

FITC-Dextran

Catalog # 4009

For Research Use Only - Not Human or Therapeutic Use

DESCRIPTION:	Fluorescein isothiocyanate labeled dextran
APPLICATIONS:	Use to assess the permeability of semi-permeable membranes either in vivo or in vitro (1, 2, 3).
	Note: It is possible to simultaneously use FITC-dextran and TRITC-dextran of different sizes as FITC and TRITC fluoresce at different wavelengths.
QUANTITY:	FITC-dextran, 5 ml Note: 96-well plates (Catalog # 9045 are available upon request).
FORM:	25 mg/ml solution in PBS (green color).
MOLECULAR WEIGHT:	40 kDa
FLUORESCENT PROPERTIES:	Excitation: 490 nm, Emission: 520 nm
IN VIVO PROTOCOL:	1. Fast mice 4 hours before the oral feeding, and for the duration of the experiment.
	2. Feed 20 ml/kg by oral gavage.
	3. Maintain fasting conditions and wait 3 hours (may vary depending on individual animals).
	4. Collect blood by retro-orbital bleeding, then spin and collect plasma. Dilute plasma 1:2 (or more) with PBS.
	5. Optional: Prepare a standard curve by making dilutions of the stock FITC-dextran with normal mouse plasma (12.5 ug/ml is a recommended starting point). <u>Caution: Protein in the samples may intefere and reduce the fluorescence intensity (FI)</u> . Therefore, to ensure an accurate determination of FITC-dextran permeability, the Plasma to PBS ratio must be consistent throughout the standard curve, as well as with the dilution factor of the sample. Use normal mouse serum diluted in PBS for the blank.
	6. Transfer 50-100 ul of supernatant to a 96-well plate and read on a fluorescent plate reader.
IN VITRO PROTOCOL:	Please refer to protocol of choice.
STORAGE TEMPERATURE:	4°C in the dark
STABILITY:	1 year



REFERENCES:

1. M. Vijay-Kumar et al., Deletion of TLR5 results in spontaneous colitis in mice. J Clin Invest 117, 3909-3921 (2007).

2. Q. Wang et al., Intestinal permeability is reduced and IL-10 levels are increased in septic IL-6 knockout mice. Am J Physiol Regul Integr Comp Physiol 281, R1013-1023 (2001).

3. W. Huang et al., HMGB1 increases permeability of the endothelial cell monolayer via RAGE and Src family tyrosine kinase pathways. Inflammation 35, 350-362 (2012).

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