

PLATELET-ANTIBODY SCREENING CELLS

- Pool of different donor platelets
- Typed for HPA-1, -2, -3, -4, -5, -6, -15
- All donors are of blood group O
- Reagent for the detection of anti-platelet antibodies or anti-HLA class I antibodies

PLATELET-ANTIBODY SCREENING CELLS

REF. 900001

Set of 6 vials, 1 ml per vial

PLATELET-ANTIBODY IDENTIFICATION PANEL CELLS KIT

- Panel of 6 individual platelet cells
- Typed for HPA-1, -2, -3, -4, -5, -6, -15
- All donors are of blood group O
- Reagent for the identification of anti-platelet antibodies

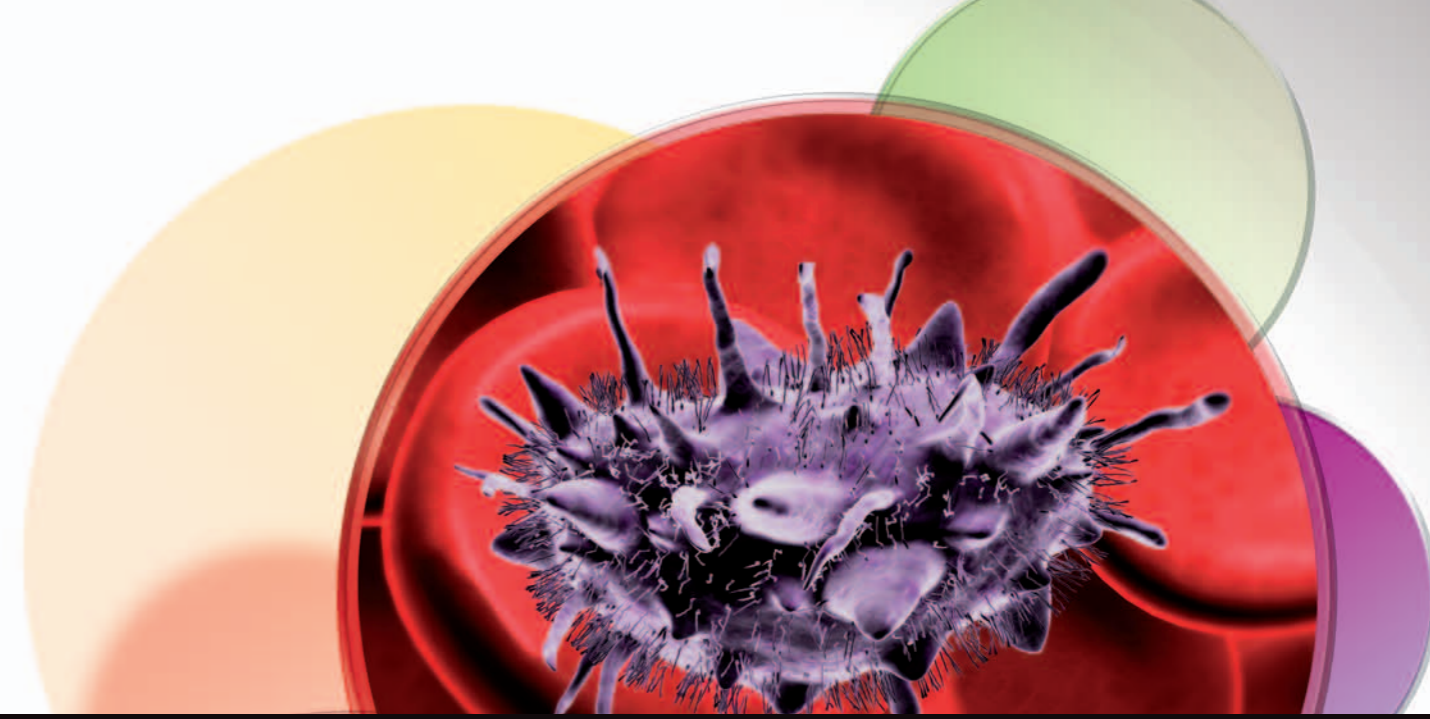
PLATELET-ANTIBODY IDENTIFICATION PANEL CELLS KIT

REF. 900002

Set of 6 cells, 1 ml per vial

RELATED PRODUCTS:

Control Plasma Kit, set of 4 controls	Ref. 900003
MAIPA Reagents Kit	Ref. 900004
MAIPA ELISA Detection Kit	Ref. 900005
Complete MAIPA Kit	Ref. 900006



**STANDARDIZED PLATELET-ANTIBODY SCREENING
AND IDENTIFICATION USING MAIPA TECHNOLOGY**

**STANDARDIZED PLATELET-ANTIBODY SCREENING
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PLATELET

Antibody Screening Cells

PLATELET

Antibody Identification Panel Cells Kit

PLATELET

Antibody Screening Cells

PLATELET

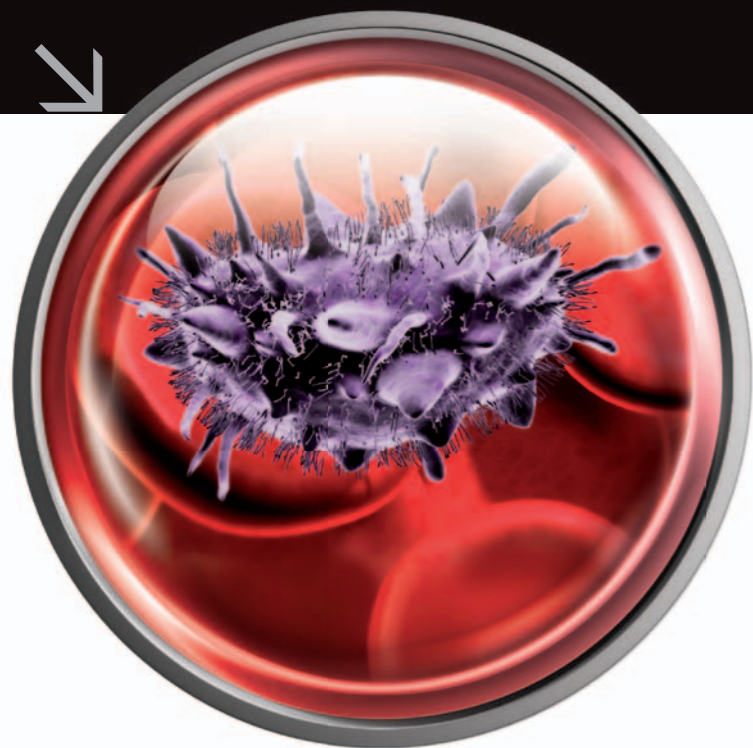
Antibody Identification Panel Cells Kit



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ISO 13485: 2003 CERTIFIED COMPANY



PLATELET

Antibody Screening Cells Antibody Identification Panel Cells Kit

ANTIBODIES TO PLATELETS GIVE RISE TO THREE CLINICAL SYMPTOMS:

- NEONATAL/FETAL ALLO-IMMUNE THROMBOCYTOPENIA (NAIT)**
- POST-TRANSFUSION PURPURA (PTP)**
- PLATELET REFRACTORINESS (PR)**

NAIT: Feto-maternal incompatibility of human platelet allo-antigens may induce antibodies to Human Platelet Antigens (anti-HPA) which may lead to a neonatal/fetal allo-immune thrombocytopenia (NAIT/FNAIT). The mother produces antibodies against fetus' antigens inherited from the father. These allo-antibodies (IgG) can cross the placenta, destroy fetal thrombocytes and may induce severe thrombocytopenia. It is most commonly caused by the HPA-1a antigen (80%). NAIT has an estimated incidence of 1/1000 pregnancies and may lead to intra-cerebral bleeding and/or ventriculomegaly. Typing the maternal platelets for the HPA-1a antigen should be performed systematically. Screening and identification of maternal antibodies has to be done for prevention and treatment of such manifestations.

PTP: Post-Transfusion Purpura is an adverse reaction to a blood transfusion due to donor platelet antigens being different from patient platelet antigens. Allo-antibodies destroy the transfused platelets and auto-antibodies destroy the patient's own platelets, leading to a severe form of thrombocytopenia that lasts for several weeks and sometimes even several months. It is most commonly caused by the HPA-1a antigen: PTP is most common in HPA-1a negative women who have had multiple pregnancies, while in men PTP may occur after having undergone previous transfusions. This adverse reaction to blood transfusion typically occurs 10 days following a transfusion. The thrombocytopenia can be treated with therapeutic intravenous immunoglobulin (IVIgG). Other platelet allo-antigens are occasionally implicated in post transfusion purpura.

PR: Long-term application of platelet concentrates may induce anti-HLA and anti-HPA antibodies. These patient antibodies destroy transfused platelets and prevent successful therapy. The use of matched platelets saves valuable resources and costs whilst minimizing concomitant risks of platelet concentrate transfusion such as bacterial or cytokine load. Characterization of the allo-antibodies is an important step in improving the efficacy of platelet transfusion. Of the platelet antigens involved in platelet refractoriness upon platelet transfusion the most prominent allo-immunization is caused by the HPA-5b platelet antigen followed by the HPA-1a allo-antigen. Typing donors and recipients for HPA-1a and HPA-5b antigen is of utmost importance, screening and identification of antibodies has to be done to achieve an effective platelet transfusion treatment.

DETECTION AND IDENTIFICATION OF ANTI-PLATELET ANTIBODIES WITH APDIA STANDARDIZED PLATELETS can be done with several technologies such as immunoblotting, immunoprecipitation, platelet immunofluorescence tests (PIFT) or the mono-

clonal antibody-specific immobilization of platelet antigens (MAIPA) assay. MAIPA is considered to be the gold standard method for platelet antibody detection. It requires the use of human thrombocytes typed for the important platelet antigens frequently observed in HPA immunizations: primarily the HPA-1, -3, -5 and secondly the HPA-2, -4, -6 and -15 antigens. The HPA-1 and HPA-3 platelet antigens are located on glycoprotein GpIIb/IIIa (CD41/CD61) fibrin receptor, while GpIa (CD49b/CD31) collagen receptor bears the HPA-5 system and GpIbIX (CD42a/CD42b) platelet receptor for von Willebrand factor also carries relevant antigens. Besides the platelet specific glycoproteins, the HLA class I found on platelets and nucleated cells is also a major allo-antigen giving rise to antibodies reacting with HLA on the platelets.

Detection and identification of allo- or auto-antibodies against platelets is indispensable for a targeted therapy of NAIT, PTP and PR in platelet transfusions.

PRODUCTS

The apDia ready-to-use human Platelet-Antibody Screening Cells & Platelet-Antibody Identification Panel Cells are manufactured by employing a special proprietary production process. The standardized antibody screening panel allows the sensitive detection of anti-HPA antibodies, while the platelet antibody identification panel offers the ability to reliably identify the antibodies. These reagents are especially recommended for the MAIPA procedure.

ADVANTAGES

The apDia thrombocyte reagents are advantageous for standardization, handling and workflow organization in the platelet immunology laboratory. The use of well-characterized thrombocytes offers the ability to standardize the MAIPA: the apDia thrombocyte reagents allow the use of typed cells expressing even rare antigen combinations such as HPA-1 [a-,b+] [2.5%] and HPA-5 [a-,b+] [less than 1%] for an extended period of time. The stability of the platelet preparations and platelet antigens is guaranteed until indicated expiry date if stored at 2-8 °C.

Donor	Blood group	HPA-1a	HPA-1b	HPA-2a	HPA-2b	HPA-3a	HPA-3b	HPA-4a	HPA-4b	HPA-5a	HPA-5b	HPA-6a	HPA-6b	HPA-15a	HPA-15b
Screening cells	0	+	+	+	+	+	+	+	-	+	+	+	-	+	+
Identification platelet 1	0	-	+	+	-	+	-	+	-	-	-	+	-	+	+
Identification platelet 2	0	-	+	+	-	+	-	+	-	+	+	+	-	+	-
Identification platelet 3	0	-	+	+	+	-	+	+	-	+	+	+	-	-	+
Identification platelet 4	0	+	-	+	-	-	+	+	-	-	-	+	-	-	+
Identification platelet 5	0	+	-	+	+	+	-	+	-	+	+	+	-	+	-
Identification platelet 6	0	+	-	+	-	-	+	+	-	+	+	+	-	+	+

DONOR AND ANTIGENS OF EACH IDENTIFICATION CELL PANEL MAY CHANGE FROM LOT TO LOT.

BIBLIOGRAPHY

1. Monoclonal antibody-specific immobilization of platelet antigens (MAIPA): a new tool for the identification of platelet-reactive antibodies. Kiefel V, Santos S, Weisheit M, Müller-Eckhardt C. *Blood*. 1987 Dec; 70(6):1722-6.
2. A modified rapid monoclonal antibody-specific immobilization of platelet antigen assay for the detection of human platelet antigen (HPA) antibodies: a multicentre evaluation. Campbell K, Rishi K, Howkins G, Gilby D, Mushens R, Ghevaert C, Metcalfe P, Ouweland WH, Lucas G. *Vox Sang*. 2007 Nov; 93(4):289-97.
3. Report on the 13th International Society of Blood Transfusion Platelet Immunology Workshop. Foxcroft Z, Campbell K, Mérieux Y, Urbaniak S, Briertley M, Rigal D, Ouweland WH, Metcalfe P. *Vox Sang*. 2007 Nov; 93(4):300-5.
4. The detection of platelet antibodies by simultaneous analysis of specific platelet antibodies and the monoclonal antibody-specific immobilization of platelet . Nguyen XD, Goebel M, Schober M, Klüter H, Panzer S. *Transfusion*. 2010 Jul; 50(7):1429-34. E pub 2010 Apr 23.
5. Human platelet antigen frequencies of platelet donors in the French population determined by polymerase chain reaction with sequence-specific primers. Mérieux Y, Debost M, Bernaud J, Raffin A, Meyer F, Rigal D. *Pathol. Biol. (Paris)* 1997 Nov; 45(9):697-700.