dia-chem s.r.l.

TECNOLOGIE BIOMEDICHE
BIOLOGIA MOLECOLARE

NEW CATALOGUE

Molecular diagnostics kit

Company Portrait

Dia-chem S.r.l. was founded in 1989 by a group of managers with significant experience in the world of diagnostics and collaborates with a team of researchers for development and standardization of molecular biology kits. Dia-chem S.r.l. focuses on innovation and collaboration with the international scientific community since its inception. This multinational presence strengthens the ability to offer solutions in the field of clinical diagnostics, anticipating needs of each customer. Dia-chem S.r.l. is present on the market with following diagnostic lines: Pharmacogenetics, Oncohematology, Oncology and Epigenetics, Hemostasis and Thrombosis, Hemochromatosis, Virology, Neurology, Infertility and Panels for Predisposition to Inflammation, Food Intolerance and Recurrent Abortion. Our proposal focuses on quality, practicality and implementation of international reference techniques, with particular attention to management costs. The primary goal is identification and satisfaction of customer needs; this involves solving their problems and anticipating their future needs, maintaining close contacts. Customer consultancy is a key element in our strategy to develop and produce kits that meet market needs and exceed expectations. Our core business is the molecular analysis of DNA, RNA and proteins performed in molecular diagnostic laboratories. Modern and innovative analyses based on the detection of nucleic acids offer significant advantages over traditional diagnostic methods. These procedures allow the detection of genetic mutations, chromosomal rearrangements, chromosomal translocations, deletions, insertions, and viral, bacterial and parasitic diseases more quickly with greater sensitivity and specificity. The cause of a disease can be found early and specifically allowing for more suitable therapy.

Molecular diagnostics in modern medicine provides the necessary tools for the development of new strategies for treatment of various diseases. Hospitals and diagnostic laboratories that use these techniques require high sensitivity, high specificity, ease of use, fast analysis times and low costs.

Dia-chem S.r.l. offers a wide range of CE IVD devices with techniques based on Real-Time Polymerase Chain Reaction (RT-PCR), Sanger sequencing and Restriction Fragment Length Polymorphism (RFLP) and are supplied both directly to healthcare professionals and through distributors. These products are not instrumentation dependent and are used on different analytical platforms such as: Rotor-Gene™ (Qiagen), LineGeneKTM (Bioer Technologies), iQ5, CFXTM (BioRad), SmartCycler™ (Cefeide), Applied Biosystems 7300/7500, StepOne, MX3005PTM, MX3000 PTM (Agilent Technologies).

Dia-chem S.r.l. is registered in the National Research Register of the Ministry of University and Research (MUR) at N.60236RGP.

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Quality Management System

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Design, production and marketing of molecular biology kits. Distribution of in vitro diagnostic and disposable products for clinical analysis laboratories.

which fulfils the requirements of the following standard:

ISO 9001:2015

Issued on: 10/06/2024
First issued on: 26/09/2000
Expires on: 22/06/2027

Registration Number: IT- 10708 - 2114

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Design, production and placing on the market of kit for molecular biology. Distribution of in vitro diagnostic and disposable for clinical laboratories.

which fulfils the requirements of the following standard:

ISO 13485:2016

Issued on: 13/06/2024
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PHARMACOGENETICS

Study of genetic variants that influence the response to pharmacological treatments

The variability in the response to pharmacological treatment between patients has always been one of the most relevant problems in clinical practice. Individual responses to drugs, in fact, vary greatly: reduced or even absent therapeutic effects, adverse reactions or side effects can be observed in some patients compared to others, despite the same drug being administered at the same dosage. This inter-individual variability in the past was mainly attributed to the influence of non-genetic factors such as age, sex, nutritional status, renal and hepatic function, lifestyle habits, diet, alcohol abuse and smoking, concomitant intake of other drugs or the presence of comorbidities. Currently, it is believed that, in addition to the factors mentioned above, hereditary factors also play an important role in the individual response to drugs.
Results of studies on monozygotic and dizygotic twins suggest that, for certain drugs subject to intense metabolism, genetic factors play an important role in determining pharmacokinetic and pharmacodynamic variability. The clinical consequences of interindividual variability in response to pharmacological treatment may therefore be represented by therapeutic failure (lack of or only partial efficacy of the therapy), by side effects of a given active ingredient or by serious and sometimes fatal adverse reactions. Pharmacogenetics was born around the 1950s when researchers began to think that the response to drugs could also be regulated in part by genes and that the variability of reaction to a given active ingredient by different individuals was nothing more than a reflection of genetic differences. Pharmacogenetics studies inter-individual variations in the DNA sequence in relation to the response to drugs. The practical application of knowledge from research in pharmacogenetics consists in the possibility of predicting a patient's response to a given drug on the basis of a routine genetic test, to arrive at an individualization of the therapy, "the right drug for the right patient". DNA tests based on these genetic variations can predict how a patient will respond to a particular drug. Clinicians can use this information to decide on the optimal therapy and to personalize the dosage; the benefits will consist of a reduced incidence of adverse reactions, better clinical outcomes and reduced costs. These tests represent the first step towards patient-specific therapies.
Variations in the DNA sequence that are present in at least 1% of the population are called polymorphisms. These gene polymorphisms give rise to enzymes with different levels of metabolic activity or receptors with different affinity for the drug, modifying the pharmacological response of an individual. Genetic variations most often involve a single nucleotide and are therefore called single nucleotide polymorphisms (SNPs), but they can also involve multiple nucleotides or even large stretches of DNA: these are, for example, substitutions, insertions, deletions, amplifications and translocations. They refer to monogenic traits, that is, polymorphisms of a single gene encoding a protein involved in the metabolism of a drug or in its effect that cause variable individual responses to drugs. The problem of individual variability in drug response is particularly important in tumor therapy because in this case drugs characterized by a particularly narrow therapeutic index are used, with minimal variation between effective dose and toxic dose. Even limited alterations in the metabolism of an antitumor chemotherapy due to genetic variations can cause significant changes in the pharmacological effect in terms of both toxicity and efficacy. Unfortunately, this occurs frequently because the dosage of antineoplastic agents is established by the oncologist in a standardized manner based on the patient's body surface area (obviously also taking into account other non-genetic variability factors).
As with other pathologies, variations in the DNA sequence may affect the structure of genes that code for enzymes of drug metabolism and transport or for proteins involved in the action of drugs, influencing their fate in the organism, toxicity and also, as more recently highlighted, efficacy. In this regard, it is important to underline that not only the polymorphisms of the host genome, but also those of the tumor genome can influence the response to antineoplastic drugs. The polymorphisms of the host and tumor genomes both regulate the transport, retention and efflux of antitumor drugs, determining the degree of penetration into the tumor tissue; the tumor genome possesses the majority of polymorphisms that influence tumor aggressiveness and its drug sensitivity or resistance; the polymorphisms of the host genome represent the main determinants of the risk of toxicity for the patient, to which the polymorphisms of the tumor genome do not contribute substantially.

PHARMACOGENETICS with AGAROSE GEL DETECTION

Code	Description	Tests n.
1.419	**Ampli MGMT**	24
1.423	**Ampli B-RAF V600E** (RFLP)	24
1.428	**Ampli K-RAS** (RFLP)	24
1.428C	**Ampli K-RAS c**-DNA Mutations in 12,13 codons	24
1.428N	**Ampli K-RAS COLD NESTED** (RFLP) Mutations in 12,13 codons with cold-nested PCR	24
2.001	**Ampli CYP2C9-VKORC1** (RFLP) CYP2C9*2, CYP2C9*3 polymorphism in CYP2C9 gene and 1639G>A in VKORC1 gene	24
2.002	**Ampli EGFR ELREA** (RFLP)	24
2.003	**Ampli EGFR Leu858Arg** (RFLP)	24
2.015	**Ampli CDA -92 A/G** (RFLP)	100
2.020	**Ampli VDR** (RFLP) TaqI, ApaI, BmsI e FokI polymorphism in Vitamin D Receptor	24
2.022	**Ampli TYMS 28bp tandem repeat** (RFLP)	25
2.024	**Ampli ERCC2 (A751C)**	45
2.027	**Ampli DPYD** (RFLP) ISV14+1G>A in DPYD*2A	25
2.036	**Ampli LCT** (RFLP) Polymorphism in -13910 of LCT	25

PHARMACOGENETICS with AGAROSE GEL DETECTION

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PHARMACOGENETICS IN REAL-TIME PCR

Code	Description	Tests n.
1.356RT	**Ampli ABCB1** 3435 C>T polymorphism in ABCB1 Gene	25
1.419RT	**Ampli MGMT**	48
1.423RT	**Ampli B-RAF** (V600E)	25
1.428RT	**Ampli K-RAS** in Codons 12 (G12R, G1S,G12D,G12C,G12V, G12A), 13 (G13D, G13C), 59 (A59T), 61 (Q61K, Q61L, Q61R, Q61H), 117 (K117N), 146 (A146T, A146V, A146P) mutations.	12
1.433RT	**Ampli N-RAS** in Codons 12 (G12S,G1D,G12C,G12V), 13 (G13R, G13D,G13V), 59 (A59D), 61 (Q61K, Q61L,Q61R, Q61H), 117 (K117N),146 (A146T) mutations.	12
2.002RT	**Ampli EGFR ELREA**	25
2.003RT	**Ampli EGFR** Mutations: G719X es 18, Del. E19del es 19, L858R es 21, L861Q es 21, T790M es 20, S768I es 20 and Ins. E20ins. es 20 in EGFR Gene	12
2.004RT	**Ampli GSTP1 Ile105Val**	25
2.005RT	**Ampli MDR1 C3435T**	25
2.006RT	**Ampli MDR1 G2677T**	25
2.007RT	**Ampli MDR1 G2677T/A**	25
2.012RT	**Ampli CDA 451 C/T**	25
2.013RT	**Ampli CDA 79 A/C**	25
2.014RT	**Ampli DCK C28624T**	24
2.015RT	**Ampli CDA -92 AG**	25
2.016RT	**Ampli HLA B 5701**	30

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PHARMACOGENETICS IN REAL-TIME PCR

Code	Description	Tests n.
2.017RT	**Ampli IL28 b**	50
2.018RT	**Ampli CDA 435 C/T**	25
2.019RT	**Ampli CDA -31del C**	25
2.023RT	**Ampli XRCC3 (A4541G)**	25
2.024RT	**Ampli ERCC2 (A751C)**	25
2.027RT	**Ampli DPYD *2A *13 *2846** *2A (rs391829), *13(rs55886062), *2846(rs67376798) A>T polymorphism in DPYD Gene	25
2.027.1RT	**Ampli DPYD *1236G>A, *2194G>A** *1236G>A, *2194G>A polymorphism in DPYD Gene	25
2.028RT	**Ampli NAFLD** KLF-6, LPIN-1, PNPLA-3, SOD-2 genotypes	24
M2.030RT	**Multi kit Citidinadeaminasi (CDA)** 451 C/T, 79 A/C, 435 C/T, 92 S/G, -31del C polymorphism in CDA gene	24
M2.031RT	**Multi kit TYMS 28bp tandem repeat, XRCC3, ERCC2, UGT1A1**	25
2.032RT	**AMPLI UGT1A1 *28 *36 *37** *28 *36 (TA5/TA5), *37 (TA8/TA8) polymorphism in UGT1A1 gene	25
2.033RT	**Ampli CYP2C19 2 *3 *17** *2 681G>A (rs4244285), *3 636G>A (rs4986893), *17 806C>T (rs12248560) polymorphism in CYP2C19 Gene	25
2.034RT	**AMPLI TPMT** Polymorphism of Thiopurine S-metiltrasferasi (TPMT)TPMT *2, TPMT *3A e TPMT *3C gene	25
2.035RT	**Ampli CYP2C8 *3 *22**	24
2.037RT	**Ampli CYP2C19 *17**	24
2.040RT	**AMPLI HLA B27**	48

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PHARMACOGENETICS IN REAL-TIME PCR

Code	Description	Tests n.
2.041RT	**AMPLI HLA B*51**	48
2.045RT	**Ampli CYP2D6*3 Identification** *3 2549delA polymorphism in CYP2D6 Gene	24
2.046RT	**Ampli CYP2D6*4** *4 G1846A polymorphism in CYP2C6 Gene	24
2.047RT	**Ampli CYP2D6*10** *10 C100T polymorphism in CYP2D6 Gene	24
2.048RT	**Ampli CYP1A2 *1C *1F** *1C *1F (rs2069514) polymorphism in CYP1A2 Gene	50
2.049RT	**Ampli CTH** 1364G>T (rs1021737) and -1320 C>T (rs648743) polymorphism in CTH gene	25
2.050RT	**Ampli GSTA1*B** C>T (rs3957357) polymorphism in GSTA1*B Gene	25

PHARMACOGENETICS IN REAL-TIME PCR

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PHARMACOGENETICS SANGER SEQUENCING

Code	Description	Tests n.
1.423seq	**Ampli B-RAF seq V600E**	25
1.424seq	**Ampli c-Kit 9**	25
1.425seq	**Ampli c-Kit 11**	25
1.426seq	**Ampli c-Kit 13**	25
1.428nseq	**Ampli K-RAS COLD PCR** Mutations in 12, 13 codons in KRAS gene with cold PCR and sanger sequencing	25
1.428seq	**Ampli K-RAS seq** Mutations in 12, 13 e 61 codons in KRAS gene	25
1.431seq	**Ampli c-kit 17**	25
1.436seq	**Ampli p53** Gene Tp53 mutations in 2, 3, 4, 5, 6, 7, 8 and 9 exons	25
1.437seq	**Ampli PDGFR α** Most frequent mutations in exons 12, 14 e 18 of the gene PDGFR alpha	25
2.008seq	**Ampli EGFR seq ex.18**	25
2.009seq	**Ampli EGFR seq ex.19**	25
2.010seq	**Ampli EGFR seq ex.20**	25
2.011seq	**Ampli EGFR seq ex.21**	25
2.012seq	**Ampli EGFR seq** Most frequent mutations in exons 18, 19, 20 and 21 of the gene EGFR	25

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ONCOHEMATOLOGY

Identification of gene mutations involved in the development of cancer

With the advent of new biomolecular diagnosis techniques, Oncohematological diseases can avail themselves of efficient tools for diagnosis and monitoring, thanks to the Polymerase Chain Reaction (PCR) technique, for the analysis of monoclonality, rearrangements, chromosomal translocations and to perform early and differential diagnoses, in addition to the evaluation of minimal residual disease (MRD). In oncohematological diseases, specific chromosomal translocations are present, which lead to the fusion of genes that code for proteins with oncogenic properties. These molecular alterations are exploited as specific markers of the neoplastic cell; they can be searched for at the level of nucleic acids (DNA and RNA), using the Polymerase Chain Reaction (PCR) technique, which allows the amplifying of the neoplastic marker. This technique is successfully used both at diagnosis, to better characterize the pathology and specify the prognosis, and in the study of the so-called minimal residual disease (MMR), allowing to verify whether neoplastic cells are present in the body after having treated the patient.
Myeloproliferative Neoplasms (MPN) are one of the categories of myeloid neoplasms according to the classification of the World Health Organization (WHO). Philadelphia-negative MPNs are Polycythemia Vera (PV), Essential Thrombocythemia (ET), Primary Myelofibrosis (PMF) and Primary Myelofibrosis in the prefibrotic phase. These disorders are neoplastic diseases that affect hematopoietic stem cells. In 2005, a point mutation (single nucleotide substitution G>T at nucleotide 1849, resulting in the substitution of a valine residue with a phenylalanine residue at position 617) was identified in exon 14 of the JAK2 gene, encoding a tyrosine kinase (Janus Kinase 2) involved in the intracellular JAK-STAT signaling pathway. This mutation, JAK2V617F, is present in almost all patients with Polycythemia Vera (95%) and in a significant proportion of patients with Essential Thrombocythemia (55%) and Primary Myelofibrosis (65%). In the remaining percentage of patients with Polycythemia Vera, mutations of the JAK2 gene other than V617F can be found. These mutations are concentrated in exon 12.
Somatic mutations were subsequently identified in the MPL gene, which encodes the thrombopoietin receptor, involved in the same intracellular signaling pathway mediated by JAK-STAT. The mutations confer a functional gain to the mutated protein, which is constitutionally active. The MPL gene mutations found in MPNs are concentrated in exon 10, involving the codon W515 (MPLW515L and MPLW515K), and are present in approximately 4-5% of patients with Essential Thrombocythemia and in 7-10% of patients with Myelofibrosis.
Finally, in 2013, the presence of mutations in the CALR gene (encoding Calreticulin) was identified in the population of patients who had no mutations in either JAK2 or MPL. 80% of cases are constituted by the type 1 mutation (deletion of 52 base pairs) and the type 2 mutation (insertion of 5 base pairs). However, there remains a percentage of "triple negatives" for which it is not possible to identify the mutation characterizing the neoplasia.
Dia-chem S.r.l. has a complete panel of highly specific, sensitive and easy-to-perform systems for the diagnosis of oncohematological diseases.

Code	Description	Tests n.
1.400	**Ampli Lymphoma B** (VDJ) Kit made in accordance with the Biomed II Guidelines	45
1.400d4	**Ampli Lymphoma B** (VDJ) Kit made in accordance with the Biomed II Guidelines	45
1.400HS	**Ampli Lymphoma B HS** Nested PCR	45
1.400fam	**Ampli Lymphoma B** (VDJ) with capillary electrophoresis Kit made in accordance with the Biomed II Guidelines	45

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ONCOHEMATOLOGY with AGAROSE GEL DETECTION

Code	Description	Tests n.
1.400.2	**Ampli NPM/ALK t(2-5)** Anaplastic Lymphoma	45
1.400.3	**Ampli TCR β** Kit made in accordance with the Biomed II Guidelines	45
1.401	**Ampli Lymphoma T-Cell Receptor Y**	45
1.401fam	**Ampli Lymphoma T-Cell Receptor Y** with capillary electrophoresis	45
1.402	**Ampli PML-RARa bcr1, bcr2, bcr3** Translocation t(15;17) Kit made in accordance with the Biomed II Guidelines	45
1.403	**Ampli BCR-ABL p190 and p210** Translocation t(9;22) - Kit made in accordance with the Biomed II Guidelines	45
1.404	**Ampli Lymphoma Cells** Translocation t(14;18) Nested Kit made in accordance with the Biomed II Guidelines	45
1.404B	**Ampli Lymphoma Cells** Translocation t(14;18).	45
1.405	**Ampli MLL-AF4**	45
1.406	**Ampli FTL3 ITD**	45
1.407	**Ampli FTL3 D835**	45
1.408	**Ampli Mant. Lymph. t(11;14)(Q13;Q32)**	45
1.427	**Ampli JAK2 V617F**	45
1.438	**Ampli AML1ETO** Translocation t(8:21)	45

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ONCOHEMATOLOGY IN REAL TIME PCR

Code	Description	Tests n.
1.001	**Quant BCR-ABL p190** Kit made in accordance with the Biomed II Guidelines	24
1.002	**Quant BCR-ABL p210** Kit made in accordance with the Biomed II Guidelines	24
1.002/D	**Ampli CR-ABL p190/p210 Real-Time OneStep** transcripts of the m-bcr/M-bcr Fusion Gene t(9-22)	24
1.003RT	**Quant PML-RARα bcr1**	24
1.004RT	**Quant PML-RARα bcr2**	24
1.005RT	**Quant PML-RARα bcr3**	24
1.006RT	**Quant BCL2/IgH™ t(14-18)**	24
1.007RT	**Quant BCL1/IgH™ t(11-14)**	24
1.008RT	**Ampli MYCN**	24
1.009RT	**Ampli CALR** Polymorphism of the CALR (Type 1, del 52bp e Type 2, ins 5bp) gene	25
1.010RT	**Ampli MPL**	25
1.427RT	**Quant JAK2 PNA Real-Time** JAK2 V617F	24

ONCOHEMATOLOGY IN REAL TIME PCR

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ONCOLOGY AND EPIGENETICS

Genetic tests for the early diagnosis of tumors and genetic evaluation of predisposition to hereditary tumors

Today, tumors can be considered a pathology with a genetic component characterized by uncontrolled cellular growth. The cells of our body receive signals that tell them when to grow and multiply and when such growth must stop. In tumors, these cells, due to alterations in their genetic heritage, do not respond to the control signals and grow and multiply irregularly, spreading to different parts of the body. The event that determines the alteration of the function of genes is called mutation. When a gene undergoes a mutation for biological, chemical, physical causes, the information that reaches the cell will be inappropriate for the functions for which it is responsible. Most tumors are sporadic, that is, the alterations in DNA develop randomly at the level of somatic cells, that is, those cells that make up every organ and system in our body. These mutations originate in the DNA of a small group of cells and will determine the genetic error that will be perpetuated in the descendants of those cells, which accumulate in a certain organ and will initially replace the healthy tissue and then spread to other nearby or distant organs (metastasis). DNA methylation is a post-replicative process. Among the modifications concerning DNA, methylation is fundamentally decided during development. DNA methylation is therefore one of the mechanisms related to cellular differentiation, through the inhibition of gene expression at the transcriptional level. It is essential for the normal development of mammals, is associated with genomic imprinting, transcriptional inactivation of the X chromosome, aging and has a role in the development of pathological events, such as tumorigenesis. DNA methylation is a post-synthetic epigenetic modification that, with the transfer of a methyl group from S-adenosylmethionine to the C atom in position 5 of the Cytosine ring, introduces 5mC as a new base in DNA.
CpG islands contain CpG dinucleotides with a mathematically predetermined frequency. CpG islands are about 30,000 generally located at the 5' end of the promoter region of housekeeping genes, sometimes overlapping the coding region for a variable extension (usually the first exon). The frequency with which CpG dinucleotides are present in the genome is lower than expected, except for CpG islands. This is the result of an evolutionary mechanism linked to the presence of a spontaneous deaminase activity in the nucleus. This enzymatic reaction transforms methylated Cytosine into thymine and unmethylated Cytosine into Uracil. Subsequent controls and repair mechanisms recognize Uracil as a foreign DNA base and therefore replace it, while this substitution does not occur for Thymine, a common DNA base. Most CpGs are unmethylated in the normal cell regardless of the transcriptional state of the gene, while during tumor development, CpGs outside CpG islands become hypomethylated, and CpG islands in the promoter region of tumor suppressor genes become hypermethylated. This hypermethylation is associated with chromatin condensation and loss of transcription. The most recent data indicate that epigenetic and genetic events interact to aid progressive tumor development.

Code	Description	Tests n.
1.410	**Ampli GSTP1** With DNA modification	24
1.411	**Ampli Lung Cancer** With DNA modification	24
1.412	**Ampli Breast Carcer** With DNA modification	24
1.413	**Ampli Colon Cancer** With DNA modification	24
1.414	**Ampli BRCA 1-2** 185delAG, 188del-11bp e 5382insC mutations in BRCA1, and 6174delT mutation in BRCA2.	24

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ONCOLOGY AND EPIGENETICS IN REAL TIME PCR

ONCOLOGY AND EPIGENETICS with AGAROSE GEL DETECTION

Code	Description	Tests n.
1.414-7	**Ampli BRCA 1-2** 185delAG, 188del11bp, 5382insC, 1499insA, 5083del19 mutation in BRCA1 and 6174delT, 5445del4 in BRCA2.	25
1.415	**Ampli BRCA1** 1499insA, 3596del-4, 4172insT, 5083del-19, 5677insA mutation in BRCA1.	20
1.416	**Ampli BRCA2** 544del4, 6696delTC, 9189del4 mutation in BRCA2.	20
1.417	**Ampli p16** With DNA modification	24
1.419	**Ampli MGMT** With DNA modification	24
1.420	**Ampli hMLH1** With DNA modification	24
1.421	**Ampli BRCA1** With DNA modification	24
1.422	**Ampli DAP-Kinase** With DNA modification	24
1.429	**Ampli PARP-1 Val762Ala**	45

ONCOLOGY AND EPIGENETICS IN REAL TIME PCR

Code	Description	Tests n.
1.410RT	**Ampli GSTP1** With DNA modification	24
1.417RT	**Ampli p16** With DNA modification	24
1.418RT	**Ampli CDKN2A**	24
1.419RT	**Ampli MGMT** With DNA modification	24
1.420RT	**Ampli hMLH 1** With DNA modification	24
1.421RT	**Ampli BRCA1** With DNA modification	24
1.422RT	**Ampli DAP-kinase** With DNA modification	24
1.422pRT	**Ampli DAPK1**	24
1.900RT	**Ampli hMLH1/hMSH2** Mutations in exon 16 (CCG>CTG) of the hMLH1 gene and in exon 7 (ctt>ttt) of the hMSH2 gene	25

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HEMOSTASIS AND THROMBOSIS

Study of genetic variants involved in thrombotic and cardiovascular risk

Thrombosis is a multifactorial disorder, with both congenital and acquired risk factors. There are many genetic abnormalities that cause an increase in the risk of thrombophilia and the presence of several abnormalities gives rise to a further increase in thrombotic risk. Hemostasis is a set of biological processes, including blood coagulation, which aim to stop a hemorrhage following the rupture of a vessel. The different cellular and biochemical factors of the hemostatic process also have implications in several physiological processes including inflammation. In hemostasis there is a balance between procoagulant factors and anticoagulant proteins. The first genetic thrombotic disorders described were deficiencies of natural procoagulants, such as antithrombin, Protein C and Protein S, but these abnormalities are rare and are caused by many different mutations. More recently, single polymorphisms that are relatively common in the population have been described in procoagulant factors such as Factor V and Prothrombin, which result in an increased risk of venous thrombosis. By analyzing the haemostatic system, other polymorphisms have come to light, such as the genetic polymorphisms associated with thrombosis and arteriovascular disease, which have been found in many procoagulant proteins, including Factor V, Prothrombin, Fibrinogen, Factor VII, Factor XI and Factor XIII. In addition, polymorphisms of platelet glycoproteins, fibrinolytic proteins and enzymes of the transsulfuration pathway, leading to elevated homocysteine levels, have been described in association with thrombotic vascular diseases. In general, the risk factors that favor venous thrombosis are abnormalities of the anticoagulant pathway, while polymorphisms of procoagulant proteins and of the transsulfuration metabolic pathway are associated mainly with arterial diseases. Therefore, it is appropriate to analyze the association between the polymorphisms recently studied in the literature, for the evaluation of thromboembolic risk.

Code	Description	Tests n.
1.300	**Ampli MTHFR C677T**	45
1.301	**Ampli MTHFR A1298C**	45
1.302	**Ampli MTRR A66G**	45
1.310	**Ampli Factor II G20210A**	45
1.311	**Ampli Factor V Leiden**	45
1.312	**Ampli Factor VII R353Q**	45
1.313	**Ampli Factor VII 401 G/T 402 G/A**	45
1.315	**Ampli Factor V H1299R**	45
1.316	**Ampli Factor V Y1702C**	45
1.317	**Ampli Factor XIII Val34Leu**	45

The products in the catalogue are all CE marked IVD, compliant with directive 98/79.

HEMOSTASIS AND THROMBOSIS with AGAROSE GEL DETECTION

Code	Description	Tests n.
1.330	**Ampli CBS A114V**	45
1.331	**Ampli CBS I278T**	45
1.332	**Ampli CBS 844ins68**	45
1.340	**Ampli GpIIb/IIIa** P1A1/P1A2 (Leu33Pro) polymorphism	45
1.350	**Ampli ACE I/D**	45
1.351	**Ampli PAI-1 4G/5G**	45
1.352	**Ampli β Fibrinogen**	45
1.353	**Ampli AGT T174M**	45
1.354	**Ampli AGT M235T**	45
1.355	**Ampli Apo B** R3500Q mutations	45

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HEMOSTASIS AND THROMBOSIS IN REAL TIME PCR

Code	Description	Tests n.
1.300RT	**Ampli MTHFR C677T**	50
1.301RT	**Ampli MTHFR A1298C**	50
1.302RT	**Ampli MTRR A66G**	50
1.310RT	**Ampli Factor II G20210A**	50
1.311RT	**Ampli Factor V Leiden**	50
1.312RT	**Ampli Factor VII R353Q**	50
1.315RT	**Ampli Factor V H1299R**	50
1.316RT	**Ampli Factor V Y1702C**	50
1.330RT	**Ampli CBS A114V**	50
1.331RT	**Ampli CBS I278T**	50
1.332RT	**Ampli CBS 844ins68**	50
1.333RT	**Ampli Apo E** C112R e R158C polymorpfism	50
1.334RT	**Thrombosis Profile** Mutations: FII 20210 G>A, FV Leiden 1691 G>A (Arg506Gln), MTHFR C677T and MTHFR A1298G	25
1.340RT	**Ampli Gp IIb/IIIa** P1A1/P1A2 (Leu33Pro) polymorphism	25
1.352RT	**Ampli β Fibrinogeno** -455 G/A polymorphism	25
1.355RT	**Ampli Apo B** R3500Q mutation	25
1.356RT	**Ampli ABCB1** 3435 C>T polymorphism of the Gene ATP-binding cassette 1	25
1.357RT	**Ampli AGTR1** A1166C polymorphism	25
1.358RT	**Ampli ACE II** rs2074192	50
1.359RT	**Ampli ACE II** rs2106809	50

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INFERTILITY

Genes involved in the genetic diagnosis of infertility

Y chromosome microdeletions are the second most common cause of male infertility and are present in 1 in 4000 men in the general population but their frequency increases significantly among infertile men. In azoospermic men the incidence of microdeletions is higher than in oligospermic men (frequency 2-10%). Due to the results of an external quality control, the need arose to provide guidelines for the analysis of Y chromosome microdeletions. For this reason the European Academy of Andrology (EAA) and the Molecular Genetics Quality Network (EMQN) supported the publication of two "guidelines for the molecular analysis of Y chromosome microdeletions" (Simoni et al, 1999-2004) and offer an external quality assessment (EQA) in accordance with the EAA/EMQN guidelines. The Ampli Y Chromosome UE and Ampli Y Chromosome UE FAM kits allow the analysis of Y chromosome microdeletions in the three regions AZFa, AZFb and AZFc using 2 multiplex PCR. The STS (Sequenced Tagged Sites) are tested following the EAA guidelines, as are the internal controls ZFX/ZFY and SRY. The detection is performed by agarose gel electrophoresis or fragment analysis (cat.1.501FAM). The Ampli Set Y Chromosome Extension kit allows to confirm the microdeletions identified in the first step of the kit described above and to analyze the entire deleted region, evaluating whether the deletion is partial or total. The choice of markers refers to the EAA/EMQN guidelines.

Ampli Set Y Chromosome gr/gr: the AZFc region is particularly subject to homologous intrachromosomal recombination events, due to its repetitive structure, which can lead to deletions. New types of deletions, called partial deletions or gr/gr, have been described. They remove about half of the AZFc content, including two DAZ genes (CDY1 and BPY2). In Italy, gr/gr carriers have a 7.9-fold increased risk of reduced spermatogenesis compared to men without these deletions. The Ampli Set Y Chromosome gr/gr kit allows the analysis of the presence of partial gr/gr deletions using specific primers for SY1291 and SY1191 and for ß-globin as an internal control of multiplex PCR. Ampli Set FSH ß-FSHR: FSH or follicle stimulating hormone is a glycoprotein hormone produced and secreted by the adenohypophysis and contributes, in both sexes, to the regulation of development and pubertal maturation of the reproductive process. The -211 G>T polymorphism (rs10.835.638) influences FSH levels in serum. This polymorphism, which causes the substitution of a G with a T, is located in the promoter of the gene -211 bp upstream the start site of mRNA transcription. A statistically significant association has been highlighted between serum FSH levels and the FSH ß genotype: heterozygotes (GT) or homozygotes (TT) have significantly lower serum FSH ß values than WT subjects (GG). Several studies, including that of Tüttelmann in 2012, have found a significant correlation between T carriers and serum FSH levels (24% lower in TT than in GG), the correlation between FSH/LH, testicular volume and sperm concentration and count (36% and 34% lower in TT than in GG). FSH is able to exert its stimulatory effects on gametogenesis only if it binds to a specific receptor (FSHR). This receptor is located on the surface of Sertoli cells in the testes and on the surface of the ovarian granulosa. The variant 2039 A>G (rs6166) characterizes the exon 10 haplotype. In the protein, the substitution 2039A>G causes the replacement of an Asparagine with a Serine. In women, the FSHR genotype related to these SNPs is the factor that most influences the ovarian response to FSH treatment required for ovulation induction in assisted fertilization techniques. The 2039 A>G variant reflects a reduced ovarian sensitivity in women, thus requiring a higher dose of exogenous FSH in fertilization techniques. A significant decrease in testicular volume has also been demonstrated in relation to the various genotypes of the SNP 2039A>G and -211GT -211TT of FSHβ. The studies of Selice (2011) and Ferlin (2011) have demonstrated that the analysis of the FSHβ and FSHR genes are a valid pharmacogenetic approach in men since FSH treatment is able to induce an improvement in seminal parameters only in the subgroup of oligospermic men with a specific genotype related to these two genes.

INFERTILITY with AGAROSE GEL DETECTION

Code	Description	Tests n.
1.501	**Ampli Y Chromosome UE** AZF region Kit compliant with EAA/EMQN guidelines	25
1.501FAM	**Ampli Y Chromosome UE FAM** AZF region with capillary electrophoresis. - Kit compliant with EAA/EMQN guidelines	25
1.501gr	**Ampli Y Chromosome gr-gr** Microdeletions of the AZF region of the Y chromosome and partial gr/gr deletions	25
1.502	**Ampli Y Chromosome Extension** Extension oh the Microdeletions of the AZF region - Kit compliant with EAA/EMQN guidelines	25
1.503	**Ampli Androgen Receptor** CAG polymorphism in the exon 1	45
1.504	**Ampli FSHβ - FSHR** (RFLP) -211 G>T polymorphism on the FSHβ gene and 2039 A>G on the FSHR gene	24
1.505	**Ampli HLA G** Deletion/insertion of 14bp in 3' UTR of exon 8 of the HLA-G gene by bi-PASA Molecular Biology technique.	25
1.506	**Ampli HLA G -725** Major Histocompatibility Complex, Class I, G (HLA-G) Variant 5'URR -725 (C/ G o T)	25

INFERTILITY IN REAL TIME PCR

Code	Description	Tests n.
1.505RT	**Ampli HLA G** Deletion/insertion of 14bp in 3' UTR of exon 8 of the HLA-G gene by bi-PASA Molecular Biology technique.	50
1.507RT	**Ampli FSHR T307A e N680S** rs6165 e rs6166 polymorphism of the gene FSHR	25
1.508RT	**Ampli ESR1 e ESR2** IVS1-397 T/C polymorphism of the gene ESR1 and 1730 A/G (*39 A>G) oh the gene ESR2	25
1.509RT	**Ampli FSHβ -211 G>T** -211 G>T polymorphism oh the gene FSHβ	50

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PREDISPOSITION TO INFLAMMATION

Genetic polymorphisms involved in alterations of the inflammatory panel

The tests are based on the analysis of genetic polymorphisms that have been found to be associated with an increased risk of disease where the inflammatory component is a controlling factor. The tests are based on the analysis of five genetic polymorphisms, located on four genes. IL-6 is a pleiotropic cytokine, capable of performing many functions, especially pro-inflammatory. It contains various polymorphisms including one present in position -174 in the promoter which consists in the substitution of a guanine with a cytosine. IL-10 is an anti-inflammatory molecule that inhibits the release of pro-inflammatory cytokines during the development of inflammatory responses. It is secreted by T lymphocytes, monocytes and macrophages. This molecule regulates inflammatory responses and has immunosuppressive activity. IL-10, with immunosuppressive action, plays an important role in the pathogenesis of cardiovascular diseases. Many studies have investigated the polymorphism in the promoter region of the IL-10 gene at position -1082 G>A. The presence of the A allele is associated with a lower production of the IL-10 molecule. It has been found that the presence of the AA genotype increases the risk of developing myocardial infarction and cardiovascular disease compared to the genotype (GG. Murakozy 2001 J Mol Med79, 665) Recently, the AA haplotype has been associated with chronic periodontitis. (Loo et al 2012 J Translational Medicine (10):58). The interleukin-1 (IL-1) gene is a pluripotent cytokine, that is, it is able to perform and regulate many immune functions and is mainly involved in the activation of inflammatory responses. IL-1α in particular is also released into the bloodstream, exerting widespread actions in the body. In fact, it is one of the factors capable of inducing fever, sleep, anorexia and hypotension. IL-1 β is important in the pathogenesis of myocardial infarction because it stimulates macrophages and endothelial cells to release tissue factor (TF), a potent thrombus inducer. Furthermore, the presence of mutated alleles also correlates with periodontal diseases. The polymorphism on the IL-1b promoter in position -511 consists in the substitution of an A (adenine) with a G (guanine). The presence of an allele in combination with other polymorphisms in other genes increases the risk of developing thromboembolic diseases. On the contrary, the polymorphism in subjects with protective IL-1 beta causes blood clotting to be induced to a much lesser extent, thus reducing the probability of being exposed to the risk of infarction or stroke. (Mattila 2002 J Med Genet 39, 400; Rizzi C, carcinogenesi. 2011 Dec, 32 (12): 1849-1854).

Code	Description	Tests n.
1.800RT	**Ampli IL 6** G-174C (rs1800795) polymorphism	25
1.801RT	**Ampli IL 10 1082 AG** Mutation 1082A-G (rs1800896)	25
1.802RT	**Ampli IL 1α** A114S 4845 C/A (rs17561) polymorphism	25
1.803RT	**Ampli IL 1β** -511 CT (rs16944) polymorphism	25
1.804RT	**Ampli IL 22** +1046 T/A (rs1182844) and +1995 A/C (rs1179246) polymorphisms	25
1.805RT	**Ampli IL - 8** -251A/T (rs4073) polymorphism	25
1.806RT	**Ampli IL - 6 Receptor GP130/GP80**	25

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PREDISPOSITION TO INFLAMMATION IN REAL TIME PCR

Code	Description	Tests n.
1.807RT	**Ampli C Reactive Protein** Genotype	25
1.808RT	**Ampli Expression IL - 6α** Genotype	25
1.809RT	**Ampli TNFα** T857C (rs1777724) polymorphism	25
1.810RT	**Ampli IL - 32** C/T (rs12934561) and A/T (rs28372698)	25
1.811RT	**Ampli IL - 17** Polymorphism (rs2275913) of the Gene IL 17* G197A.	25
M1.800RT	**Multi kit IL** C4845A IL-1α, C-511T IL-1b, G-174C IL-6, T-592G e A-1082G IL-10 Poly-morphisms.	20

HEMOCHROMATOSIS

Study of the genes involved in excessive accumulation of iron (Fe) resulting in tissue damage

Hemochromatosis (HC) is a disease caused by the accumulation of an excessive amount of iron in the body, which causes damage to some important organs, such as the liver, pancreas and heart. HC was once considered a rare disease, but today we know that it is a common disease in white people, affecting approximately 1 in 300-350 people in Northern Europe; The gene responsible for HC, called HFE, is located on the short arm of chromosome 6. The HFE gene contains the information for the production of a protein important in regulating iron absorption, although its exact function is still being studied. In people with HC, this gene contains mutations, which alter its function. Two main mutations have been identified in this gene, and are called: C282Y and H63D.

Code	Description	Tests n.
1.320	**Ampli EMO C282Y and H63D**	45
1.321	**Ampli EMO A77D**	45
1.322	**Ampli EMO N144H**	45
1.323	**Ampli EMO C282Y, H63D e S65C Cap. Elect.** with capillary electrophoresis	45

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RECURRENT ABORTION

Genetic analysis of couples with a history of recurrent miscarriages

The miscarriage events are unfortunately not rare. While hormonal, immune, uterine and chromosomal alterations are now included as possible causes of repeated miscarriages, recent studies are moving in a new direction: the genetics of blood clotting factors. Women suffering from hereditary thrombophilia, excessive clotting caused by a genetic anomaly, are in fact the category most at risk of miscarriage. In most cases, the death of the fetus is caused by genetic alterations of one or more blood clotting factors that determine the onset of placental thrombosis, characterized by an obstruction of the placental blood vessels. A thrombotic event occurs, venous or arterial, thus preventing the flow of blood. The clot is called a thrombus. The genes known today for susceptibility to thrombosis are gene variants that are so frequent in the population that they are considered polymorphic variants. The genes under consideration are those related to factor V Leiden, factor II of coagulation, β-Fibrinogen, PAI, AGT and MTHFR. In pregnancy, a genetic condition of heterozygosity or homozygosity for one or more of these genes is considered predisposing to spontaneous abortion. The study of the gene variants of these three genes is indicated in: Subjects with previous episodes of venous thromboembolism or arterial thrombosis, women who intend to take oral contraceptives, women with previous episodes of thrombosis during pregnancy, women with recurrent miscarriages, women with a previous child with DTN (neural tube defect), pregnant women with IUGR, thrombophlebitis or placental thrombosis and diabetic subjects.

Code	Description	Tests n.
1.300	**Ampli MTHFR C677T**	45
1.300RT	**Ampli MTHFR C677T** in Real Time PCR	50
1.301	**Ampli MTHFR A1298C**	45
1.301RT	**Ampli MTHFR A1298C** in Real Time PCR	50
1.310	**Ampli FACTOR II G20210A**	45
1.310RT	**Ampli FACTOR II G20210A** in Real Time PCR	50
1.311	**Ampli FACTOR V LEIDEN**	45
1.311RT	**Ampli FACTOR V LEIDEN** in Real Time PCR	50
1.351	**Ampli PAI-1 4G/5G**	45
1.352	**Ampli β-FIBRINOGEN**	45
1.352RT	**Ampli β-FIBRINOGEN** in Real Time PCR	50

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PREDISPOSITION TO FOOD INTOLERANCES

Study of genetic variants that influence the body's food intake

Celiac disease is a chronic inflammation of the small intestine, triggered by the ingestion of gluten in genetically predisposed subjects. It is one of the most common autoimmune diseases in Western countries and is a clinical condition that persists throughout life. In 90% of cases it is associated with the presence of HLA DQ2 antigens and in the remaining cases with HLA DQ8. Celiac disease is also associated with various extra-intestinal manifestations, such as headaches or anemia, and can have significant effects on the reproductive system.

Lactose intolerance is the most common of the enzymatic intolerances and is determined by the absence of the enzyme necessary for the metabolization of the food. This intolerance, in fact, occurs in case of deficiency of the lactase enzyme capable of breaking down lactose (the main sugar in milk) into glucose and galactose and the symptoms most frequently appear between the ages of seven and thirty. If it is not digested correctly, the lactose that remains in the intestine is fermented by the intestinal bacterial flora with consequent clinical manifestations such as colic, cramps, meteorism and diarrhea. It is a very common pathology in adults and very widespread in Italy, especially in the southern regions.

Code	Description	Tests n.
1.701RT	**Ampli DQ2/DQ8** HLA II DQ2/DQ8 Variant in Real-Time PCR.	25
2.036	**Ampli LCT rs 4988235** Identificazione del Polimorfismo C/T polymorphism in Position -13910 with agarose gel detection	25

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VIROLOGY

Viral respiratory infections are highly contagious and can be caused by several pathogens such as: influenza virus, parainfluenza virus, respiratory syncytial virus, coronaviruses, rhinovirus, coxsackivirus, adenovirus, bocavirus, metapneumovirus.
A virus is a small infectious organism, much smaller than a fungus or bacterium, that must invade a living cell to reproduce. The virus adheres to a host cell, enters it and releases its DNA or RNA inside the cell itself. The DNA or RNA of the virus is the genetic material that contains the information needed by the virus to replicate. The genetic material of the virus takes control of the cell and forces it to replicate the virus. Typically, the infected cell dies because it prevents it from carrying out its normal functions. Before dying, the cell releases new viruses, which continue to infect other cells.

Among the emerging viruses that can cause respiratory infections we find:
SARS-CoV first identified in 2002;
Avian influenza caused by viruses H5N1, H7N9;
MERS-CoV identified in 2012;
the new Coronavirus called SARS-CoV-2.

The viral particles of SARS-CoV-2 have a spheroid shape with a diameter of about 100-160 nanometers. They have a lipid envelope in which the surface glycoproteins of the virus are anchored, giving the viral particle a characteristic crown shape, hence the name coronavirus. The genome is made up of single-stranded RNA with positive polarity of about 30 kb. The virus contains 4 structural proteins and 16 non-structural proteins. The virus attack on the cell is mediated by the interaction of the Spike protein with the cellular receptor constituted by the enzyme angiotensin convertase (ACE 2) which is followed by its internalization and fusion with the endosome membrane through protease activities and subsequent release of genomic RNA into the cytoplasm. This is immediately translated into the polyproteins pp1a and pp1ab then processed to give the replicase and other non-structural proteins responsible for genome replication and expression of structural and accessory proteins preceded by a discontinuous transcription of negative polarity subgenomic RNAs on which the relative messenger RNAs (mRNAs) are synthesized. Subsequently, the viral proteins assemble with the genomic RNA which is followed by the release of new viral particles. Coronaviruses belong to the Coronaviridae family. In December 2019, the new coronavirus was identified and on 7/1/2020, the nucleotide sequence made available in a few days, showed a genetic identity of about 80% with SARS-CoV-1 previously identified in 2002. This virus, defined precisely as SARS-CoV-2, is highly contagious for humans, so much so that the World Health Organization declared the state of Pandemic.
The analysis of patients for the SARS-CoV-2 virus will be carried out on nucleic acid (RNA), appropriately isolated from a swab of the upper/lower respiratory tract, using a Real-Time RT PCR Simplex that identifies the agent of the new coronavirus disease (COVID-19) using as a target two regions of the N gene (N1 and N2) that codes for the viral nucleocapsid and the human RNase P gene (RP) is identified as a control.
Real-Time RT PCR is therefore based on three main processes: isolation of the virus RNA from the samples, reverse transcription of the RNA, real-time amplification of the cDNA.
The SARS-Cov-2 kit provides for the identification of the SARS-CoV-2 virus using the Real-Time RT-PCR technique, in particular with the FAM fluorophore N_1 is identified, with the VIC fluorophore N2 is identified and with the FAM fluorophore the Mix RP. The Ampli SARS-CoV-2 Kit has a specificity and sensitivity of 99%

VIROLOGY IN REAL TIME PCR

Code	Description	Tests n.
1.108RT-DX	**QUANT– HCV DX**	48
1.110RT-DX	**QUANT- HBV DX**	48
1.113RT	**QUANT-CMV DNA**	50
1.115RT	**QUANT-EBV DNA**	50
1.116RT	**Ampli HDV RNA**	50
1.208RT-DX	**QUANT-HIV DX**	48
1.240RT	**Ampli Virus HERPES SIMPLEX I-II**	50

SARS-COV-2 IDENTIFICATION KIT

Code	Description	Tests n.
102T	**RNA Extraction from biological fluids** RNA Extraction Kit Valid for Nasopharyngeal Swabs, Biological Fluids, Sputum, Broncho Aspirate and Pleural Effusion, to Identify the COVID-19 Virus and the Influenza Virus. Execution Time: 50 minutes for 10 samples.	200
1.220mRT	**Ampli SARS-CoV-2 in Multiplex Real-Time RT-PCR**	100
1.220RT	**Ampli SARS-CoV-2**	100
1.220.1RT	**Ampli SARS-CoV-2 in Simplex Real-Time RT-PCR**	100
1.221RT	**Ampli SARS-CoV-2** Sars COV2, Gene N, E,ORF 1ab	100

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SEXUALLY TRANSMITTED DISEASES

Molecular tests for the identification, quantitative analysis and genotyping of infectious agents

Sexually Transmitted Diseases (STDs), with over 300 million cases/year, are now a global health problem. Freer customs and lifestyles, ease of travel between continents, migrations of entire populations in search of better socio-economic conditions, have contributed decisively to the expansion of this phenomenon. The emergence of new infectious agents such as Chlamydia trachomatis, HSV-2, HPV, HIV and others have broadened the spectrum of venereal diseases that only a few years ago seemed, if not on the way to extinction, certainly in a phase of containment. Today we have gone from the few classic agents of the 80s to more than 30 etiological causes of STDs. All this has led to the onset of new morbid conditions as well as complications and sequelae that were unusual until a few years ago. Among these: sterility, ectopic pregnancies, abortions, perinatal mortality and risk of involvement of other organs.

Italian data on the real situation of the phenomenon are not easily traceable or are distributed unevenly across the national territory. However, from the data set and in light of the authoritative interventions of the major Italian scholars on the subject, it can be stated that both the classic STDs, in particular syphilis and gonococcus, and those that can be defined as "second generation" have been in constant and worrying increase since the beginning of the third millennium, so much so that, in particular for syphilis, in the diagnosis and treatment centers of the main Italian cities there are increases of up to 30% compared to previous years. All these factors confirm a certain recrudescence of the phenomenon of STDs which today represent an important aspect of modern Medicine and in particular require different diagnostic, therapeutic and prevention interventions compared to the past. It is obvious therefore that, in the face of such an important phenomenon, the need has arisen to develop new diagnostic methods, among which an important place has been taken by the techniques of Molecular Biology, which offer in daily practice the possibility of a rapid and reliable diagnosis without thereby setting aside traditional methods, often still valid. As regards the treatment of many sexually transmitted diseases, this can be problematic. In fact, these diseases pose important therapeutic problems linked to resistance phenomena, such as for HIV, where resistance to antiretroviral drugs and their combinations rapidly develops. Other factors that limit the possibility of treatment especially in developing countries are the cost of therapy (a serious problem especially for the fight against AIDS), the subclinical nature of infections and the reluctance to seek and/or follow prevention and treatment programs.

Code	Description	Tests n.
1.111	**Ampli HPV Screening** HPV genome of L1 region 6, 11, 13, 16, 18, 26, 31, 33, 35, 39, 42, 43, 44, 45, 51, 52, 53, 54, 56, 57, 58, 59)	55
1.111G	**Ampli HPV Genotype High/Low Risk** Genotypes 6, 11, 16, 18 e 33 in E6 region	45
1.600	**Ampli Chlamidia Trachomatis**	50
1.601	**Ampli Ureaplasma Urealyticum**	50
1.604	**Ampli Neisseria Gonorrhoeae**	50
1.605	**Ampli Trichomonas Vaginalis**	50
1.611	**Ampli Mycoplasma Hominis**	55
1.612	**Ampli Mycoplasma Genitalium**	50
1.622	**Ampli Gardnerella Vaginalis**	50

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SEXUALLY TRANSMITTED DISEASES with AGAROSE GEL DETECTION

Code	Description	Tests n.
1.630	**Ampli Treponema Pallidum**	50
1.640	**Ampli Candida Albicans**	50
1.643	**Ampli Pseudomonas Vaginalis**	50

SEXUALLY TRANSMITTED DISEASES IN REAL TIME PCR

Code	Description	Tests n.
1.111HRG/RT	**Ampli HPV Typing High Risk** Genotypes 16, 18, 31, 33, 35, 39, 45, 52, 56, 58, 59, 66	50
1.600RT	**Ampli Chlamidia Trachomatis**	50
1.602RT	**Ampli Ureaplasma Urealyticum**	50
1.604RT	**Ampli Neisseria Gonorrhoeae**	50
1.605RT	**Ampli Trichomonas Vaginalis**	50
1.607RT	**Ampli Toxoplasma Gondii**	50
1.608RT	**Ampli Helicobacter Pylori**	50
1.611RT	**Ampli Mycoplasma Hominis**	50
1.612RT	**Ampli Mycoplasma Genitalium**	50
1.620RT	**Ampli MTB Complex DNA**	50
1.622RT	**Ampli Gardnerella Vaginalis**	50
1.630RT	**Ampli Treponema Pallidum**	50
1.643RT	**Ampli Pseudomonas Vaginalis**	50
1.645RT	**Quant Steptococcus B**	50
1.711RT	**Ampli Helicobacter Pylori VacA/CagA**	50

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NEUROLOGY

NEUROLOGY with AGAROSE GEL DETECTION

Code	Description	Tests n.
2.100	**Ampli DRD2-A1/A2**	**25**
2.113	**Ampli SERT (SLC6A4)** Gene 5-HTT	**25**

OTHER GENETIC TESTS

OTHER GENETICS TESTS

Code	Description	Tests n.
1.299	**Ampli AMD** Y402HI polymorphism of the gene CFH and A69S of the gene ARMS2 with agarose gel detection	25
1.402-403-405	**Reverse Transcriptase**	50
1.409	**Ampli Beta 2 Microglobulin** Agarose gel detection the Quality and Efficiency of RNA and Reverse Transcription	45
1.430	**Ampli Beta Globin** Agarose gel detection DNA Quality and Efficiency	45
1.440RT	**Quant DNA Real Time** Quantification of DNA in plasma, blood, tissue, paraffin-embedded tissue, and biological fluids with Real Time PCR	100
1.700	**Ampli Connexin 26** Identification of the 35delG mutation of Connexin 26 gene with agarose gel detection	24

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EXTRACTION OF NUCLEIC ACIDS

Nucleic acids are polymeric macromolecules made up of repeating units called nucleotides. The latter are formed by a pentose sugar, a nitrogenous base (purine or pyrimidine) and phosphate groups. Two types of nucleic acids are found in living organisms: DNA and RNA. Watson and Crick's hypothesis on the three-dimensional structure of DNA was that DNA was composed of 2 purines: adenine (A) and guanine (G) and two pyrimidines: cytosine (C) and thymine (T), linked by phosphorylated sugars. Since DNA plays a central role as genetic material, clarification of its three-dimensional structure was considered essential to understanding its functions. Watson and Crick's deductions were profoundly influenced by Linus Pauling's description of hydrogen bonds and the alpha-helix. The fundamental characteristics of the model are that DNA is a double helix with the sugar and phosphate backbone arranged on the outside of the molecule while the bases face inwards, oriented in such a way that hydrogen bonds form between the purines and pyrimidines of the opposite chains. The base pairing is highly specific: A always binds to T and G to C. For this specific base pairing, the two chains of a DNA molecule are complementary: each chain contains all the information necessary to determine the sequence of the bases in the other chain. The three-dimensional structure of RNA is represented by a simple chain made up of two purines: A – G and two pyrimidines: C and Uracil (U). RNA decodes the information present in DNA, used to synthesize specific proteins. The difference between DNA and RNA is that while DNA essentially performs the function of containing information, the RNA present in the cell performs different functions, in fact it is present in different forms: mRNA, tRNA, rRNA. The idea of searching for circulating DNA in plasma or serum came to a Swiss physiologist a few years ago. Having found it in plant sap, he wanted to find out if it was also present in the plasma of patients with advanced cancer. He demonstrated its presence with radioimmunoassays. Later, other American laboratories published studies that indicated the tumor origin of the DNA circulating in plasma, demonstrating the presence of genetic and epigenetic alterations typical of tumor DNA in plasma DNA in patients with various types of neoplasia such as head and neck, lung, colon, liver, pancreas, breast and prostate cancers. The first to introduce the term mutation in the field of genetics was Hugo de Vries in 1901, observing how in the progeny of a strain of the plant Oenothera Lamarckiana some unexpectedly giant individuals could be obtained. The concept of mutation as it is understood today, however, was only used starting in 1927. In general, it can be said that genetic mutations have played an essential role even before, since the dawn of genetics; already in the famous works of the father of genetics, Gregor Mendel, in fact, phenotypes such as the white color of petals or the yellow of ripe seeds, used to formulate his laws, were only due to inactivating mutations of the corresponding genes. Genetic mutation means any stable and heritable modification in the nucleotide sequence of a genome or more generally of genetic material (DNA and RNA) due to external agents or interactions with the environment, but not to genetic recombination. A mutation therefore modifies the genotype of an individual and can possibly modify its phenotype.

EXTRACTION OF NUCLEIC ACIDS

Code	Description	Tests n.
101	**DNA Extra Kit** DNA Extraction from Whole Blood, Biological Fluids, Lymphocytes and Cell Concentrates Salting out method. Biological sample volume: from 0.5 ml to 10 ml	50
101C	**DNA Extra Kit cf DNA Nucleo Spin L Plasma/Serum** Extraction of Free Circulating DNA for Pharmacogenetic Analysis of Liquid Biopsy. Biological sample volume: 1.0/2.0 ml	20
101DGC	**DNA Extra-NucleoSpin DNA on Silica Membrane Columns** DNA extractions from 200 µl of Whole Blood, Biological Fluids and Cells. Execution time: 40 minutes	50/100
101M	**DNA Extra Kit - Magnetic Separation** from Whole Blood with Magnetic Separation. Biological sample volume: 100 µl	45
101SM	**DNA Extra Kit - Magnetic Separation** From Serum or plasma with Magnetic Separation. Sample volume: 200 µl. Execution time: 40 minutes	50
102	**RNA Extra Kit from Whole Blood and Cells/Pellets** from Whole Blood and Cells/Pellets, isopropanol Chloroform Technique. Sample volume: 100/200 µl. Run time: 50 minutes	50/100
102RV	**Extra Viral DNA/RNA of Viral Nucleic Acids kit Using Columns** Extraction of Viral Nucleic Acids Using Silica Membrane Columns. Sample volume: 100/200 µl	50
102S	**RNA Extra from Whole Blood** RNA Extraction from 1 ml of Whole Blood. Techniques for Oncohematology in Accordance with the Biomed II Guidelines	50
102T	**RNA Extra from Biological Fluids** RNA Extraction Valid for Naso-Pharyngeal Swabs, Biological Fluids, Sputum, Broncho Aspirate and Pleural Effusion, to identify the COVID-19 Virus and the Influenza Virus. Execution time: 50 minutes for 10 samples	200
103	**DNA Extra Kit from tissue** Genomic DNA Extraction from Paraffinized Tissue. Sample in 50 mg Tissue Slice. Execution Time: Overnight	50/100
105	**DNA Extra Kit from swab** Genomic DNA extraction from Cervical, Urethral, Ocular Swabs, Placed in 0.2 ml of Transport Medium (provided in the kit). Capture of Nucleic Acids with Chelating Resin. Execution time: 50 minutes	50
105R	**RNA Extra Kit from biological materials** RNA extraction from biological fluids, sputum, bronchial aspirate, pleural effusion and urine. Capture of nucleic acids with chelating resin. Execution time: 50 minutes	50

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TERMS AND CONDITIONS

VALIDITY
This catalog is valid from May 1, 2025.

ORDERS
Orders must include:
-the buyer's full name and company name including VAT number and unique code.
-the order date and number.
-the indication of the place and address of delivery of the goods and, if applicable, the delivery times.
Orders must be sent by email: info@diachem-srl.it directly to Dia-chem S.r.l..

USE
The products sold by Dia-chem S.r.l. are laboratory reagents and are used exclusively for research and diagnostics. The products must be stored as recommended in the individual manuals supplied with each product and used by qualified personnel.
Dia-chem S.r.l. will not be liable for any accidental, indirect or contingent damages that result from the incorrect use of its products.

SHIPPING
The general delivery term for all our products is 1-3 weeks from receipt of the order/date of the official order confirmation by Dia-chem S.r.l.. The shipment of the products will take place in the best way decided by Dia-chem S.r.l., unless otherwise specified.
The goods travel on behalf and at the risk and peril of the customer.

NOTES
The delivery term is intended as the period of time that passes from receipt of the order or advance payment, if requested, until the shipment of the products by Dia-chem S.r.l..

COMPLAINTS and RESPONSIBILITIES
All complaints must be submitted in writing to Dia-chem S.r.l. within 15 days of receipt of the goods, enclosing the delivery note. In the event that the complaint is recognized as valid, the supplier is obliged to only replace the goods with other faultless ones, excluding any other charges.
Even partial returns not previously communicated will not be accepted.

COMPETENT COURT
For any dispute, the Court of Naples is competent.

Our sales network will take care of following you: contact us for any further information at the telephone numbers and e-mail addresses indicated below.

DIA-CHEM S.r.l.
Registered Office: Via G. Pergolesi n.1B,
80122 Naples - ITALY
Operational headquarters: Via Grotta dell'olmo, 67
80014 Giugliano in Campania (NA) - ITALY
Tel. (0039) 0815870231
e-mail: info@diachem-srl.it
Web site: www.diachem-srl.it

DIA-CHEM S.r.l.

Registered Office: Via G. Pergolesi n.1B, 80122 Naples ITALY
Operational headquarters: Via Grotta dell'olmo, 67
80014 Giugliano in Campania (NA) ITALY
Tel. (+39) 0815870231
e-mail: info@diachem-srl.it
ordini@diachem-srl.it
assistenza@diachem-srl.it
Sito web: www.diachem-srl.it