

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Kell in direct ELISAs and Western blots. In direct ELISAs, approximately 25% cross-reactivity with recombinant mouse Kell is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human Kell aa 68-732
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

CyTOF-ready	Optimal dilution of this antibody should be experimentally determined.
Western Blot	Optimal dilution of this antibody should be experimentally determined.
Flow Cytometry	Optimal dilution of this antibody should be experimentally determined.
Immunoprecipitation	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Kell, a type II membrane glycoprotein, is linked through a single disulfide bond to XK, a putative membrane transporter. The two proteins constitute the Kell blood group antigens (1). Kell is a zinc endopeptidase of the neprilysin (NEP) family which also includes endothelin converting enzymes (ECE-1 and ECE-2), PEX, XCE, DINE and several NEP-like proteins (2). It has been shown to cleave big endothelin-3 (ET-3) at Trp21-Ile22, yielding ET-3, and to a much lesser extent, big ET-1 and big ET-2 at Trp21-Val22, yielding ET-1 and ET-2 (3). Several different molecular defects cause the Kell null phenotype, which has no obvious clinical outcome (4).

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