

# Recombinant Human SP-A1 Protein(Fc Tag)

Catalog Number: PDMH100321



**Note:** Centrifuge before opening to ensure complete recovery of vial contents.

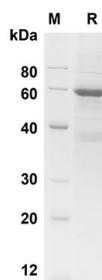
## Description

<b>Species</b>	Human
<b>Source</b>	Mammalian-derived Human SP-A1 proteins Glu21-Phe248,with an C-terminal Fc
<b>Mol_Mass</b>	49.9 kDa
<b>Accession</b>	Q8IWL2
<b>Bio-activity</b>	Not validated for activity

## Properties

<b>Purity</b>	> 90% as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 1.0 EU/mg of the protein as determined by the LAL method
<b>Storage</b>	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 °C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
<b>Reconstitution</b>	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

## Data



SDS-PAGE analysis of Human SP-A1 proteins , 2µg/lane of Recombinant Human SP-A1 proteins was resolved with SDS-PAGE under reducing conditions , showing bands at 60 KD

## Background

Surfactant protein A (SFTPA1), a member of the collagenous lectin (collectin) family , was first described as a major constituent of lung surfactant but has recently also been found in the female genital tract. Various microorganisms colonize this area and may cause intrauterine infection or trigger preterm labor. The Human SFTPA1 and SFTPA2 genes encode SP-A1 and SP-2 proteins , and each gene has been identified with numerous genetic variants. SP-A1 and SP-A2 differentially enhance bacterial phagocytosis. Sex differences have been observed in pulmonary disease and survival of wild type and SP-A knockout (KO) mice. SP-A interacts and regulates many of the functions of alveolar macrophages (AM). It is shown that SP-A variants differ in their ability to regulate the AM miRNome in response to oxidative stress (OxS).

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