## **Product Data Sheet**

## PE/Cy7 anti-human CD9

Catalog # / Size: 2160575 / 25 tests

2160580 / 100 tests

Clone: HI9a

**Isotype:** Mouse IgG1, κ

Reactivity: Human, Non-human primate

**Preparation:** The antibody was purified by affinity

chromatography and conjugated with PE/Cy7 under optimal conditions. The solution is free of unconjugated PE/Cy7

and unconjugated antibody.

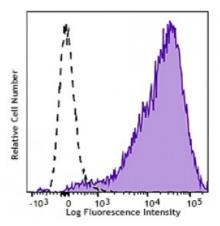
Formulation: Phosphate-buffered solution, pH 7.2,

containing 0.09% sodium azide and

0.2% (w/v) BSA (origin USA).

Workshop Number: V P018

Concentration: Lot-specific



Human platelets were stained with CD9 (clone HI9a) PE/Cy7 (filled histogram) or mouse IgG1,  $\kappa$  PE/Cy7 isotype control (open histogram).

## **Applications:**

**Applications:** Flow Cytometry

Recommended

**Usage:** 

Each lot of this antibody is quality control tested by immunofluorescent staining with flow cytometric analysis. For flow cytometric staining, the suggested use of this reagent is 5  $\mu$ l per million cells or 5  $\mu$ l per 100  $\mu$ l of whole blood. It is recommended that the reagent be titrated for optimal performance for each

application.

**Application** 

1. Miao WM, et al. 2001 Blood 97:1689.

**References:** 

2. Ellerman DA, et al. 2003 Mol. Biol Cell. (Epub ahead of print).

3. Schlossman S, et al. Eds. 1995. Leucocyte Typing V. Oxford University Press.

New York.

**Description:** 

CD9 is a 24 kD type III transmembrane protein also known as tetraspanin, MRP-1 and DRAP-24. It is a member of the tetraspan family (spanning the membrane four times) found on platelets, B cell progenitors, activated lymphocytes, granulocytes, endothelial cells and epithelial cells. CD9 induces adhesion, platelet aggregation, and B cell development. CD9 has been shown to associate with CD63, CD81, CD82, and CD36 and to bind to  $\beta_1$  integrins.

Antigen References:

1. Miao WM, et al. 2001 Blood 97:1689.

2. Ellerman DA, et al. 2003 Mol. Biol Cell. (Epub ahead of print).

3. Schlossman S, et al. Eds. 1995. Leucocyte Typing V. Oxford University Press.

New York.