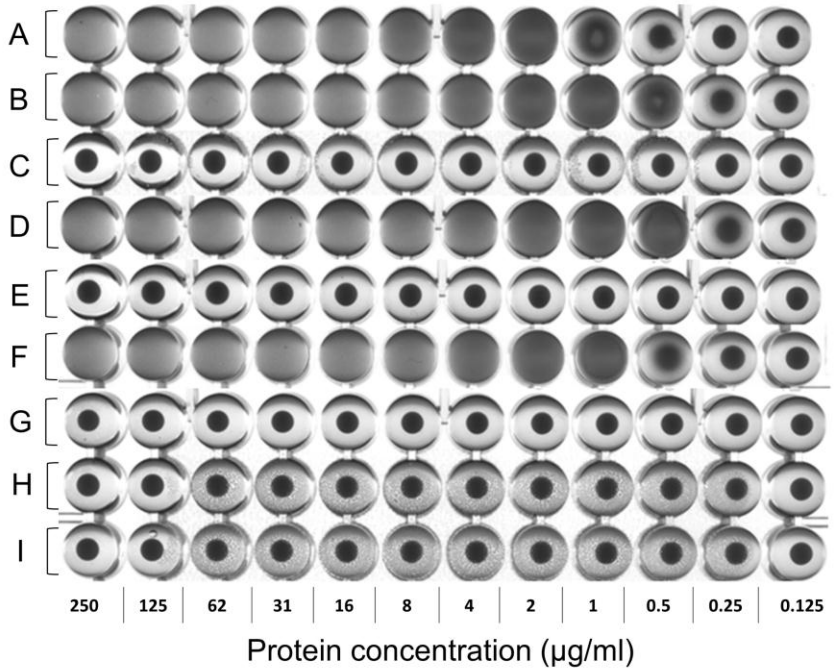

Figure S1 Hemagglutination activities of native FIP-*fve*, wild type and site-directed rFIP-*fve* mutants. Hemagglutination activity toward 100 μl/ml of 2% human red blood cells in PBS of native FIP-*fve* (**A**), wild type rFIP-*fve*, (**B**), W24G (**C**), T28N (**D**), D34G (**E**), T90A (**F**), I91A (**G**) and W111G (**H**) mutants rFIP-*fve* in 100 μl, respectively. Reaction mixtures were placed in 96-well microtiter plates and incubated at 37 ℃. Hemagglutination activities of serially diluted mixtures ranges from 250 to 0.125 (μg/ml) were recorded following 1.5 h incubation. Serially diluted GST (**I**) was added instead of rFIP-*fve* to human red blood cells as a control.

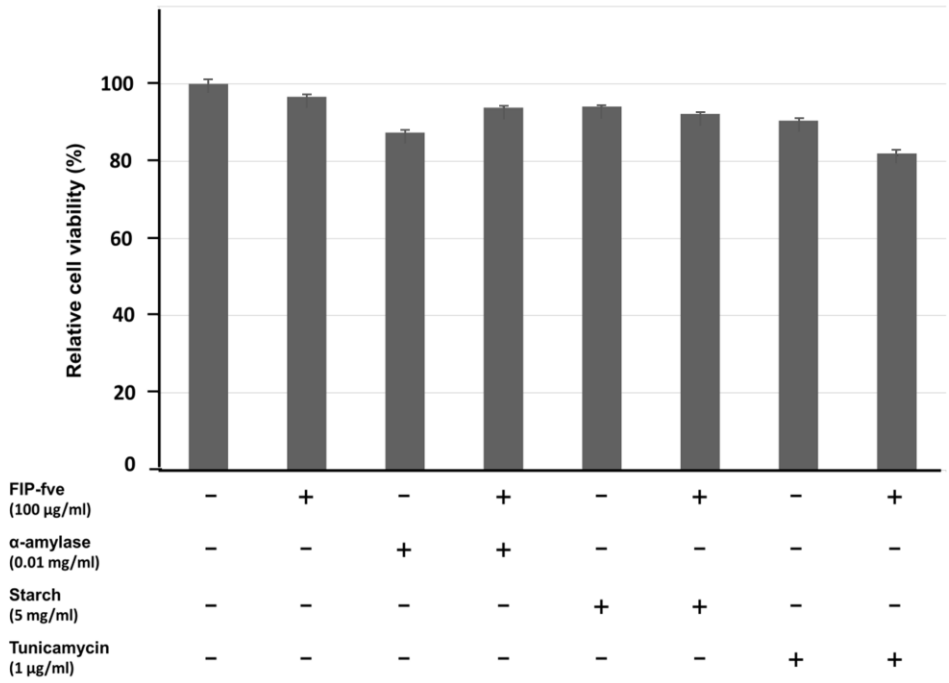

Figure S2 Effects of FIP-*fve*, α-amylase, Starch and Tunicamycin treatments on hPBMCs cell viability. hPBMCs (2×10^6/well) cells were treated with concentrations of 100 µg/ml FIP-*fve*, 0.01 mg/ml α-amylase, 5 mg/ml starch and 1 µg/ml Tunicamycin for 48 h followed by MTT assay to estimate the cell viability. The data are presented as mean ± SD of triplicate experiments.

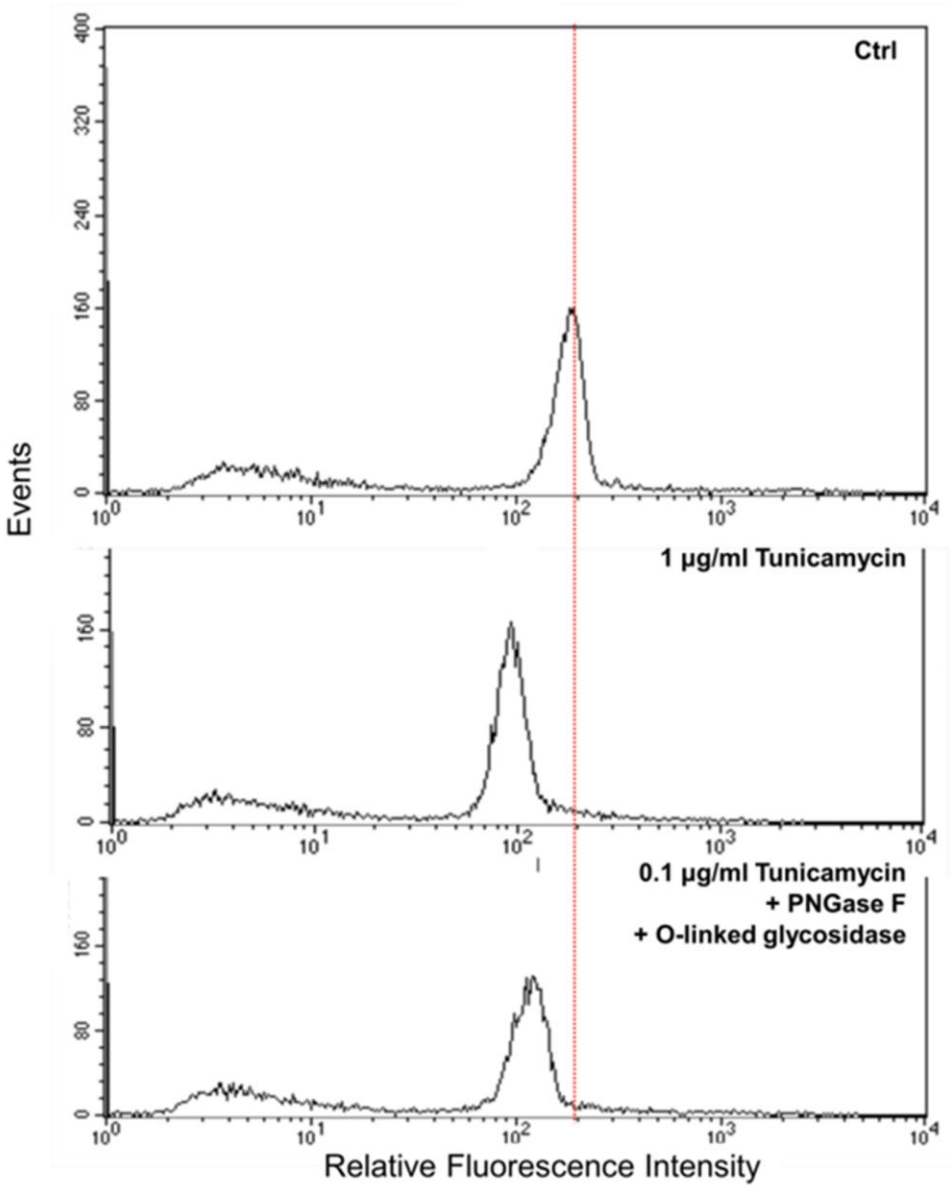

Figure S3 Influence of tunicamycin and glycosidase enzymes treatment to be confirmed the hPBMC cell surface glycoproteins in ability to recognize *O*-linked and *N*-linked via FITC-labeled ConA. Cultured hPBMCs ($2x10^6$ cells/mL, 1 mL/well) were treated with 1 µg/ml tunicamycin or pretreated with 0.1, µg/ml tunicamycin for 16 h before treated with the deglycosylation enzymes *O*-linked glycosidase ($8x10^4$ units/ml) and PNGase F ($1x10^3$ units/ml) for 4 h. At the end of treatment, Con A-FITC (5 µg/ml) was added to the medium and incubated for 30 m. The fluorescence-activated cells were analyzed by flow cytometry.

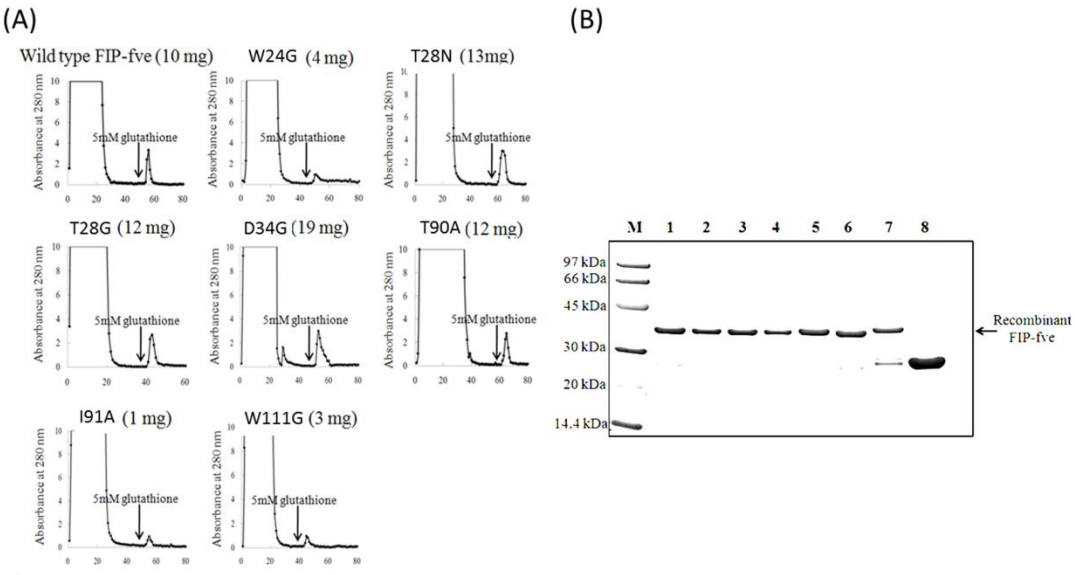

Figure S4 Verify the purity of the recombinant proteins. The fusion proteins were purified more than 85% by affinity chromatograph with Glutathione Sepharose^{TM} 4 Fast Flow. (**A**) The yields of wild type rFIP-*fve*: 10 mg; W24G: 4 mg; T28N: 13 mg; T28G: 12 mg; D34G: 19 mg; T90A: 12 mg; I91A: 1 mg and W111G: 3 mg. (**B**) 12% SDS PAGE analysis of purified rFIP-*fve*. M: marker; lane 1: wild type rFIP-*fve*; lane 2: rFIP-*fve* mutation W24G; lane 3: rFIP-*fve* mutation T28N; lane 4 rFIP-*fve* mutation D34G; lane 5: rFIP-*fve* mutation T90A; lane 6: rFIP-*fve* mutation I91A; lane 7: rFIP-*fve* mutation W113G; lane 8: GST.

Table S1 Nucleotide sequences of oligonucleotide primers used for introduction of mutation by site-directed mutagenesis for specific amino acid residues in the putative CBM family 34 of the rFIP-*fve*

Amino acid substitution	Mutagenic PCR primer (5' -> 3') [a]
W24G	CACCCCCAAC**G**GGGGCCGTGGTACC
T28G	CTGGGGCCGTGGTA**A**CCCAAGCAGCTAC
T28N	CTGGGGCCGTGGT**GG**CCCAAGCAGCTAC
D34G	GGTACCCCAAGCAGCTACATCG**G**CAACC
T90A	CGCGGACACCAAA**G**CGATTCAAGTTTTCG
I91A	CGCGGACACCAAAACG**GC**TCAAGTTTTCG
W111G	GGAGTACATCATCGCTGA**G**GGGAAGAAGAC

a. Mutations are indicated in *bold and underlined*