	2629	A	LOC654342 (-)	9	9q21.11	22817	D	APBA1 (-)
2	2p11.1	131379	A	FKSG73 (+)	9	9q22.31	14697	A	DIRAS2 (-)
2	2p11.1	163696	A	LOC654342 (-)	9	9q22.31	14697	D	DIRAS2 (-)
2	2p11.1	44791	D	FKSG73 (+)	9	9q21.13	10412	A	PCSK5 (+)
2	2p11.2	30077	A	LOC285074 (-)	9	9q21.13	10412	D	PCSK5 (+)
2	2p11.2	251976	A	NCRNA00152 (+)	9	9p24.2	11939	A	RFX3 (-)
2	2p11.2	11539	D	POLR1A (-)	9	9q31.1	12305	A	COL15A1 (+)
2	2p12	9832	A	SNAR-H (-)	10	10p11.1	23884	A	LOC399744 (+)
2	2p12	25600	A	LOC388965 (+)	10	10p11.21	1097	A	ZNF248 (-)
2	2p13.3	14195	A	MPHOSPH10 (+)	10	10p11.21	2796	A	ZNF248 (-)
2	2p16.1	10997	A	SMEK2 (-)	10	10p11.21	28174	D	ANKRD30A (+)
2	2p16.1	1788	D	RTN4 (-)	10	10p11.22	2937	D	C10orf68 (+)
2	2p16.2	17580	A	ASB3 (-)	10	10p12.1	1207	A	KIAA1217 (+)
2	2p16.2	4909	D	SPTBN1 (+)	10	10p12.1	33173	D	MYO3A (+)
2	2p16.3	2493	D	NRXN1 (-)	10	10p12.32	2099	A	PLXDC2 (+)
2	2p16.3	13139	D	NRXN1 (-)	10	10p12.33	11632	D	NSUN6 (-)
2	2p16.3	27759	D	FSHR (-)	10	10p13	7197	A	CUBN (-)
2	2p21	32158	A	HAAO (-)	10	10p13	12758	D	RSU1 (-)
2	2p21	37296	A	TTC7A (+)	10	10p14	6397	A	CUGBP2 (+)
2	2p22.2	9267	A	CRIM1 (+)	10	10p15.1	3416	A	PRKCQ (-)

2	2p22.3	9347	A	LTBP1 (+)	10	10p15.1	3470	A	LOC100216001 (-)
2	2p22.3	11086	A	CRIM1 (+)	10	10p15.1	5304	A	PRKCQ (-)
2	2p22.3	12912	A	RASGRP3 (+)	10	10p15.1	8701	A	PRKCQ (-)
2	2p22.3	23651	A	CRIM1 (+)	10	10p15.1	22779	A	ASB13 (-)
									LOC100216001
2	2p22.3	3913	D	CRIM1 (+)	10	10p15.1	6376	D	(-)
2	2p23.3	5988	A	OTOF (-)	10	10p15.3	2956	A	ADARB2 (-)
2	2p23.3	9892	A	ITSN2 (-)	10	10p15.3	19168	A	DIP2C (-)
2	2p23.3	56154	D	ATAD2B (-)	10	10p15.3	1944	D	ADARB2 (-)
2	2p24.1	26406	A	OSR1 (-)	10	10p15.3	5259	D	ADARB2 (-)
2	2p24.1	28952	A	OSR1 (-)	10	10p15.3	8488	D	PFKP (+)
2	2p24.1	2062	D	OSR1 (-)	10	10p15.3	9635	D	ADARB2 (-)
2	2p24.2	4857	A	KCNS3 (+)	10	10p15.3	16027	D	ADARB2 (-)
2	2p24.2	23636	A	KCNS3 (+)	10	10p15.3	57331	D	TUBB8 (-)
2	2p24.3	8612	A	FAM49A (-)	10	10q11.21	16944	A	ZNF33B (-)
2	2p25.2	3046	D	LOC400940 (+)	10	10q11.21	195657	A	LOC441666 (-)
2	2p25.2	8766	D	CMPK2 (-)	10	10q11.21	39530	D	LOC441666 (-)
2	2p25.3	1163	A	ALLC (+)	10	10q11.21	195657	D	LOC441666 (-)
2	2p25.3	1202	A	ALLC (+)	10	10q11.22	61390	A	GDF10 (-)
2	2p25.3	10099	A	ALLC (+)	10	10q11.22	1580	D	PPYR1 (+)
2	2p25.3	10684	A	ALLC (+)	10	10q11.22	10008	D	SYT15 (-)
2	2p25.3	15560	A	PXDN (-)	10	10q11.22	101731	D	FAM21C (+)
2	2p25.3	1163	D	ALLC (+)	10	10q11.23	14097	D	AGAP7 (-)
2	2p25.3	11284	D	TMEM18 (-)	10	10q21.1	1712	A	IPMK (-)
2	2q11.2	38264	D	ANKRD36B (-)	10	10q21.1	7696	D	ZWINT (-)
2	2q11.2	114341	D	ANKRD36 (+)	10	10q21.2	1385	A	SLC16A9 (-)
2	2q11.2	233732	D	LOC729234 (+)	10	10q21.3	1910	A	CTNNA3 (-)
2	2q12.3	79329	A	RGPD4 (+)	10	10q21.3	5431	A	CTNNA3 (-)
2	2q12.3	20532	D	UXS1 (-)	10	10q21.3	1910	D	CTNNA3 (-)
2	2q13	42034	A	ACOXL (+)	10	10q21.3	11888	D	CTNNA3 (-)
2	2q14.1	8174	A	DDX18 (+)	10	10q21.3	14398	D	TSPAN15 (+)
2	2q14.1	23484	A	DDX18 (+)	10	10q22.1	33778	A	COL13A1 (+)
2	2q14.1	65472	D	DPP10 (+)	10	10q22.1	88248	A	C10orf27 (-)
2	2q14.2	7929	A	GLI2 (+)	10	10q22.3	5105	A	DLG5 (-)
2	2q14.3	5794	A	HS6ST1 (-)	10	10q22.3	20330	A	C10orf11 (+)
2	2q14.3	18919	D	CNTNAP5 (+)	10	10q22.3	70338	A	KCNMA1 (-)
2	2q21.1	1523	A	HS6ST1 (-)	10	10q23.1	7748	D	NRG3 (+)
2	2q21.1	4344	A	LOC389033 (-)	10	10q23.31	18984	A	HTR7 (-)
2	2q21.1	17627	A	LOC389033 (-)	10	10q23.31	8795	D	KIF20B (+)
2	2q21.1	52315	D	LOC389033 (-)	10	10q23.31	19496	D	CH25H (-)
2	2q21.2	21073	D	MIR663B (-)	10	10q23.32	55025	D	MARCH5 (+)
2	2q21.2	22627	D	TUBA3D (+)	10	10q23.33	1909	A	CYP2C8 (-)
2	2q21.2	31181	D	NCRNA00164 (-)	10	10q23.33	1960	A	CYP2C8 (-)
2	2q21.3	48892	D	ZRANB3 (-)	10	10q23.33	2872	D	CYP2C8 (-)
2	2q22.1	21938	A	LRP1B (-)	10	10q23.33	3868	D	CYP2C8 (-)
2	2q22.1	7165	D	CXCR4 (-)	10	10q23.33	6878	D	CYP2C8 (-)
2	2q23.3	4030	A	RBM43 (-)	10	10q24.1	7449	A	SLIT1 (-)

2	2q23.3	25150	D	ARL6IP6 (+)	10	10q24.2	23858	A	C10orf28 (+)
2	2q31.1	3262	A	SPC25 (-)	10	10q24.31	20564	A	PAX2 (+)
2	2q31.1	4258	A	RAPGEF4 (+)	10	10q24.31	37938	D	C10orf75 (+)
2	2q31.1	5400	A	MIR1246 (-)	10	10q24.33	142225	A	SH3PXD2A (-)
2	2q31.1	15176	A	RAPGEF4 (+)	10	10q25.1	1328	D	SORCS1 (-)
2	2q31.1	7640	D	NOSTRIN (+)	10	10q25.1	8834	D	XPNPEP1 (-)
2	2q31.1	34244	D	UBR3 (+)	10	10q25.1	12506	D	SORCS1 (-)
2	2q31.2	44109	A	PDE11A (-)	10	10q26.13	3815	D	ATE1 (-)
2	2q31.3	9356	D	ZNF385B (-)	10	10q26.13	32216	D	METTL10 (-)
2	2q32.1	10549	A	ZNF804A (+)	10	10q26.2	25357	D	FANK1 (+)
2	2q32.1	7043	D	NUP35 (+)	10	10q26.3	6237	A	GLRX3 (+)
2	2q33.1	21897	A	PLCL1 (+)	10	10q26.3	7832	A	TCERGIL (-)
2	2q33.1	17716	D	BMP2 (+)	10	10q26.3	7858	A	GLRX3 (+)
2	2q33.1	107843	D	SNORD11 (+)	10	10q26.3	8068	A	MGMT (+)
2	2q33.3	27845	D	CREB1 (+)	10	10q26.3	10392	A	GLRX3 (+)
2	2q34	13919	A	PTH2R (+)	10	10q26.3	4964	D	JAKMIP3 (+)
2	2q35	62391	A	GMPPA (+)	10		80553	D	DUX4 (+)
2	2q36.1	22318	A	SLC4A3 (+)	10	10p11.21	1059	A	ZNF248 (-)
2	2q37.1	1406	A	ALPP (+)	10	10p11.21	11491	A	ZNF248 (-)
2	2q37.1	2170	A	ALPP (+)	10	10q25.2	22040	D	GPAM (-)
2	2q37.1	2530	A	ALPP (+)	10	10p15.3	6803	D	ADARB2 (-)
2	2q37.1	3158	D	SPP2 (+)	10	10q25.3	17501	D	HABP2 (+)
2	2q37.3	14098	A	MGC16025 (-)	10	10q26.12	16229	A	SEC23IP (+)
2	2q37.3	71942	A	CAPN10 (+)	10	10q23.33	30501	A	LGII (+)
2	2q37.3	26030	D	AGAP1 (+)	10	10q26.11	12857	D	CASC2 (+)
2		51797	A	LOC728323 (+)	10	10q26.11	8962	A	GRK5 (+)
2		5288	D	LOC728323 (+)	10	10p15.1	4828	A	KLF6 (-)
2	2p12	13488	A	REG3G (+)	10	10q23.31	4190	A	PTEN (+)
2	2q14.2	11893	A	GLI2 (+)	10	10q23.31	3017	A	PTEN (+)
2	2q36.3	4802	A	PID1 (-)	10	10q23.31	3017	D	PTEN (+)
2	2q24.3	5500	A	FIGN (-)	10	10q26.12	14171	D	PPAPDC1A (+)
2	2p24.1	22833	D	OSR1 (-)	10	10q25.1	33209	D	SORCS1 (-)
2	2q13	9246	D	SH3RF3 (+)	10	10q23.31	11710	D	PTEN (+)
2	2p14	15663	A	SPRED2 (-)	10	10q26.13	2064	D	FGFR2 (-)
2	2q14.1	22433	A	LOC654433 (+)	10	10q21.3	15284	D	CTNNA3 (-)
2	2p14	10808	A	ETAA1 (+)	10	10q26.13	10654	D	FGFR2 (-)
2	2q33.3	17416	D	KLF7 (-)	10	10q23.31	11509	A	KIF20B (+)
2	2q36.1	11553	D	EPHA4 (-)	10	10q26.13	19415	D	ATE1 (-)
2	2q12.1	21695	D	MAP4K4 (+)	10	10p15.1	6426	D	KLF6 (-)
2	2p25.1	1414	A	ASAP2 (+)	10	10q23.31	6140	A	PTEN (+)
2	2q14.2	18055	A	LOC84931 (-)	10	10q23.31	6140	D	PTEN (+)
2	2p25.1	12470	A	ASAP2 (+)	10	10q26.13	1583	D	FGFR2 (-)
2	2p16.2	15611	D	ASB3 (-)	10	10q23.31	4449	A	PTEN (+)
2	2p12	12803	D	LRRTM1 (-)	10	10q23.31	4449	D	PTEN (+)
2	2q34	9827	A	LOC29034 (+)	10	10q21.1	13008	A	IPMK (-)
2	2q33.1	14682	D	PLCL1 (+)	10	10q23.31	5766	A	PTEN (+)
2	2q31.2	26771	A	OSBPL6 (+)	10	10q23.31	5043	D	PTEN (+)
2	2q33.2	27214	D	NBEAL1 (+)	10	10p13	8473	A	FRMD4A (-)

2	2q36.3	29518	D	KIAA1486 (+)	10	10q11.21	1279	A	RET (+)
2	2q36.1	24984	D	EPHA4 (-)	10	10p11.21	26128	A	ANKRD30A (+)
2	2p21	23105	D	EPCAM (+)	10	10q11.21	20626	A	RET (+)
2	2q31.1	52121	A	HOXD10 (+)	10	10q23.33	36530	A	CYP2C9 (+)
2	2q37.3	21774	A	MGC16025 (-)	10	10q11.23	14501	D	SGMS1 (-)
2	2p25.1	6879	D	PDIA6 (-)	10	10q23.33	10414	D	CEP55 (+)
2	2q11.2	20490	D	REV1 (-)	10	10p14	15844	A	GATA3 (+)
2	2q13	18764	A	ACOXL (+)	10	10q26.13	3916	D	FGFR2 (-)
2	2p12	18550	A	LRRTM1 (-)	10	10q26.13	15542	A	FGFR2 (-)
2	2p23.1	16937	A	GALNT14 (-)	10	10q11.23	1741	D	SGMS1 (-)
2	2q14.1	23294	A	DPP10 (+)	10	10q11.21	1730	A	RET (+)
2	2q34	30267	D	LOC29034 (+)	10	10q25.1	26781	D	SORCS3 (+)
2	2q34	22977	A	MAP2 (+)	10	10q22.1	30709	A	PCBD1 (-)
2	2q34	21458	D	MAP2 (+)	10	10q26.11	23709	A	GRK5 (+)
2	2p23.1	11297	A	CAPN13 (-)	10	10q11.23	15511	D	C10orf72 (-)
2	2p24.1	18941	A	C2orf43 (-)	10	10q25.1	14962	D	SORCS1 (-)
2	2p25.1	18038	A	ID2 (+)	10	10q26.11	17335	A	EMX2 (+)
2	2p13.2	26064	D	EXOC6B (-)	10	10p14	44473	D	USP6NL (-)
2	2q14.2	16899	D	TFCP2L1 (-)	10	10p11.23	12390	A	WAC (+)
2	2q11.2	27893	D	VWA3B (+)	10	10q21.1	16282	A	TFAM (+)
2	2q24.1	21545	A	NR4A2 (-)	10	10q22.3	22892	D	RPS24 (+)
2	2q11.2	40096	A	VWA3B (+)	10	10q26.13	11702	A	GPR26 (+)
2	2p13.3	29689	A	DYSF (+)	10	10p11.22	11998	D	PARD3 (-)
2	2q22.1	32999	D	LRP1B (-)	10	10p11.23	17628	D	WAC (+)
2	2q36.2	24317	D	CUL3 (-)	10	10p14	9824	D	SFTA1P (-)
2	2q24.3	14612	A	B3GALT1 (+)	10	10q23.33	7060	D	ALDH18A1 (-)
2	2q22.2	45361	D	ARHGAP15 (+)	10	10q26.11	26291	D	C10orf119 (-)
2	2q33.1	15242	A	ANKRD44 (-)	10	10q26.11	22574	A	C10orf119 (-)
2	2q22.1	24539	D	LRP1B (-)	10	10q26.11	29655	A	C10orf46 (-)
2	2q34	16695	D	MAP2 (+)	11	11p11.12	14742	A	OR4A5 (-)
2	2q22.1	63015	D	SPOPL (+)	11	11p11.12	28968	A	OR4A5 (-)
2	2q14.3	7181	A	TSN (+)	11	11p11.2	1951	A	OR4A47 (+)
2	2q24.1	23071	D	GALNT5 (+)	11	11p11.2	4835	A	OR4A47 (+)
2	2q14.2	15453	D	EPB41L5 (+)	11	11p11.2	8181	A	OR4A47 (+)
2	2p16.1	20442	D	CCDC88A (-)	11	11p11.2	11116	A	OR4A47 (+)
2	2q13	23949	D	SEPT10 (-)	11	11p11.2	24884	A	EXT2 (+)
2	2q13	14245	D	POLR1B (+)	11	11p11.2	49425	A	CHST1 (-)
2	2q36.1	23660	D	SCG2 (-)	11	11p11.2	1951	D	OR4A47 (+)
2	2q24.3	19391	A	XIRP2 (+)	11	11p11.2	5161	D	OR4A47 (+)
2	2p16.3	18519	D	NRXN1 (-)	11	11p11.2	8181	D	OR4A47 (+)
2	2q31.1	8809	A	ITGA6 (+)	11	11p13	11410	A	WT1 (-)
2	2p24.3	10347	A	MYCNOS (-)	11	11p13	16899	D	DCDC1 (-)
2	2q34	13336	D	LANCL1 (-)	11	11p14.3	1878	A	LUZP2 (+)
2	2p11.2	29264	A	C2orf89 (-)	11	11p14.3	4804	A	LUZP2 (+)
2	2q34	28972	D	MYL1 (-)	11	11p14.3	5300	A	LUZP2 (+)
3	3p12.1	9363	D	CADM2 (+)	11	11p14.3	6920	A	LUZP2 (+)
3	3p12.1	42563	D	CADM2 (+)	11	11p14.3	6920	D	LUZP2 (+)
3	3p12.3	15214	A	ROBO2 (+)	11	11p14.3	11375	D	NELL1 (+)

3	3p12.3	423903	A	MIR1324 (+)	11	11p14.3	24021	D	LUZP2 (+)
3	3p12.3	16745	D	ROBO1 (-)	11	11p14.3	26028	D	SVIP (-)
3	3p12.3	22596	D	GBE1 (-)	11	11p15.1	9590	A	MRGPRX1 (-)
3	3p14.1	1220	A	FAM19A4 (-)	11	11p15.1	19407	A	KCNC1 (+)
3	3p14.1	19197	A	FAM19A4 (-)	11	11p15.1	21010	A	USH1C (-)
3	3p14.1	2004	D	MIR548A2 (+)	11	11p15.4	1441	A	TRPC2 (+)
3	3p14.2	7120	D	PTPRG (+)	11	11p15.4	2557	A	LOC650368 (+)
3	3p14.2	8841	D	FHIT (-)	11	11p15.4	3537	A	UBQLNL (-)
3	3p14.2	14035	D	FHIT (-)	11	11p15.4	4380	A	OR51A4 (-)
3	3p14.3	1938	D	C3orf51 (-)	11	11p15.4	5855	A	OR52N5 (-)
3	3p21.1	4149	A	SFMBT1 (-)	11	11p15.4	6911	A	SYT9 (+)
3	3p21.1	14340	D	SFMBT1 (-)	11	11p15.4	14539	A	OR52N5 (-)
3	3p21.31	3100	A	CCR5 (+)	11	11p15.4	22320	A	LMO1 (-)
3	3p21.31	23397	A	SACMIL (+)	11	11p15.4	29429	A	OR56A1 (-)
3	3p21.31	32395	A	CACNA2D2 (-)	11	11p15.4	218145	A	LOC650368 (+)
3	3p21.31	33214	A	CACNA2D2 (-)	11	11p15.4	14238	D	OR51Q1 (+)
3	3p21.31	72384	A	APEH (+)	11	11p15.4	14254	D	OR52N2 (+)
3	3p21.31	73760	A	CACNA2D2 (-)	11	11p15.5	41058	A	TMEM80 (+)
3	3p21.31	5718	D	XCR1 (-)	11	11q11	67136	A	OR4C6 (+)
3	3p21.31	8266	D	DOCK3 (+)	11	11q11	13092	D	OR5AS1 (+)
3	3p21.31	103604	D	SMARCC1 (-)	11	11q12.1	15745	A	OR9I1 (-)
3	3p22.1	4229	D	LYZL4 (-)	11	11q12.1	1126793	aUPD	FAM111B (+)
3	3p22.1	10190	D	ULK4 (-)	11	11q12.1	1202925	aUPD	CTNND1 (+)
3	3p22.1	15143	D	ULK4 (-)	11	11q12.1 - 11q12.2	1725023	aUPD	MS4A4A (+)
3	3p22.1	23390	D	LYZL4 (-)	11	11q12.2 - 11q12.3	2201657	aUPD	SLC3A2 (+)
3	3p22.1	25318	D	ULK4 (-)	11	11q12.3	20741	D	SLC22A25 (-)
3	3p22.1	48710	D	ULK4 (-)	11	11q13.1	2961301	aUPD	SLC22A20 (+)
3	3p22.3	16898	D	MIR128-2 (+)	11	11q13.1 - 11q13.2	1274397	aUPD	TMEM134 (-)
3	3p24.1	16578	D	TGFBR2 (+)	11	11q13.2	21077	D	CHKA (-)
3	3p24.1	25464	D	RBMS3 (+)	11	11q13.2 - 11q13.4	2968996	aUPD	SHANK2 (-)
3	3p24.3	8603	A	UBE2E2 (+)	11	11q13.4	17947	A	SHANK2 (-)
3	3p24.3	32878	A	KCNH8 (+)	11	11q13.4	1289507	aUPD	RELT (+)
3	3p24.3	19534	D	SGOL1 (-)	11	11q13.4	1527537	aUPD	SHANK2 (-)
3	3p24.3	26417	D	KCNH8 (+)	11	11q13.4	24480	D	PGM2L1 (-)
3	3p25.1	16478	D	RAF1 (-)	11	11q13.4 - 11q13.5	1436063	aUPD	SLCO2B1 (+)
3	3p25.3	7188	A	IRAK2 (+)	11	11q13.5 - 11q14.1	4109334	aUPD	PAK1 (-)
3	3p26.1	3633	A	GRM7 (+)	11	11q14.1	1389675	aUPD	FAM181B (-)
3	3p26.1	6759	D	GRM7 (+)	11	11q14.1 - 11q14.2	5416342	aUPD	PRSS23 (+)
3	3p26.1	8659	D	GRM7 (+)	11	11q14.2	9210	D	RAB38 (-)
3	3p26.1	10442	D	GRM7 (+)	11	11q14.2 - 11q21	5434225	aUPD	FOLH1B (+)
3	3p26.1	11743	D	GRM7 (+)	11	11q14.3	24794	A	FAT3 (+)
3	3p26.1	22575	D	GRM7 (+)	11	11q21 - 11q22.1	5988235	aUPD	ENDOD1 (+)
3	3p26.2	7081	A	LRRN1 (+)	11	11q22.1	1678979	aUPD	TRPC6 (-)
3	3p26.3	11403	A	CNTN6 (+)	11	11q22.1	14587	D	CNTN5 (+)
3	3p26.3	44691	A	CHL1 (+)	11	11q22.1	5639	D	CNTN5 (+)
3	3p26.3	13202	D	CNTN6 (+)	11	11q22.1	5819	D	CNTN5 (+)
3	3p26.3	19643	D	CNTN6 (+)	11	11q22.1	6201	D	TRPC6 (-)
3	3p26.3	35691	D	CHL1 (+)	11	11q22.1	9769	D	JRKL (+)

3	3q11.2	11100	A	DCBLD2 (-)	11	11q22.1	35373	D	TRPC6 (-)
3	3q11.2	25992	A	DCBLD2 (-)	11	11q22.2 - 11q22.3	2222125	aUPD	MMP1 (-)
3	3q11.2	3354	D	DCBLD2 (-)	11	11q22.3	3061	A	CWF19L2 (-)
3	3q12.3	1044	A	MIR548A3 (-)	11	11q22.3	3163	A	CWF19L2 (-)
3	3q12.3	10739	A	MIR548A3 (-)	11	11q22.3	3387187	aUPD	GRIA4 (+)
3	3q12.3	37668	D	ZPLD1 (+)	11	11q22.3	3061	D	CWF19L2 (-)
3	3q13.11	1832	D	ALCAM (+)	11	11q22.3	7609	D	CWF19L2 (-)
3	3q13.11	11951	D	ALCAM (+)	11	11q22.3	9181	D	PDGFD (-)
3	3q13.11	20627	D	LOC100302640 (-)	11	11q22.3 - 11q23.1	2786884	aUPD	NPAT (-)
3	3q13.11	20746	D	LOC100302640 (-)	11	11q23.1 - 11q23.2	2689222	aUPD	IL18 (-)
3	3q13.13	5841	A	SLC9A10 (-)	11	11q23.2 - 11q23.3	4804631	aUPD	CADM1 (-)
3	3q13.13	11475	D	PVRL3 (+)	11	11q23.3	16178	A	TRIM29 (-)
3	3q13.2	23725	D	ATP6V1A (+)	11	11q23.3	31457	A	FXDY2 (-)
3	3q13.31	2241	A	LOC285194 (+)	11	11q23.3	1315879	aUPD	ABCG4 (+)
3	3q13.31	10163	A	LOC285194 (+)	11	11q24.1	10892	A	GRAMD1B (+)
3	3q13.31	6120	D	LOC285194 (+)	11	11q24.1	5597	D	BSX (-)
3	3q21.2	1883	A	SLC41A3 (-)	11	11q24.2	1328547	aUPD	DCPS (+)
3	3q21.2	25924	A	LOC100125556 (+)	11	11q24.2	2418	D	KIRREL3 (-)
3	3q21.2	68178	A	PLXNA1 (+)	11	11q24.2	9312	D	CDON (-)
3	3q21.2	8214	D	SLC41A3 (-)	11	11q24.2 - 11q24.3	1173834	aUPD	ETS1 (-)
3	3q21.3	25764	A	ATP2C1 (+)	11	11q24.3	1531351	aUPD	TP53AIP1 (-)
3	3q21.3	18429	D	CPNE4 (-)	11	11q24.3	14827	D	APLP2 (+)
3	3q21.3	156272	D	RPN1 (-)	11	11q25	1413369	aUPD	NTM (+)
3	3q22.2	2472	A	IL20RB (+)	11	11q25	17413	D	ADAMTS15 (+)
3	3q22.3	27360	A	CLSTN2 (+)	11	11q25 -	2434655	aUPD	IGSF9B (-)
3	3q25.1	15154	A	AADACL2 (+)	11		45956	A	B3GAT1 (-)
3	3q25.1	1432	D	P2RY1 (+)	11		45940	D	B3GAT1 (-)
3	3q25.1	2932	D	P2RY1 (+)	11	11p15.4	19588	A	OR10A6 (-)
3	3q25.1	23136	D	MBNL1 (+)	11	11q24.3	1220	D	ETS1 (-)
3	3q25.2	1391	A	C3orf33 (-)	11	11q24.3	1338	D	ETS1 (-)
3	3q25.33	13308	D	ARL14 (+)	11	11q24.3	7168	A	ETS1 (-)
3	3q26.1	1673	A	SI (-)	11	11p15.3	11129	A	DKK3 (-)
3	3q26.1	2042	A	OTOL1 (+)	11	11q24.3	3252	D	ETS1 (-)
3	3q26.1	3849	A	OTOL1 (+)	11	11p11.2	17510	A	PHF21A (-)
3	3q26.1	4583	A	OTOL1 (+)	11	11p15.1	18523	D	TSG101 (-)
3	3q26.1	7273	A	SI (-)	11	11q21	23682	D	CCDC67 (+)
3	3q26.1	12512	A	OTOL1 (+)	11	11p15.4	20755	D	OVCH2 (-)
3	3q26.1	18515	A	OTOL1 (+)	11	11p13	3589	D	ELF5 (-)
3	3q26.1	22761	A	OTOL1 (+)	11	11p14.3	28852	D	LUZP2 (+)
3	3q26.1	23126	A	BCHE (-)	11	11q13.5	39646	A	DGAT2 (+)
3	3q26.1	60725	A	OTOL1 (+)	11	11p13	8647	D	ELF5 (-)
3	3q26.1	3642	D	SLITRK3 (-)	11	11p12	10877	A	API5 (+)
3	3q26.1	4123	D	BCHE (-)	11	11q23.3	6275	A	DSCAML1 (-)
3	3q26.1	22761	D	OTOL1 (+)	11	11q13.4	46306	A	ARAP1 (-)
3	3q26.31	1096	A	TBL1XR1 (-)	11	11p15.1	20565	A	PTPN5 (-)
3	3q26.31	18646	D	NAALADL2 (+)	11	11q23.3	8311	D	TMPRSS4 (+)
3	3q27.2	3155	A	MASP1 (-)	11	11q23.3	17785	A	DSCAML1 (-)
3	3q27.2	6773	A	MASP1 (-)	11		2146	A	B3GAT1 (-)

3	3q27.2	17601	A	MASP1 (-)	11	11q12.2	13082	A	SYT7 (-)
3	3q27.2	29464	D	SNORA63 (+)	11	11q14.1	19517	D	FAM181B (-)
3	3q28	1609	A	HRASLS (+)	11	11q23.3	8021	D	MLL (+)
3	3q28	2130	A	HRASLS (+)	11	11p15.3	19837	A	CTR9 (+)
3	3q28	3047	A	HRASLS (+)	11	11q13.5	26544	A	LRRC32 (-)
3	3q28	8442	A	C3orf59 (-)	11	11q24.1	9747	A	TECTA (+)
3	3q28	2905	D	HRASLS (+)	11	11q22.1	9795	A	PGR (-)
3	3q28	3047	D	HRASLS (+)	11	11q23.3	9905	D	MLL (+)
3	3q28	4442	D	HRASLS (+)	11	11q12.2	28717	A	RPLP0P2 (+)
3	3q28	4718	D	ATP13A4 (-)	11	11q24.2	23719	D	PANX3 (+)
3	3q29	1015	A	MUC20 (+)	11	11q13.5	11712	D	MOGAT2 (+)
3	3q29	7258	A	MUC20 (+)	11	11q23.3	1292	A	MLL (+)
3	3q29	11096	A	MUC20 (+)	11	11q23.3	6880	D	MLL (+)
3	3q29	34778	A	MIR570 (+)	11	11q22.2	17205	A	ANGPTL5 (-)
3	3q29	7522	D	MUC20 (+)	11	11q22.1	17590	D	CNTN5 (+)
3	3p14.1	3669	A	FAM19A4 (-)	11	11p15.2	16329	D	CYP2R1 (-)
3	3q22.1	12702	D	BFSP2 (+)	11	11q24.1	7351	A	UBASH3B (+)
3	3p25.1	7321	A	WNT7A (-)	11	11p15.1	24779	A	NAV2 (+)
3	3q26.32	2978	D	PIK3CA (+)	11	11p15.2	35088	A	CALCB (+)
3	3p25.1	15678	A	FBLN2 (+)	11	11p15.1	14135	A	NAV2 (+)
3	3q21.3	53011	D	TMCC1 (-)	11	11q25	26206	D	OPCML (-)
3	3p12.2	20687	D	GBE1 (-)	11	11p15.4	13164	D	WEE1 (+)
3	3q21.3	7892	D	RAB7A (+)	11	11q13.5	23168	A	TSKU (+)
3	3p22.2	17330	D	XIRP1 (-)	11	11p15.3	9916	A	GALNTL4 (-)
3	3p24.1	20998	D	RBMS3 (+)	11	11q25	13720	A	SNX19 (-)
3	3q28	16407	D	FGF12 (-)	11	11q23.3	11854	A	GRIK4 (+)
3	3p14.1	16263	A	MITF (+)	11	11q23.1	12562	D	RDX (-)
3	3p26.1	8295	A	GRM7 (+)	11	11q23.2	15489	D	NCAM1 (+)
3	3q21.3	3544	D	RAB7A (+)	11	11q23.1	1728	D	RDX (-)
3	3p24.1	56820	A	SLC4A7 (-)	11	11p13	18400	A	LMO2 (-)
3	3q26.31	15854	D	TBL1XR1 (-)	11	11p15.3	18517	A	GALNTL4 (-)
3	3q28	18194	D	SNAR-I (+)	11	11q23.3	11768	A	PVRL1 (-)
3	3q13.2	21027	A	ZBTB20 (-)	11	11p13	22660	D	TRIM44 (+)
3	3p14.2	20677	D	C3orf67 (-)	11	11q24.1	14265	A	MIRLET7A2 (-)
3	3q22.3	8212	A	MRPS22 (+)	12	12p11.1	13497	A	SYT10 (-)
3	3q21.3	16751	D	TMCC1 (-)	12	12p11.1	16224	A	ALG10 (+)
3	3p22.2	26236	D	XYLB (+)	12	12p11.1	25990	A	ALG10 (+)
3	3p25.3	16827	A	ATP2B2 (-)	12	12p11.1	40523	A	ALG10 (+)
3	3p22.1	33658	A	SLC25A38 (+)	12	12p11.1	47833	A	ALG10 (+)
3	3q11.2	10469	D	DCBLD2 (-)	12	12p11.21	8009	A	TSPAN11 (+)
3	3q26.32	4837	D	PIK3CA (+)	12	12p11.21	18964	A	BICD1 (+)
3	3p24.1	30493	D	LRRC3B (+)	12	12p11.23	2768	A	C12orf70 (+)
3	3p22.1	9857	D	CTNBN1 (+)	12	12p12.1	11874	D	BCAT1 (-)
3	3q13.31	18309	A	IGSF11 (-)	12	12p13.1	9172	D	C12orf36 (-)
3	3q13.13	19416	A	FLJ25363 (+)	12	12p13.2	2923	A	ETV6 (+)
3	3q26.32	1907	A	PIK3CA (+)	12	12p13.2	12599	A	ETV6 (+)
3	3p12.3	18493	D	ROBO1 (-)	12	12p13.2	17952	A	PRB2 (-)
3	3p22.1	5333	A	CTNBN1 (+)	12	12p13.2	18068	A	ETV6 (+)

3	3p22.1	1210	A	CTNNB1 (+)	12	12p13.2	16556	D	LRP6 (-)
3	3p22.1	3416	A	CTNNB1 (+)	12	12p13.31	2043	A	LOC389634 (-)
3	3p14.1	21917	D	FOXP1 (-)	12	12p13.31	2496	A	LOC389634 (-)
3	3q24	24110	D	PLOD2 (-)	12	12p13.31	7532	A	LOC389634 (-)
3	3p24.1	3277	D	LRRC3B (+)	12	12p13.31	11117	A	CLEC2D (+)
3	3q11.2	28711	D	OR5K3 (+)	12	12p13.31	23056	A	LOC374443 (+)
3	3p12.3	26391	D	ROBO2 (+)	12	12p13.31	28957	A	CLEC6A (+)
3	3p21.33	31928	D	MIR138-1 (+)	12	12p13.31	69210	A	DDX12 (-)
3	3p21.31	60906	D	SETD2 (-)	12	12p13.31	106385	A	LOC389634 (-)
3	3q13.13	32879	D	PVRL3 (+)	12	12p13.31	2043	D	LOC389634 (-)
3	3p14.3	16175	A	SPATA12 (+)	12	12p13.31	2496	D	LOC389634 (-)
3	3p12.1	14977	D	CADM2 (+)	12	12p13.31	7532	D	LOC389634 (-)
3	3q13.12	14359	D	DPPA2 (-)	12	12p13.31	38240	D	CLEC4C (-)
3	3q27.1	17514	D	DGKG (-)	12	12p13.33	3602	A	CACNA1C (+)
3	3q12.1	26221	D	IMP2 (-)	12	12p13.33	27377	A	CACNA1C (+)
3	3p13	24899	D	FLJ10213 (+)	12	12p13.33	95697	A	IQSEC3 (+)
3	3q22.3	18035	D	MRAS (+)	12	12p13.33	6908	D	WNK1 (+)
3	3q13.31	12014	A	IGSF11 (-)	12	12q13.11	2693	A	ANO6 (+)
3	3p24.2	12764	A	RARB (+)	12	12q13.11	3190	A	ANO6 (+)
3	3p21.1	7504	D	CACNA1D (+)	12	12q13.11	6289	A	ANO6 (+)
3	3q26.33	17478	A	MCCC1 (-)	12	12q13.11	44521	D	ARID2 (+)
3	3q26.2	3485	A	TNIK (-)	12	12q13.13	12126	D	SNORA2B (-)
3	3q26.33	33538	A	DCUN1D1 (-)	12	12q13.2	34152	D	SOAT2 (+)
4	4p11	74273	A	CWH43 (+)	12	12q14.1	1152	A	XRCC6BP1 (+)
4	4p12	21209	A	FRYL (-)	12	12q14.1	2956	A	LRIG3 (-)
4	4p13	7448	A	GNPDA2 (-)	12	12q14.1	1986	D	XRCC6BP1 (+)
4	4p14	27671	D	KLHL5 (+)	12	12q14.1	2956	D	LRIG3 (-)
4	4p15.1	1935	A	STIM2 (+)	12	12q14.1	37057	D	RBMS2 (+)
4	4p15.1	3746	A	ARAP2 (-)	12	12q14.3	13644	D	DPY19L2 (-)
4	4p15.1	13009	A	ARAP2 (-)	12	12q21.1	31661	D	CNOT2 (+)
4	4p15.1	34108	A	ARAP2 (-)	12	12q21.2	12950	A	NAV3 (+)
4	4p15.1	41534	A	PCDH7 (+)	12	12q21.2	13337	A	LOC552889 (+)
4	4p15.1	17262	D	PCDH7 (+)	12	12q21.2	6052	D	NAV3 (+)
4	4p15.1	20979	D	PCDH7 (+)	12	12q21.2	15817	D	LOC552889 (+)
						12q21.31			
4	4p15.1	34360	D	PCDH7 (+)	12	12q21.32	18856	D	SLC6A15 (-)
4	4p15.1	40939	D	PCDH7 (+)	12	12q21.33	16737	A	KITLG (-)
4	4p15.2	11483	D	RBPJ (+)	12	12q23.1	4949	A	RMST (+)
4	4p15.2	15945	D	C4orf52 (+)	12	12q23.1	50432	A	NEDD1 (+)
4	4p15.31	6434	D	KCNIP4 (-)	12	12q24.11	54450	A	SSH1 (-)
4	4p15.33	7022	D	HSP90AB2P (+)	12	12q24.13	26773	D	CCDC63 (+)
4	4p15.33	8760	D	HS3ST1 (-)	12	12q24.31	14240	A	TCTN2 (+)
4	4p16.1	3155	A	WDR1 (-)	12	12q24.31	12290	D	SRRM4 (+)
									LOC100128554
4	4p16.1	4772	A	WDR1 (-)	12	12q24.32	4121	D	(+)
									LOC100128554
4	4p16.1	14041	A	WDR1 (-)	12	12q24.32	8401	D	(+)
4	4p16.1	17280	A	PSAPL1 (-)	12	12q24.33	2807	A	TMEM132C (+)

4	4p16.1	22919	A	PSAPL1 (-)	12	12q24.33	10290	A	SFRS8 (+)
4	4p16.1	3155	D	WDR1 (-)	12	12q24.33	10673	A	LOC116437 (+)
4	4p16.1	4772	D	WDR1 (-)	12	12q24.33	5382	D	LOC116437 (+)
4	4p16.2	9064	A	LOC348926 (-)	12		9003	A	ZNF26 (+)
4	4p16.2	285126	A	LOC348926 (-)	12		27421	A	GALNT9 (-)
4	4p16.2	19023	D	OTOP1 (-)	12		3980	D	ZNF26 (+)
4	4p16.2	285126	D	LOC348926 (-)	12		4392	D	ZNF26 (+)
4	4p16.3	12272	A	ZNF595 (+)	12	12q12	24855	D	LRRK2 (+)
4	4p16.3	14173	A	ZNF718 (+)	12	12p13.2	1815	A	ETV6 (+)
4	4p16.3	27625	A	ZNF595 (+)	12	12p11.21	22733	A	IPO8 (-)
4	4p16.3	28232	A	ZFYVE28 (-)	12	12q12	10730	A	CNTN1 (+)
4	4p16.3	5602	D	ZNF595 (+)	12	12q24.13	19747	D	ATXN2 (-)
4	4p16.3	6671	D	ZNF595 (+)	12	12p13.2	27010	A	KLRA1 (-)
4	4p16.3	7989	D	ZNF718 (+)	12	12q24.23	8969	D	MAP1LC3B2 (+)
4	4p16.3	14173	D	ZNF718 (+)	12	12q24.33	9265	D	GLT1D1 (+)
4	4p16.3	15978	D	SLBP (-)	12	12q21.1	7854	D	PTPRR (-)
4	4p16.3	20833	D	RNF212 (-)	12	12q12	23949	D	PDZRN4 (+)
4	4p16.3	27625	D	ZNF595 (+)	12	12q24.21	4977	A	OAS2 (+)
4	4q12	10173	D	LNX1 (-)	12	12q24.33	9055	D	GLT1D1 (+)
4	4q13.1	17846	D	TECRL (-)	12	12q14.1	3487	A	ERBB3 (+)
									LOC100128554
4	4q13.1	35953	D	TECRL (-)	12	12q24.32	9570	A	(+)
4	4q13.2	3234	A	YTHDC1 (-)	12	12q14.1	5537	D	ERBB3 (+)
4	4q13.2	10827	A	TMPRSS11E (+)	12	12q24.33	3836	D	GLT1D1 (+)
4	4q13.2	11154	A	UGT2B7 (+)	12	12p11.22	2071	D	FAR2 (+)
4	4q13.2	16632	A	UGT2B15 (-)	12	12q21.31	29797	A	PTPRQ (+)
4	4q13.2	22905	A	YTHDC1 (-)	12	12p12.1	21480	A	ETNK1 (+)
4	4q13.2	50118	A	TMPRSS11E (+)	12	12q23.3	33344	A	GLT8D2 (-)
4	4q13.2	16452	D	UGT2B15 (-)	12	12q13.13	20309	A	KRT71 (-)
4	4q13.2	44308	D	TMPRSS11E (+)	12	12q23.2	19715	D	CCDC53 (-)
4	4q13.3	9997	A	UGT2A2 (-)	12	12p12.1	7632	D	KRAS (-)
4	4q13.3	9997	D	UGT2A2 (-)	12	12q21.1	14178	A	BEST3 (-)
4	4q13.3	18985	D	ODAM (+)	12	12q24.31	27585	D	GCN1L1 (-)
4	4q21.1	8085	A	SHROOM3 (+)	12	12q24.31	16067	A	SRRM4 (+)
4	4q21.22	15966	D	HNRNPD (-)	12	12q13.13	5625	A	KRT71 (-)
4	4q21.23	1129	D	MAPK10 (-)	12	12q23.3	10313	A	CHST11 (+)
4	4q22.1	2605	A	IBSP (+)	12	12q22	12507	A	EAA1 (-)
4	4q22.1	3097	A	IBSP (+)	12	12q24.11	15713	A	WSCD2 (+)
4	4q22.1	2519	D	IBSP (+)	12	12p11.23	26669	D	SSPN (+)
4	4q22.3	1733	A	PDHA2 (+)	12	12q13.11	12903	A	ANO6 (+)
4	4q22.3	13196	D	C4orf37 (-)	12	12q24.21	5001	A	LHX5 (-)
4	4q24	14805	A	TBCK (-)	12	12q21.31	13759	D	TMTC2 (+)
4	4q24	2722	D	TACR3 (-)	12	12q23.1	17282	D	LTA4H (-)
4	4q24	7199	D	TACR3 (-)	12	12q13.2	10261	D	KRT76 (-)
4	4q24	14498	D	INTS12 (-)	12	12q12	18391	D	PDZRN4 (+)
4	4q24	14922	D	DKK2 (-)	12	12q24.11	9038	A	WSCD2 (+)
									LOC100128191
4	4q24	15236	D	SLC39A8 (-)	12	12q23.1	37833	D	(-)

4	4q25	1133	A	DKK2 (-)	12	12q21.2	17087	D	LOC552889 (+)
4	4q25	1524	A	DKK2 (-)	12	12q24.31	13149	D	SRRM4 (+)
4	4q25	1600	A	DKK2 (-)	12	12q15	21846	A	DYRK2 (+)
4	4q25	2429	A	DKK2 (-)	12	12q12	12625	D	PDZRN4 (+)
4	4q25	17131	A	LEF1 (-)	12	12q13.13	42313	D	SPATS2 (+)
4	4q25	23741	A	C4orf21 (-)	12	12q24.23	32592	D	SUDS3 (+)
4	4q25	37485	A	MIR302B (-)	13	13q12.11	26169	A	XP04 (-)
4	4q25	4028	D	DKK2 (-)	13	13q12.11	422652	A	LOC284232 (-)
4	4q26	2222	A	ARSJ (-)	13	13q12.11	29620	D	LOC284232 (-)
4	4q26	2328	A	NDST4 (-)	13	13q12.11	422652	D	LOC284232 (-)
4	4q26	2630	A	ARSJ (-)	13	13q12.12	47621	A	SACS (-)
4	4q26	3268	A	NDST3 (+)	13	13q12.13	5680	A	SPATA13 (+)
4	4q26	4880	A	ARSJ (-)	13	13q12.13	20197	A	ATP8A2 (+)
4	4q26	11477	A	MIR1973 (+)	13	13q12.3	1678	A	FLT3 (-)
4	4q26	22790	A	TRAM1L1 (-)	13	13q13.1	12949	A	B3GALTL (+)
4	4q26	2773	D	NDST3 (+)	13	13q13.1	25691	A	B3GALTL (+)
4	4q26	3268	D	NDST3 (+)	13	13q13.3	37187	D	NBEA (+)
4	4q26	16147	D	NDST3 (+)	13	13q14.11	17467	D	DNAJC15 (+)
4	4q26	22995	D	MIR1973 (+)	13	13q14.11	19681	D	DNAJC15 (+)
4	4q28.1	2394	A	HSPA4L (+)	13	13q14.11	21878	D	DNAJC15 (+)
4	4q28.1	2500	A	HSPA4L (+)	13	13q14.2	16298	A	CPB2 (-)
4	4q28.2	12651	D	C4orf33 (+)	13	13q14.3	15007	A	CYSLTR2 (+)
4	4q28.3	5073	A	PCDH18 (-)	13	13q21.1	36063	A	FLJ37307 (-)
4	4q28.3	7707	A	PCDH18 (-)	13	13q21.33	3868	A	PCDH9 (-)
4	4q28.3	23161	A	PCDH10 (+)	13	13q21.33	6221	A	KLHL1 (-)
4	4q28.3	29062	A	SLC7A11 (-)	13	13q21.33	20932	A	KLHL1 (-)
4	4q28.3	13332	D	PCDH18 (-)	13	13q21.33	22458	D	DACH1 (-)
4	4q28.3	17315	D	PCDH10 (+)	13	13q22.2	14477	D	LOC647288 (-)
4	4q31.1	83804	D	NAA15 (+)	13	13q22.3	23869	A	MYCBP2 (-)
4	4q31.1 - 4q31.21	1120165	aUPD	IL15 (+)	13	13q31.1	20735	D	SPRY2 (-)
4	4q31.21	17157	A	GAB1 (+)	13	13q31.1	25192	D	SLITRK6 (-)
4	4q31.21	1073	D	INPP4B (-)	13	13q31.2	40011	D	SLITRK5 (+)
4	4q31.22	12284	D	ZNF827 (-)	13	13q33.1	3479	A	FGF14 (-)
4	4q31.3	4410	D	LRBA (-)	13	13q33.1	15154	A	A2LD1 (-)
4	4q32.2	6762	A	NAF1 (-)	13	13q33.1	25984	A	PCCA (+)
4	4q32.2	7930	A	FSTL5 (-)	13	13q33.1	16095	D	TMTC4 (-)
4	4q32.3	7607	A	ANXA10 (+)	13	13q33.2	22510	D	DAOA (+)
4	4q32.3	8790	D	ANXA10 (+)	13	13q21.33	1193	A	PCDH9 (-)
4	4q34.1	1632	A	GALNTL6 (+)	13	13q14.11	27428	A	TNFSF11 (+)
4	4q34.1	12563	A	GALNTL6 (+)	13	13q21.33	5990	A	PCDH9 (-)
4	4q34.1	4265	D	GALNTL6 (+)	13	13q32.1	32798	A	HS6ST3 (+)
4	4q34.2	24712	D	ASB5 (-)	13	13q22.1	15798	A	KLF5 (+)
4	4q34.3	2512	D	MGC45800 (-)	13	13q14.13	20496	D	LOC100190939 (+)
4	4q34.3	17872	D	LOC285501 (+)	13	13q14.3	4453	A	RNASEH2B (+)
4	4q35.1	5803	A	MGC45800 (-)	13	13q32.3	17114	D	STK24 (-)
4	4q35.1	3519	D	ENPP6 (-)	13	13q13.3	17304	D	TRPC4 (-)
4	4q35.2	5168	A	FRG1 (+)	13	13q14.11	15630	D	KIAA0564 (-)

4	4q35.2	11602	A	FRG1 (+)	13	13q14.3	28094	A	RNASEH2B (+)
4	4q35.2	18889	A	ZFP42 (+)	13	13q14.3	28094	D	RNASEH2B (+)
4	4q35.2	215941	A	LOC653544 (+)	13	13q33.3	14261	D	ABHD13 (+)
4	4q35.2	12841	D	TRIML2 (-)	13	13q31.2	25192	A	SLITRK6 (-)
4	4q35.2	16231	D	ZFP42 (+)	13	13q21.2	28403	A	DIAPH3 (-)
4	4q28.1	22183	D	SLC25A31 (+)	13	13q22.1	11600	D	C13orf34 (+)
4	4q21.3	19131	A	PTPN13 (+)	13	13q14.11	17213	A	LHFP (-)
4	4p12	1214	D	GABRG1 (-)	13	13q21.31	36062	D	TDRD3 (+)
4	4q28.1	12194	A	HSPA4L (+)	13	13q14.3	16761	A	DLEU7 (-)
4	4q28.2	1492	D	C4orf33 (+)	13	13q21.31	9550	A	OR7E156P (+)
4	4p12	20398	D	GABRG1 (-)	13	13q12.3	6459	D	FLT3 (-)
4	4q13.3	2335	D	MTHFD2L (+)	13	13q14.3	15902	A	RB1 (+)
4	4q26	17222	A	MIR1973 (+)	13	13q31.3	16506	A	LOC144776 (-)
4	4q21.21	19685	A	PRKG2 (-)	13	13q32.1	6065	A	ABCC4 (-)
4	4q28.1	14415	A	MIR2054 (+)	13	13q21.1	3277	D	MIR1297 (-)
4	4q28.3	27786	D	PABPC4L (-)	13	13q14.11	22702	D	SUGT1L1 (-)
4	4q31.21	1452	A	GAB1 (+)	13	13q13.1	14682	D	FRY (+)
4	4q31.3	8558	A	ARFIP1 (+)	13	13q31.3	17364	D	LOC144776 (-)
4	4q31.23	21949	A	NR3C2 (-)	13	13q32.1	17510	D	CLDN10 (+)
4	4p16.3	27686	A	ZFYVE28 (-)	13	13q12.11	18196	A	IFT88 (+)
4	4q34.3	27257	D	MGC45800 (-)	13	13q14.3	10757	A	RNASEH2B (+)
4	4p15.2	7262	A	LGI2 (-)	13	13q13.3	27265	D	C13orf36 (+)
4	4q31.3	12357	A	TIGD4 (-)	13	13q21.31	22972	A	DIAPH3 (-)
4	4q21.1	24131	A	NUP54 (-)	13	13q31.1	21775	A	SPRY2 (-)
4	4p14	23895	D	ARAP2 (-)	13	13q32.3	19202	D	SLC15A1 (-)
4	4q34.3	12508	D	VEGFC (-)	13	13q12.3	32417	A	POMP (+)
4	4q13.3	12026	A	COX18 (-)	13	13q14.11	27711	D	ENOX1 (-)
4	4p15.2	14568	D	STIM2 (+)	13	13q22.2	30417	D	LOC647288 (-)
4	4q35.1	11557	D	ODZ3 (+)	13	13q14.2	10450	A	RB1 (+)
4	4q32.3	26311	D	KLHL2 (+)	13	13q14.2	1335	D	SUCLA2 (-)
4	4q25	16060	D	C4orf32 (+)	13	13q14.2	14947	A	HTR2A (-)
4	4q31.3	11795	D	KIAA0922 (+)	13	13q21.31	12993	D	PCDH20 (-)
4	4q34.1	14335	D	GALNTL6 (+)	13	13q32.2	17318	D	HS6ST3 (+)
4	4q31.22	39904	D	SMAD1 (+)	13	13q22.2	15888	A	KLF12 (-)
4	4p12	18315	D	GABRA2 (-)	13	13q21.2	11946	A	DIAPH3 (-)
4	4q28.1	10940	D	INTU (+)	13	13q13.3	14429	D	TRPC4 (-)
4	4q21.21	18597	D	C4orf22 (+)	13	13q13.1	11519	D	FRY (+)
4	4p14	22616	D	ARAP2 (-)	13	13q21.31	35895	A	PCDH20 (-)
4	4q34.3	34521	D	LOC285501 (+)	13	13q13.2	1154	D	BRCA2 (+)
4	4q25	37021	D	C4orf32 (+)	14	14q11.2	1207	A	DAD1 (-)
4	4p14	12976	D	DTHD1 (+)	14	14q11.2	1237	A	DAD1 (-)
4	4q23	9519	D	PPP3CA (-)	14	14q11.2	1373	A	DAD1 (-)
4	4p15.2	9048	A	LGI2 (-)	14	14q11.2	1691	A	DAD1 (-)
4	4q12	16766	D	KIT (+)	14	14q11.2	1694	A	DAD1 (-)
4	4q21.21	12253	D	ANXA3 (+)	14	14q11.2	1749	A	DAD1 (-)
4	4q27	27828	D	ANXA5 (-)	14	14q11.2	1754	A	DAD1 (-)
4	4q12	1243	A	PDGFRA (+)	14	14q11.2	2051	A	DAD1 (-)
4	4q32.2	15896	D	NPY5R (+)	14	14q11.2	2342	A	DAD1 (-)

4	4q12	17243	A	PDGFRA (+)	14	14q11.2	2557	A	DAD1 (-)
4	4q28.1	19255	D	MIR2054 (+)	14	14q11.2	2758	A	DAD1 (-)
4	4q31.1	16455	A	C4orf49 (-)	14	14q11.2	2836	A	DAD1 (-)
4	4q21.22	14221	A	HNRNPD (-)	14	14q11.2	3483	A	DAD1 (-)
4	4q12	1099	A	PDGFRA (+)	14	14q11.2	3589	A	DAD1 (-)
4	4q12	1032	A	PDGFRA (+)	14	14q11.2	3668	A	DAD1 (-)
4	4q12	1032	D	PDGFRA (+)	14	14q11.2	3824	A	OR4E2 (+)
4	4q12	13272	A	PDGFRA (+)	14	14q11.2	3870	A	OR4E2 (+)
4	4p15.33	21397	D	MIR572 (+)	14	14q11.2	3904	A	DAD1 (-)
4	4q28.2	34104	D	C4orf33 (+)	14	14q11.2	4381	A	DAD1 (-)
4	4q32.1	30599	D	GLRB (+)	14	14q11.2	4458	A	DAD1 (-)
4	4p15.2	6946	A	STIM2 (+)	14	14q11.2	4606	A	DAD1 (-)
4	4q12	38635	A	PDGFRA (+)	14	14q11.2	4775	A	DAD1 (-)
4	4q23	11325	A	TSPAN5 (-)	14	14q11.2	4808	A	DAD1 (-)
4	4q32.3	13421	D	PALLD (+)	14	14q11.2	5805	A	DAD1 (-)
4	4q25	24632	D	C4orf32 (+)	14	14q11.2	7184	A	DAD1 (-)
4	4q31.1	16862	D	MAML3 (-)	14	14q11.2	7795	A	DAD1 (-)
4	4q28.3	17254	D	PCDH10 (+)	14	14q11.2	7903	A	DAD1 (-)
4	4q23	41573	D	ADH6 (-)	14	14q11.2	8790	A	OR4E2 (+)
4	4q13.2	13121	D	EPHA5 (-)	14	14q11.2	9642	A	DAD1 (-)
4	4p15.1	33507	D	ARAP2 (-)	14	14q11.2	9938	A	DAD1 (-)
4	4q21.23	11363	D	NKX6-1 (-)	14	14q11.2	11382	A	DAD1 (-)
4	4q13.3	35081	A	SLC4A4 (+)	14	14q11.2	11712	A	DAD1 (-)
4	4p16.1	14788	D	CLNK (-)	14	14q11.2	11727	A	DAD1 (-)
4	4q32.1	23241	A	RAPGEF2 (+)	14	14q11.2	11901	A	OR4E2 (+)
5	5p11	29201	A	HCN1 (-)	14	14q11.2	12421	A	DAD1 (-)
5	5p11	37568	A	HCN1 (-)	14	14q11.2	12474	A	DAD1 (-)
5	5p13.3	28783	A	C1QTNF3 (-)	14	14q11.2	13173	A	DAD1 (-)
5	5p14.3	24558	D	LOC728411 (+)	14	14q11.2	13881	A	OR4E2 (+)
5	5p14.3	46967	D	LOC728411 (+)	14	14q11.2	18299	A	DAD1 (-)
5	5p15.1	3363	A	FBXL7 (+)	14	14q11.2	20133	A	DAD1 (-)
5	5p15.2	15216	A	TRIO (+)	14	14q11.2	21399	A	DAD1 (-)
5	5p15.31	21485	D	ADCY2 (+)	14	14q11.2	24124	A	DAD1 (-)
5	5p15.33	8663	A	IRX2 (-)	14	14q11.2	25173	A	DAD1 (-)
5	5p15.33	16120	A	IRX1 (+)	14	14q11.2	26099	A	DAD1 (-)
5	5p15.33	26485	A	IRX4 (-)	14	14q11.2	27542	A	DAD1 (-)
5	5p15.33	16601	D	TPPP (-)	14	14q11.2	32565	A	DAD1 (-)
5	5q11.1	18577	A	PARP8 (+)	14	14q11.2	1056	A	DAD1 (-)
5	5q11.1	104685	A	PARP8 (+)	14	14q11.2	1207	A	DAD1 (-)
5	5q11.2	1850	A	PLK2 (-)	14	14q11.2	1237	A	DAD1 (-)
5	5q11.2	2386	A	PLK2 (-)	14	14q11.2	1244	A	OR4E2 (+)
5	5q11.2	3347	A	PLK2 (-)	14	14q11.2	1373	A	DAD1 (-)
5	5q11.2	4106	A	PLK2 (-)	14	14q11.2	1691	A	DAD1 (-)
5	5q12.1	37605	D	KIF2A (+)	14	14q11.2	1694	A	DAD1 (-)
5	5q13.2	53915	D	BDP1 (+)	14	14q11.2	1749	A	DAD1 (-)
5	5q13.3	11315	D	SV2C (+)	14	14q11.2	1754	A	DAD1 (-)
5	5q14.1	24825	A	MSH3 (+)	14	14q11.2	2051	A	DAD1 (-)
5	5q14.3	9639	D	COX7C (+)	14	14q11.2	2342	A	DAD1 (-)

5	5q15 - 5q21.1	3553931	D	CAST (+)	14	14q11.2	2454	A	DAD1 (-)
5	5q21.1	41496	A	RGMB (+)	14	14q11.2	2557	A	DAD1 (-)
5	5q21.1	9270	D	LOC100133050 (-)	14	14q11.2	2613	A	DAD1 (-)
5	5q21.1 - 5q21.2	4322057	D	GIN1 (-)	14	14q11.2	2757	A	OR4E2 (+)
5	5q21.2	6996	A	RAB9P1 (+)	14	14q11.2	2758	A	DAD1 (-)
5	5q21.2	6996	D	RAB9P1 (+)	14	14q11.2	2780	A	OR4E2 (+)
5	5q21.2	7035	D	RAB9P1 (+)	14	14q11.2	2836	A	DAD1 (-)
5	5q21.2	7592	D	RAB9P1 (+)	14	14q11.2	2980	A	DAD1 (-)
5	5q21.2	14411	D	NUDT12 (-)	14	14q11.2	3221	A	DAD1 (-)
5	5q21.2	219938	D	RAB9P1 (+)	14	14q11.2	3483	A	DAD1 (-)
5	5q21.2	324984	D	RAB9P1 (+)	14	14q11.2	3552	A	DAD1 (-)
5	5q21.2 - 5q22.2	7815565	D	C5orf13 (-)	14	14q11.2	3589	A	DAD1 (-)
5	5q22.2	2141	A	APC (+)	14	14q11.2	3796	A	DAD1 (-)
5	5q22.2	2538	A	APC (+)	14	14q11.2	3824	A	OR4E2 (+)
5	5q22.2	10674	A	APC (+)	14	14q11.2	3870	A	OR4E2 (+)
5	5q22.2	2426	D	APC (+)	14	14q11.2	3904	A	DAD1 (-)
5	5q22.2	3122	D	APC (+)	14	14q11.2	3921	A	DAD1 (-)
5	5q22.2	48305	D	APC (+)	14	14q11.2	4048	A	OR4E2 (+)
5	5q22.2	75776	D	APC (+)	14	14q11.2	4381	A	DAD1 (-)
5	5q22.2	550160	D	APC (+)	14	14q11.2	4458	A	DAD1 (-)
5	5q22.2 - 5q23.1	2664087	D	KCNN2 (+)	14	14q11.2	4606	A	DAD1 (-)
5	5q22.3	13917	A	CCDC112 (-)	14	14q11.2	4624	A	DAD1 (-)
5	5q23.1	11530	D	DTWD2 (-)	14	14q11.2	4704	A	DAD1 (-)
5	5q23.1	22721	D	COMMD10 (+)	14	14q11.2	4775	A	DAD1 (-)
5	5q23.1	25926	D	FTMT (+)	14	14q11.2	4808	A	DAD1 (-)
5	5q23.1	690807	D	SEMA6A (-)	14	14q11.2	5805	A	DAD1 (-)
5	5q23.1	1269005	D	SEMA6A (-)	14	14q11.2	7184	A	DAD1 (-)
5	5q23.1	3605231	D	HSD17B4 (+)	14	14q11.2	7795	A	DAD1 (-)
5	5q23.1 - 5q23.3	9087112	D	GRAMD3 (+)	14	14q11.2	7903	A	DAD1 (-)
5	5q23.3	18163	A	ADAMTS19 (+)	14	14q11.2	8044	A	OR4E2 (+)
5	5q23.3	24223	D	HINT1 (-)	14	14q11.2	8176	A	DAD1 (-)
5	5q23.3 - 5q31.1	2167780	D	LEAP2 (+)	14	14q11.2	8790	A	OR4E2 (+)
5	5q31.1	19761	A	CSF2 (+)	14	14q11.2	9220	A	DAD1 (-)
5	5q31.1	38087	A	FSTL4 (-)	14	14q11.2	9642	A	DAD1 (-)
5	5q31.1	46475	A	LOC153328 (+)	14	14q11.2	9938	A	DAD1 (-)
5	5q31.1	25932	D	MIR1289-2 (-)	14	14q11.2	11049	A	DAD1 (-)
5	5q31.1	366044	D	HSPA4 (+)	14	14q11.2	11382	A	DAD1 (-)
5	5q33.1	32473	D	FLJ41603 (+)	14	14q11.2	11561	A	OR4E2 (+)
5	5q33.2	23305	A	SGCD (+)	14	14q11.2	11712	A	DAD1 (-)
5	5q33.3	15767	D	CLINT1 (-)	14	14q11.2	11837	A	OR4E2 (+)
5	5q33.3	22165	D	CLINT1 (-)	14	14q11.2	12063	A	DAD1 (-)
5	5q34	21512	A	ATP10B (-)	14	14q11.2	12421	A	DAD1 (-)
5	5q35.1	35864	D	UBTD2 (-)	14	14q11.2	12474	A	DAD1 (-)
5	5q35.2	1027	A	HMP19 (+)	14	14q11.2	13019	A	OR4E2 (+)
5	5q35.2	1027	D	HMP19 (+)	14	14q11.2	13173	A	DAD1 (-)
5	5q35.2	9635	D	HMP19 (+)	14	14q11.2	13881	A	OR4E2 (+)
5	5q35.3	28158	A	BTNL3 (+)	14	14q11.2	14252	A	DAD1 (-)
5	5q35.3	32036	A	LMAN2 (-)	14	14q11.2	14905	A	DAD1 (-)

5	5q35.3	36292	A	BTNL3 (+)	14	14q11.2	17936	A	OR4E2 (+)
5	5q35.3	43695	A	F12 (-)	14	14q11.2	19847	A	OR4E2 (+)
5	5q35.3	8321	D	GRK6 (+)	14	14q11.2	20133	A	DAD1 (-)
5	5q35.3	17584	D	ADAMTS2 (-)	14	14q11.2	21399	A	DAD1 (-)
5	5q35.3	19600	D	NSD1 (+)	14	14q11.2	21427	A	OR4E2 (+)
5	5q35.3	43695	D	F12 (-)	14	14q11.2	21812	A	OR4E2 (+)
5	5q35.2	8228	A	HMP19 (+)	14	14q11.2	22262	A	OR4E2 (+)
5	5q11.2	2462	A	FST (+)	14	14q11.2	23678	A	OR4E2 (+)
5	5q31.2	23332	D	SPOCK1 (-)	14	14q11.2	25173	A	DAD1 (-)
5	5p12	24291	A	C5orf39 (-)	14	14q11.2	26099	A	DAD1 (-)
5	5q22.2	12275	A	MCC (-)	14	14q11.2	27542	A	DAD1 (-)
5	5q11.2	6902	A	FST (+)	14	14q11.2	32565	A	DAD1 (-)
5	5q11.2	22393	A	PELO (+)	14	14q11.2	35909	A	OR4E2 (+)
5	5q32	40383	D	TCERG1 (+)	14	14q11.2	44775	A	OR4E2 (+)
5	5q11.2	10165	A	ESM1 (-)	14	14q12	5025	A	NOVA1 (-)
5	5p15.1	31378	D	FBXL7 (+)	14	14q12	5747	A	DHRS4L1 (+)
5	5q11.2	3075	A	FST (+)	14	14q12	13869	D	NOVA1 (-)
5	5q22.2	13866	D	APC (+)	14	14q12	28204	D	NOVA1 (-)
5	5q35.2	6541	A	CPEB4 (+)	14	14q21.1	19034	A	FBXO33 (-)
5	5q33.3	25895	A	CLINT1 (-)	14	14q21.1	2832	D	CLEC14A (-)
5	5q33.3	25895	D	CLINT1 (-)	14	14q21.3	16145	A	C14orf106 (-)
5	5p13.1	33086	A	PTGER4 (+)	14	14q22.1	32101	A	POLE2 (-)
5	5q23.1	1733	A	SEMA6A (-)	14	14q22.1	32676	D	SDCCAG1 (-)
5	5q23.1	4760	D	SEMA6A (-)	14	14q22.3	11323	D	CDKN3 (+)
5	5q35.2	16040	A	CPEB4 (+)	14	14q22.3	17204	D	CDKN3 (+)
5	5q23.1	3028	A	SEMA6A (-)	14	14q23.1	14606	A	DACT1 (+)
5	5q15	13174	D	PCSK1 (-)	14	14q23.3	6508	A	SPTB (-)
5	5q23.1	17261	A	SEMA6A (-)	14	14q24.2	6916	A	FLJ44817 (+)
5	5q22.2	8798	D	MCC (-)	14	14q24.2	15374	A	SLC8A3 (-)
5	5q35.2	1589	A	CPEB4 (+)	14	14q24.3	26472	A	ACOT1 (+)
5	5q14.3	13929	D	LOC100129716 (+)	14	14q24.3	20570	D	C14orf179 (+)
5	5p12 - 5p11	19430	D	HCN1 (-)	14	14q24.3	22231	D	LIN52 (+)
5	5q33.3	35794	D	FABP6 (+)	14	14q31.1	4252	A	NRXN3 (+)
5	5q35.1	18942	A	GABRP (+)	14	14q31.1	15904	D	ADCK1 (+)
5	5q11.2	27224	A	ACTBL2 (-)	14	14q31.1	18568	D	NRXN3 (+)
5	5q12.1	11519	A	PART1 (+)	14	14q32.12	20250	D	TTC7B (-)
5	5p13.1	15165	A	GHR (+)	14	14q32.12	39195	D	ATXN3 (-)
5	5q21.3	18018	A	RAB9P1 (+)	14	14q32.13	27170	A	ITPK1 (-)
5	5p12	34109	D	HCN1 (-)	14	14q32.2	13488	A	C14orf49 (-)
5	5q35.2	11370	A	ERGIC1 (+)	14	14q32.31	40703	A	MIR411 (+)
5	5q34	14944	A	LOC285629 (-)	14	14q32.33	16078	D	KIAA0125 (+)
5	5q14.3	30734	D	MEF2C (-)	14		24807	A	KIAA0125 (+)
5	5q13.3	20833	D	FAM169A (-)	14		41266	A	ADAM6 (-)
5	5q31.1	6595	D	MIR1289-2 (-)	14		41108	D	KIAA0125 (+)
5	5q31.2	26311	D	SIL1 (-)	14	14q32.33	30310	D	KLC1 (+)
5	5q32	13330	D	HMHB1 (+)	14	14q23.2	6345	D	RHOJ (+)
5	5q22.3	7589	A	KCNN2 (+)	14	14q11.2	29393	D	ACIN1 (-)
5	5q31.1	77590	D	ZCCHC10 (-)	14	14q21.1	40664	D	SIP1 (+)

5	5q31.2	40162	D	CDC25C (-)	14	14q21.1	15479	D	NKX2-8 (-)
5	5p13.1	7365	A	DAB2 (-)	14	14q32.2	8856	A	BCL11B (-)
5	5q34	21913	D	GABRG2 (+)	14	14q32.31	25874	D	SNORD113-2 (+)
5	5q14.3	36562	A	CETN3 (-)	14	14q24.2	21136	D	RGS6 (+)
5	5q31.3	23331	D	ARHGAP26 (+)	14	14q21.3	17840	D	LRFN5 (+)
5	5p13.1	15494	A	DAB2 (-)	14	14q12	27948	D	STXBP6 (-)
5	5q31.1	9226	D	TCF7 (+)	14	14q32.11	42790	D	FOXN3 (-)
5	5q35.1	12199	A	KCNIP1 (+)	14	14q32.2	10357	A	VRK1 (+)
5	5q12.1	15103	A	ZSWIM6 (+)	14	14q24.2	18501	A	ACTN1 (-)
5	5q14.1	20350	D	WDR41 (-)	14	14q24.2	19341	D	PCNX (+)
5	5q23.3	17126	A	ADAMTS19 (+)	14	14q22.3	18691	A	CNIH (-)
5	5q34	22487	D	ODZ2 (+)	14	14q31.1	12248	A	DIO2 (-)
6	6p11.2	2004	A	PRIM2 (+)	14	14q32.2	21511	A	DICER1 (-)
6	6p11.2	14228	A	PRIM2 (+)	14	14q32.12	44830	D	SNORA11B (+)
6	6p11.2	14228	D	PRIM2 (+)	14	14q31.3	17441	A	FLRT2 (+)
6	6p12.1	2144	A	C6orf142 (+)	14	14q13.3	21607	D	RALGAPA1 (-)
6	6p12.1	2485	A	C6orf142 (+)	14	14q23.3	3537	A	ESR2 (-)
6	6p12.1	5982	A	C6orf142 (+)	14	14q23.2	12183	A	TMEM30B (-)
6	6p12.3	15189	A	GPR116 (-)	14	14q24.2	19542	A	RGS6 (+)
6	6p21.1	11721	A	TFEB (-)	14	14q24.2	13686	A	RGS6 (+)
6	6p21.1	36883	A	NCR2 (+)	14	14q23.3	53484	A	FUT8 (+)
6	6p21.2	7942	A	MDGA1 (-)	14	14q23.1	10187	D	DAAM1 (+)
6	6p21.2	22484	A	BTBD9 (-)	14	14q21.1	10554	A	SLC25A21 (-)
6	6p21.31	16639	A	SPDEF (-)	14	14q23.1	25122	D	GPR135 (-)
6	6p21.31	3055	D	MIR1275 (-)	15	15q11.2	19549	A	NF1P1 (-)
6	6p21.32	36387	A	HLA-DRB5 (-)	15	15q11.2	86080	A	GOLGA6L6 (-)
6	6p21.32	38233	A	HLA-DRB5 (-)	15	15q11.2	127203	A	GOLGA6L6 (-)
6	6p21.32	40062	D	BRD2 (+)	15	15q12	9545	A	SNORD115-29 (+)
6	6p21.33	19551	A	PSORS1C1 (+)	15	15q12	11204	A	SNORD115-44 (+)
6	6p22.1	26302	A	ZFP57 (-)	15	15q12	22101	A	PWRN2 (-)
6	6p22.1	30031	A	HLA-H (+)	15	15q12	27285	A	SNORD115-1 (+)
6	6p22.1	60937	A	GUSBL1 (-)	15	15q12	38727	A	SNORD116-16 (+)
6	6p22.1	13964	D	GUSBL1 (-)	15	15q12	70502	A	SNORD115-31 (+)
6	6p22.1	40749	D	C6orf41 (+)	15	15q12	11204	A	SNORD115-44 (+)
6	6p22.1	60937	D	GUSBL1 (-)	15	15q12	17977	D	SNORD115-48 (+)
6	6p22.2	27249	A	FAM65B (-)	15	15q13.2	24897	D	WHAMML2 (+)
6	6p22.3	1124	A	MIR548A1 (+)	15	15q13.3	31379	D	DKFZP434L187 (+)
6	6p22.3	1493	A	MIR548A1 (+)	15	15q13.3	191804	D	ARHGAP11B (+)
6	6p22.3	3589	A	MIR548A1 (+)	15	15q15.1	43996	A	C15orf23 (+)
6	6p22.3	6134	A	MIR548A1 (+)	15	15q21.2	67890	D	CTXN2 (+)

6	6p22.3	15904	D	ID4 (+)	15	15q21.3	11456	D	WDR72 (-)
6	6p24.1	8789	A	HIVEP1 (+)	15	15q21.3	17771	D	WDR72 (-)
6	6p24.3	8719	D	DSP (+)	15	15q23	17998	D	GLCE (+)
6	6p25.2	118748	D	BPHL (+)	15	15q24.1	6599	D	UACA (-)
6	6p25.3	5425	A	DUSP22 (+)	15	15q24.1	12833	D	ARIH1 (+)
6	6p25.3	11224	D	LOC285768 (-)	15	15q24.2	7198	A	PML (+)
6	6q11.1	76762	D	KHDRBS2 (-)	15	15q24.3	1804	A	CCDC33 (+)
6	6q12	4209	A	MCART3P (+)	15	15q24.3	65078	D	CYP1A1 (-)
6	6q12	18568	A	EYS (-)	15	15q25.1	3993	A	SCAPER (-)
6	6q12	12752	D	EYS (-)	15	15q25.1	34071	D	AGPHD1 (+)
6	6q12	16617	D	EYS (-)	15	15q25.1	38530	D	C15orf27 (+)
6	6q14.1	8758	A	BCKDHB (+)	15	15q25.1	53132	D	C15orf5 (-)
6	6q14.1	14524	A	IMPG1 (-)	15	15q25.3	15504	A	AGBL1 (+)
6	6q14.1	23173	A	IRAK1BP1 (+)	15	15q25.3	20904	D	CPEB1 (-)
6	6q14.1	2047	D	IRAK1BP1 (+)	15	15q26.1	15173	A	AGBL1 (+)
6	6q16.1	3360	D	EPHA7 (-)	15	15q26.1	10283	D	NCRNA00052 (+)
6	6q16.3	15319	A	GRIK2 (+)	15	15q26.1	13740	D	AGBL1 (+)
6	6q16.3	17760	D	GRIK2 (+)	15	15q26.1	15173	D	AGBL1 (+)
6	6q21	21074	A	FOXO3 (+)	15	15q26.2	1248	A	SLCO3A1 (+)
6	6q21	32479	A	RFPL4B (+)	15	15q26.2	2108	D	SLCO3A1 (+)
6	6q21	22188	D	C6orf186 (-)	15	15q26.2	5830	D	SLCO3A1 (+)
6	6q21	23412	D	RFPL4B (+)	15	15q26.2	11398	D	SLCO3A1 (+)
6	6q21	50211	D	LACE1 (+)	15	15q26.3	13608	D	NR2F2 (+)
6	6q22.1	26997	A	HS3ST5 (-)	15		16564	A	ADAMTS17 (-)
6	6q22.31	12976	A	NCOA7 (+)	15		69249	A	OR4F4 (-)
6	6q22.31	29055	D	C6orf170 (-)	15		20285	D	PCSK6 (-)
6	6q22.31	33725	D	GJA1 (+)	15	15q12	5105	A	SNORD116-23 (+)
6	6q22.33	7281	D	LAMA2 (+)	15	15q26.2	52804	D	FAM174B (-)
6	6q23.3	13409	D	BCLAF1 (-)	15	15q25.1	27879	D	SCAPER (-)
6	6q23.3	20107	D	FAM54A (-)	15	15q25.2	18081	A	FAH (+)
6	6q23.3	35277	D	BCLAF1 (-)	15	15q12	49561	A	SNORD116-28 (+)
6	6q23.3	325157	D	MAP3K5 (-)	15	15q26.3	16174	D	LOC91948 (-)
6	6q23.3	532558	D	FAM54A (-)	15	15q23	23826	D	DPP8 (-)
6	6q24.3	4118	A	SAMD5 (+)	15	15q24.2	21014	A	C15orf59 (-)
6	6q25.1	9796	D	ESR1 (+)	15	15q14	18313	D	MEIS2 (-)
6	6q25.1	11563	D	LATS1 (-)	15	15q26.3	23448	D	LOC145820 (+)
6	6q25.3	14720	A	ARID1B (+)	15	15q12	23059	D	SNORD109A (+)
6	6q25.3	15720	A	SLC22A3 (+)	15	15q26.2	20554	D	LOC145820 (+)
6	6q25.3	18203	A	IGF2R (+)	15	15q23	13675	A	CORO2B (+)
6	6q25.3	23705	A	TULP4 (+)	15	15q25.3	28551	D	TM6SF1 (+)
6	6q25.3	13050	D	SLC22A1 (+)	15	15q26.3	18309	A	ARRDC4 (+)
6	6q25.3	19178	D	SLC22A3 (+)	15	15q15.1	15166	D	C15orf54 (+)
6	6q26	1072	A	LPA (-)	15	15q22.2	19520	D	FAM81A (+)
6	6q26	2011	A	PLG (+)	15	15q14	23541	D	ZNF770 (-)
6	6q26	4803	A	PLG (+)	15	15q24.1	8112	D	THSD4 (+)

6	6q26	9302	A	PARK2 (-)	15	15q12	14013	A	SNRPN (+)
6	6q26	16392	A	PLG (+)	15	15q15.3	37930	D	RTF1 (+)
6	6q26	15199	D	PARK2 (-)	15	15q26.2	10845	A	SLCO3A1 (+)
6	6q26	19128	D	PARK2 (-)	15	15q26.1	14372	A	NTRK3 (-)
6	6q27	23099	A	MIR1913 (-)	15	15q21.1	17092	D	EIF3J (+)
6	6q27	18722	D	WDR27 (-)	15	15q24.2	2148	A	C15orf60 (+)
6	6p23	7925	A	JARID2 (+)	15	15q24.2	23888	A	C15orf60 (+)
6	6q21	6808	D	RFPL4B (+)	15	15q26.3	15079	D	TTC23 (-)
6	6p21.1	1605	A	KLC4 (+)	15	15q24.2	4830	A	C15orf60 (+)
6	6q22.33	18985	D	LAMA2 (+)	15	15q22.2	16498	D	AQP9 (+)
6	6p24.2	30103	D	SYCP2L (+)	15	15q26.1	10829	D	ABHD2 (+)
6	6q21	40709	D	CDK19 (-)	15	15q21.2	17755	D	COPS2 (-)
6	6q25.1	6598	D	ESR1 (+)	15	15q26.1	19879	A	ABHD2 (+)
6	6p22.3	18122	A	HDGFL1 (+)	15	15q26.3	13773	D	IGF1R (+)
6	6p22.3	23566	A	ID4 (+)	15	15q21.1	35213	D	CASC4 (+)
6	6q14.1	41439	D	MYO6 (+)	15	15q22.2	17684	A	RORA (-)
6	6q24.3	53348	D	SHPRH (-)	15	15q22.31	65401	D	HERC1 (-)
6	6q25.2	18313	D	OPRM1 (+)	15	15q23	26075	D	AAGAB (-)
6	6q15	27229	D	MDN1 (-)	16	16p11.1	28611	D	LOC283914 (+)
6	6q21	17017	D	FIG4 (+)	16	16p11.1	28675	D	LOC146481 (-)
6	6p21.1	13705	A	UNC5CL (-)	16	16p11.2	3251	A	SLC6A10P (-)
6	6q22.31	16527	A	NKAIN2 (+)	16	16p11.2	16946	A	SLC6A10P (-)
6	6q16.1	16929	D	EPHA7 (-)	16	16p11.2	30047	A	SLC6A10P (-)
6	6p12.3	16478	D	TFAP2B (+)	16	16p11.2	63267	D	HERC2P4 (-)
6	6q22.31	11445	A	RNF217 (+)	16	16p12.1	10365	D	HS3ST4 (+)
6	6p21.2	13799	D	TDRG1 (+)	16	16p12.1	21423	D	LOC653786 (+)
6	6p22.1	54155	D	HIST1H2AH (+)	16	16p12.1	33642	D	C16orf65 (-)
6	6q21	31299	A	FYN (-)	16	16p12.3	3528	A	XYLT1 (-)
6	6p21.1	25793	A	MED20 (-)	16	16p12.3	6125	A	XYLT1 (-)
6	6q14.1	11782	D	IRAK1BP1 (+)	16	16p12.3	22332	A	XYLT1 (-)
6	6q14.3	31137	D	SYNCRIP (-)	16	16p12.3	5475	D	XYLT1 (-)
6	6q25.1	7096	A	UST (+)	16	16p13.11	24955	D	MYH11 (-)
6	6q25.1	14837	A	UST (+)	16	16p13.11	88692	D	MPV17L (+)
6	6q14.1	9812	A	HMGN3 (-)	16	16p13.12	8115	A	SNX29 (+)
6	6p22.3	18368	D	DEK (-)	16	16p13.12	13075	A	SNX29 (+)
6	6p22.3	13474	A	DEK (-)	16	16p13.12	10428	D	MKL2 (+)
6	6p21.31	8172	A	ETV7 (-)	16	16p13.13	35716	A	TEKT5 (-)
6	6q21	28560	A	PPIL6 (-)	16	16p13.2	16208	A	A2BP1 (+)
6	6q23.3	5319	A	MYB (+)	16	16p13.2 - 16p13.13	10833	D	GRIN2A (-)
6	6p21.32	6288	A	SYNGAP1 (+)	16	16p13.3	6420	A	SRL (-)
6	6q23.2	11793	D	SNORA33 (+)	16	16p13.3	35362	A	TCEB2 (-)
6	6p22.3	8719	A	MIR548A1 (+)	16	16p13.3	5182	D	A2BP1 (+)
6	6q14.3	16032	A	TBX18 (-)	16	16p13.3	33052	D	TESSP1 (+)
6	6p21.1	37226	D	USP49 (-)	16	16q12.1	13571	A	CBLN1 (-)
6	6q14.1	24526	A	UBE2CBP (-)	16	16q12.2	12292	A	FTO (+)
6	6q12	13865	D	EYS (-)	16	16q12.2	14556	A	RBL2 (+)
6	6p22.1	2515	D	PRSS16 (+)	16	16q12.2	21566	A	CHD9 (+)
6	6p22.1	22211	D	PRSS16 (+)	16	16q12.2	5317	D	CHD9 (+)

6	6p22.3	17780	D	ID4 (+)	16	16q12.2	9011	D	CHD9 (+)
6	6q22.33	23728	A	L3MBTL3 (+)	16	16q12.2	10416	D	CHD9 (+)
6	6q24.2	33984	A	AIG1 (+)	16	16q12.2	11151	D	CHD9 (+)
6	6q24.3	37119	A	EPM2A (-)	16	16q12.2	16725	D	CHD9 (+)
6	6q24.1	20663	D	GPR126 (+)	16	16q21	34699	D	CCL22 (+)
6	6q23.3	72439	A	HBS1L (-)	16	16q22.1	15912	A	TMC07 (+)
6	6q13	10187	A	COL9A1 (-)	16	16q22.1	33519	A	AGRP (-)
6	6q14.1	14624	D	HMG3 (-)	16	16q22.1	43330	A	NRN1L (+)
6	6q22.31	13652	A	HSF2 (+)	16	16q22.3	28504	A	HYDIN (-)
6	6p24.3	14653	D	HULC (+)	16	16q22.3	41648	A	CLEC18C (+)
6	6p12.1	13139	D	DST (-)	16	16q22.3	24467	D	PMFBP1 (-)
6	6p22.3	7786	A	ATXN1 (-)	16	16q22.3	36490	D	DDX19B (+)
7	7p11.1	5086	A	ZNF716 (+)	16	16q23.1	1168	A	CNTNAP4 (+)
7	7p11.2	12384	A	LANCL2 (+)	16	16q23.1	18436	D	ADAMTS18 (-)
7	7p11.2	23144	A	HPVC1 (-)	16	16q23.1	19059	D	ADAMTS18 (-)
7	7p12.1	14558	A	POM121L12 (+)	16	16q23.3	12927	D	C16orf61 (-)
7	7p12.1	14558	D	POM121L12 (+)	16	16q24.1	1235	D	KIAA0182 (+)
7	7p12.1	22361	D	COBL (-)	16	16q24.2	3986	A	FBXO31 (-)
7	7p12.1	32334	D	POM121L12 (+)	16	16q24.2	5683	A	FBXO31 (-)
7	7p13	28855	D	SEPT13 (-)	16	16q24.2	12834	A	FOX1L (+)
7	7p13	37556	D	IGFBP1 (+)	16	16q24.2	12911	A	FOXF1 (+)
7	7p14.1	1646	A	TARP (-)	16	16q24.2	13911	A	IRF8 (+)
7	7p14.1	1935	A	TARP (-)	16	16q24.3	14216	A	ZNF469 (+)
7	7p14.1	3559	A	TARP (-)	16		10806	A	SPG7 (+)
7	7p14.1	4352	A	TARP (-)	16		74439	A	ACSF3 (+)
7	7p14.1	4872	A	TARP (-)	16		47950	D	CBFA2T3 (-)
7	7p14.1	4874	A	TARP (-)	16	16p12.1	12539	A	PRKCB (+)
7	7p14.1	7791	A	TARP (-)	16	16q24.1	19128	A	CDH13 (+)
7	7p14.1	8437	A	TARP (-)	16	16q23.3	12336	D	MPHOSPH6 (-)
7	7p14.1	13197	A	TARP (-)	16	16q12.1	35572	A	N4BP1 (-)
7	7p14.1	1580	A	TARP (-)	16	16q24.3	11574	A	FBXO31 (-)
7	7p14.1	1716	A	AMPH (-)	16	16q12.2	5007	A	CHD9 (+)
7	7p14.1	1858	A	TARP (-)	16	16q22.3	18921	A	MARVELD3 (+)
7	7p14.1	1868	A	TARP (-)	16	16p13.13	15652	D	ZC3H7A (-)
7	7p14.1	2142	A	TARP (-)	16	16q23.2	4138	A	WWOX (+)
7	7p14.1	2225	A	TARP (-)	16	16q24.2	6213	A	FOXF1 (+)
7	7p14.1	2225	A	AMPH (-)	16	16q23.1	15157	D	PSMD7 (+)
7	7p14.1	2730	A	TARP (-)	16	16q12.2	10208	D	RBL2 (+)
7	7p14.1	3396	A	AMPH (-)	16	16p12.1	17111	A	HS3ST4 (+)
7	7p14.1	3559	A	TARP (-)	16	16q23.2	4805	D	WWOX (+)
7	7p14.1	4352	A	TARP (-)	16	16q24.2	13280	D	IRF8 (+)
7	7p14.1	4872	A	TARP (-)	16	16q24.1	2114	D	KIAA0513 (+)
7	7p14.1	4874	A	TARP (-)	16	16q23.1	8060	A	HTA (+)
7	7p14.1	4900	A	AMPH (-)	16	16q23.2	7922	A	WWOX (+)
7	7p14.1	5181	A	TARP (-)	16	16q12.1	2043	D	BRD7 (-)
7	7p14.1	6706	A	TARP (-)	16	16q23.1	31536	D	ADAT1 (-)
7	7p14.1	7791	A	TARP (-)	16	16q12.1	6471	A	N4BP1 (-)
7	7p14.1	7803	A	AMPH (-)	16	16q21	21128	D	CDH8 (-)

7	7p14.1	8127	A	TARP (-)	16	16q12.1	22878	D	BRD7 (-)
7	7p14.1	8437	A	TARP (-)	16	16p13.2	33277	D	C16orf72 (+)
7	7p14.1	14243	A	C7orf10 (+)	16	16q21	23396	D	ARL2BP (+)
7	7p14.1	24837	A	AMPH (-)	16	16q23.3	8624	A	MPHOSPH6 (-)
7	7p14.3	26371	D	MIR550-2 (+)	16	16q22.1	37076	A	CMTM3 (+)
7	7p15.1	30115	A	GARS (+)	16	16q22.1	9160	D	CDH5 (+)
7	7p15.2	21396	A	C7orf71 (+)	16	16q21	13923	A	LOC644649 (-)
7	7p15.3	5017	A	TWISTNB (-)	16	16p13.12	9007	D	ERCC4 (+)
7	7p21.2	3711	A	ETV1 (-)	16	16p13.12	7103	D	ERCC4 (+)
7	7p21.2	9742	A	ETV1 (-)	17	17p11.2	205983	A	C17orf51 (-)
7	7p21.2	11029	D	ETV1 (-)	17	17p11.2	18532	D	RAII (+)
7	7p21.2	14398	D	DGKB (-)	17	17p11.2	36511	D	ZNF624 (-)
7	7p21.2	20446	D	ETV1 (-)	17	17p11.2	57995	D	CCDC144B (-)
7	7p21.3	11964	A	NXPH1 (+)	17	17p11.2 - 17p11.1	36784	D	FLJ36000 (+)
7	7p21.3	2314	D	THSD7A (-)	17	17p12	5300	A	PMP22 (-)
7	7p21.3	9777	D	ARL4A (+)	17	17p13.1	15312	A	SHISA6 (+)
7	7p21.3	24752	D	COL28A1 (-)	17	17p13.2	21882	D	UBE2G1 (-)
7	7p22.1	24764	A	C1GALT1 (+)	17	17p13.3	27114	A	RAP1GAP2 (+)
7	7p22.1	13373	D	ZNF12 (-)	17	17p13.3	72227	A	RPH3AL (-)
7	7p22.3	19704	A	FAM20C (+)	17	17q11.2	26781	A	WSB1 (+)
7	7q11.21	1587	A	INTS4L2 (+)	17	17q11.2	94486	D	WSB1 (+)
7	7q11.21	19946	A	LOC643955 (-)	17	17q12	30850	D	RFFL (-)
7	7q11.21	20220	A	LOC643955 (-)	17	17q12	33592	D	LRRC37B2 (+)
7	7q11.21	24163	A	LOC728927 (+)	17	17q21.2	12510	D	TADA2A (+)
7	7q11.21	27528	A	VKORC1L1 (+)	17	17q21.31	2357	A	KRTAP9-4 (+)
7	7q11.21	33012	A	ZNF107 (+)	17	17q21.31	2822	A	KRTAP9-4 (+)
7	7q11.21	49528	A	LOC643955 (-)	17	17q21.31	3334	A	KRTAP9-4 (+)
7	7q11.21	73212	A	LOC643955 (-)	17	17q21.31	4823	A	KRTAP9-4 (+)
7	7q11.21	96534	A	INTS4L2 (+)	17	17q21.31	7669	A	KRTAP9-4 (+)
7	7q11.21	6835	D	ZNF92 (+)	17	17q21.31	8788	A	ETV4 (-)
7	7q11.21	12639	D	INTS4L2 (+)	17	17q21.31	2357	D	KRTAP9-4 (+)
7	7q11.21	12853	D	ZNF92 (+)	17	17q21.31	25464	D	KRTAP4-1 (-)
7	7q11.21	34036	D	LOC641746 (+)	17	17q21.32	16599	A	MAPT (+)
7	7q11.21	77062	D	INTS4L2 (+)	17	17q21.32	27640	D	DBF4B (+)
7	7q11.22	11192	A	AUTS2 (+)	17	17q22	26029	A	CA10 (-)
7	7q11.23	71828	A	SPDYE7P (-)	17	17q22	110612	D	SPAG9 (-)
7	7q11.23	1010	D	POMZP3 (-)	17	17q23.2	24148	A	RPS6KB1 (+)
7	7q11.23	6065	D	CCL26 (-)	17	17q24.1	18672	D	TBC1D3P2 (-)
7	7q11.23	19410	D	HIP1 (-)	17	17q24.1	113905	D	TANC2 (+)
7	7q21.3	11297	A	TAC1 (+)	17	17q24.3	8938	A	CACNG5 (+)
7	7q21.3	86404	A	MGC72080 (-)	17	17q25.1	1268	A	RPL38 (+)
7	7q22.1	19995	A	MUC17 (+)	17	17q25.1	8159	A	RPL38 (+)
7	7q22.1	17850	D	CYP3A7 (-)	17	17q25.1	34636	A	RPL38 (+)
7	7q22.1	24126	D	SMURF1 (-)	17	17q25.1	31998	D	CDC42EP4 (-)
7	7q22.1	29627	D	LRRC17 (+)	17	17q25.3	9325	A	TBC1D16 (-)
7	7q22.1	47545	D	CUX1 (+)	17	17p11.2	18183	D	ULK2 (-)
7	7q22.2	26580	D	SRPK2 (-)	17	17p13.1	6201	D	NTN1 (+)
7	7q22.3	37961	D	COG5 (-)	17	17q24.1	11991	D	BRIP1 (-)

7	7q31.1	1653	A	EIF3IP1 (-)	17	17p13.1	1392	A	SHISA6 (+)
7	7q31.1	1665	A	EIF3IP1 (-)	17	17p13.3	5431	D	SGSM2 (+)
7	7q31.1	1792	A	IMMP2L (-)	17	17q21.31	13411	A	ERBB2 (+)
7	7q31.1	1884	A	EIF3IP1 (-)	17	17q24.3	19308	D	PSMD12 (-)
7	7q31.1	16062	A	EIF3IP1 (-)	17	17p13.3	67238	D	FAM101B (-)
7	7q31.1	4022	D	EIF3IP1 (-)	17	17q22	24911	A	STXBP4 (+)
7	7q31.1	5661	D	EIF3IP1 (-)	17	17q21.32	32924	D	G6PC3 (+)
7	7q31.1	6587	D	NRCAM (-)	17	17q25.3	27068	D	MGAT5B (+)
7	7q31.1	11868	D	DOCK4 (-)	17	17q21.33	13326	A	ZNF652 (-)
7	7q31.1	15247	D	C7orf60 (-)	17	17q12	33687	A	CCL1 (-)
7	7q31.33	12132	D	GRM8 (-)	17	17q22	18539	A	TOM1L1 (+)
7	7q31.33	17185	D	POT1 (-)	17	17q21.33	16447	D	ZNF652 (-)
7	7q32.1	22708	A	RBM28 (-)	17	17p13.2 - 17p13.1	25847	A	ALOX12P2 (+)
7	7q32.1	58542	D	METTL2B (+)	17	17q24.2	37731	D	CCDC46 (-)
7	7q33	1190	A	LRGUK (+)	17	17q12	39522	A	TMEM132E (+)
7	7q33	3290	A	LRGUK (+)	17	17q21.31	9189	A	RARA (+)
7	7q33	3295	A	LRGUK (+)	17	17q22	20462	A	HLF (+)
7	7q33	7662	A	EXOC4 (+)	17	17p13.3	11277	A	SMG6 (-)
7	7q33	20383	A	PTN (-)	17	17q21.31	53536	A	RARA (+)
7	7q33	33182	A	SLC35B4 (-)	17	17q11.2	61979	D	LOC645851 (-)
7	7q33	3295	D	LRGUK (+)	17	17q22	6079	D	CA10 (-)
7	7q34	1034	A	TRY6 (+)	17	17q22	5420	A	HLF (+)
7	7q34	12225	A	PRSS1 (+)	17	17q11.2	30374	D	NUFIP2 (-)
7	7q34	13862	A	PRSS1 (+)	17	17q22	19101	A	SPOP (-)
7	7q34	25835	A	PRSS1 (+)	17	17p13.2	13028	A	TEKT1 (-)
7	7q34	26905	A	PRSS1 (+)	17	17q23.3	17585	A	BCAS3 (+)
7	7q34	48653	A	PRSS1 (+)	18	18p11.21	20743	A	SLMO1 (+)
7	7q34	73065	A	PRSS1 (+)	18	18p11.21	65554	A	ZNF519 (-)
7	7q34	1034	A	TRY6 (+)	18	18p11.21	192724	A	LOC644669 (-)
7	7q34	1162	A	TRYX3 (-)	18	18p11.21	56427	D	ANKRD30B (+)
7	7q34	3275	A	PRSS2 (+)	18	18p11.21	89640	D	LOC644669 (-)
7	7q34	7160	A	PRSS2 (+)	18	18p11.32	2891	A	C18orf2 (-)
7	7q34	10402	A	PRSS2 (+)	18	18q12.1	1313	A	FAM59A (-)
7	7q34	10549	A	TRYX3 (-)	18	18q12.1	1738	A	FAM59A (-)
7	7q34	12069	A	PRSS1 (+)	18	18q12.1	5697	A	C18orf34 (-)
7	7q34	12225	A	PRSS1 (+)	18	18q12.1	6538	A	AQP4 (-)
7	7q34	13862	A	PRSS1 (+)	18	18q12.1	12306	A	FAM59A (-)
7	7q34	13879	A	ZC3HAV1 (-)	18	18q12.1	15411	A	FAM59A (-)
7	7q34	14254	A	TRYX3 (-)	18	18q12.1	8917	D	FAM59A (-)
7	7q34	25835	A	PRSS1 (+)	18	18q12.1	15311	D	CDH2 (-)
7	7q34	26905	A	PRSS1 (+)	18	18q12.1	18906	D	CDH2 (-)
7	7q34	28471	A	PRSS1 (+)	18	18q12.2	2317	A	FHOD3 (+)
7	7q34	36585	A	PRSS1 (+)	18	18q12.3	1359	A	KC6 (-)
7	7q34	44595	A	PRSS1 (+)	18	18q12.3	4537	A	KC6 (-)
7	7q34	52624	A	TRYX3 (-)	18	18q12.3	17467	A	SYT4 (-)
7	7q34	80126	A	TRYX3 (-)	18	18q12.3	6842	D	PIK3C3 (+)
7	7q34	93625	A	PRSS1 (+)	18	18q21.1	14145	A	RNF165 (+)
7	7q34	10402	D	PRSS2 (+)	18	18q21.2	6504	A	MAPK4 (+)

7	7q35	7964	A	OR6B1 (+)	18	18q21.2	14935	D	DCC (+)
7	7q36.1	32241	D	ATP6V0E2 (+)	18	18q21.33	3094	D	MC4R (-)
7	7q36.1	77731	D	MLL3 (-)	18	18q21.33	3105	D	PMAIP1 (+)
7	7q36.3	16586	D	LOC100132707 (+)	18	18q22.1	41674	A	C18orf20 (-)
7		9737	A	VIPR2 (-)	18	18q22.1	1288	D	CDH19 (-)
7	7q31.31	11556	A	ING3 (+)	18	18q22.1	16040	D	CDH19 (-)
7	7q32.3	27822	A	PLXNA4 (-)	18	18q22.2	22891	A	TMX3 (-)
7	7p14.1	9192	D	INHBA (-)	18	18q22.2	4702	D	DSEL (-)
7	7q11.23	32195	A	PTPN12 (+)	18	18q22.2	6594	D	DSEL (-)
7	7q21.2	12012	D	CDK6 (-)	18	18q22.2	21703	D	TMX3 (-)
7	7p15.3	9987	A	MGC87042 (-)	18	18q22.3	36550	A	SOCS6 (+)
7	7p21.3	8013	D	ARL4A (+)	18	18q22.3	9633	D	NETO1 (-)
7	7q21.2	17259	A	CDK6 (-)	18		3216	D	SALL3 (+)
7	7q31.31	18361	D	ANKRD7 (+)	18	18p11.32	12934	A	NDC80 (+)
7	7q31.32	19200	A	PTPRZ1 (+)	18	18q12.3	4499	D	KC6 (-)
7	7q11.22	40051	A	AUTS2 (+)	18	18p11.31	13526	D	LOC642597 (-)
									LOC100130522
7	7q35	17701	D	MIR548F4 (-)	18		88428	A	(+)
7	7q36.1	7850	D	TMEM176A (+)	18	18q21.1	2776	A	SLC14A2 (+)
7	7q35	19887	D	TPK1 (-)	18	18q21.1	13931	A	SLC14A2 (+)
7	7q31.32	24845	D	PTPRZ1 (+)	18	18q21.1	1225	D	SMAD2 (-)
7	7q36.2	11665	A	DPP6 (+)	18	18q21.2	1382	A	MBD2 (-)
7	7q36.1	20441	D	LOC100128542 (+)	18	18q21.2	12915	A	MBD2 (-)
7	7q34	6540	D	BRAF (-)	18	18p11.31	14692	A	LOC642597 (-)
7	7p12.3	9883	A	TNS3 (-)	18	18q21.1	14487	A	SLC14A2 (+)
7	7q34	7318	D	BRAF (-)	18	18q22.3	18922	D	FBXO15 (-)
7	7p15.1	11202	A	JAZF1 (-)	18	18q12.3	11066	D	SYT4 (-)
7	7p21.3	14903	A	THSD7A (-)	18	18q12.2	25015	D	GALNT1 (+)
7	7q21.2	30445	D	AKAP9 (+)	18	18p11.22	10325	A	VAPA (+)
7	7p15.2	14221	D	OSBPL3 (-)	18	18p11.22	13659	D	FAM38B (-)
7	7q34	15930	D	BRAF (-)	18	18q21.1	28242	A	KIAA0427 (+)
7	7p14.2	30403	A	ELMO1 (-)	18	18q21.1	10624	A	LOXHD1 (-)
7	7p14.1	21840	A	VPS41 (-)	18	18p11.21	12834	A	MC2R (-)
7	7p15.3	23316	D	MPP6 (+)	18	18q21.1	21734	A	SETBP1 (+)
7	7p15.3	5171	A	SP4 (+)	18	18p11.31	10730	A	TMEM200C (-)
7	7p15.2	15867	D	NPVF (-)	18	18q21.32	3648	A	GRP (+)
7	7p12.3	20517	A	ABCA13 (+)	18	18q12.1	16170	A	KCTD1 (-)
7	7q31.1	18135	A	NRCAM (-)	18	18q21.2	3703	A	SMAD4 (+)
7	7p15.3	19416	A	RAPGEF5 (-)	18	18q21.33	9064	D	CDH20 (+)
7	7p15.2	18589	D	SKAP2 (-)	19	19p12	11355	A	RPSAP58 (+)
7	7p14.1	32266	A	GLI3 (-)	19	19p12	19277	A	ZNF676 (-)
8	8p11.1	92286	D	POTEA (+)	19	19p12	28659	D	ZNF676 (-)
8	8p11.21	1853391	A	ANK1 (-)	19	19p12 - 19p11	4290670	aUPD	LOC148189 (-)
8	8p11.21 - 8q11.1	4782349	A	FNTA (+)	19	19p13.2	3294	A	MUC16 (-)
8	8p11.23	75905	A	ADAM3A (-)	19	19p13.2	10705	A	MUC16 (-)
8	8p11.23	78598	A	ADAM5P (+)	19	19p13.2	25837	A	LASS4 (+)
8	8p11.23 - 8p11.21	854644	A	ADAM2 (-)	19	19p13.2	35553	A	MUC16 (-)
8	8p12	1298	A	NRG1 (+)	19	19p13.2	14162	D	MBD3L1 (+)

8	8p12	1873	A	NRG1 (+)	19	19q11	11814	D	LOC148145 (+)
8	8p12	2321	A	NRG1 (+)	19	19q11	11865	D	LOC148145 (+)
8	8p12	3915	A	NRG1 (+)	19	19q12	21993	A	CHST8 (+)
8	8p12	4067	A	NRG1 (+)	19	19q12	63801	A	TSHZ3 (-)
8	8p12	12555	A	NRG1 (+)	19	19q12	22944	D	NPHS1 (-)
8	8p12	5587208	A	RNF122 (-)	19	19q13.11	25718	D	LGALS4 (-)
8	8p12	1298	D	NRG1 (+)	19	19q13.2	40095	A	PSG6 (-)
8	8p12	2321	D	NRG1 (+)	19	19q13.2	17005	D	PSG5 (-)
8	8p12	3915	D	NRG1 (+)	19	19q13.2	40148	D	IRGC (+)
8	8p12	12410	D	NRG1 (+)	19	19q13.31	20221	A	MEIS3 (-)
8	8p12 - 8p11.23	942066	A	FGFR1 (-)	19	19q13.31	19783	D	NUCB1 (+)
8	8p21.2	1236	A	EBF2 (-)	19	19q13.31	25794	D	MEIS3 (-)
8	8p21.2	5504	A	ADAM7 (+)	19	19q13.31	26634	D	PPP1R15A (+)
8	8p21.2	23875	A	EBF2 (-)	19	19q13.31	34199	D	PLEKHA4 (-)
8	8p21.2	1148194	A	KCTD9 (-)	19	19q13.32	29801	D	SIGLEC11 (-)
8	8p21.2	5504	D	ADAM7 (+)	19	19q13.33	30604	A	LILRA2 (+)
8	8p21.2 - 8p12	6973463	A	ADRA1A (-)	19	19q13.32	11819	D	ZNF677 (-)
8	8p21.3	525833	A	SLC18A1 (-)	19	19q12	15190	A	(+)
8	8p21.3	3067439	A	EPB49 (+)	19	19p13.3	26640	A	TMEM146 (+)
8	8p21.3	9267	D	LZTS1 (-)	19	19q13.41	25972	A	ZNF671 (-)
8	8p21.3 - 8p21.2	1250006	A	ADAMDEC1 (+)	19	19q12	21280	A	ZNF536 (+)
8	8p22	17964	A	C8orf48 (+)	19	19p13.12	43305	D	LPHN1 (-)
8	8p22	3422	A	C8orf79 (+)	19	19q13.2	20612	A	CALM3 (+)
8	8p22	666303	A	C8orf79 (+)	19	19q11	17253	D	UQCRFS1 (-)
8	8p22	8634	D	SGCZ (-)	19	19q13.2	12352	A	PNMAL2 (-)
8	8p22 - 8p21.3	6071064	A	NAT1 (+)	19	19q11	1301	D	UQCRFS1 (-)
8	8p23.1	2842	A	FAM66A (+)	19	19q11	7130	D	UQCRFS1 (-)
8	8p23.1	3913	A	MIR598 (-)	19	19p13.13	35299	A	NFIX (+)
8	8p23.1	5482	A	MIR598 (-)	20	20p11.21	25642	A	ZNF337 (-)
8	8p23.1	6302	A	FAM86B2 (-)	20	20p11.21	26282	A	CST5 (-)
8	8p23.1	11249	A	MIR598 (-)	20	20p12.1	14252	A	MACROD2 (+)
8	8p23.1	17163	A	PPP1R3B (-)	20	20p12.3	1547	A	FERMT1 (-)
8	8p23.1	28233	A	DEFA3 (-)	20	20p12.3	2037	A	FERMT1 (-)
8	8p23.1	88548	A	FAM86B2 (-)	20	20p12.3	15241	A	C20orf196 (+)
8	8p23.1	171990	A	LONRF1 (-)	20	20p12.3	2037	D	FERMT1 (-)
8	8p23.1	1247512	A	CTSB (-)	20	20p13	1704	A	SIRPB1 (-)
8	8p23.1	1802811	A	MIR1322 (-)	20	20p13	3192	A	SIRPB1 (-)
8	8p23.1	2287528	A	FAM90A10 (+)	20	20p13	3658	A	SIRPB1 (-)
8	8p23.1	3861	D	MTMR9 (+)	20	20p13	6399	A	SIRPB1 (-)
8	8p23.1	3986	D	PPP1R3B (-)	20	20p13	10561	A	SIRPB1 (-)
8	8p23.1	6057	D	MIR598 (-)	20	20p13	12287	A	SIRPB1 (-)
8	8p23.1	9191	D	MCPHI (+)	20	20p13	2042	D	SIRPB1 (-)
8	8p23.1	21995	D	FAM86B2 (-)	20	20p13	6399	D	SIRPB1 (-)
8	8p23.1 - 8p22	289053	A	LOC340357 (-)	20	20p13	6591	D	TGM3 (+)
8	8p23.2	1753	A	MCPHI (+)	20	20q11.21	51713	D	COMMD7 (-)
8	8p23.2	2380	A	CSMD1 (-)	20	20q11.22	19181	A	BPIL1 (+)
8	8p23.2	5455	A	CSMD1 (-)	20	20q11.22	19738	D	TRPC4AP (-)

8	8p23.2	5846	A	CSMD1 (-)	20	20q12	3491	A	CHD6 (-)
8	8p23.2	7449	A	CSMD1 (-)	20	20q12	11026	A	MAFB (-)
8	8p23.2	13026	A	MCPH1 (+)	20	20q12	18253	A	MAFB (-)
8	8p23.2	21010	A	CSMD1 (-)	20	20q13.11	3439	D	PTPRT (-)
8	8p23.2	50283	A	CSMD1 (-)	20	20q13.12	23333	D	PLTP (-)
8	8p23.2	215393	A	CSMD1 (-)	20	20q13.13	16264	A	UBE2V1 (-)
8	8p23.2	349553	A	CSMD1 (-)	20	20q13.13	40095	A	LOC284749 (+)
8	8p23.2	510528	A	MCPH1 (+)	20	20q13.13	40858	A	CEBPB (+)
8	8p23.2	522396	A	CSMD1 (-)	20	20q13.13	41626	D	LOC100131496 (+)
8	8p23.2	730380	A	CSMD1 (-)	20	20q13.32	76148	D	HMGB1L1 (-)
8	8p23.2	1379863	A	CSMD1 (-)	20	20q13.33	13794	A	GMEB2 (-)
8	8p23.2	1021	D	MYOM2 (+)	20	20q13.33	24816	A	CDH4 (+)
8	8p23.2	1753	D	MCPH1 (+)	20	20q13.33	33537	D	ZBTB46 (-)
8	8p23.2	3492	D	MCPH1 (+)	20	20p12.1	7925	D	BFSP1 (-)
8	8p23.2	21010	D	CSMD1 (-)	20	20q11.23	27129	A	GDF5 (-)
8	8p23.2 - 8p23.1	752846	A	ANGPT2 (-)	20	20q13.33	28926	D	CDH4 (+)
8	8p23.3	4891	A	ERICH1 (-)	20	20p13	18959	D	SIRPA (+)
8	8p23.3	9589	A	ERICH1 (-)	20	20q13.12	32736	D	WFDC11 (-)
8	8p23.3	14485	A	DLGAP2 (+)	20	20p13	1053	A	SCRT2 (-)
8	8p23.3	557849	A	ZNF596 (+)	20	20p13	4282	A	SCRT2 (-)
8	8p23.3	844070	A	ERICH1 (-)	20	20p13	4282	D	SCRT2 (-)
8	8p23.3 - 8p23.2	827700	A	KBTBD11 (+)	20	20p13	10710	D	C20orf54 (-)
8	8q11.1	43653	A	BEYLA (+)	20	20q11.23	18022	D	PHF20 (+)
8	8q11.1	221608	A	BEYLA (+)	20	20p13	19641	A	ADRA1D (-)
8	8q11.1	439010	A	BEYLA (+)	20	20p12.1	39917	D	SEL1L2 (-)
8	8q11.1	43653	D	BEYLA (+)	20	20p13	23843	A	SCRT2 (-)
8	8q11.1 - 8q11.21	1184671	A	PRKDC (-)	20	20p11.21	79365	A	GINS1 (+)
8	8q11.21	1056429	A	PRKDC (-)	20	20q13.2	10911	D	DOK5 (+)
8	8q11.21	23224	D	PRKDC (-)	20	20p12.3	19277	D	MCM8 (+)
8	8q11.21 - 8q11.22	2517386	A	C8orf22 (+)	20	20q13.33	11751	A	EDN3 (+)
8	8q11.22	11253	D	SNTG1 (+)	20	20q13.31	23966	A	TFAP2C (+)
8	8q11.22 - 8q11.23	918766	A	PCMTD1 (-)	20	20q11.23	5186	A	RBL1 (-)
8	8q11.23	9624	A	NPBWR1 (+)	20	20p12.3	4235	D	PLCB1 (+)
8	8q11.23	580167	A	FAM150A (-)	20	20q11.23	1321	A	RBL1 (-)
8	8q11.23 - 8q12.1	2814154	A	MRPL15 (+)	20	20q11.23	3410	A	RBL1 (-)
8	8q12.1	1506	A	UBXN2B (+)	20	20q11.23	3410	D	RBL1 (-)
8	8q12.1	4022	A	CA8 (-)	20	20q11.23	31104	D	RBL1 (-)
8	8q12.1	8063	A	C8orf71 (+)	20	20q13.2	16887	D	PFDN4 (+)
8	8q12.1	15156	A	LYN (+)	20	20p12.3	37154	D	FERMT1 (-)
8	8q12.1	167355	A	C8orf71 (+)	20	20p12.1	43729	A	SEL1L2 (-)
8	8q12.1	732528	A	FAM110B (+)	20	20q12	27212	D	LOC339568 (-)
8	8q12.1	1552522	A	CHCHD7 (+)	20	20q13.33	10918	D	C20orf197 (+)
8	8q12.1	1698176	A	NSMAF (-)	20	20q11.23	4802	A	CTNBNB1 (+)
8	8q12.1	4022	D	CA8 (-)	20	20p11.23	8605	D	RIN2 (+)
8	8q12.1	6877	D	UBXN2B (+)	20	20q13.32	17659	D	C20orf85 (+)
8	8q12.1 - 8q13.1	6052769	A	TRIM55 (+)	20	20q13.31	18413	D	CBLN4 (-)
8	8q13.1 - 8q13.2	3124729	A	TRIM55 (+)	20	20q13.13	12197	A	PTPN1 (+)

8	8q13.2	12294	A	SULF1 (+)	20	20q11.23	3589	A	RBL1 (-)
8	8q13.2 - 8q13.3	3610855	A	LACTB2 (-)	20	20q11.23	2131	A	RBL1 (-)
8	8q13.3	6957	D	PRDM14 (-)	20	20q11.22	26554	D	MMP24 (+)
8	8q13.3	122592	D	PRDM14 (-)	20	20q11.23	9232	A	RBL1 (-)
8	8q13.3 - 8q21.11	2479637	A	STAU2 (-)	20	20q11.23	24208	A	RPRD1B (+)
8	8q21.11	9833	D	CRISPLD1 (+)	20	20p12.2	19374	D	SNAP25 (+)
8	8q21.12	29121	D	PKIA (+)	20	20q13.12	1563	A	WFDC2 (+)
8	8q21.13	15538	A	SNX16 (-)	20	20q13.12	23249	A	WFDC2 (+)
8	8q21.13	1533	D	FABP9 (-)	20	20p12.2	18640	D	BTBD3 (+)
8	8q21.2	3110	A	REXO1L2P (-)	20	20q13.13	7904	D	PTPN1 (+)
8	8q21.2	30748	A	RALYL (+)	20	20q11.21	28078	D	TPX2 (+)
8	8q21.3	8232	D	RIPK2 (+)	20	20q13.13	14922	A	KCNB1 (-)
8	8q21.3 - 8q22.1	2597902	A	SLC26A7 (+)	20	20q13.2	15643	A	TSHZ2 (+)
8	8q22.1	21450	A	C8orf38 (+)	20	20p11.22	21424	D	PAX1 (+)
8	8q22.1	47016	A	C8orf83 (-)	20	20q13.2	14987	D	TSHZ2 (+)
8	8q22.1	522067	A	TMEM67 (+)	20	20p11.21	37566	A	ENTPD6 (+)
8	8q22.1	1174392	A	CDH17 (-)	20	20q13.12	15009	A	RBPJL (+)
8	8q22.1 - 8q22.2	3225169	A	POP1 (+)	21	21q11.2	4662	A	POTED (+)
8	8q22.2 - 8q22.3	3632843	A	NCALD (-)	21	21q21.1	18237	A	NRIP1 (-)
8	8q22.3	7324	A	ODF1 (+)	21	21q21.1	24170	A	NCAM2 (+)
8	8q22.3	8168	A	ODF1 (+)	21	21q21.1	18757	D	PRSS7 (-)
8	8q22.3	8355	A	ODF1 (+)	21	21q21.1	49604	D	PRSS7 (-)
8	8q23.3	2338	A	CSMD3 (-)	21	21q21.2	4994	A	NCRNA00158 (-)
8	8q23.3	2548	A	CSMD3 (-)	21	21q21.2	8403	A	NCRNA00158 (-)
8	8q23.3	2666	A	TRPS1 (-)	21	21q21.2	12426	A	NCAM2 (+)
8	8q23.3	23440	A	TRPS1 (-)	21	21q21.2	8403	D	NCRNA00158 (-)
8	8q23.3	26567	A	MIR2053 (+)	21	21q21.2	8742	D	NCRNA00158 (-)
8	8q23.3	570414	A	MIR2053 (+)	21	21q22.12	153883	D	RUNX1 (-)
8	8q23.3	1708	D	TRPS1 (-)	21	21q22.12	303019	D	C21orf96 (-)
8	8q23.3	3838	D	TRPS1 (-)	21	21q22.12	-		
8	8q23.3	16973	D	TRPS1 (-)	21	21q22.13	43327	D	C21orf96 (-)
8	8q23.3	69488	D	EIF3H (-)	21	21q22.13	13969	A	HLCS (-)
8	8q23.3	69488	D	TRPS1 (-)	21	21q22.13	42061	A	CLDN14 (-)
8	8q23.3 - 8q24.21	13469893	A	TRMT12 (+)	21	21q22.13	205336	D	MIR802 (+)
8	8q24.21	8719	A	MIR1208 (+)	21	21q22.13	171730	D	MIR802 (+)
8	8q24.21 - 8q24.22	5961468	A	PHF20L1 (+)	21	21q22.2	19586	D	SH3BGR (+)
8	8q24.22	12612	A	ZFAT (-)	21	21q22.2	19505	A	DSCAM (-)
8	8q24.22	432735	A	ZFAT (-)	21	21q22.3	19505	A	DSCAM (-)
8	8q24.22	1753	D	WISP1 (+)	21	21q22.12	30930	A	CLIC6 (+)
8	8q24.22 - 8q24.23	1395235	A	KHDRBS3 (+)	21	21q22.3	1114	D	TMPRSS2 (-)
8	8q24.23	1906218	A	FAM135B (-)	21	21q21.2	13535	A	NCAM2 (+)
8	8q24.23	4968	D	KHDRBS3 (+)	21	21q22.12	12944	A	ITSN1 (+)
8	8q24.23 - 8q24.3	1564067	A	COL22A1 (-)	21	21q22.3	24146	A	DSCAM (-)
8	8q24.3	50341	A	TRAPPC9 (-)	21	21q22.12	7508	A	RCAN1 (-)
8	8q24.3	50341	A	TRAPPC9 (-)	21	21q21.3	22195	A	N6AMT1 (-)

8	8q24.3	52513	A	HEATR7A (+)	21	21q22.12	1118	D	C21orf96 (-)
8	8q24.3	71506	A	TRAPPC9 (-)	21	21q22.12	26225	D	C21orf96 (-)
8	8q24.3	546277	A	TRAPPC9 (-)	21	21q22.3	16708	A	SIK1 (-)
8	8q24.3	3925155	A	FLJ43860 (-)	21	21q22.12	34544	D	RUNX1 (-)
8	8q24.3	6580	D	GPIHBP1 (+)	21	21q22.13	11966	D	SETD4 (-)
8	8q24.3 -	1069134	A	MIR939 (-)	21	21q22.2	1369	A	ERG (-)
									NCRNA00159
8	8q13.3	6373	D	PRDM14 (-)	21	21q22.11	16034	D	(+)
8	8q21.13	1306	A	FABP9 (-)	21	21q21.2	11569	D	NCAM2 (+)
8	8q21.13	1306	D	FABP9 (-)	21	21q22.13	181673	D	MIR802 (+)
8	8q11.22	24311	A	PXDNL (-)	21	21q22.11	10896	D	TCP10L (-)
8	8q23.3	2933	A	CSMD3 (-)	21	21q22.2	23071	A	ETS2 (+)
8	8q24.23	6894	A	KHDRBS3 (+)	21	21q22.13	42563	D	MIR802 (+)
8	8q21.3	46901	A	MMP16 (-)	21	21q21.1	20994	D	NCAM2 (+)
8	8p21.3	7011	A	CSGALNACT1 (-)	21	21q22.2	26246	D	BRWD1 (-)
8	8p21.3	7011	D	CSGALNACT1 (-)	21	21q22.11	23632	D	KRTAP22-1 (+)
8	8q11.23	25666	D	MRPL15 (+)	22	22q11.1 - 22q11.21	308454	A	POTEH (-)
8	8q22.2	20523	A	NIPAL2 (-)	22	22q11.1 - 22q11.21	308454	D	POTEH (-)
8	8q11.22	16003	D	SNTG1 (+)	22	22q11.21	5023	A	DGCR6 (+)
8	8p21.2	14314	A	EBF2 (-)	22	22q11.21	11191	A	COMT (+)
8	8q24.22	9776	A	ZFAT (-)	22	22q11.21	31429	A	GNB1L (-)
8	8q24.3	26724	A	TRAPPC9 (-)	22	22q11.21	32350	A	DGCR8 (+)
8	8q24.3	10008	A	TRAPPC9 (-)	22	22q11.21	36995	A	DGCR2 (-)
8	8q21.11	15223	D	PXMP3 (-)	22	22q11.21	37703	A	TRMT2A (-)
8	8q24.23	14149	A	FAM135B (-)	22	22q11.21	16905	D	CLTCL1 (-)
8	8q24.22	87320	A	ZFATAS (+)	22	22q11.21	663188	D	OR11H1 (-)
8	8p11.21	21056	A	ZMAT4 (-)	22	22q11.22	1974	D	LOC400891 (+)
8	8p21.3	14837	A	LZTS1 (-)	22	22q11.22	303755	D	POM121L8P (+)
8	8q12.3	26105	D	NKAIN3 (+)	22	22q11.23	16670	A	VPREB1 (+)
8	8q24.3	17478	A	TRAPPC9 (-)	22	22q11.23	17069	A	GNAZ (+)
8	8q24.3	17478	D	TRAPPC9 (-)	22	22q11.23	26509	A	VPREB1 (+)
8	8p21.3	15112	A	LOXL2 (-)	22	22q11.23	6595	D	TOP3B (-)
8	8q23.3	16506	D	EIF3H (-)	22	22q11.23	11887	D	TOP3B (-)
8	8q13.3	26915	A	KCNB2 (+)	22	22q11.23	13191	D	LOC96610 (+)
8	8q13.1	14092	A	TRIM55 (+)	22	22q12.1	5406	A	GSTTP1 (-)
8	8p11.21	24154	A	IKBKB (+)	22	22q12.1	11518	A	LRP5L (-)
8	8q22.3	11882	D	NACAP1 (+)	22	22q12.1	16421	A	TPST2 (-)
8	8q24.22	4238	A	ZFATAS (+)	22	22q12.1	19055	A	SEZ6L (+)
8	8q24.22	5414	A	ZFATAS (+)	22	22q12.1	29550	A	GSTTP2 (-)
8	8p21.3	21542	A	LZTS1 (-)	22	22q12.1	29700	A	MIAT (+)
8	8q21.11	12626	D	HNF4G (+)	22	22q12.1	14831	D	SEZ6L (+)
8	8q23.1	31218	D	ZFPM2 (+)	22	22q12.1	16385	D	MIR548J (-)
8	8q24.22	1412	A	ZFATAS (+)	22	22q12.2	22277	D	RFPL1S (-)
8	8q24.3	11750	D	COL22A1 (-)	22	22q12.2	26157	D	MTMR3 (+)
8	8q21.3	1001	A	TMEM64 (-)	22	22q12.3	12278	A	ISX (+)
8	8q21.3	32583	A	TMEM64 (-)	22	22q12.3	27945	A	PATZ1 (-)
8	8q22.3	21686	D	NCALD (-)	22	22q12.3	5134	D	LARGE (-)
8	8q21.13	23846	A	RALYL (+)	22	22q12.3	27333	D	DRG1 (+)

8	8q24.3	7858	A	KCNK9 (-)	22	22q13.1	12572	A	C1QTNF6 (-)
8	8q24.11	21645	D	MED30 (+)	22	22q13.1	42498	D	MYH9 (-)
8	8p12	4638	A	FGFR1 (-)	22	22q13.31	12815	A	TTC38 (+)
8	8p12	11164	D	FGFR1 (-)	22	22q13.31	12890	A	NFAM1 (-)
8	8q21.3	1293	A	TMEM64 (-)	22	22q13.31	19038	A	SCUBE1 (-)
8	8p12	17596	D	FUT10 (-)	22	22q13.31	19402	A	NFAM1 (-)
8	8q22.1	12207	D	C8orf37 (-)	22	22q13.31	3372	D	SAMM50 (+)
8	8q21.3	59068	D	WWP1 (+)	22	22q13.31	8251	D	KIAA1644 (-)
8	8q24.12	8646	D	DEPDC6 (+)	22	22q13.31	12890	D	NFAM1 (-)
8	8p12	31935	D	UNC5D (+)	22	22q13.31	25388	D	PRR5 (+)
8	8q12.1	24534	A	FAM110B (+)	22	22q13.31	46897	D	LOC730668 (+)
8	8q21.13	55589	D	ZBTB10 (+)	22	22q13.32	57470	A	GRAMD4 (+)
8	8q11.21	24775	A	SNAI2 (-)	22	22q13.33	14375	A	FAM19A5 (+)
8	8q22.1	13483	A	PDP1 (+)	22	22q13.33	7848	D	FAM19A5 (+)
8	8q11.23	22903	A	FAM150A (-)	22	22q13.33	14026	D	FAM19A5 (+)
9	9p11.2	8315	A	FAM27A (+)	22	22q13.1	10140	D	RBM9 (-)
9	9p11.2	63848	A	FAM27A (+)	22	22q13.31	4537	D	NFAM1 (-)
9	9p11.2	243315	A	FAM27A (+)	22	22q11.22	24158	D	CRKL (+)
9	9p11.2 - 9q12	23013442	A	FAM75A7 (-)	22	22q13.33	7497	D	FAM19A5 (+)
9	9p13.1	10656	A	CNTNAP3 (-)	22	22q12.3	27801	A	SYN3 (-)
9	9p13.1 - 9p11.2	6115844	A	ZNF658B (-)	22	22q12.3	22065	A	TCN2 (+)
9	9p13.2	7005	A	PAX5 (-)	22	22q12.3	19880	D	LARGE (-)
9	9p13.2 - 9p13.1	2378032	A	ALDH1B1 (+)	22	22q13.2	21216	D	ENTHD1 (-)
9	9p13.3	6006	A	PRSS3 (+)	22	22q13.2	6522	A	EP300 (+)
9	9p13.3	71328	A	UBE2R2 (+)	22	22q13.2	12955	D	EP300 (+)
9	9p13.3	715278	A	SUGT1P1 (-)	22	22q11.22	1748	D	SNAP29 (+)
					22	22q11.22	4214	D	SNAP29 (+)

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