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PRODUCT CATALOGUE

Discovering Life, Enriching Futures

DIOGNASTIC KITS

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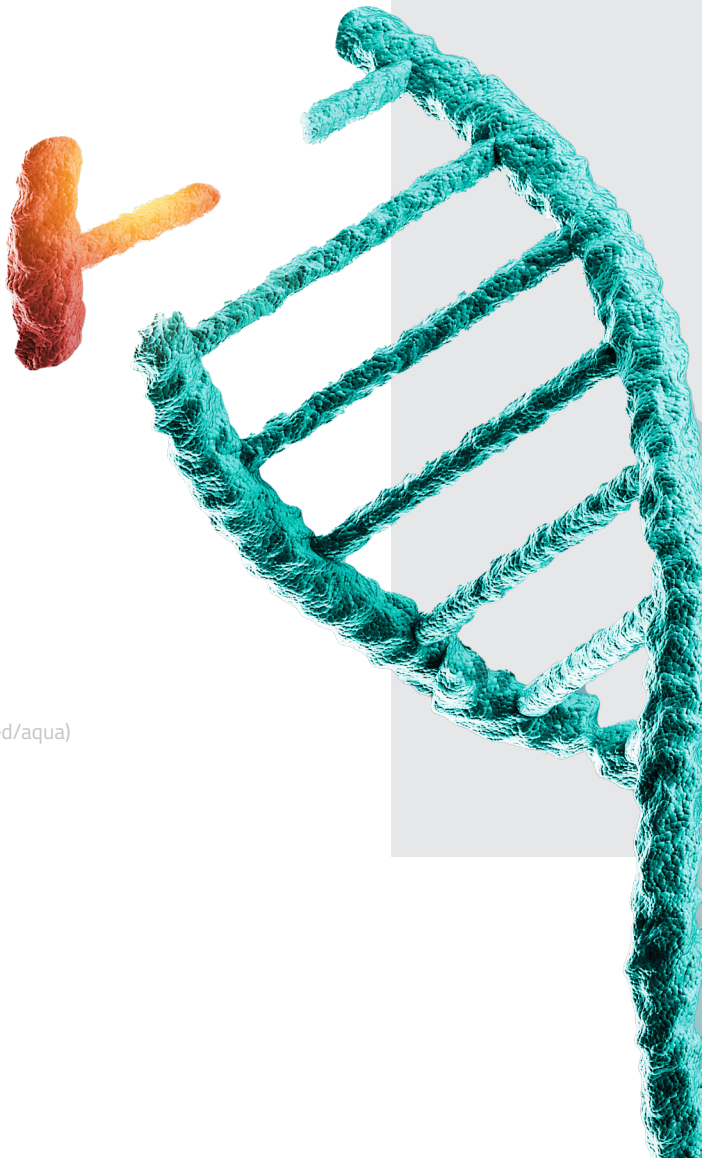


COMPANY PROFILE

Diagen Biotechnology Inc. has been established in 1998. At the year of 2008 it has launched R&D Department with cooperation with National Programmes and Universities which let company to start developing diagnostic kits on Molecular Genetics and Cytogenetic Diagnostic field.

Diagen has a frame which following the latest studies with latest technologies which allows us to have dynamic structure tor developing products on the fields of Medicine, Vet and Food safety.Now with high-tech end products which numbers are exceeding 200 Diagen exporting to vary of countries and supporting national economical growth with including national and international scientists. Since Covid-19 pandemic started Diagen focused on diagnostic kits and with development of Real Time PCR kits at 2020 company received confirmation from Ministiry of Health and launched sales and marketing activities abroad.

Developed Products

- SARS-CoV-2 OneStep RT-PCR Kit (S-gene) SARS-CoV-2 OneStep RT-PCR Kit (S-ORFlab)
- Sars-Cov-2 (Covid-19) Direct OneStep RT-PCR kit (S/ORFlab) (including fast extraction kit) VTM Solution (Viral Transport Medium)
- Extraction kits which takes 10 mins (Thermal/Centrifugal/Benchtop)
- SMA Real Time PCR kit
- Thrombophilia Panel Real Time PCR kit
- FMF Panel Real Time PCR kit
- Y microdeletion PCR diagnostic kit (13 microdeletion recognition)
- Species Designation tor Animals Real Time PCR kit
- Bacterial Menengitis Diagnosis kit (Pneumococcus, Meningococus, and Influenza)
- With FISH probe panels (Prenatal, Postnatal, Microdeletion, Hematologic and Pathologic probes) numbers are exceeding 200.

Certification

- ISO 9001:2015 Quality Management System
- ISO 14001:2015 Environmental Management System
- ISO 13485:2016 Medical Equipment Quality Management System TSE-HYB

Brands

- primeFISH
- AzoPlex

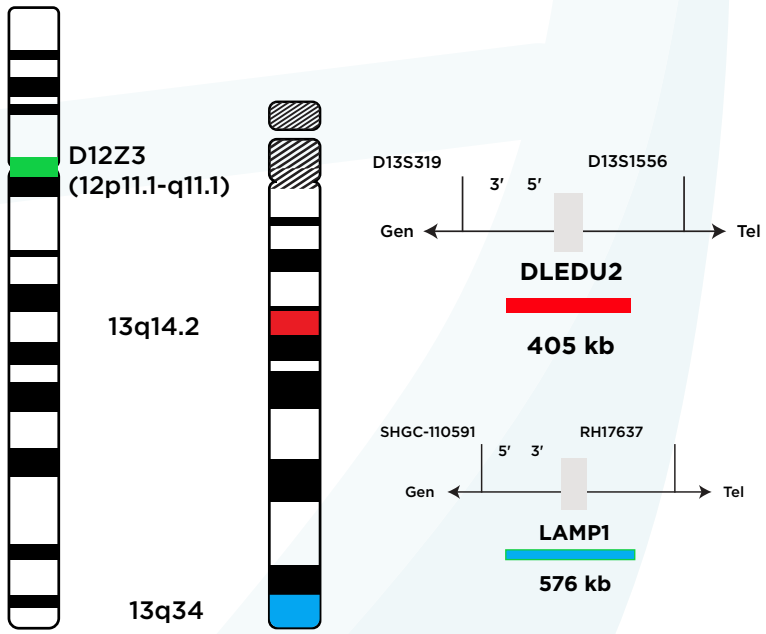
primeFISH Products

HEMATOLOGY PANEL

17-001-A del (13q14) (D13S319) /LAMP1 (13q34) / Cent 12

Deletions covering the 13ql4 region are frequently seen in a wide range of hema tological disorders. Chromosome 13q de letions occur in 16-40% of multiple myelo ma (MM) cases, and most are complete ly monosomy 13 (85%); the remaining 15% constitutes the deletion of 13ql4. A case study of patients with multiple myeloma narrowed the critically deleted region to 13ql4. Historically, deletions of 13q have been associated with poor prognosis in MM, but its prognostic relevance is now believed to be related to its association with other concomitant genetic lesions. Deletions in the long arm of chromo some 13 are also frequently detected in patients with aggressive nonHodgkin lymphoma (NHL).

Trisomy 12 is another common chromo somal abnormality seen in CLL and is de tected in approximately 20% of CLL cases. Trisomy 12 is associated with a moderate prognostic outcome when there is only one abnormality. Therefore, when used in conjunction with other biomarkers, mor phology, and clinical information, FISH is a valuable tool far predicting disease pro gression and overall survival in CLL pa tients.



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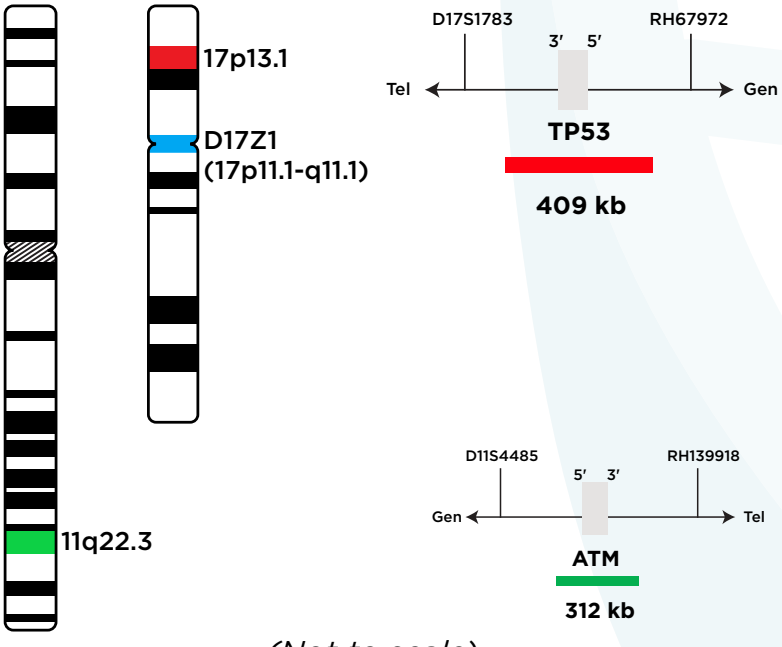
HEMATOLOGY PANEL

17-052 ATM/ TP53 / Cent 17

The tumor suppressor TP53 gene in the l7pl3.l region and the protein kinase ATM gene in the llq22.3 region are frequently deleted in chronic lymphocytic leukemia (CLL) conditions. Deletions of the TP53 (tumor protein 53; alsa known as p53) gene have been detected in patients with CLL, multiple myeloma (MM), and acute myeloid leuke mia (AML). Allelic loss of the short arm of chromosome 17 in CLL patients is associated with failure of treatment with alkylating agents and shorter survival times. The ATM (ataxia telangiectasia mutated) gene is located at llq22.3, and encodes a protein kinase involved in celi cycle regulation, including TP53 activation. CLL patients with llq deletion show rapid disease progression and low-grade survival.



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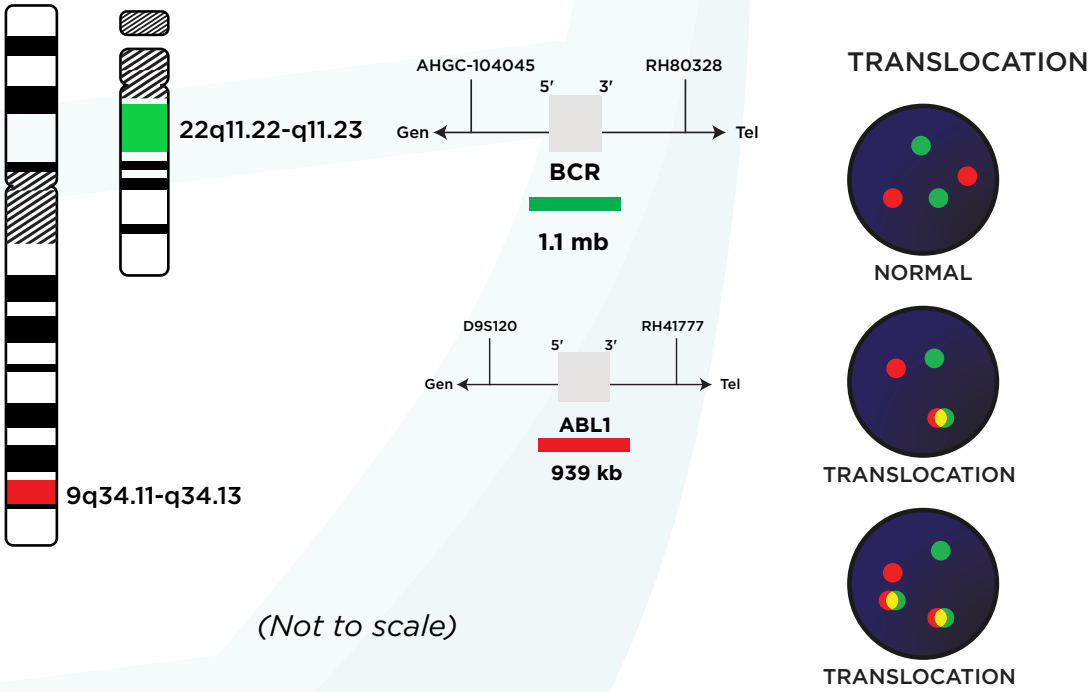
HEMATOLOGY PANEL

17-040 BCR-ABL t(9;22)

Rearrangements involving t(9;22)(q34.l;qll.2) are observed in approximately 90% of adults with chronic myeloid leukemia (CML) and approximately 25% of adults with acute lymphobalstic leukemia (ALL). The presence ofa BCR-ABLl fusion has important diagnostic and prognostic implications in various hematological diseases. Rearrange ments are characterized cytogenetically by the presence of the Philadelphia (Ph) chro mosome.

This translocation often results in a chimeric BCR/ ABLl fusion gene on the derivative 22nd chromosome. The product of this gene is the BCR/ ABLl protein with abnor-mal tyrosine kinase activity. in normal cells, ABLl kinase activity is well regulated in response to growth factors and other stimuli.



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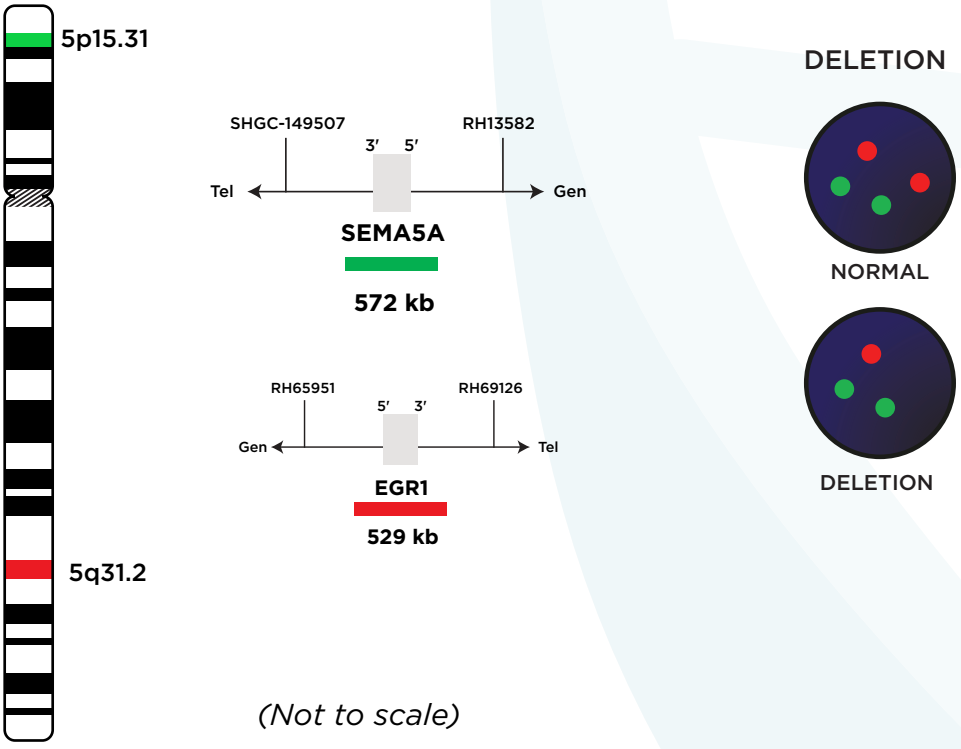
HEMATOLOGY PANEL

17-068 EGR1 del(5q31.2)

Deletions covering the 5q3l.2 region are one of the most common karyotypic abnor malities in myelodysplastic syndrome (MDS) and acute myeloid leukemia (AML) with changes due to myelodysplasia. in therapy-associated MDS or AML, 40% of patients show a 5q deletion. The fact that EGRl deletion is associated with higher tumor grade in estrogen receptor negative (ER-negative) breast carcinomas suggests that loss of the EGRl gene may contribute to the pathogenesis of ER-negative breast carcinomas. Transfusion-independent, lower-risk MDS patients with a 5q deletion are treated with the FDA-approved lenalidomide, a thalidomide analogue. Dicentric chromosomes, including chromosome 5, have often been observed in patients with de nova or thera py-associated MDS and AML. These patients often show a complex karyotype. in such conditions, characterization of rearrangement by conventional cytogenetics is hardly feasible. Therefore, FISH can be a useful tool far diagnosis and treatment decisions.



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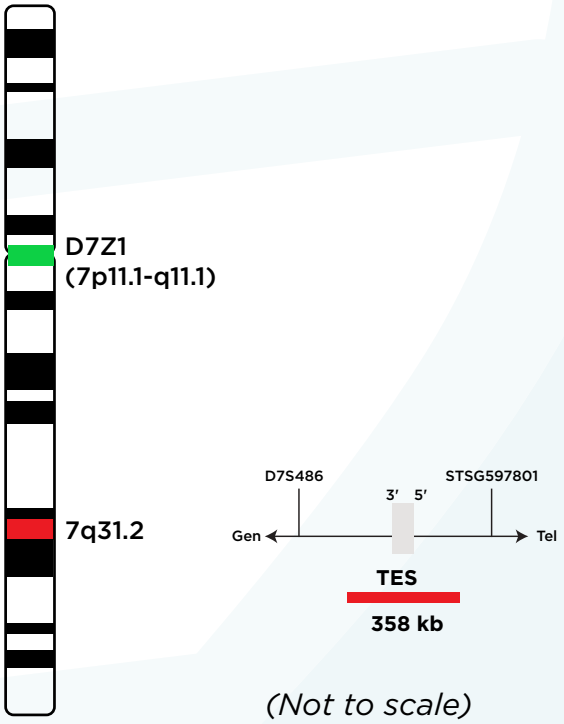
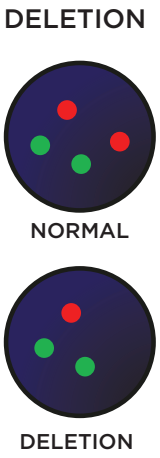
HEMATOLOGY PANEL

17-054 del(7q31.2)

Chromosome 7 monosomy and deletion of the long arm of chromosome 7 are recurrent chromosomal abnormalities frequently seen in myeloid diseases. Monosomy 7 and de1(7q); it is seen in many myeloid diseases, including myelodysplastic syndrome (MDS), acute myeloid leukemia (AML), and juvenile myelomonocytic leukemia (JMML). The pres ence of Monosomy 7 or del (7q) as karyotypic changes is associated with worse out comes in myeloid malignancies.





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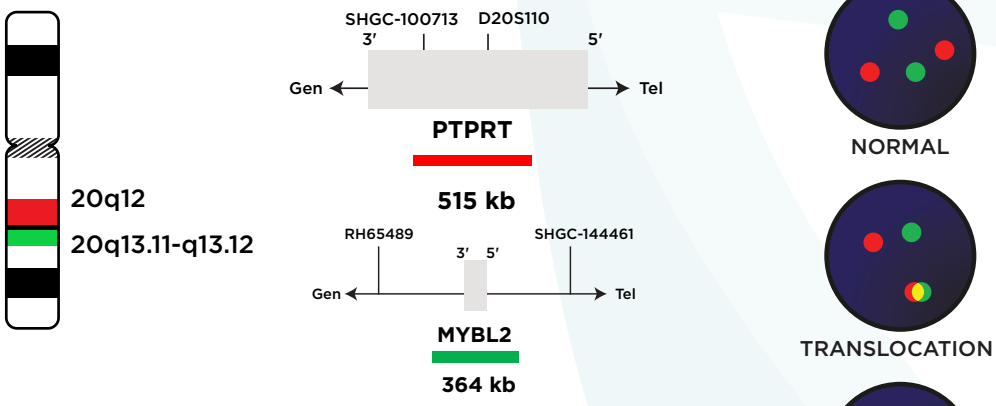
HEMATOLOGY PANEL

17-059 del 20q12-13

Chromosome 20q deletions can occur in a variety of myeloid disorders; for example, myelodysplastic syndromes (MDS), acute myeloid leukemia (AML) and myeloprolifer ative neoplasms (MPNs). in MDS, de1(20q) as the only cytogenetic abnormality is asso ciated with a favorable prognosis, better survival, anda lower risk of conversion to AML. De1(20q) with additional cytogenetic abnormalities predicts a poor prognosis. The 20q deletion is seen in approximately 2% of MDS cases. Patients with del (20q) base have an advantageous outcome compared to patients with additional abnormalities such as del (5q), del (7q), monosomy 7 and trisomy 8. Most de1(20q) patients have intersti tial deletion between 20qll.2 and 20ql3.3.



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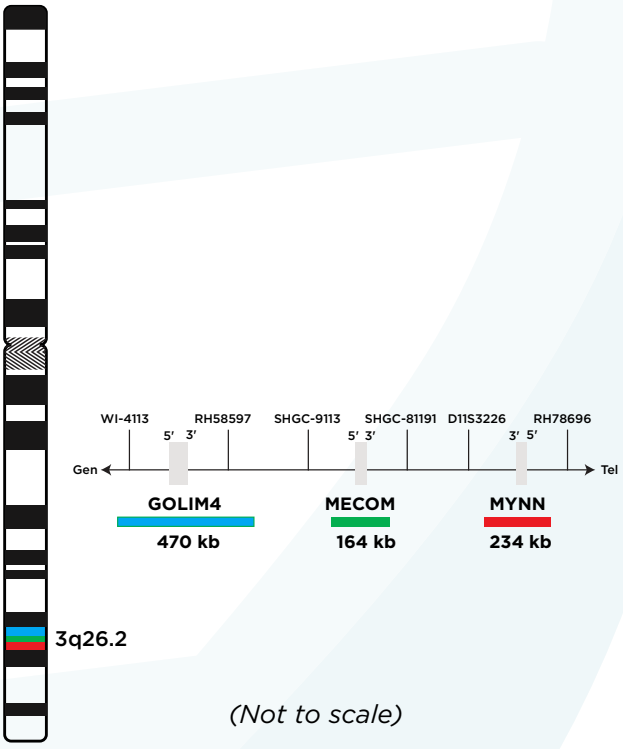
HEMATOLOGY PANEL

17-091 EV/1 (3q26.2) Breakapart

ınv(3)/t(3;3) and less commonly ins(3;3)(q26.2;q2lq26.2) occur in l-2.5% of acute myeloid leukemia (AML) and occur in myelodysplastic syndromes and chronic it is alsa observed in the blastic stage of myeloid leukemia. Various other MECOM translocations involving other fusion partner genes have alsa been reported in various types of myeloid malig nancies. 3q26.2 rearrangements are associated with minimal or no response to chemo therapy and poor clinical outcome. Chromosomal rearrangements involving the 3q26.2 region are associated with myeloid malignancies, abnormal expression of the MECOM gene, an unfavorable prognosis, and an aggressive clinical course.



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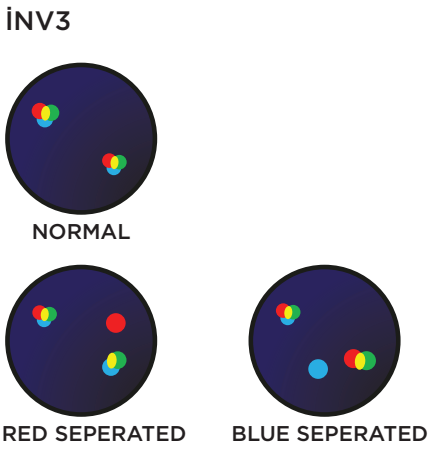

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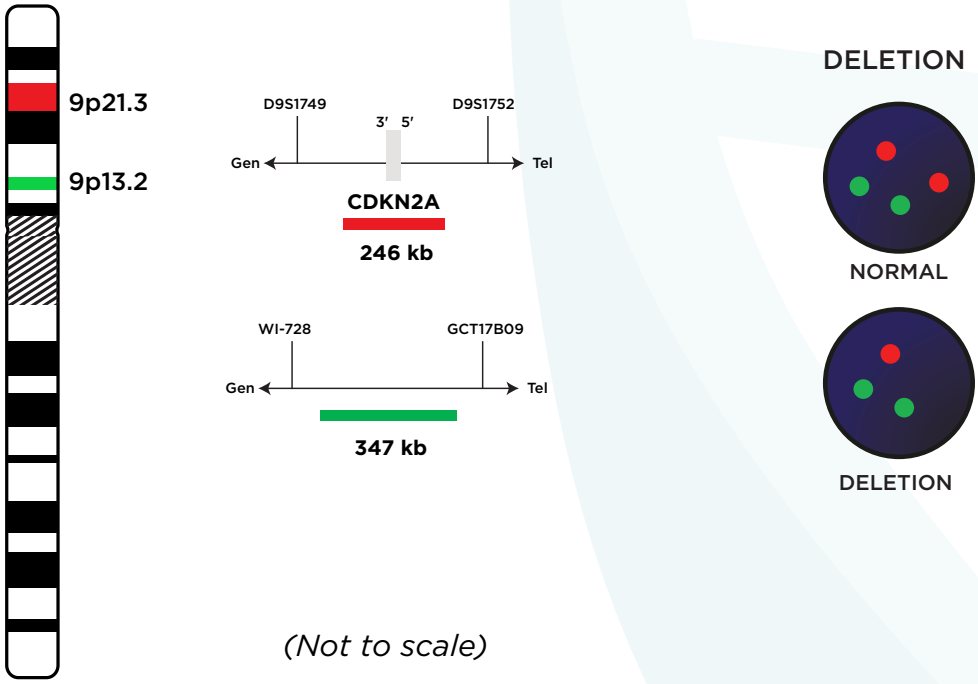
HEMATOLOGY PANEL

17-004 p16 (9p21

The CDKN2A (cyclin dependent kinase inhibitor 2A) gene, often referred to as pl6 or INK4a/ ARF, is located in the 9p2l.3 chromosome region. Using alternative first exons and an alternative reading frame, the gene encodes two different tumor suppressor proteins pl61NK4a and pl4ARF, both of which are involved in celi cycle regulation. CDK-N2A has been identified as a major susceptibility gene for melanoma. The tumor suppressor gene CDKN2A is inactivated by homozygous deletions with high frequency in various human primary tumors such as bladder and renal celi carcinomas, prostate and ovarian adeno car-cinomas, non-small celi lung cancer, sarcoma, glioma, mesothelioma, and melano ma. Furthermore, deletion of the CDKN2A gene is found in up to 80% of T-cell acute lym phoblastic leukemia cases and is associated with poor prognosis and disease relapse.



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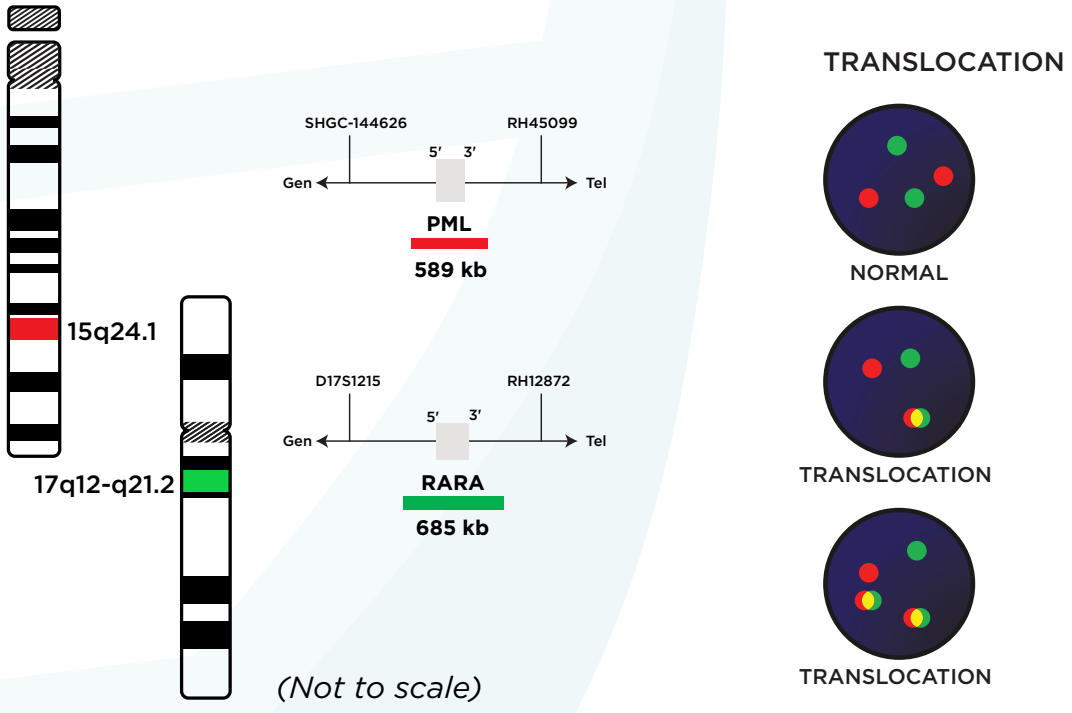
HEMATOLOGY PANEL

17-043 PML-RARA t(15;17

Translocations involving the PML (promyelocytic leukemia, also known as MYL) gene and the PARA (retinoic acid re-ceptor alpha, RARa) gene are thought to be characteristic of acute promyelocyt ic leukemia (APL), a subtype of acute myeloid leukemia. The PML-RARA fusion gene is created by the t(15;17)(q24;q21) translocation found in 90% of APL cases, a leukemia that accounts for 5-8% of acute myeloid leukemia (AML) cases. Variant RARA translocations may be observed in a subset of cases. Known fusion partners are NPMl at 5q35, NUMAl at llql3, ZBTB16 (PLZF) at llq23, STAT5B at 17q21, PRKARlA at 17q24, FIPlll at 4ql2, and BCOR at Xpll. Because the PML/RARA fusion is responsible for the response of these neoplasms to ali-trans retinoic acid (ATRA) therapy and other conven tional chemotherapy, it is important to correctly distinguish between t(15;17) translocations and those involving other RARA partners.



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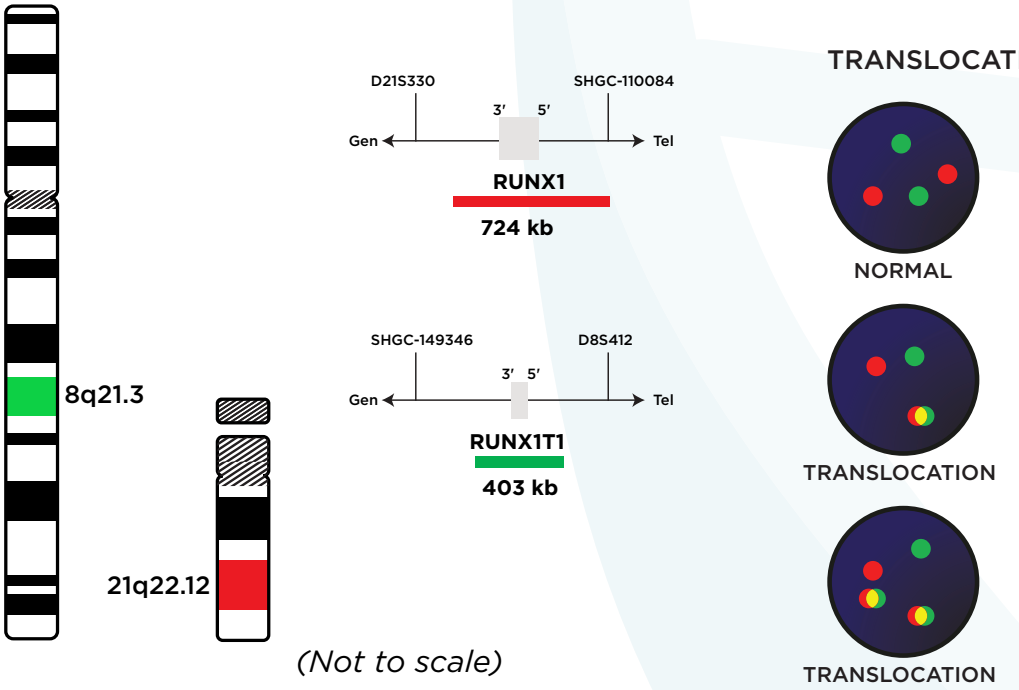
HEMATOLOGY PANEL

17-032 AML1-ETO t(8;21)

The t(8;21) balanced chromosomal translocation is found in approximately 90% of patients with acute myeloid leukemia (AML). AML is a heterogeneous clonal disorder of hematopoietic progenitor cells and is one of the most common malignant myeloid disorders in adults. Runt-associated transcription factor 1 gene (RUNXl) and RUNXl translocation partner 1 gene (RUNXlTl) are involved in the transcriptional regulation of genes during normal hematopoiesis. Translocation occurs in 10% to 22% of patients with AML FAB type M2 and in 5% to 10% of AML cases; it is most common in children and young adults2 and is a good prognostic indicator.



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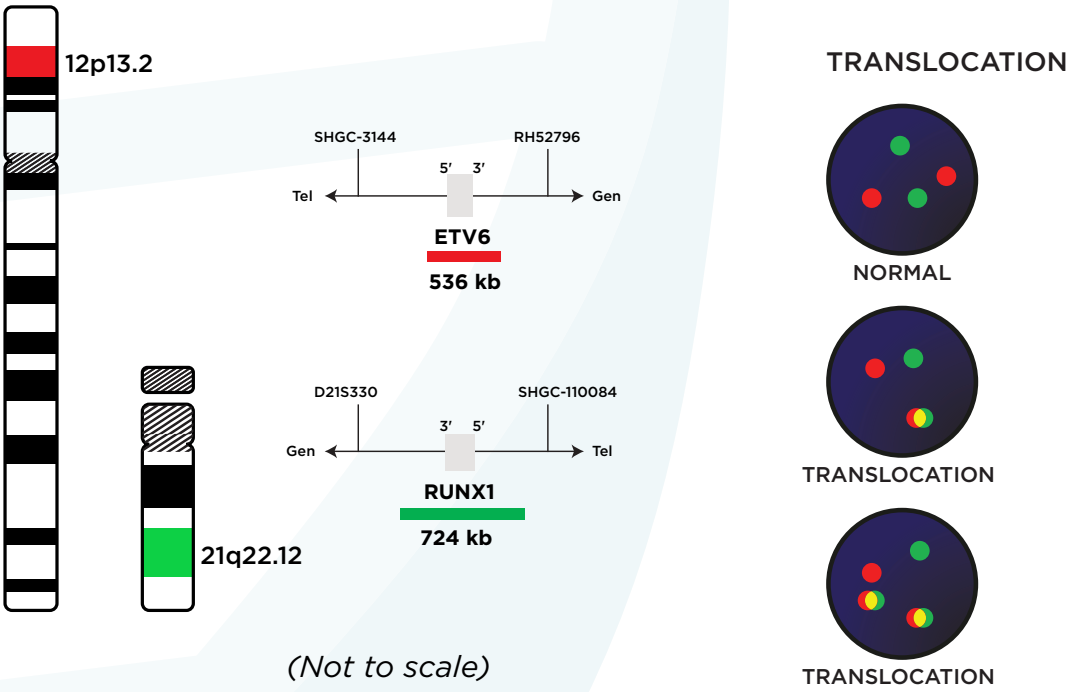
HEMATOLOGY PANEL

17-033 TEL-AML t(12;21)

t(12;21)(p13.2;q22.1) balanced chromosome translocation childhood onset B-cell precursor (BCP) is the most common genetic rearrangement in acute lymphoblastic leukemia (ALL) (19-27% of BCP-ALL) occurs in the early stages of pregnancy) and has been associated with a good progno sis. in ETV6/RUNX1 positive ALL, three secondary abnormalities have been found to impair the clini cal course: deletion of the second non-translocation ETV6 allele, gains of the RUNXl gene, and duplication of derivation 21. Detection of t(12;21) by fluorescent in situ hybridization (FISH) allows simultaneous detection of the most common secondary changes and provides additional infor mation about the likely outcome of the disease in ALL patients.



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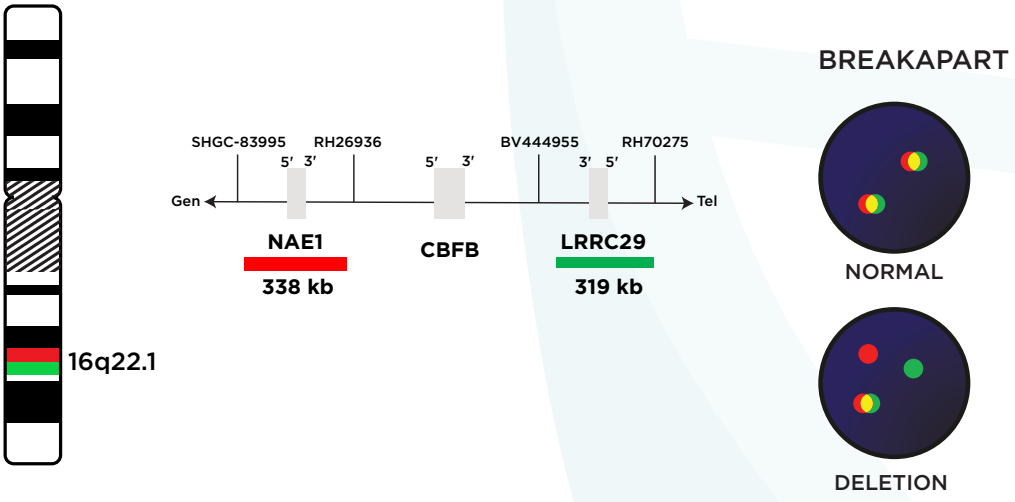
HEMATOLOGY PANEL

17-050 CBFB Breakapart

CBFB encodes the beta subunit of the CBFA/CBFB transcription factor complex involved in myeloid differ entiation. Chromosomal abnormalities of inv(16)(pl3.lq22.l) and its associated t(l6;16)(pl3.l;q22.l) translo cation detected in approximately 10% of AML (acute myeloblastic leukemia) patients it causes its fusion with the MYHll (smooth muscle myosin heavy chain) gene at l6pl3.l. Rearrangements of the CBFB gene are frequently found in patients with the myelomonocytic subtype with increased bone marrow eosinophils, termed AML FAB (French-American-British classification) type M4Eo, and are found in 5-8% of AMLs. This rearrangement may also occur in cases of treatment-related AML. lnversion inv(16)(pl3.llq22.l) or translo cation t(l6;16)(pl3.ll;q22.l) produces CBFB-MYHll gene rearrangements and is classified as a favorable cytogenetic risk group in patients with AML.



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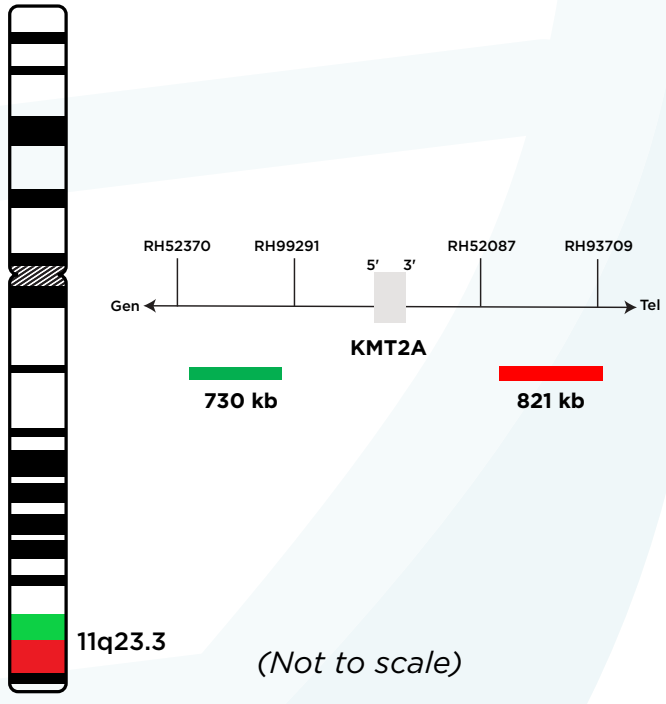
HEMATOLOGY PANEL

17-002 MLL (11q23) Breakapart

Up to 1-15% of cancer patients treated with KMT2A gene rearrangements with a DNA topoisomer ase il inhibitor develop therapy-associated leukemia (t-AML) associated with KMT2A transloca tions. Translocations involving the KMT2A gene are detected in 5-6% of all acute myeloid leuke mias (AML) and 5-10% of ali acute lymphoblastic leukemias (ALL). The frequency of translocations involving the KMT2A gene is significantly higher in infants with AML (50%) and ALL (80%). Over 30 fusion partners have been documented tor KMT2A; the most common translocations are t(4;11) and t(ll;l9) in ALL patients, t(6;11), t(9;11) and t(ll;l9) in AML patients. Overall, the finding of KMT2A re-arrangements in patients with acute leukemia indicates a less favorable prognosis. However, recent studies suggest that the specific KMT2A translocation partner may have an impact on response to therapy and overall prognosis, depending on the clinical concept.



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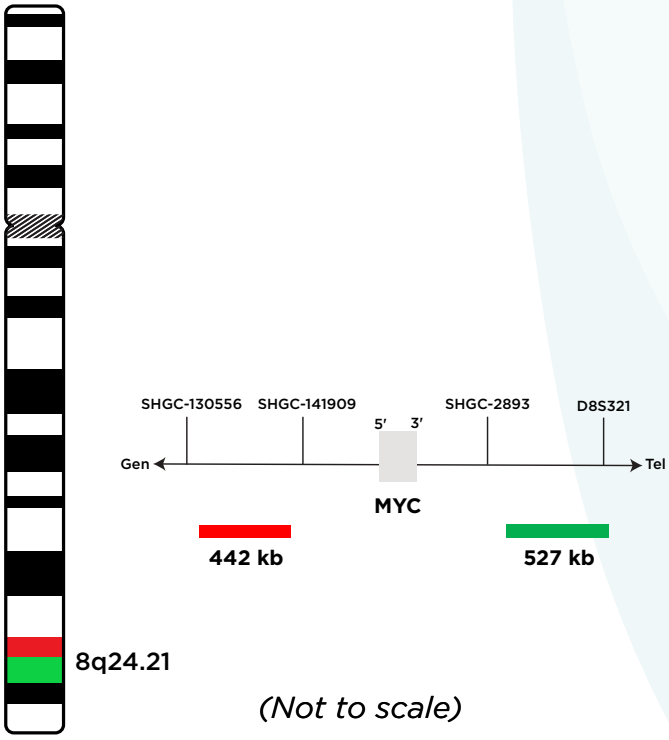
HEMATOLOGY PANEL

17-066 c-MYC (Bq24) Breakapart

The MYC proto-oncogene (MYC proto-oncogene, bHLH transcription factor, a.k.a. CMYC) encodes a transcription factor required tor celi growth and proliferation and is extensively involved in tumorigenesis. Translocations involving the MYC gene are thought to be the cyto genetic hallmark of Burkitt's lymphoma, but are also found in other lymphoma types. MYC rearrangements that activate MYC by translocation with one of the three immunoglobulin loci (IGH, IGL, or IGK) are detected in nearly all cases of Burkitt lymphoma at diagnosis. They are also seen in diffuse large B-cell lymphoma (DLBCL), multiple myeloma, and plasmablastic lymphoma, among other diseases.



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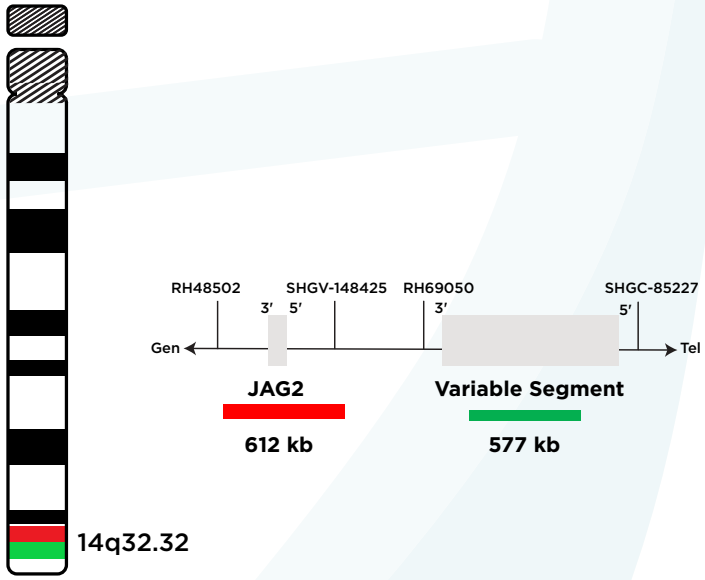
HEMATOLOGY PANEL

17-073 IGH (14q32) Breakapart

Rearrangements involving the IGH (immunoglobulin heavy chain) gene are considered to be cytogenetic markers of non-Hodgkin lymphoma (NHL). NHLs represent 50% of all hematologi cal malignancies. IGH gene rearrangements have been identified in approximately 50% of NHLs and are associated with specific subtypes of NHLs. Translocation of t(ll;l4)(ql3.3;q32.3) in approximately 95% of mantle celi lymphoma (MCL), 80% of t(l4;18)(q32.3;q2l.3) follicular lym phoma (FL) in , t(3;14)(q27;q32.3) is found in diffuse large B-cell lymphoma (DLBCL) and t(8;14)(q24.2l;q32.3) Burkitt's lymphoma. in all of these translocations, an oncogene located near the breakpoint of the translocation partner is activated by passing near the IGH regula tory sequences.



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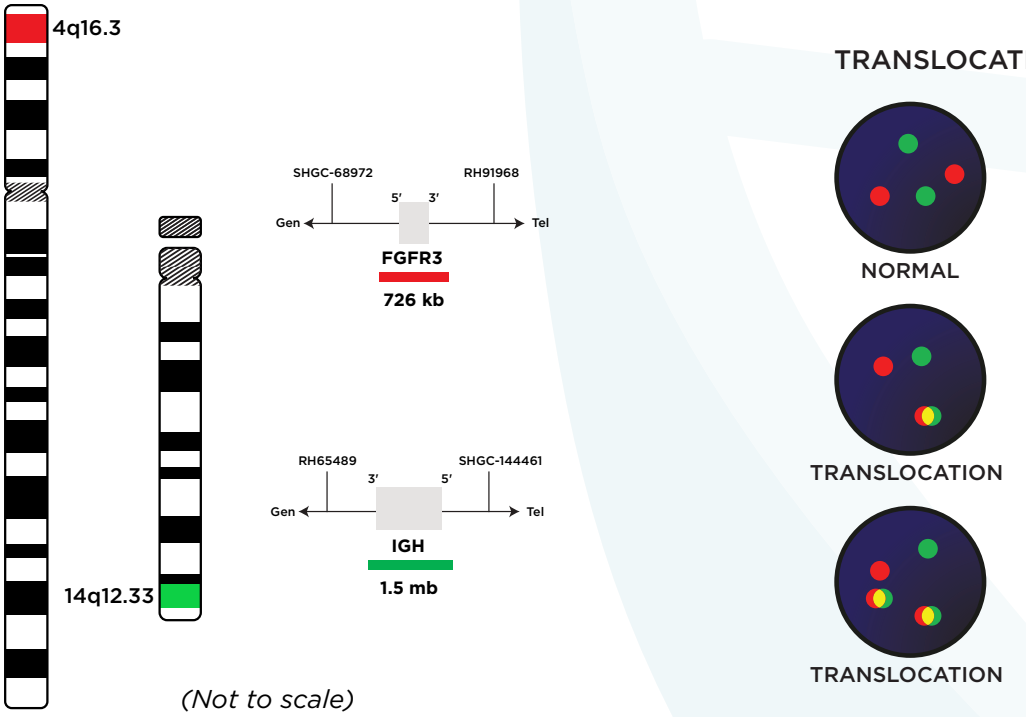
HEMATOLOGY PANEL

17-069 IGH-FGFR3 t(4;14)

The FGFR3 (fibroblast growth factor receptor 3) gene is located in the 4pl6.3 region, and the IGH (immunoglobulin heavy locus) is located in the 14q32.33 region. FGFR3 encodes a receptor tyrosine kinase that regulates the downstream signaling chain after ligand binding. Fusion of several part ner genes (including the IGH locus), often found in multiple myeloma (MM), can lead to ligand-in dependent activation of the tyrosine kinase of the re-sulting FGFR3 fusion protein. FGFR3/IGH trans locations are observed in approximately 15-20% of MM patients. Breakpoints for the 4Pl6.3 locus are located between the FGFR3 gene and the 5′ end of the NSD2 gene. The t(4;14)(pl6.3;q32.3) translo cation is associated with up-regulation of FGFR3 and myeloma NSD2 (also known as MMSET) portion protein. Patients with the FGFR3/IGH translocation show an overall poor prognosis, which is only partially alleviated by the use of the newer agents bortezomib and lenalidomide.



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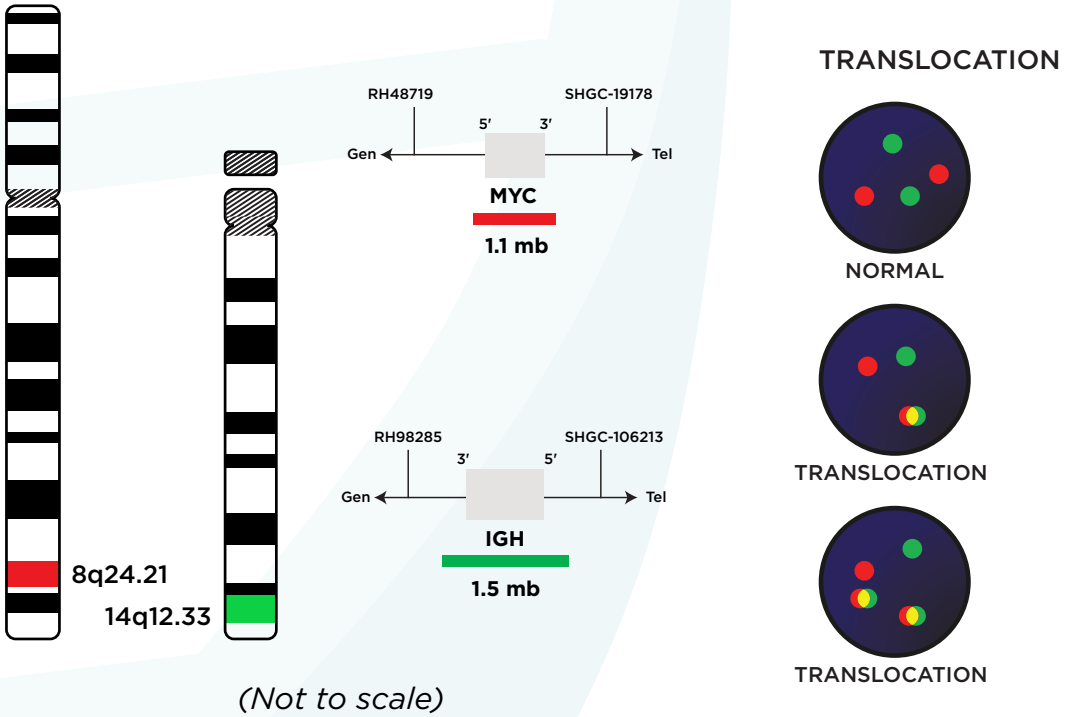
HEMATOLOGY PANEL

17-074 IGH-cMYC t(B;14)

MYC proto-oncogene (MYC proto-oncogene, bHLH transcription factor) encodes a transcrip tion factor required for celi growth and proliferation and is extensively involved in tumorigene sis. Burkitt lymphoma has the cMYC/IGH translocation, t(8;14)(q24;q32) and variant forms t(2;8)(pl3;q24) and t(8;22)(q24;qll) and is a mature B celi or Burkitt type Acute Lymphoblastic Leukemia (ALL) is also observed.The most common translocation t(8;14)(q24.2l;q32.3) involving the MYC gene region can be found in approximately 80% of Burkitt lymphoma cases and moves the MYC gene near the lgH (immunoglobulin heavy chain) locus. Other translocations affecting the MYC gene are t(8;22)(q24.2l;qll.2) and t(2;8)(pll.2;q24.21), both of which involve one of two immunoglobulin light chain loci.



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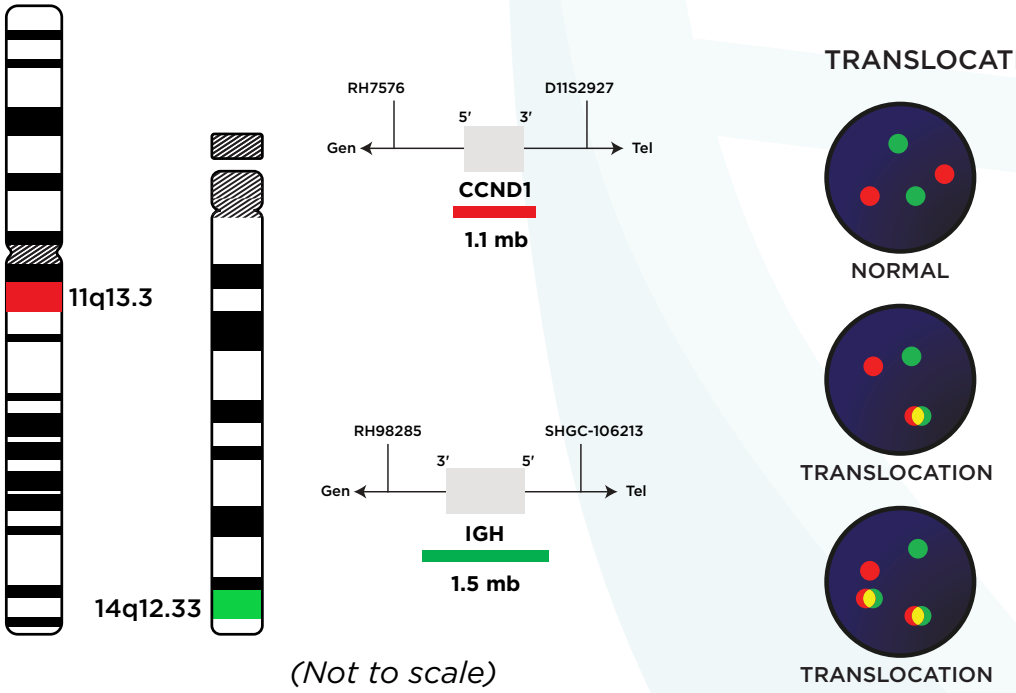
HEMATOLOGY PANEL

17-077 IGH-CCNDl t(11;14)

The translocation moves the CCNDl gene (also known as cyclin Dl; PRADl and BCLl) near the IGH (immunoglobulin heavy locus, also known as IGH®) locus, resulting in constitutive overexpression of CCNDl. The t(ll;l4)(ql3;q32) rearrangement involving CCNDl and IGH is considered indicative of mantle celi lymphoma (MHL), whose presence can be used to aid in the differential diagnosis of CD5+ B-cell lymphoproliferative diseases.

The t(ll;l4)(ql3.3;q32.3) translocation containing the CCNDl and IGH gene regions is detected in up to 95% of patients with mantle cell lymphomas (MCL) and is the genetic variant of this subtype of low-grade peripheral B-cell neoplasms considered to be an indicator. However, t(ll;l4) has also been detected in other lymphoproliferative disorders (LPDs) such as B-prolymphocytic leukemia (PLL) and less frequently in plasma celi myelomas, B-cell chronic lymphocytic leukemia, and villous lymphocyte splenic lymphomas (SLVL).



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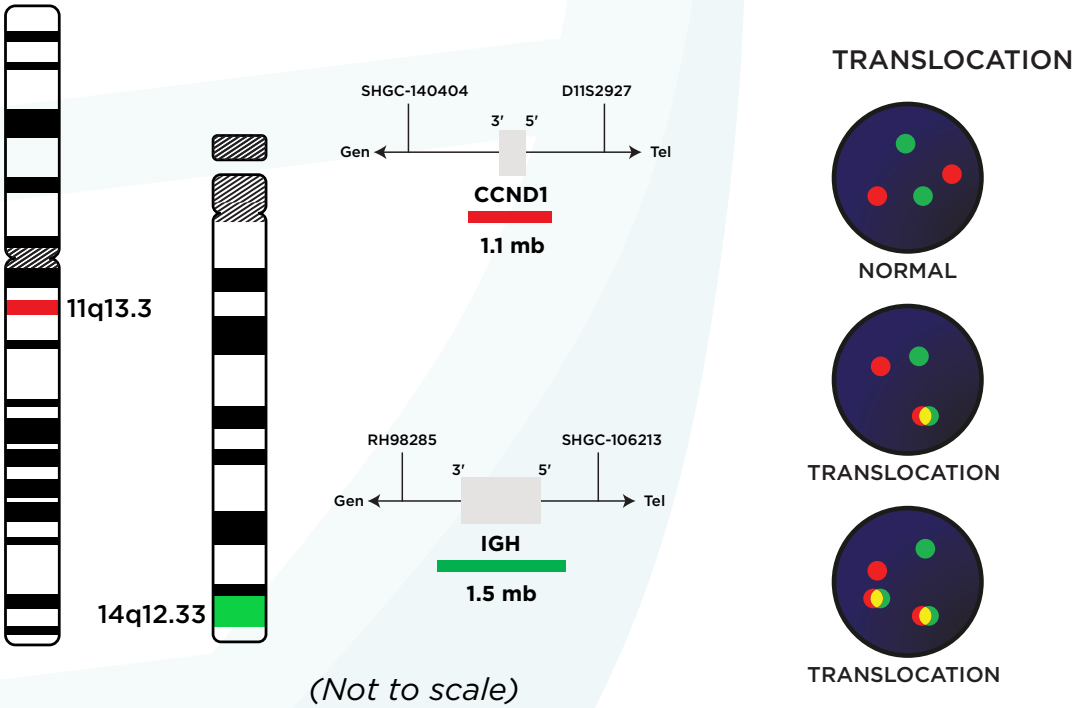
HEMATOLOGY PANEL

17-093 IGH-MYEOV t(11;14)

Mantle cell lymphoma (MCL) is a B-cell non-Hodgkin lymphoma (NHL) with an aggressive clini cal course. it is genetically characterized by t(ll;l4)(ql3;q32) found in approximately 95% of MCL patients. Less frequently, t(ll;l4) can also be detected in B-cell prolymphocytic leukemia, my elomas, and chronic lymphocytic leukemia. The t(ll;l4)(ql3;q32) translocation is the most common translocation in MM, occurring in approximately 15% of cases. Unlike IGH rearrange ments in other neoplasms, those found in MM have IGH breakpoints predominantly in the C/J region, placing the MYEOV gene under the control of the 3' Eal enhancer when in the MYEOV state. in contrast to CCNDl translocations, the Eμ enhancer controls CCNDl expression. MYEOV overexpression may be a possible prognostic factor in MM.



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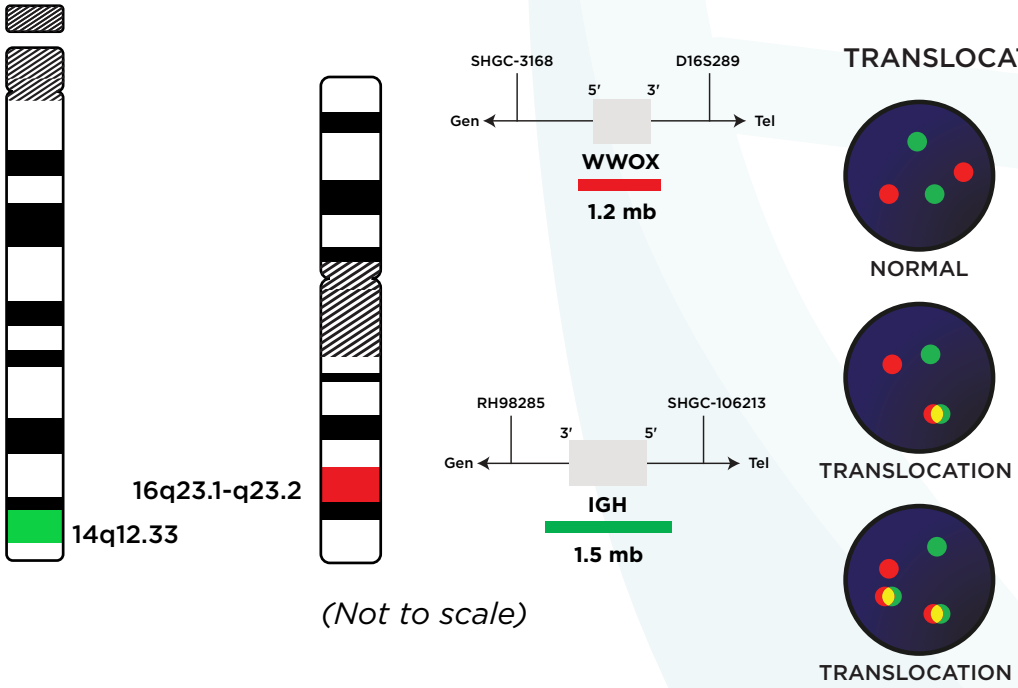
HEMATOLOGY PANEL

17-011 IGH-MAF t(14;16)

The MAF (MAF bZIP transcription factor) gene is located in the IGH (immunoglobin heavy locus) at 16q23 and 14q32.3. Approximately 50-60% of multiple myeloma (MM) cases involve translocations involving IGH and one of several partners such as CCNDl, NSD2 (WHSCl) and FGFR3, CCND3, MAF or MAFB. The t(l4; 16) (q32.3; q23) translocation is a recurrent translocation seen in 2-10% of MM. The t(l4;16) (q32.3;q23) translocation is frequently found in multiple myeloma (MM). MM is a malignant post-germinal center tumor of somatic-mutated, isotype-transformed plasma cells that accu mulate in the bone marrow.



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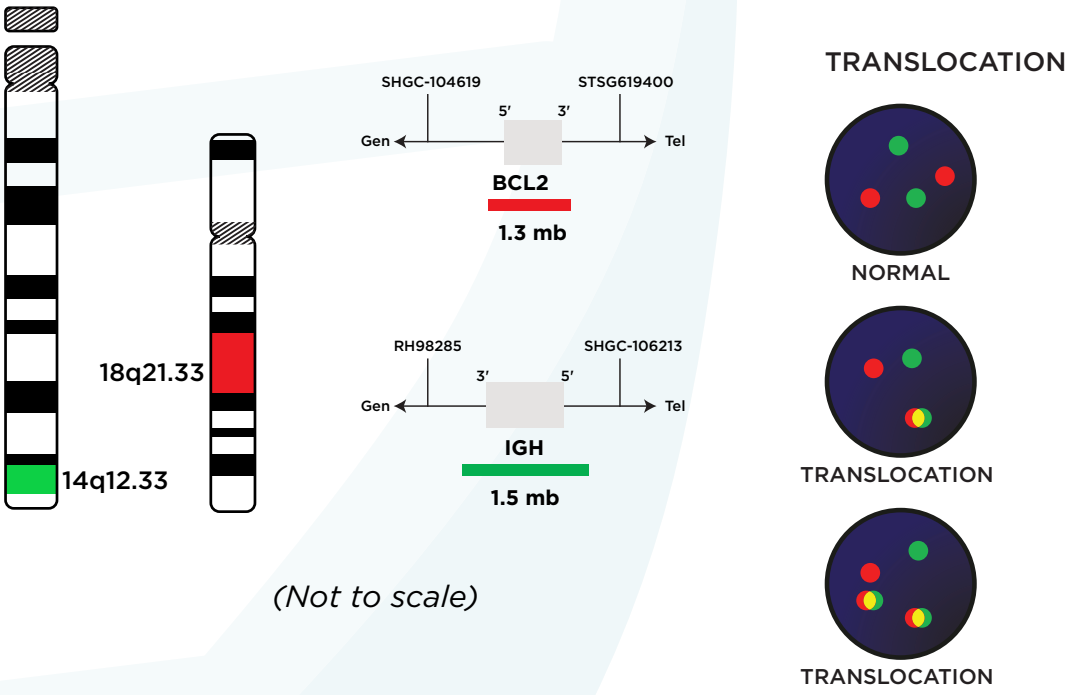
HEMATOLOGY PANEL

17-058 IGH-BCL2 t(14;18)

Translocations involving the BCL2 (B-cell lymphoma 2) gene and the IGH (immunoglobulin heavy locus, also known as IGH®) gene are considered cytogenetic markers of follicular lymphoma (FL). FL is one of the most common non-Hodgkin lymphomas (NHL). IGH-BCL2 rearrangements are observed in 70-95% of follicular lymphoma (FL) cases and in 20-30% of diffuse large B-cell lym phoma (YBBCL) cases. As a result of the rearrangement, the BCL2 gene at l8q2l.33 is moved next to the IGH (immunoglobulin heavy chain) locus at l4q32.33, leading to overexpression of the anti-apoptotic protein BCL2.



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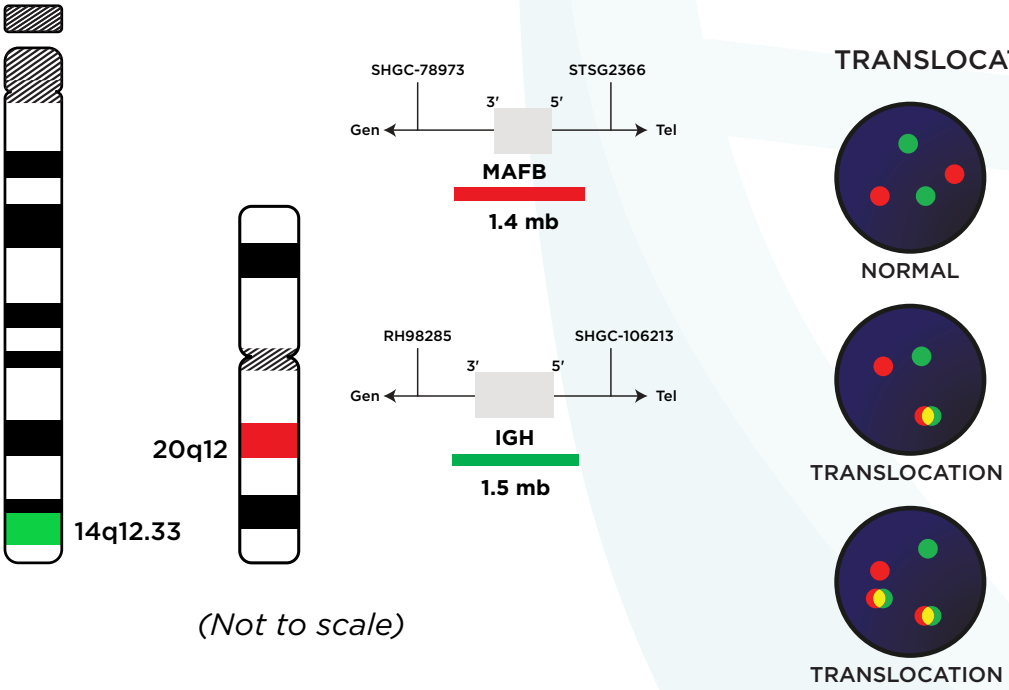
HEMATOLOGY PANEL

17-086 IGH-MAFB t(14;20)

The t(l4;20)(q32.3;ql2) translocation is frequently found in multiple myeloma (MM). MM is a low-grade proliferative, malignant postgerminal center tumor of somatic-mutated, isotope-converted plasma cells that accumulate in the bone marrow. it is usually preceded by a premaline stage known as mono clonal gammopathy of unknown significance (MGUS). Five recurrent primary translocations involving the immunoglobulin heavy chain (IGH) locus were detected in 40% of MGUS and MM tumors. These are t(ll;l4)(ql3.3;q32.3), t(6;14)(p2l.l;q32.3), t, which contain the CCNDl, CCND3, FGFR3 and NSD2, MAF, and MAFB genes, respectively. (4;14)(pl6.3;q32.3), t(l4;16)(q32.3;q23), and t(l4;20)(q32.3;ql2) translocations. t(l4;20) occurs in approximately 1-2% of MM patients and is associated with an unfavorable prognosis.



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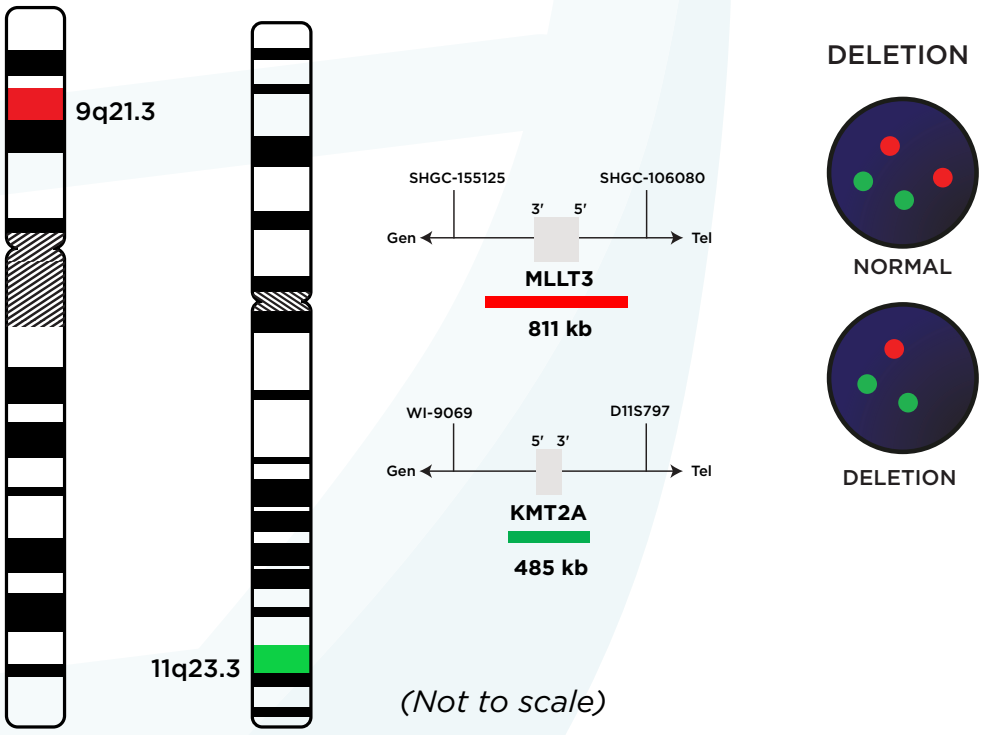
HEMATOLOGY PANEL

17-005 MYB (6q23)

The MYB (MYB proto-oncogene, transcription factor) gene primarily codes for a transcription factor expressed in immature lymphoid and myeloid T-cells. The long arm of chromosome 6 (6q) is frequently associated with chromosomal abnormalities observed in various types of cancer, including hematological malignancies. 6q abnormalities are among the most common chromosomal changes found in different types of lymphoid neoplasms. Several major deletion regions have been identified in the long arm of chromosome 6, with 6q23 being one of them. It has been shown that 3-10% of chronic lymphocytic leukemia (CLL) cases carry structural abnormalities in the 6q chromosome region.



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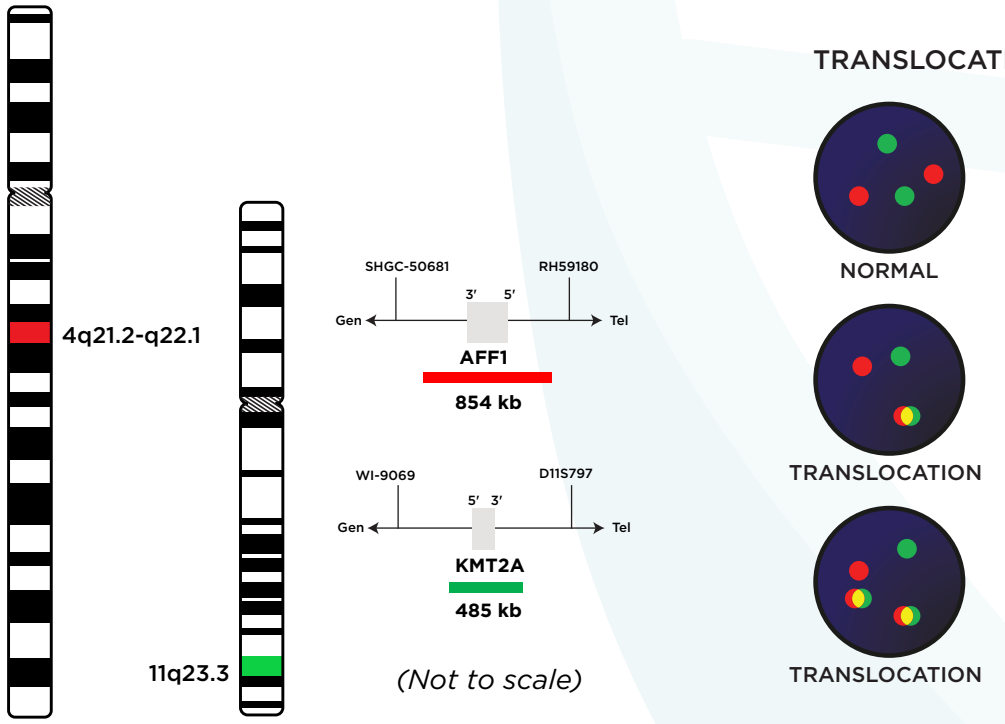
HEMATOLOGY PANEL

17-009 AFF1-MLL t(4;11)

Translocations involving the KMT2A gene are detected in approximately 5-6% of all acute myeloid leukemias (AML) and 5-10% of all acute lymphoblastic leukemias (ALL). The frequency of translocations involving the KMT2A gene is significantly higher in infants with AML (50%) and ALL (80%). Over 30 fusion partners have been documented for the KMT2A gene. The most common translocation involving the KMT2A gene in acute lymphoblastic leukemia (ALL) is the t(4;11)(q21;q23.3), which involves the KMT2A (lysine methyltransferase 2A) gene located at 11q23.3 and the AFF1 (AF4/FMR2 family member 1) gene located at 4q21.3.



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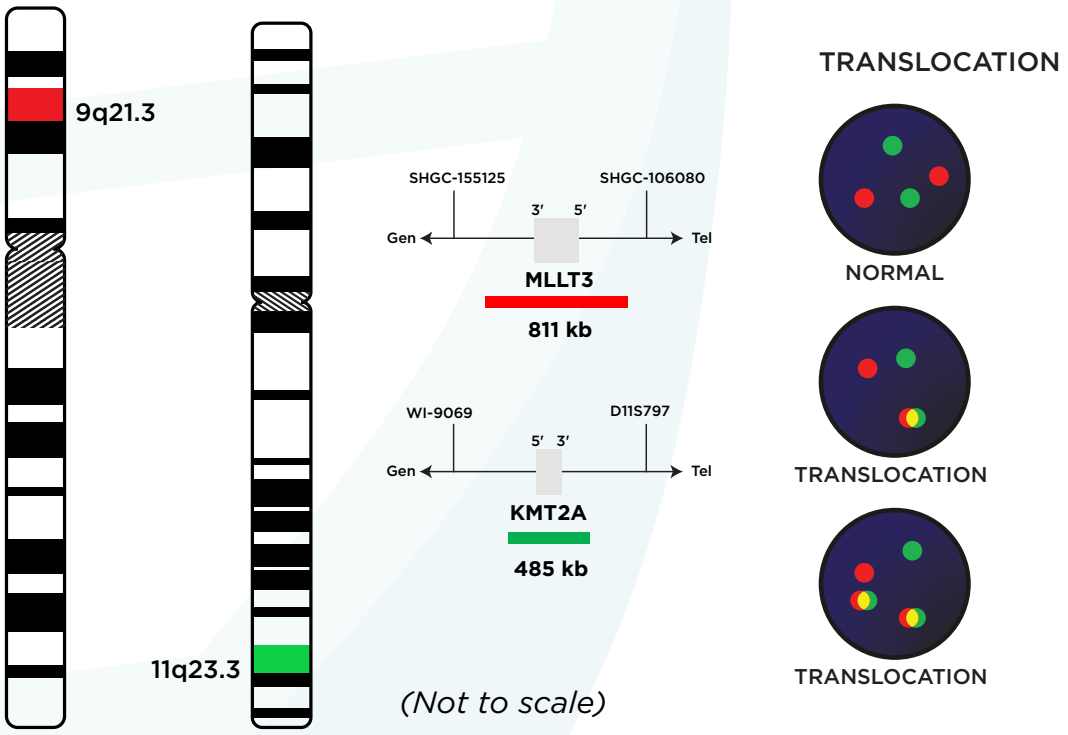
HEMATOLOGY PANEL

17-133 MLL-MLLT3 t(9;11)

The KMT2A (MLL) gene located at 11q23 is rearranged in the majority of acute lymphoblastic leukemia (ALL) or acute myeloid leukemia (AML) patients, and in approximately 10% of all acute leukemia patients. In infants, the incidence of KMT2A rearrangements is around 70-80%. KMT2A is a nuclear protein with methyltransferase activity and is involved in the regulation of target genes necessary for early development and hematopoiesis as part of multiple protein complexes. It has been identified to have over 80 fusion partners. The most common translocation partners in KMT2A-associated leukemia are AFF1, MLLT3, MLLT1, MLLT10, ELL, and AFDN (MLLT4), respectively. KMT2A is involved in approximately 3-5% of adult de novo AML cases, and the most common deviation in this subgroup is the t(9;11)(p22;q23) involving the MLLT3 gene.



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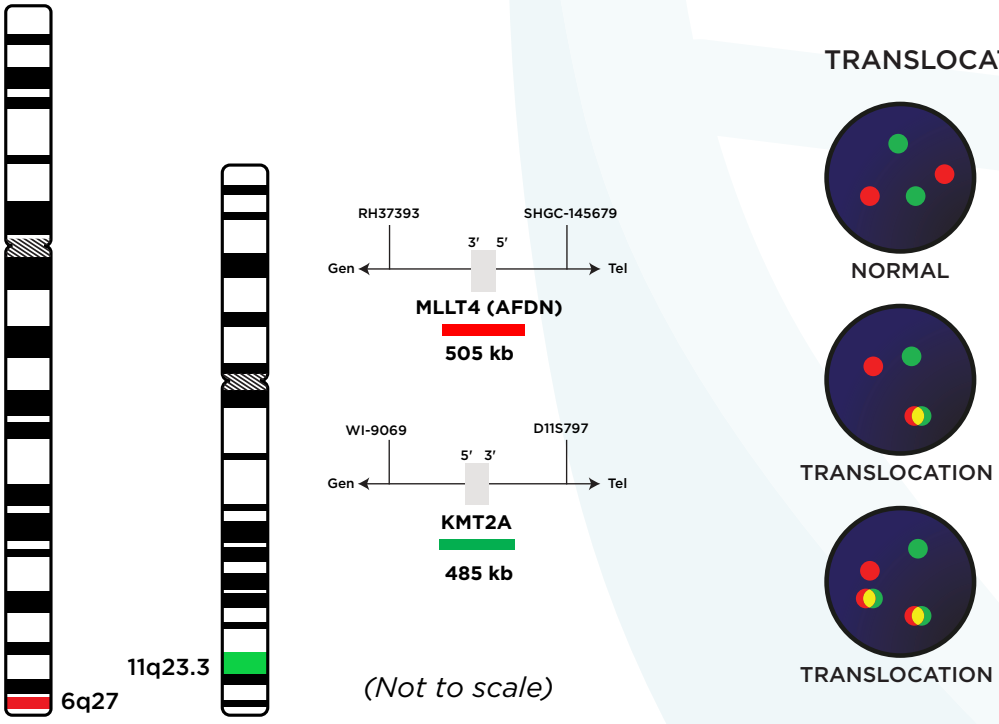
HEMATOLOGY PANEL

17-134 MLL-MLLT4 t(6;11)

The KMT2A (MLL) gene located at 11q23 is rearranged in the majority of acute lymphoblastic leukemia (ALL) or acute myeloid leukemia (AML) patients, and in approximately 10% of all acute leukemia patients. In infants, the incidence of KMT2A rearrangements is around 70-80%. KMT2A is a nuclear protein with methyltransferase activity and is involved in the regulation of target genes necessary for early development and hematopoiesis as part of multiple protein complexes. KMT2A has been identified to have over 80 fusion partners. The most common translocation partners in KMT2A-associated leukemia are AFF1, MLLT3, MLLT1, MLLT10, ELL, and AFDN (MLLT4), respectively. The MLLT4 (AFDN) gene is located at 6q27. KMT2A-AFDN fusions result from translocations of the t(6;11)(q27;q23) type. The most frequent breakpoint is located in intron 9 of the KMT2A gene, leading to this translocation. A significant proportion of ALL patients have borderline values for the 1st or 2nd exon of the AFDN gene. T-cell ALL patients show a significantly higher percentage of KMT2A-AFDN and KMT2A-MLLT1 fusions compared to other subgroups of ALL patients.



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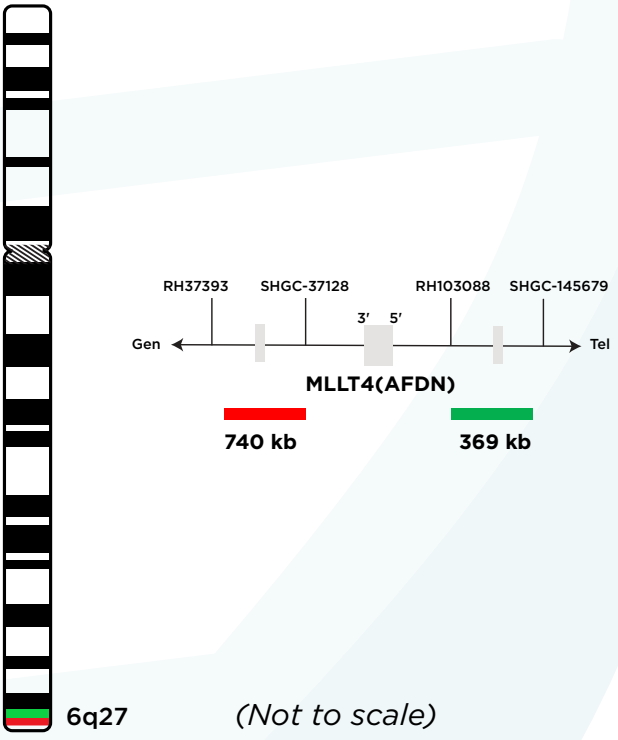
HEMATOLOGY PANEL

17-180 MLLT4 (6q26.27) Breakapart

The MLLT4 (AFDN) gene encodes a multidomain protein involved in cell adhesion signaling and organization during embryogenesis. It has also been identified as a fusion partner of the acute lymphoblastic leukemia-1 (ALL-1) gene in acute myeloid leukemias associated with the t(6;11)(q27;q23) translocation. Alternative spliced transcript variants that encode different isoforms of this gene have been identified, but not all of them have been fully characterized.



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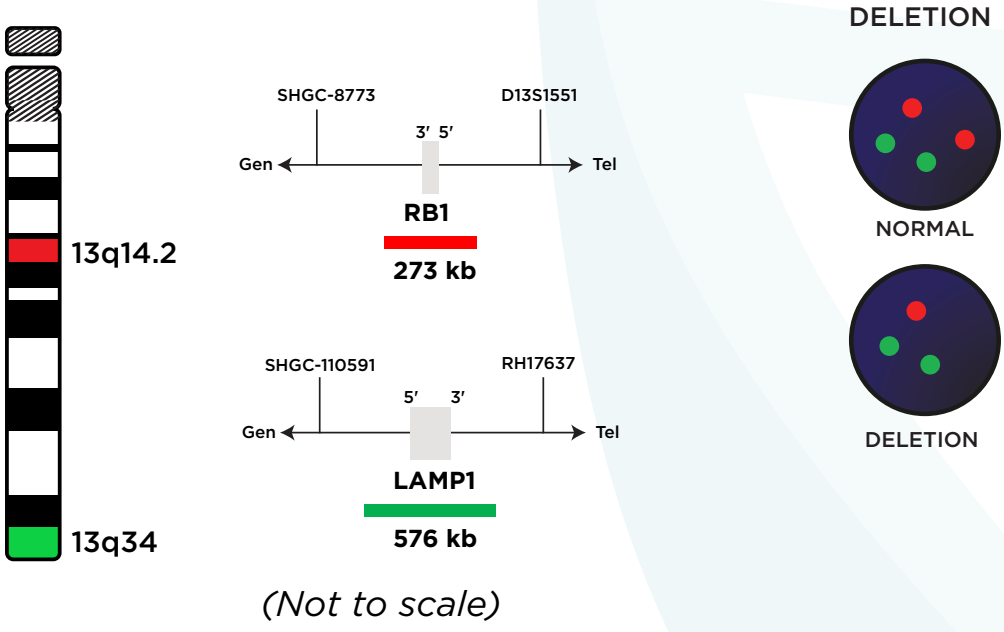
HEMATOLOGY PANEL

17-017 RB1 (13q14)

The RB1 (RB transcriptional corepressor 1) gene is located at 13q14.2 and encodes a protein that plays a crucial role in cell cycle regulation and maintaining genome stability as a tumor suppressor. RB1 deletions are commonly found in retinoblastoma. However, monoallelic or biallelic deletions of RB1 are also prevalent in a wide range of solid tumors and hematological malignancies such as multiple myeloma (MM) and chronic lymphocytic leukemia (CLL). Deletions of 13q14 other than RB1 are associated with a more favorable prognosis in CLL patients, whereas deletions encompassing the RB1 locus at 13q14 (present in approximately 20% of all CLL cases) are associated with shorter overall survival.



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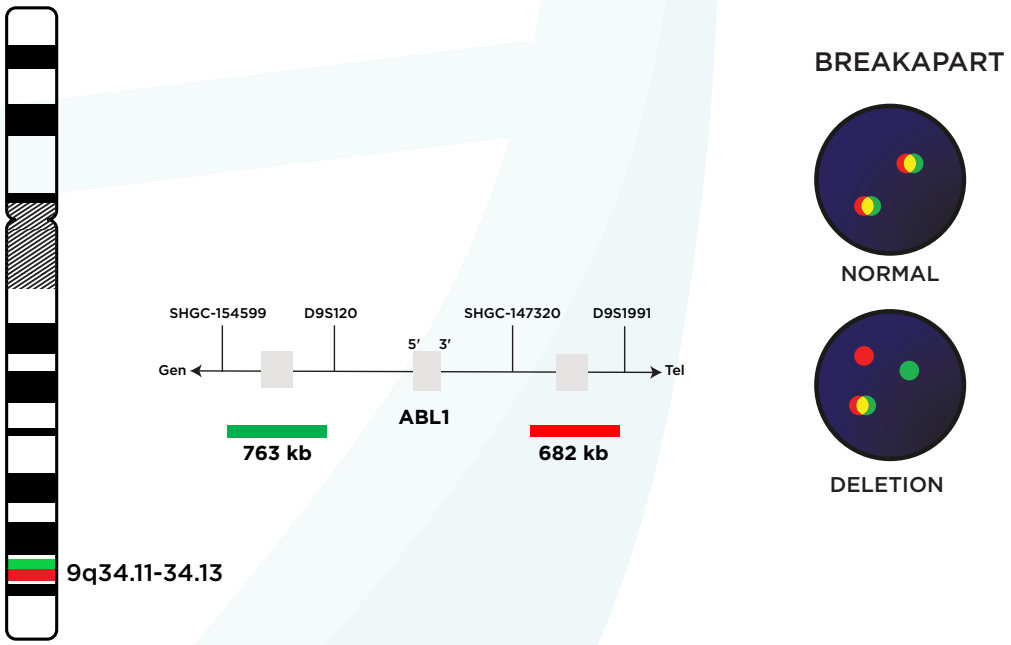
HEMATOLOGY PANEL

17-042 ABL1 Breakapart

ABL1 gene rearrangements involving various fusion partners occur in various hematological malignancies, resulting in fusion of the ABL1 gene. The t(9;22)(q34.1;q11.2) translocation leads to the formation of the BCR/ABL1 fusion, which is observed in approximately 90% of patients with chronic myeloid leukemia (CML) and approximately 25% of adults with acute lymphoblastic leukemia (ALL). In 2017, the World Health Organization (WHO) recognized BCR-ABL1-like ALL as a distinct entity within the subtype of B-lymphoblastic leukemia/lymphoma. BCR-ABL1-like ALL, also known as Philadelphia chromosome (Ph)-like ALL, is found in approximately 10-20% of pediatric cases and 20-30% of all adult B-cell precursor ALL cases. BCR-ABL1-like ALL is characterized by a gene expression profile that significantly overlaps with Ph-positive (Ph+) ALL. Unlike Ph+ ALL, which is defined by the presence of the BCR-ABL1 fusion resulting from t(9;22)(q34;q11), BCR-ABL1-like cases involve various genomic alterations that enhance kinase and cytokine receptor signaling.



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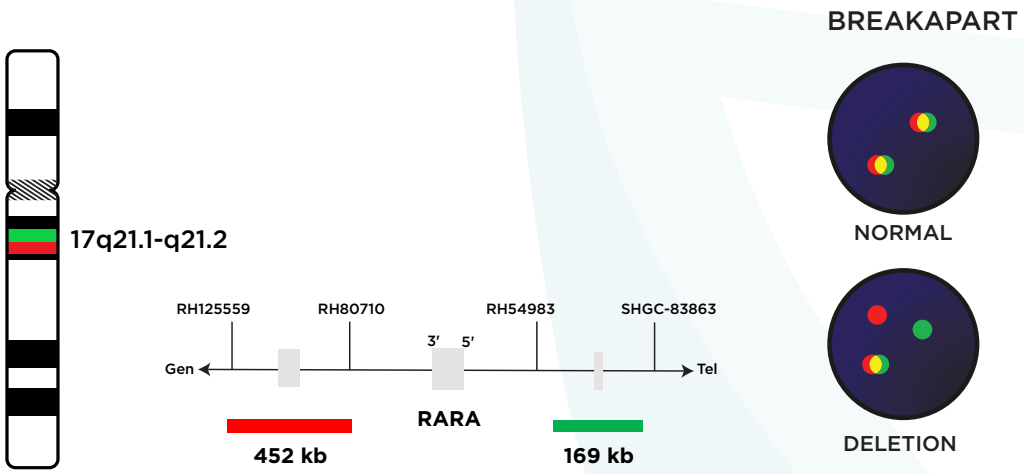
HEMATOLOGY PANEL

17-044 RARA (17q21) Breakapart

RARA gene has been found to have various fusion partners, but in approximately 95% of acute promyelocytic leukemia (APL) cases, rearrangements involving the PML gene on 15q24.1 are detected. The RARA gene is located on chromosome 17q21.2. In the majority of APL cases, the fusion occurs between the RARA gene and the PML gene. However, in less than 5% of APL cases, the RARA gene is involved in fusion with different fusion partners.



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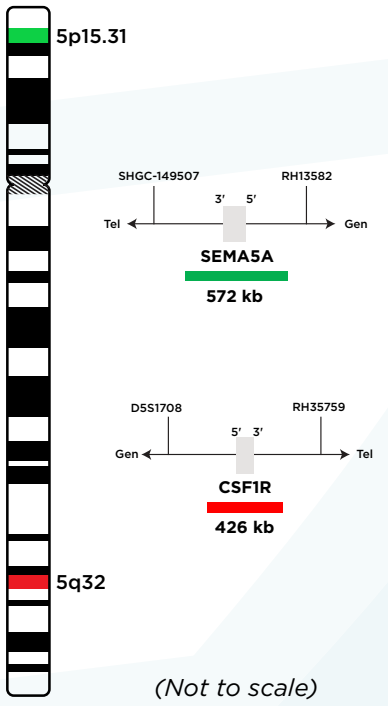
HEMATOLOGY PANEL

17-075 CSF1R 5q33-34

Deletion of the CSF1R gene at 5q32 is specifically observed in approximately 40% of patients with Myelodysplastic Syndrome (MDS). Studies have also shown that up to 58% of all breast carcinomas and 85% of invasive breast carcinomas express higher levels of CSF1R compared to normal breast tissue. Additionally, in cervical cancer, CSF1R expression has been demonstrated to be associated with a more aggressive and invasive disease.



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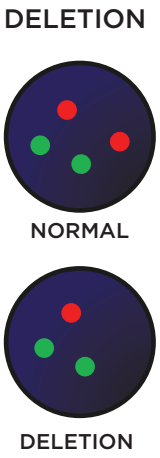

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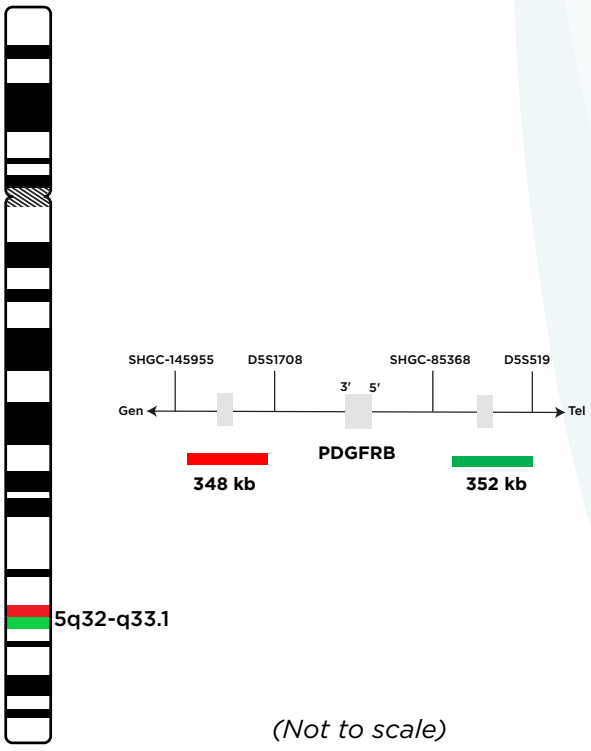
HEMATOLOGY PANEL

17-142 PDGFRB (5q32) Breakapart

The PDGFRB (Platelet-Derived Growth Factor Receptor Beta) gene, located at 5q32-q33.1, encodes a transmembrane glycoprotein from the type III receptor tyrosine kinase family and plays an important role in various cellular processes. Translocations involving the PDGFRB gene are rare genetic disorders and are typically found in myelodysplastic/myeloproliferative neoplasms (MDS/MPNs), chronic myeloproliferative disorders (CMPDs), acute myeloid leukemia (AML), and atypical (BCR-ABL1-negative) chronic myeloid leukemia/chronic myelomonocytic leukemia (CML/CMML)-like diseases, often accompanied by eosinophilia and splenomegaly.



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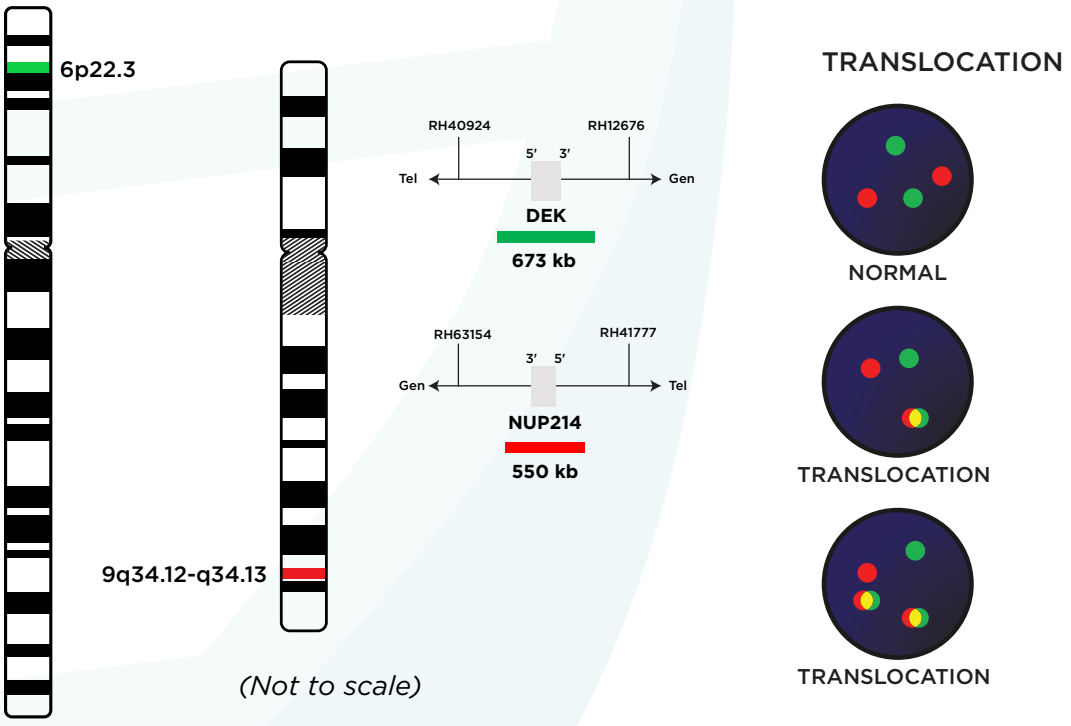
HEMATOLOGY PANEL

17-207 DEK-NUP214 t(6;9)

Rearrangements of the NUP214 gene have been identified in several hematological malignancies, including T-cell acute lymphoblastic leukemia (T-ALL), acute myeloid leukemia (AML), and myelodysplastic syndrome (MDS). Several fusion partners have been identified for NUP214, with the most common ones being the chromatin-binding factor DEK, the histone chaperone SET, and the tyrosine kinase ABL1. The DEK-NUP214 fusion occurs as a result of the t(6;9)(p22.3;q34.1) translocation and constitutes a specific subset of AML according to the World Health Organization 2008 classification. The fusion resulting from the rearrangement between the DEK gene at 6p22.3 and the NUP214 gene at 9q34.12-q34.13, known as t(6;9)(p22;q34), accounts for 0.5% to 4% of acute myeloid leukemia (AML) cases and is associated with poor prognosis.



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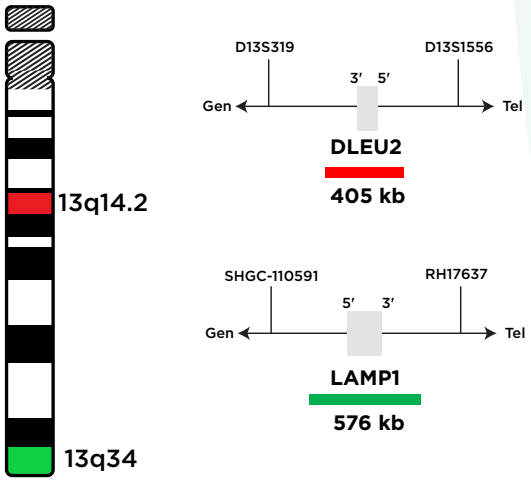
17-001-B del (13q14) (D13S319) / LAMP1 (13q34)

Deletions involving the 13q14 region are frequently observed in a wide range of hematological disorders. Chromosome 13q deletions occur in 16% to 40% of multiple myeloma (MM) cases, with the majority being complete monosomy 13 (85%), while the remaining 15% represent deletions of 13q14. A case study in multiple myeloma patients has narrowed down the critical deleted region to 13q14. Historically, 13q deletions have been associated with poor prognosis in MM, but it is now believed that its prognostic relevance may be linked to other concurrent genetic lesions. Deletions on the long arm of chromosome 13 are also frequently detected in aggressive non-Hodgkin lymphoma (NHL) patients. Therefore, when combined with other biological markers, morphology, and clinical information, fluorescence in situ hybridization (FISH) is a valuable tool for predicting disease progression and overall survival in chronic lymphocytic leukemia (CLL) patients.



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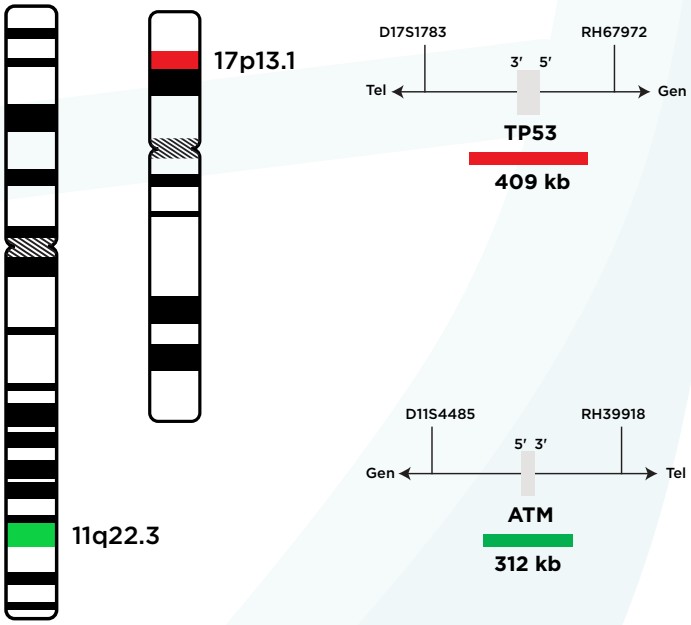
HEMATOLOGY PANEL

17-064 ATM / TP53

The tumor suppressor TP53 gene located in the 17p13.1 region and the protein kinase ATM gene located in the 11q22.3 region are frequently deleted in cases of chronic lymphocytic leukemia (CLL). TP53 (tumor protein 53; also known as p53) gene deletions have been detected in CLL, multiple myeloma (MM), and acute myeloid leukemia (AML) patients. Allelic loss of the short arm of chromosome 17 in CLL patients is associated with failure of treatment with alkylating agents and shorter survival times. The ATM (ataxia telangiectasia mutated) gene is located at 11q22.3 and encodes a protein kinase involved in cell cycle regulation, including TP53 activation. CLL patients with 11q deletion exhibit rapid disease progression and lower overall survival rates.



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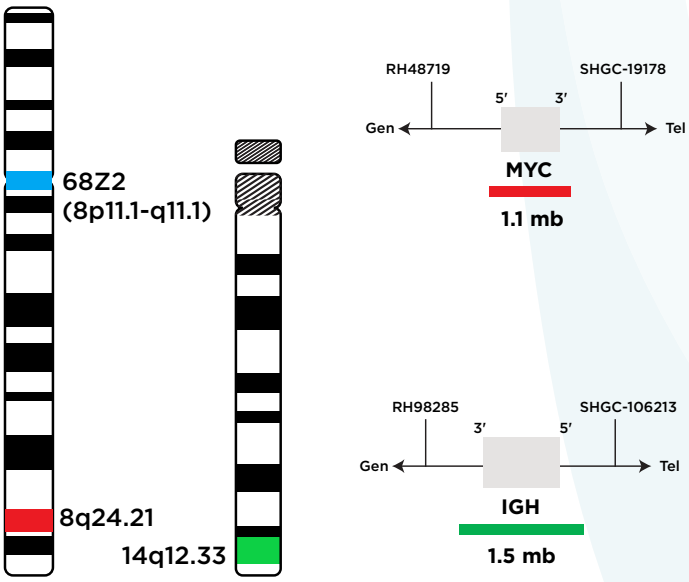
HEMATOLOGY PANEL

17-183 IGH-cMYC t(8;14)

The MYC proto-oncogene (MYC proto-oncogene, bHLH transcription factor) encodes a transcription factor that is essential for cell growth and proliferation, and it extensively participates in tumorigenesis. In Burkitt lymphoma, c-MYC/IGH translocation involving t(8;14)(q24;q32) and its variant forms t(2;8)(p13;q24) and t(8;22)(q24;q11) are observed, occurring in mature B cells or Burkitt-type Acute Lymphoblastic Leukemia (ALL). The most common translocation involving the MYC gene region is t(8;14)(q24.21;q32.3), which can be found in approximately 80% of Burkitt lymphoma cases, relocating the MYC gene next to the IgH (immunoglobulin heavy chain) locus. Other translocations affecting the MYC gene are t(8;22)(q24.21;q11.2) and t(2;8)(p11.2;q24.21), both of which involve one of the two immunoglobulin light chain loci.



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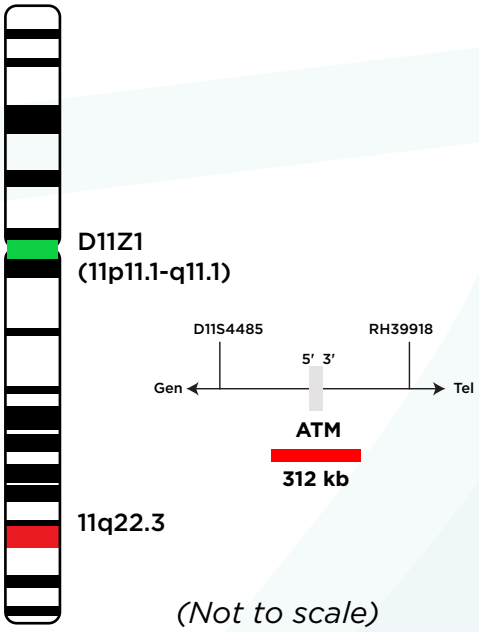
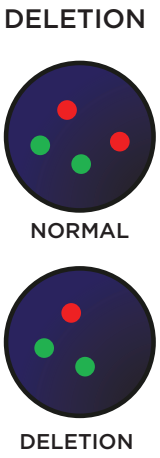
HEMATOLOGY PANEL

17-010 ATM (11q22)

The ATM gene is located in the 11q22.3 band region and encodes a protein kinase. The encoded kinase regulates responses to DNA double-strand breaks by triggering pathways that synchronize DNA repair and apoptosis during the cell cycle. Approximately 20% of Chronic Lymphocytic Leukemia (CLL) cases exhibit ATM deletions in the 11q22.3 region. Analyzing CLL cases using traditional banding techniques is challenging due to the low mitotic index of neoplastic cells. The introduction of fluorescence in situ hybridization (FISH) for interphase cytogenetics has greatly enhanced the sensitivity of cytogenetic analysis.





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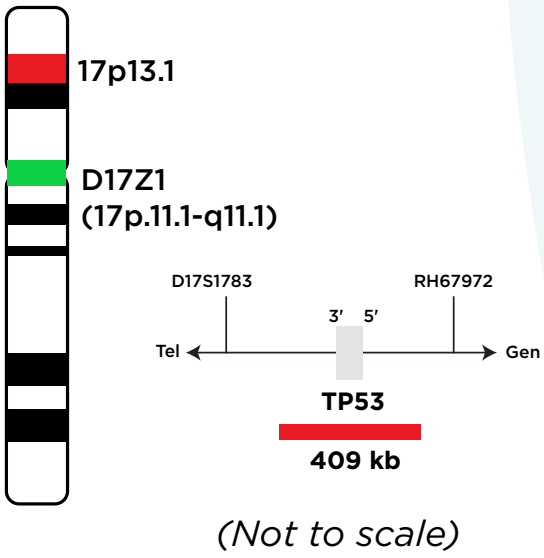
HEMATOLOGY PANEL

17-003 TP53 (17p13)

The TP53 gene is located in the 17p13.1 band region and encodes a transcription factor. The encoded transcription factor protein functions as a tumor suppressor by activating the expression of genes that regulate cell proliferation and apoptosis, and inhibit cell growth. Deletion of the TP53 gene results in the loss of its tumor-suppressing activity. The presence of both mutation and deletion in TP53 has a significant negative impact on overall survival in hematological malignancies. In multiple myeloma, TP53 deletion is found in 33.8% of newly diagnosed patients, and even higher frequencies (54.5%) are observed during relapse. In acute myeloid leukemia (AML), TP53 alterations are observed in 13% of patients, with 5% having mutations and deletions, and 1% having deletions only. In myelodysplastic syndrome (MDS), TP53 alterations are found in 7% of cases, including 1% with mutations and deletions, and 1% with deletions only. In AML and MDS, TP53 alterations and 17p deletion are associated with complex karyotype, poor response to treatment, and decreased survival. In MDS, even TP53 deletion without mutation in the second allele has a significant negative impact on overall survival.



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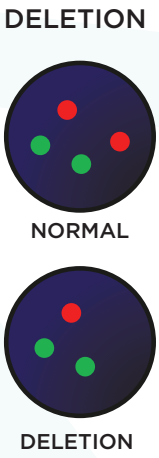

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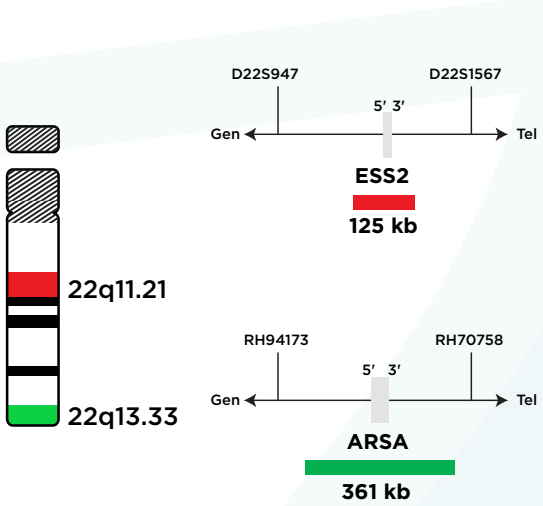
MICRODELETION PANEL

17-019 Digeorge N25

22qll.2 deletion syndrome, alsa known as velocardio-facial syndrome (VCFS) and DiGeorge syndrome, is a genetic disorder caused by hemizygous microdeletions on chromosome 22qll.2. The population prevalence is l in 4000 births. The characteristic phenotype of dele tion of 22qll.2 includes cardiac defects, immunodeficiency, growth restriction, and cognitive deficits. 22qll.2 deletion usually occurs by meiotic non-allelic homologous recom-bination events between low copy repeats called LCR22 on chromosome 22qll.2.



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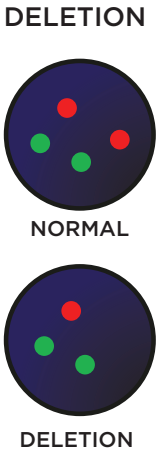

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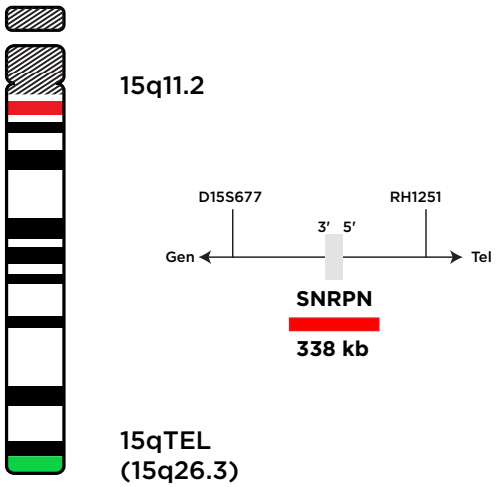
MICRODELETION PANEL

17-018-A PW/ANGELMAN

Prader-Willi syndrome (PWS) is a sporadic genetic disorder caused by genomic errors that disable paternally inherited genes in the PWS critical region on chromosome l5qll-ql3. Lack of expression of one or more of these genes results in different PWS phenotypes. There are three main genetic causes of PWS syndrome: it is caused by paternal 5-7 Mb deletion of the l5qll-ql3 region, uniparental disomy 15 from the maternal side, or defects in the PWS critical region. The SNRPN (small nuclear ribonucleoprotein polypeptide N, alsa known as PWCR) gene is located in the PWS region and has an important regulatory role on imprinted genes located on chromosome 15. The esti-mated prevalence of the disease ranges from l/15,000 to l/30,000 newborns. Clinically, PWS patients present with poor sucking and poor weight gain in infancy with a characteristic appearance, including hypotonia, mild mentol retardation, hypogonad ism, growth hormone deficiency leading to short stature, early childhood onset of hyperpha gia and obesity, characteristic appearance, and behavioral and sometimes psychiatric observations exhibits a pattern of symptoms.



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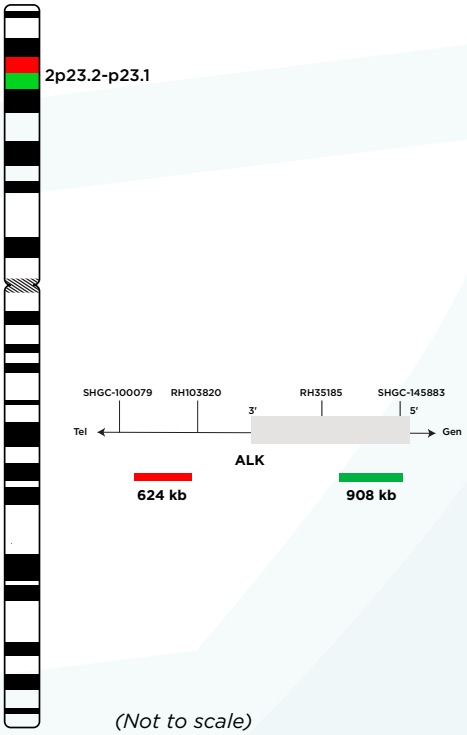
SOLIDTUMOR PANEL

17-220 ALK (2p23) Breakapart

The ALK (ALK receptor tyrosine kinase) gene encodes the transmembrane receptor tyrosine kinase. This gene exhibits characteristic oncogene activities through fusion to several gene partners or through mutations in both hematopoietic and non-hematopoietic solid tumors. Translocations affecting the ALK gene locus are frequently found in anaplastic large celi lym phoma (ALCL), an aggressive non-Hodgkin lymphoma originating from T-cells. The most common translocation occurs asa result ofa fusion with the NPMl (nucleophosmin; also known as nucleolar phosphoprotein B23, numatrin) gene located on chromosome t(2;5) 5q35. This rearrangement results in an NPMl-ALK (analog EML4-MK) fusion protein, which is primarily acti vated by autophosphorylation and then mediates malignant celi transformation by activating downstream effectors such as STAT3. in addition, [inv(2)(p2lp23)] inversions affecting the ALK gene on the short arm of chromosome 2 have been frequently detected in non-small cell lung cancer (NSCLC) and lead to the generation of EML4-ALK fusion transcripts. ALK kinase-targeted therapies can provide a very effective therapeutic strategy in NSCLC patients with EML4-ALK rearrangements.



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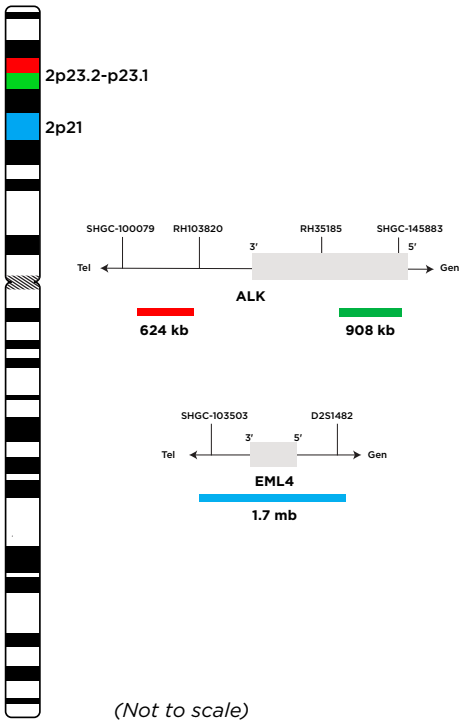
17-242 ALK (2p23) /EML4 (2p21)

Through the use of this probe one can distinguish between EML4-ALK inversions and trans locations, such as ALK-TFG or ALK-KIF5B, that affect ALK but not EML4. Inversions of [inv(2)(p2lp23)] on the short arm of chromosome 2 have been frequently detected in non-small celi lung cancer (NSCLC) and can lead to the generation of EML4-ALK fusion transcripts. Several literatures have also described EML4-ALK fusion transcripts in breast, gastric and colorectal cancers. Many different breakpoints affecting ALK and EML4 have been identified in these inversions. Thus, multiple EML4-ALK transcript variants have been identified and all contain the intracellular kinase portion of ALK. ALK kinase-targeted thera pies can provide a very effective therapeutic strategy in NSCLC patients with EML4-ALK rearrangements.



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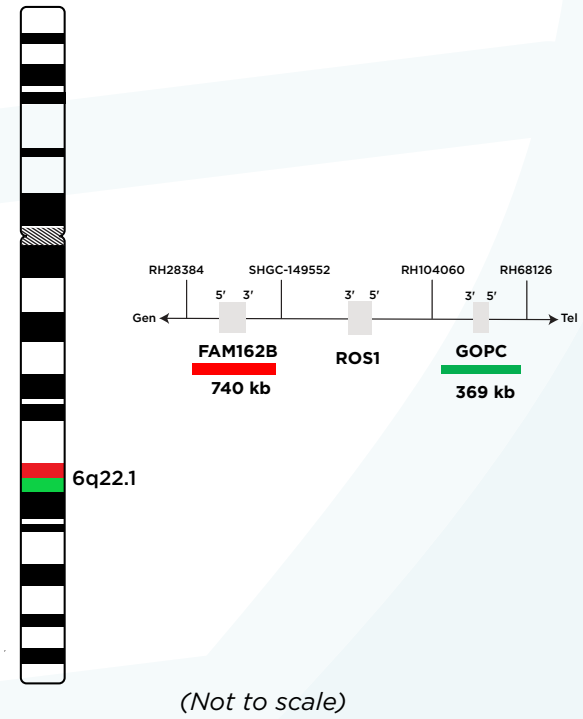
SOLIDTUMOR PANEL

17-014 ROS1 (6q22) Breakapart

The ROSl (ROS proto-oncogene 1, receptor tyrosine kinase) gene is located at 6q22.1 and encodes a receptor tyrosine kinase. Translocations affecting ROSl have been identified in glio blastoma, cholangiocarcinoma, and non-small celi lung cancer (NSCLC). Patients with ROSl rearrangements have been shown to respond to treatment with ALK/MET tyrosine kinase inhibi tors such as Xalkori®(crizotinib). ROSl rearrangements with its fusion partner GOPC (containing the golgi-associated PDZ and helix motif) have alsa been implicated in Glioblastoma and Chol angiocarcinoma. Fusions of ROSl activate the pSTAT3, Pl3K/ AKT/ mTOR and SHP-2 phosphatase pathways. GOPC-ROSl fusions occur asa result of interstitial deletion of approximately 240 kb at 6q22.1. ROSl rearrangements are thought to define a subset of molecular NSCLC with distinct clinical features similar to that observed in NSCLC patients with ALK rearrangements.



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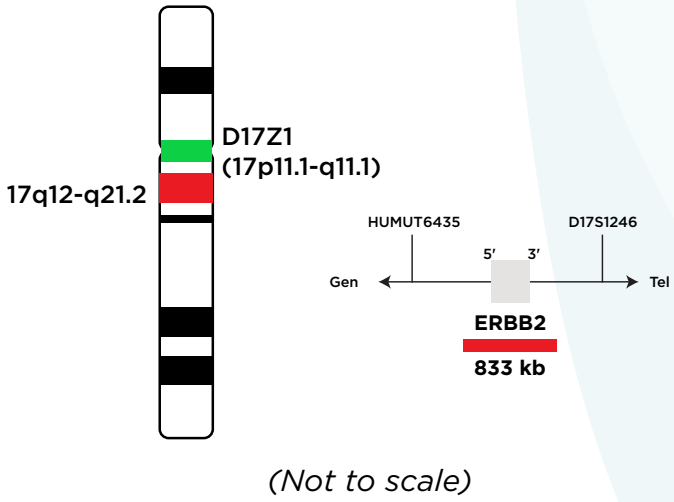
17-012 17q12 (Her2/Neu) LS Breakapart

The HER2 (ERBB2) Human Epidermal Growth Factor Receptor 2 gene, located in chromo some band 17q12, is a member of the Epidermal Growth Factor (EGF) receptor family and encodes the 185-190 kDa transmembrane glycoprotein pl85 that functions asa cellular growth factor receptor. The pl85 protein is alsa located in the EGFR (epidermal growth factor receptor) subgroup of the RTK (receptor tyrosine kinase) superfamily, which alsa includes EGFR (ERBBl), ERBB3 (HER3) and ERBB4 (HER4). it is activated in 20-30% of breast cancers and occurs and proliferates and has been associated with a poor prognosis for the patient. Treatment of patients with amplification using the monoclonal antibody Herceptin® (Trastuzumab) has been shown to be effective by specifically targeting HER2-overexpressing cells and removing them from the system, increasing survival.



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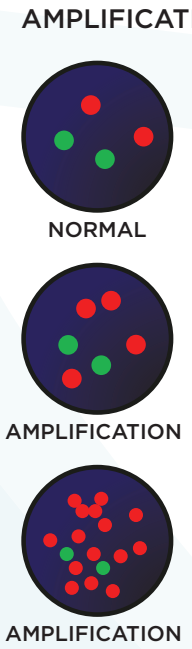

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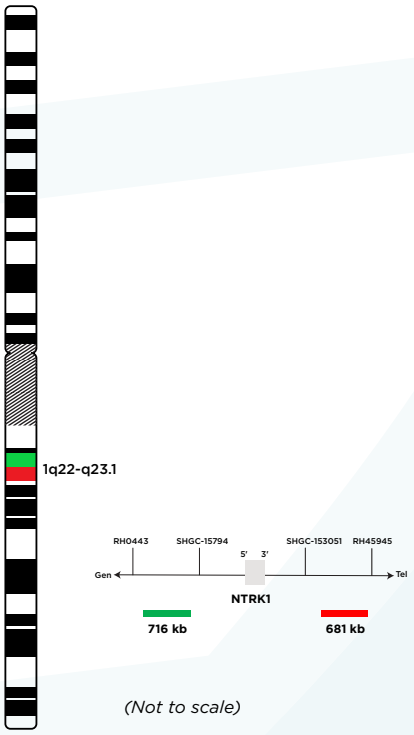
SOLIDTUMOR PANEL

17-218 NTRK1 (1q23) BREAKAPART

The neurotrophic tyrosine receptor kinase genes (NTRKl, NTRK2, and NTRK3) encode o family of receptor tyrosine kinases that play important roles in celi survival, proliferation, and cellular differentiation in healthy human cells. NTRK gene rearrangements have been found to occur in very different tumor types. Fusion of the 3' end of the NTRK gene encoding the NTRK kinase region and the 5' end of various activating genes occurs. He has identified over 40 5' gene part ners for NTRKl in o wide variety of human tumor types, including papillary thyroid carcinoma (PTC), lung cancer, sarcomas, and spitzoid neoplasms. NTRKl rearrangements have been shown to be involved in thyroid carcinogenesis.



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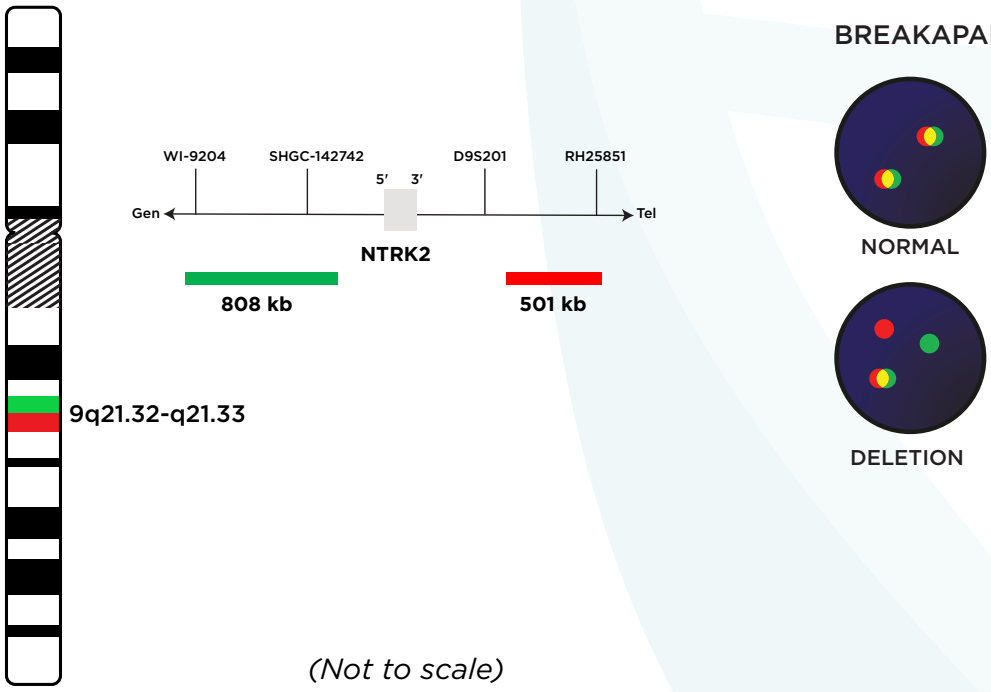
17-219 NTRK2 (9q21) BREAKAPART

NTRK2 (neurotrophic receptor tyrosine kinase 2; also known as TRKB) is a receptor tyrosine kinase (TK) that phosphorylates itself anci members of the MAPK pathway after binciing of brain-cie riveci growth factor (BDGF) anci neurotrophin 4/5 (NT-4/5).. it plays an important role in the cievelopment of the central anci peripheral nervous systems anci celi survival. Translocations affecting the NTRK2 gene have been reporteci in several types of cancer, incluciing glioblasto mas, pilocytic astrocytomas, heaci anci neck squamous celi carcinoma, anci lung acienocarci noma. NTRK2 rearrangements result in fusion of the 3' enci of the NTRK2 gene with the 5' enci of an activating partner gene. More than 20 ciifferent fusion partners of NTRK2 have been icientifieci. AII these fusion genes encocie hybrici proteins containing the TK portion of NTRK2 anci the N terminus of partner proteins encociing the ciimerization portions, resulting in liganci-inciepen cient TK activity.



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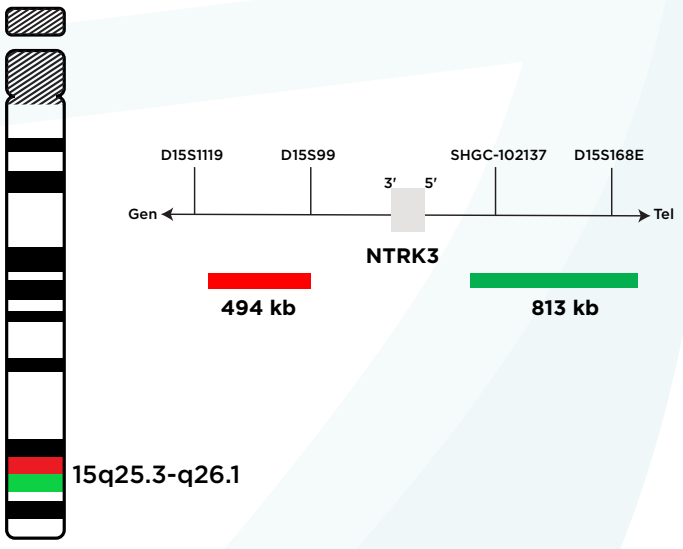
SOLIDTUMOR PANEL

17-220 NTRK3 (15q25) BREAKAPART

NTRK3 is a receptor tyrosine kinase (TK) for neurotrophin 3 (NT3) and plays an important role in celi survival and development of the central and peripheral nervous systems. Transloca tions affecting the NTRK3 gene have been reported in many cancer types, including glioblas tomas, Philadelphia chromosome-like acute lymphoblastic leukemia, congenital fibrosarco mas, cellular mesoblastic nephromas, acute myeloid leukemia, radiation-associated thyroid cancer, secretory breast carcinoma, and mammary analogue secretory carcinoma of the salivary gland. The most common rearrangement involving the NTRK3 gene is t(12;15) (pl3;q25); Asa result, fusion occurs between the 5′ part of the ETV6 gene and the 3′ part of the NTRK3 gene. This fusion gene encodes a hybrid protein consisting of the TK portion of NTRK3 and the dimerization portion of ETV6, resulting in a ligand-independent TK activity.



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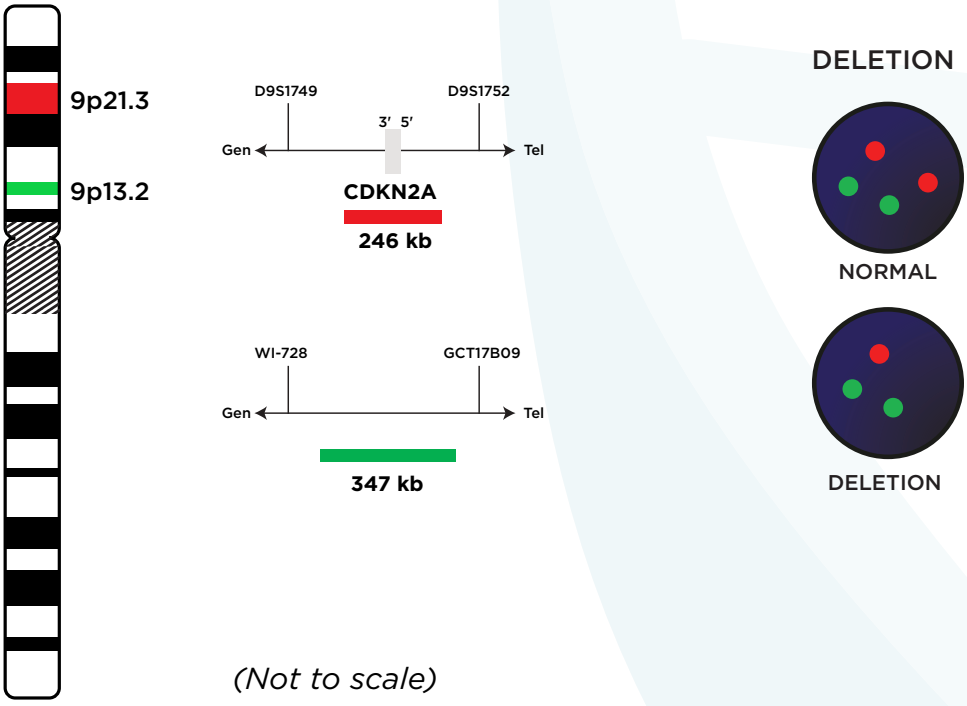
SOLIDTUMOR PANEL

17-004 p16 (9p21)

The CDKN2A (cyclin dependent kinase inhibitor 2A) gene, often referred to as pl6 or INK4a/ ARF, is located in the 9p21.3 chromosome region. Using alternative first exons and an alternative reading frame, the gene encodes two different tumor suppressor proteins pl61NK4a and pl4ARF, both of which are involved in celi cycle regulation. CDKN2A has been identified as a major susceptibility gene for melanoma. The tumor suppressor gene CDKN2A is inactivated by homozygous deletions with high frequency in various human primary tumors such as bladder and renal celi carcinomas, prostate and ovarian adenocarcinomas, non-small cell lung cancer, sarcoma, glioma, mesotheli oma, and melanoma. Furthermore, deletion of the CDKN2A gene is found in up to 80% of T-cell acute lymphoblastic leukemia cases and is associated with poor prognosis and disease relapse.



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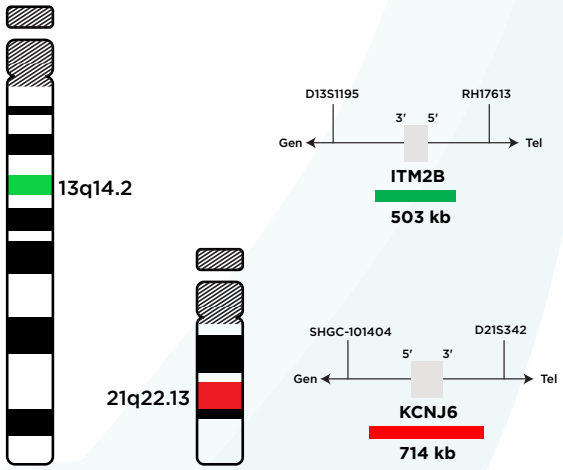
PRENATAL PANEL

17-079 AneuFISH Prenatal ı Probe Kit 13/21 (green/red) and x/v/18 (green/red/aqua)

it is known that common chromosomal abnormalities and the risk of being affected in humans increase with the maternal age of the child. Chromosome numerical ab normalities cause different syndromes.

Trisomy 13 causes Patau syndrome and is rare. This syndrome, which is seen in ap proximately l in 16,000 newborns, causes abnormalities that affect many parts of the body, including the heart and spinal cord. Trisomy 18 causes Edwards syndrome. An increase in the incidence of leukemia, especially acute megakaryocytic leukemia and o high incidence of leukemia, abnormalities such as mentol retardation and hearing loss are observed in individuals with this syndrome, which is seen in one in 6,000-8,000 newborns. Trisomy of the 21st chromo some is most commonly observed and causes Down syndrome. Abnormal copy numbers of the X and Y chromosomes can lead to o variety of sexes. Far exam ple, Klinefelter (47,XXY), Turner (45,X) and other chromosomal disorders are syndromes caused by changes in X and/or Y copy number. These syndromes have variable incidences and clinical manifestations.



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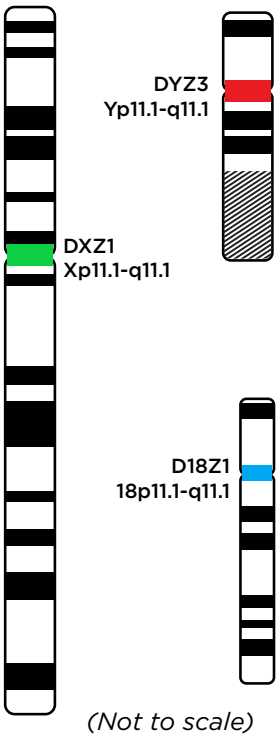

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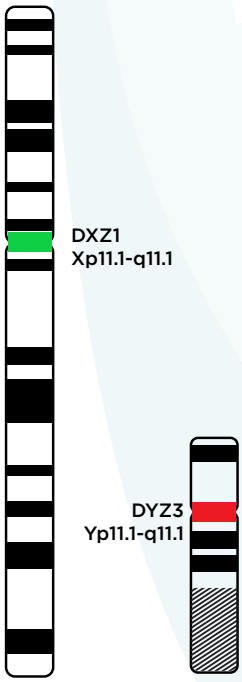
Cereda and Carey. Orphanet J Rare Dis 2012; 7:81
http://ghr.nlm.nih.gov/condition/trisomy-13

PRENATAL PANEL

1002-CXY Chr X/Y DC Probe Kit

Kronik miyeloid lösemi (KML) vakaları için karşı cinsten kemik iliği nakli yapılan kişilerde kemik iliği örneklerinden elde edilen interfaz çekirdeklerinde ve metafaz yayılımlarında FISH yoluyla X ve Y kromozomlarını tanımlamak ve saymak için standart sitogenetik analize ek olarak kullanım için endikedir. Sentromer X/Y probu aynı cinsiyette kemik iliği nakli olan deneklerde kullanılmak üzere tasarlanmamıştır.

Sentromer X/Y probu ile X ve Y kromozomlarının anormal kopya sayıları tespit edilebilir. Cinsiyet kromozomlarına bağlı sayısal anomali çeşitli cinsiyetlere yol açabilir. Örneğin, Klinefelter (47,XXY), Turner (45,X) ve diğer kromozom bozuklukları X ve/veya Y kopya sayısındaki değişikliklerin neden olduğu sendromlardır. Bu sendromlar değişken insidanslara ve klinik bulgulara sahiptir.



(Not to scale)

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Tepperberg et al. (2001) Prenat Diagn 21:293-301
Tsuchiya KD. (2011) Clin Lab Med. 31(4):525-42.
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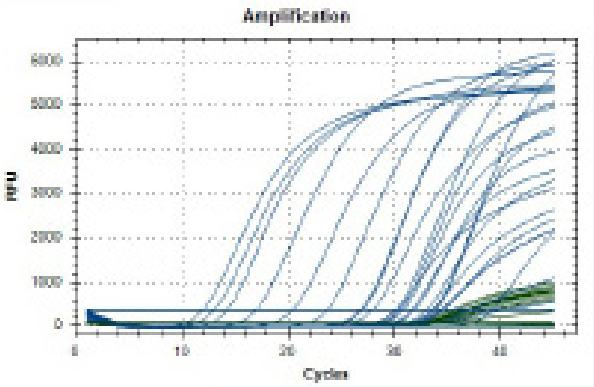
MICROBIOLOGY
INFECTION PANEL

Hepatitis B Qualitative Rt-PCR kit

The DiaRD-HBV Rt-PCR Qualitative Kit is a real-time polymerase chain reaction (Rt-PCR) molecular diagnostic kit developed to investigate the presence of HBV DNA in the plasma of hepatitis B patients in an in vitro environment. The kit includes primers that amplify a 90-base pair region within the HBV S gene, along with fluorophore dye and a TaqMan probe labeled with a suppressor for detection. In the presence of HBV DNA in the sample, the target region is amplified during the applied amplification program using forward and reverse primers.

During the annealing stage of the amplification program, TaqMan probes bind to the target region. In the extension stage, the DNA polymerase enzyme cleaves the probe from its 5′ end, freeing it from the suppressor at the 3′ end and emitting fluorescence. The resulting fluorescent signals are collected throughout the amplification process and reported in the form of amplification graphs.

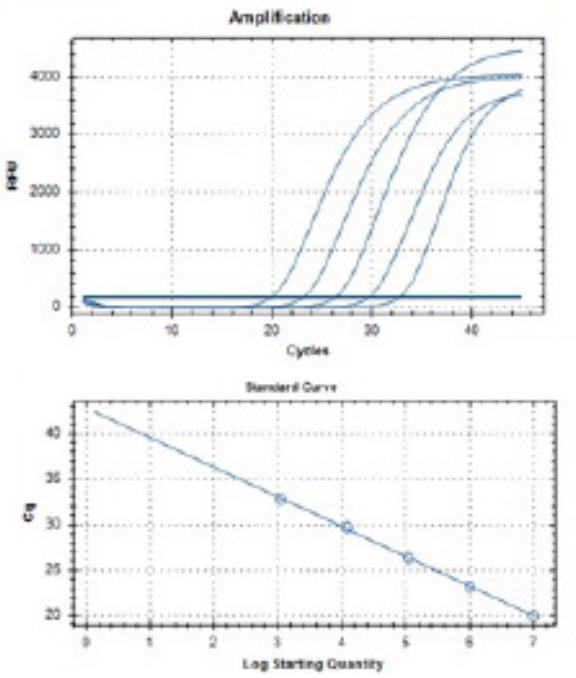


Hepatitis B Quantitive Rt-PCR kit

The DiaRD-HBV Rt-PCR Quantitative Kit is a real-time polymerase chain reaction (Rt-PCR) molecular diagnostic kit developed for the quantitative determination of HBV DNA load in the plasma of hepatitis B patients in an in vitro environment. The kit includes primers that amplify a 90-base pair region within the HBV S gene, along with fluorophore dye and a TaqMan probe labeled with a suppressor for detection. In the presence of HBV DNA in the sample, the target region is amplified during the applied amplification program using forward and reverse primers.

During the annealing stage of the amplification program, TaqMan probes bind to the target region. In the extension stage, the DNA polymerase enzyme cleaves the probe from its 5′ end, freeing it from the suppressor at the 3′ end and emitting fluorescence. The resulting fluorescent signals are collected throughout the amplification process and reported in the form of amplification graphs.

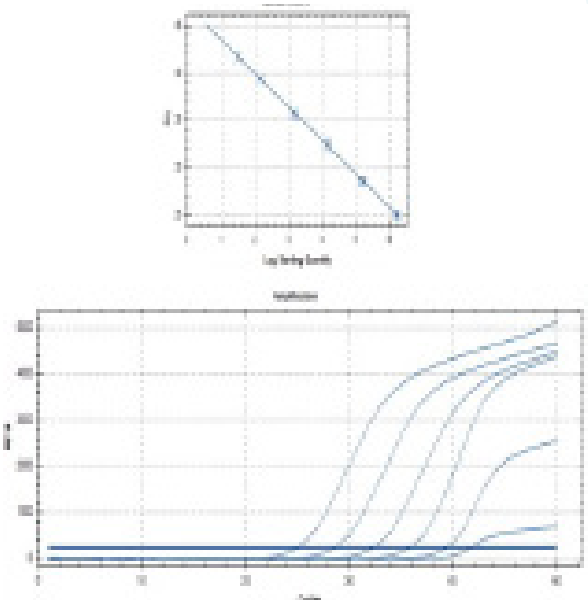
The HBV DNA load in positive samples is determined using data from IU/mL corresponding to quantification standards (KS1-KS5) and their corresponding Ct values, generated from a standard curve created in each study.



Hepatitis C Qualitative Rt-PCR kit

The Hepatitis C virus primarily causes Hepatitis C disease, affecting the liver through transmission, mainly via blood, as well as through sexual contact and transmission from mother to child via the placenta. The DiaRD-HCV RT-qPCR Qualitative Kit is a real-time reverse transcription polymerase chain reaction (RT-qPCR) diagnostic kit developed for the purpose of detecting the presence of HCV RNA in the plasma of Hepatitis C patients. The kit includes primers that amplify a 105-base region within the 5' untranslated region (UTR) of the HCV genome, along with a TaqMan probe for detection.

In the presence of HCV RNA in the sample, during the amplification program applied after the reverse transcription stage, the target region is amplified using forward and reverse primers. During the annealing stage of the amplification program, TaqMan probes bind to the target region. In the extension stage, the DNA polymerase enzyme cleaves the probe from its 5' end, freeing it from the suppressor at the 3' end and emitting fluorescence. The resulting fluorescent signals are collected throughout the amplification process and reported in the form of amplification graphs.

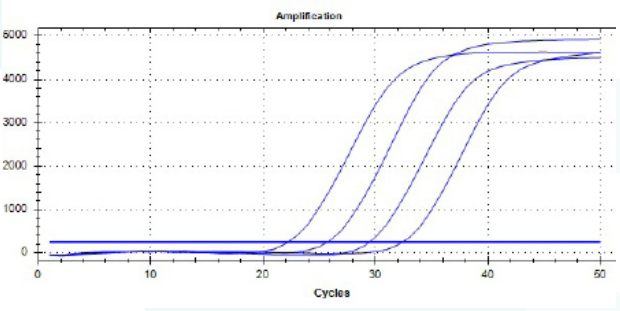


Hepatitis C Quantitive Rt-PCR kit

The Hepatitis C virus primarily causes Hepatitis C disease, affecting the liver through transmission, mainly via blood, as well as through sexual contact and transmission from mother to child via the placenta. The DiaRD-HCV RT-qPCR Quantitative Kit is a real-time reverse transcription polymerase chain reaction (RT-qPCR) diagnostic kit developed for the quantitative determination of HCV RNA load in the plasma of Hepatitis C patients in an in vitro environment. The kit includes primers that amplify a 105-base region within the 5' untranslated region (UTR) of the HCV genome, along with a TaqMan probe for detection and quantification standards used to determine RNA quantity.

In the presence of HCV RNA in the sample, during the amplification program applied after the reverse transcription stage, the target region is amplified using forward and reverse primers. During the annealing stage of the amplification program, TaqMan probes bind to the target region. In the extension stage, the DNA polymerase enzyme cleaves the probe from its 5' end, freeing it from the suppressor at the 3' end and emitting fluorescence. The resulting fluorescent signals are collected throughout the amplification process and reported in the form of amplification graphs.

The HCV RNA load in positive samples is determined using data from IU/mL corresponding to quantification standards (KS1-KS4) and their corresponding Ct values, generated from a standard curve created in each study.



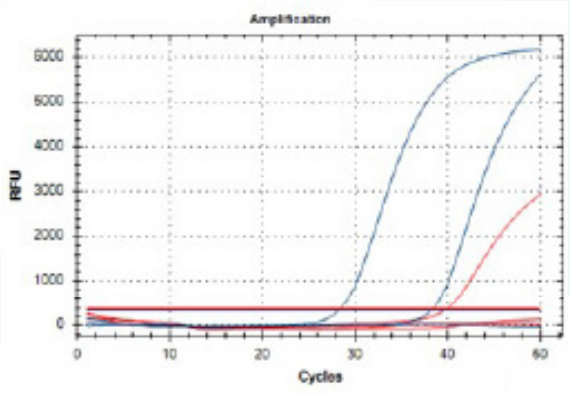
Molecular Genetics Diagnostic Products

MICROBIOLOGY
INFECTION PANEL

HIV Qualitative Rt-PCR kit

The HIV-1 Rt-PCR Qualitative Kit is a real-time reverse transcription polymerase chain reaction (RT-qPCR) molecular diagnostic kit developed for the qualitative determination of the presence of HIV-1 RNA in the plasma of AIDS patients in an in vitro environment. The kit includes all the necessary components to detect HIV-1 RNA in the plasma of patients with acquired immunodeficiency syndrome (AIDS). Within the kit, there are primers that amplify a 121-base region within the long terminal repeat (LTR) of the HIV-1 genome, along with a TaqMan probe labeled with a fluorophore dye and suppressor for detection.

In the presence of HIV-1 RNA in the sample, during the amplification program applied after the reverse transcription stage, the target region is amplified using forward and reverse primers. During the annealing stage of the amplification program, TaqMan probes bind to the target region. In the extension stage, the DNA polymerase enzyme cleaves the probe from its 5′ end, freeing it from the suppressor at the 3′ end and emitting fluorescence. The resulting fluorescent signals are collected throughout the amplification process and reported in the form of amplification graphs.



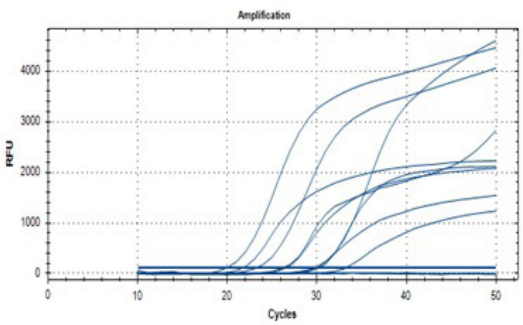
HIV Quantitive Rt-PCR kit

The HIV-1 Rt-PCR Quantitative Kit is a real-time reverse transcription polymerase chain reaction (RT-qPCR) molecular diagnostic kit developed for the quantitative determination of the HIV-1 RNA load in the plasma of AIDS (acquired immunodeficiency syndrome) patients in an in vitro environment. The kit is a quantitative test that, along with clinical findings and other laboratory data, assists in monitoring the prognosis of the disease and the effectiveness of antiviral treatment.

The kit includes primers that amplify a 121-base region within the long terminal repeat (LTR) of the HIV-1 genome, along with a TaqMan probe labeled with a fluorophore dye and suppressor for detection. Additionally, four quantification standards (KS1-KS4) are included in the kit. In the presence of HIV-1 RNA in the sample, during the amplification program applied after the reverse transcription stage, the target region is amplified using forward and reverse primers.

During the annealing stage of the amplification program, TaqMan probes bind to the target region. In the extension stage, the DNA polymerase enzyme cleaves the probe from its 5′ end, freeing it from the suppressor at the 3′ end and emitting fluorescence. The resulting fluorescent signals are collected throughout the amplification process and reported in the form of amplification graphs.

The HIV-1 RNA load in positive samples is determined using data from copy/mL corresponding to quantification standards (KS1-KS4) and their corresponding Ct values, generated from a standard curve created in each study.



MICROBIOLOGY
INFECTION PANEL

MICROBIOLOGY INFECTION PANEL
Helicobacter pylori Rt-PCR Kit
Helicobacter pylori Antibiotic Resistance Rt-PCR Kit
Malaria Screening Rt-PCR Kit
Malaria ID Rt-PCR Kit
M. tuberculosis RIF Resistane Rt-PCR Kit
Mycobacterium tuberculosis Resistance (MDR) Rt PCR kit
Cytomegalovirus Rt-PCR Kit
Epstein-Barr Virus Rt-PCR Kit
Human Papilloma Virus Rt-PCR Kit
Hepatitis D Rt-PCR Kit
Yellow Fewer Rt-PCR Kit
Morbillivirus paramyxoviridae Rt-PCR Kit
Neisseria Meningitidis Genotyping Realtime PCR HD-FRT Kit (A/B/C/W/X/Y)

MEDICAL GENETICS

Ankylosing Spondylitis HLA-8*27 Real Time hD-frt Kit

Product No: 1108-25hB

The genes encoding human leukocyte antigen (HLA) are highly poly morphic genes and are located in the Major Histocompatibility Complex (MHC) on the short arm of chromosome 6. These genes encode basically two classes of celi surface glycoproteins. The frequency of alleles of genes encoding these proteins varies from population to population. The HLA-B27 allele is approximately 9% common in Caucasians. The first clinical relationship between HLA-B27 and Ancholysing Spondylitis (AS) was demonstrated in the 1980s. After this finding, the presence of the HLA-B27 allele has been associated with many other rheumatoid diseases such as Reiter's Syndrome (RS) and Acute Anterior Uveitis (AAU). Recent studies have shown its relationship with many other diseases such as heart valve diseases, cardiac conduction diseases and immune system diseases.

Sex Determination (SRY and Y-chr. analysis)

Differences in the sexes are due to irregularities in the X and Y chromosomes. X-monosomy karyotype is seen in 50-60% of gonadal dysgenesis cases. The remaining 50-40% includes numerical and structural changes in the X and Y chromosomes.

The SRY gene plays an important role in both male sex development and testicular development. The abnormal SRY gene can cause sex reassignment. Cytogenetic analyzes may not always detect such irregularities in the Y chromosome. Numerical and structural irregular ities in the Y chromosome are associated with Turner Syndrome, Pseudohermaphroditism, Male Dysgenetic and Mixed Gonadal Dys genesıs.

Thrombophilia Panel
Sub Products:
Factor ll (Prothrombin G20210A) Real Time HYB-FRT Kit
Factor V Leiden (G1691A) Real Time HYB-FRT Kit
MTHFR (A1298C) Real Time HYB-FRT Kit
MTHFR (C677T) Real Time HYB-FRT Kit
PAl-1 (4G/5G) Real Time HYB-FRT Kit

Molecular Genetics Diagnostic Products

SPECIES DETERMINATION PANEL

Species Determination in Animal-based food

Diagen-branded Species Detection Real-Time PCR Kits detect the presence of relevant species in processed and unprocessed foods. This Real Time PCR Kit has been produced in order to determine the species in animal-derived foods that the market needs.

SPECIES DETERMINATION PANEL
Species Determination in Animal-based food
Horse - Pig- Donkey Species Identification Real Time PCR Kit /tem No:2108-25HD
Horse Species Identification Real Time PCR Kit /tem No:2101-25HD
Pig Species Identification Real Time PCR Kit /tem No:2105-25HD
Donkey Species Identification Real Time PCR Kit /tem No:2102-25HD
Turkey Species Identification Real Time PCR Kit /tem No:2106-25HD
Chicken Species Identification Real Time PCR Kit /tem No:2107-25HD
Chicken-Turkey Species Identification Multiplex Real Time PCR Kit /tem No: 2113-25HD
Goat Species Identification Multiplex Real Time PCR Kit /tem No:2110-25HD
Cattle Species Identification Real Time PCR Kit /tem No:2103-25HD
Cow Species Identification Multiplex Real Time PCR Kit /tem No:2112-25HD
Sheep Species Identification Real Time PCR Kit /tem No:2104-25HD

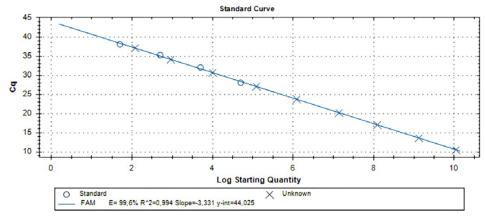
SPECIES DETERMINATION PANEL

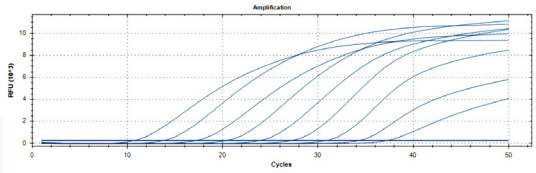
Identification of Bacterial Species

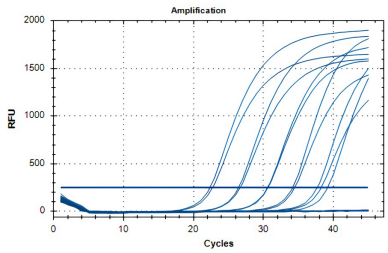
DiaRD-VDE Vancomycin resistant enterococci RT-PCR Kit

Real-time PCR Kit far vancomycin resis tant enterococci has been developed far the detection of vancomycin resistant genes. The kit has been designed to have the broadest possible detection profile while remaining specific to the vanA and vanB genes that cause vancomycin resistance. The primer sequences in this kit have 100% homology with a wide range of vancomycin-resistant enterococci as a result of extensive bioinfor matic analysis. The widespread use of antibiotics has resulted in shifts in bacterial development to overcome existing mechanisms to combat bacterial infections. Antibiotics such as penicillin and erythromycin, which used to have high activity against many bacterial species and strains, have become less effective due to the increased resistance of many bacterial strains. Vancomycin-resistant enterococci (VRE) tend to multiply rapidly due to the high frequency and continued use of vancomycin.

Enterococci are a group of gram-positive, round-shaped bacteria that usually live in the gut, although they can cause infection anywhere in the body. Vancomycin is a drug with antibacterial action by forming a complex with the D-alanyl-D-alanine terminal of the peptidoglycan precursor to block transglycosylation and transpeptidation during the synthesis of the celi wall of Gram-positive cocci.







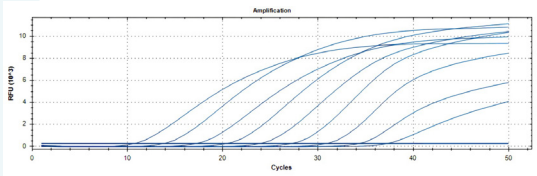
Molecular Genetics Diagnostic Products

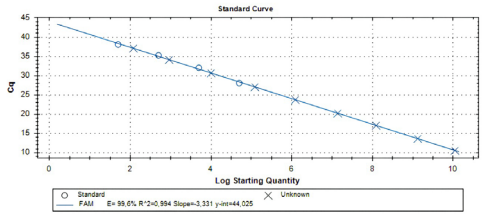
SPECIES DETERMINATION PANEL

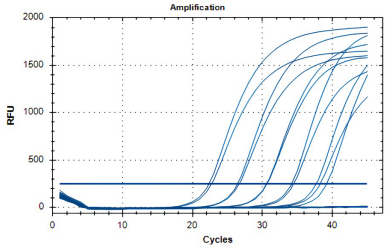
DiaRD-MRSA MRSA RT-PCR Kit

Real-time PCR Kit for methicillin (MRSA) resistance was developed far the in vitro detection of methicillin-containing Staphylococcus aureus genomes. The kit has been designed to have the broadest possible detection profile while remain ing specific to the S. aureus genome. The primer sequences in this kit have 100% homology to a wide range of MRSA-containing S. aureus sequences asa result of extensive bioinformatics analysis. The resistance gene mecA is used to detect resistance to methicillin.S. aureus is a Gram-positive bacterium from the Staphylococcaceae family. The genome of this species is about 2.8 Mb in length. Virulence factors far S. aureus are encoded by phages, pathogenicity islands, staphylococcal cassette chromosome, and plasmids that contain genes for antibiotic resistance, such as those found in Methicillin-Resistant S. aureus (MRSA). S. aureus can be found on the skin and mucous membranes of healthy individuals, but erosion of these barriers allows bacteria to enter the wound, colonize and cause infection.

Since this bacterium lives on the skin, it can easily be transmitted between individuals and is usually seen in hospital-acquired infections. S. aureus can cause a variety of different infections, from mild skin infections such as impetigo to invasive diseases and toxic-mediated diseases. Production of exotoxins known as pyrogenic toxin superantigens in some strains of S. aureus can cause toxic shock syndrome. Realtime PCR is the fastest and most reliable method of performing an accurate S. aureus detection.







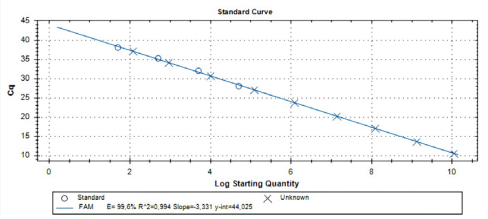
SPECIES DETERMINATION PANEL

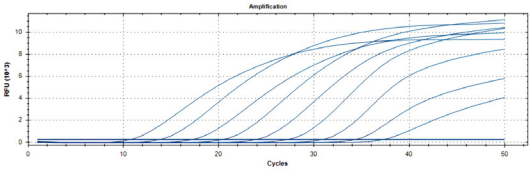
3026-100 Staphylococcus aureus RT-PCR Kit

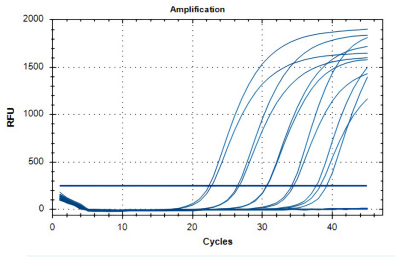
Real-time PCR Kit for Staphylococcus aureus has been developed for in vitro quantification of S. aureus genomes. The kit has been designed to have the broad estpossible detection profile while remaining specific to the S. aureus genome. The primer sequences in this kit have 100% homology with a wide range of s. aureus sequences as a result of extensive bioinformatics analysis. The chromosomal gene femA is used to detect S. aureus.

S. aureus is a Gram-positive bacterium from the Staphylococcaceae family. The genome of this species is about 2.8 Mb in length. Virulence factors for S. aureus are encoded by phages, pathogenicity islands, staphylococcal cassette chromosome, and plasmids that contain genes for antibiotic resistance, such as those found in Methicillin-Resistant S. aureus (MRSA). S. aureus can be found on the skin and mucous membranes of healthy individuals, but erosion of these barriers allows bacteria to enter the wound, colonize and cause infection. Since this bacterium lives on the skin, it can easily be transmitted between individuals and is usually seen in hospital-acquired infections.

S. aureus can cause a variety of different infections, from mild skin infections such as impetigo to invasive diseases and toxic-mediated diseases. Production of exotoxins known as pyrogenic toxin superantigens in some strains of S. aureus can cause toxic shock syndrome. Realtime PCR is the fastest and most reliable method of performing an accurate s. aureus detection.





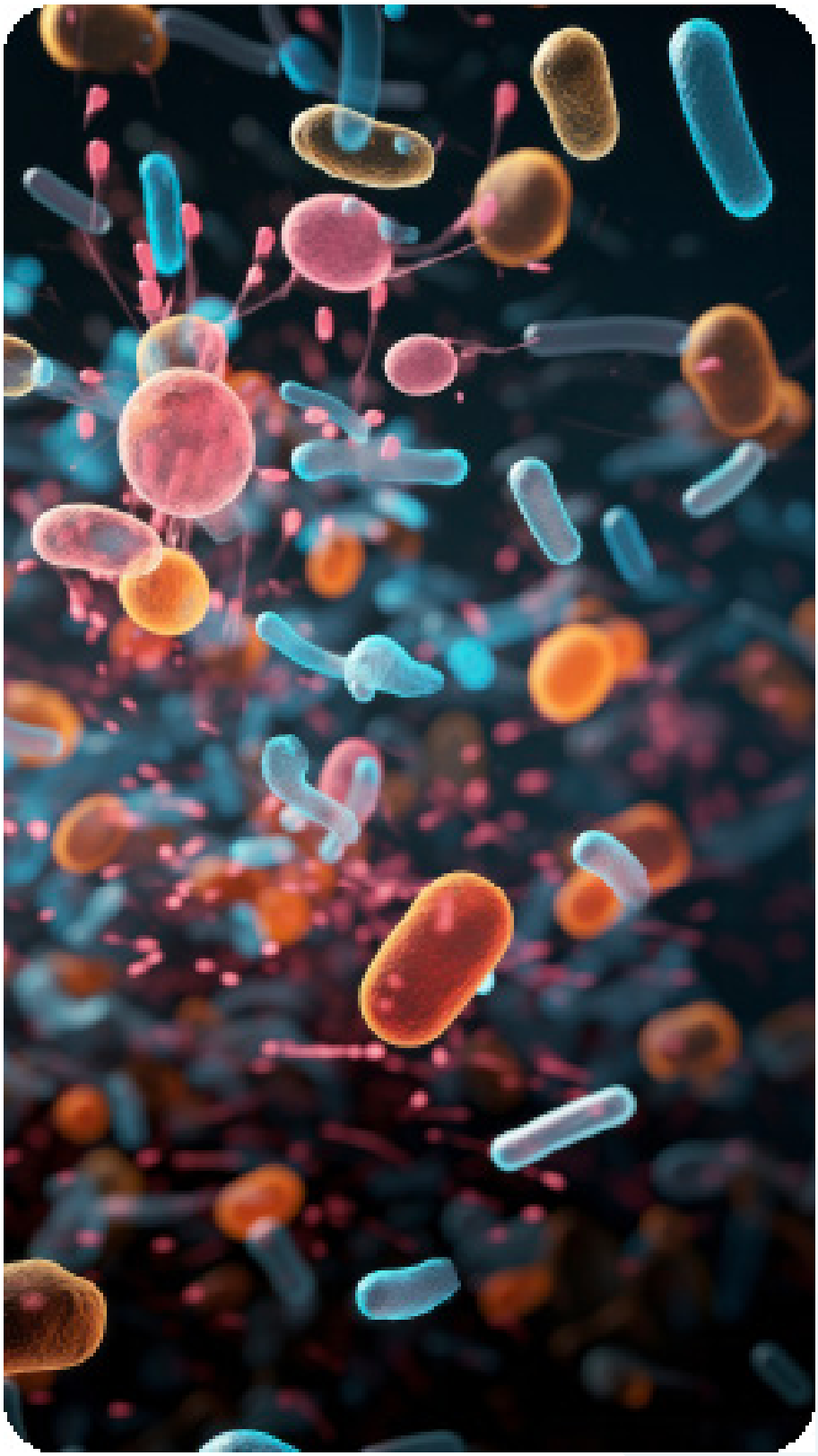


SPECIES DETERMINATION PANEL

DiaRD-MRSA MRSA RT-PCR Kit

SPECIES DETERMINATION PANEL
DiaRD-MRSA MRSA RT-PCR Kit
DiaRD-LSM Listeria monocytogenes RT-PCR Kit
3001-100 Acinetobacter baumannii RT-PCR Kit
3002-100 Akkermansia muciniphila RT-PCR Kit
3009-100 Escherichia cali RT-PCR Kit
3010-100 Eubacterium rectale RT-PCR Kit
3011-100 Faecalibacterium prausnitzii RT-PCR Kit
3012-100 Faecalibacterium RT-PCR Kit
3013-100 Kingella kingea Realtime RT-PCR Kit
3016-100 Lactobacillus acidophilus RT-PCR Kit
3017-100 Lactobacillus brevis Realtime PCR
3018-100 Lactobacillus fermentum RT-PCR Kit
3019-100 Lactobacillus plantarum RT-PCR Kit
3020-100 Lactobacillus rhamnosus RT-PCR Kit
3023-100 Salmonella enteritidis RT-PCR Kit
3024-100 Salmonella spp. RT-PCR Kit
3025-100 Salmonella typhimurium RT-PCR Kit
DiaRD-CRE CRE (VIM,IMP,NDM,KPC) Carbapenem-Resistant
Enterobacter RT-PCR Kit
3027-100 Streptococcus mutans RT-PCR Kit
3028-100 Streptococcus pyogenes RT-PCR Kit
3033-100 Lactobacillus reuteri RT-PCR Kit
3034-100 Clostrodium spp. RT-PCR Kit
3035-100 Bifidobacterium spp. RT-PCR Kit

SPECIES DETERMINATION PANEL



SERVICES

Biostatistical Analyses

Bioinformatics services are provided for PCR, Next-Generation Sequencing (NGS), and Sanger sequencing studies. Our team supports researchers from the beginning to the end of their projects, assisting in the interpretation of data.

Analyses are conducted using applications and programming languages such as SPSS, CopyCaller, R, and Python. Following the analyses, data visualization is performed, and training is provided for the interpretation of visuals.

Our team is dedicated to supporting researchers in making sense of their data, ensuring a comprehensive and insightful analysis from the initiation to the conclusion of your studies.

Microbiota Analyses

At Diagen, we conduct Microbiota projects domestically using Illumina systems and our advanced server infrastructure. Microbiota studies are performed on stool, meconium, breast milk, vaginal swabs, cervical swabs, oral swabs, and skin swab samples.

Next-Generation Sequencing (NGS) results from Illumina systems are obtained as raw data from the device and processed using the most up-to-date microbiome databases (Silva, greengenes, and others) with tools developed by us and those openly published, as used in the Human Microbiome Project (see "Sample Report" for detailed results and visuals).

Python and R programming languages are employed for analyses and data visualizations (see "Statistical Analyses" for a list of analyses conducted as part of the project).

Key Workstreams in Microbiota Projects:

Consultancy for Project Preliminary Preparation:

Providing guidance for the initial setup of the project.

Storage and Transfer of Samples under Appropriate Conditions (-80°C):

Ensuring proper preservation and transfer of samples.

Nucleic Acid Purification Processes: Conducting purification processes for nucleic acids.

Sequencing (NGS):

Performing sequencing using Illumina systems.

Bioinformatic Analysis:

- Raw Data (Sanger and NGS) Analyses.
- Cleaning of Raw Data.
- Determination of Quality Scores.
- Alignment Processes.
- Data Mining.
- Pathway Analyses.
- Statistical Modeling.

Biostatistics:

- Power Analyses.
- Alpha-Beta Diversity.
- Density Tables.
- Parametric and Non-Parametric Tests.
- Correlation and Regression Tests.

Training on the Interpretation of Results:

Providing education on interpreting the results.

Nucleic Acid Extraction Analyses

Nucleic acid extraction kits are designed for the rapid and pure extraction of nucleic acids from various tissues (blood, kidney, heart, lung, brain, muscle, liver, spleen, etc.) and body fluids, as well as viruses, bacteria, fungi, and cells cultured up to 5 x 10^6.

Nucleic acid purification kits achieve high efficiency in a short time using optimized reagents and protocols. When applied according to the protocol instructions, the optimized systems yield results with A260/280 ratios between 1.7-1.9 and A260/230 ratios between 1.6-2.1.

These kits are available in spin-column, air-flow system, and magnetic bead-based formats to suit your needs.

The obtained DNA and RNA can be directly used in PCR, qPCR, RT-PCR, Northern/Southern Blot, Next-Generation Sequencing, and enzymatic reactions.

SERVICES

PCR (Polymerase Chain Reaction) Analyses

Within the R&D unit of Diagen Biotechnology, genetic research is conducted using PCR systems, specializing in mutation analyses, molecular diagnosis, and species typing with an expert team. In addition to routine services, laboratory support is provided for projects. Our laboratory and bioinformatics unit, staffed with experts, work to provide reliable services by listening to individual needs and preferences.

In our laboratory, we use Roche LightCycler 480 II, ABI7500 Fast, ESCO 96, and Qiagen Rotor Gene Q devices, capable of providing accurate measurements for conventional PCR (ABI Veriti and Aeris) and real-time PCR studies.

Our goal is to provide personalized services by meeting individual requests such as reporting format, turnaround time, and plan changes based on the methods used in the studies.

Key Focus Areas in PCR Studies:

- Mutation Analyses
- Allele Detection
- Hydrolysis and Hybridization Probe-based QPCR
- Non-Specific qPCR (EvaGreen, SybrGreen, and Intercalating Dyes)
- Gene Expression Analyses
- Telomere Length Analyses
- miRNA Expression Analyses
  - miRNA Profiling
  - miRNA Pathway Analyses
- cDNA Synthesis
- Specific Primer and Probe Design
- Methylation Analyses
- Optimization Studies
- Specificity and Sensitivity Studies
  - Primer and Probe Optimizations
  - Device Optimization
  - Internal Laboratory Inspection
  - External Laboratory Inspection

Sequencing Service

Sanger Sequencing Analyses

Diagen Biotechnology provides services for your projects with DNA sequencing analyses. Using Sanger sequencing systems, sequence analyses of samples, nucleic acids, or PCR products are conducted, and results are delivered as quickly as possible.

Genetic material sequencing processes are carried out using the ABI Prism Genetic Analyzer.

Serological Analyses

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In ELISA diagnostic and research kit applications:

- Relevant applications for the kits
- Relevant analyses after application
- Reporting is carried out

Design of Primers and Probe Sets

Within the scope of your studies, primer and probe sets are designed according to the experimental purpose. In the design consultancy, genetic information of the desired species is examined, and designs are created for conventional PCR, dsDNA-binding dyes, hydrolysis, and hybridization probes.