Figure S1. Morphological changes in RBL-2H3 cells treated with PTFAb. RBL-2H3 cells were incubated for 24 h in the presence or absence of PTFAb (1-150 μg/ml). The final DMSO concentration was maintained at 0.1% in all experimental groups, and control cells were treated with 0.1% DMSO as the vehicle control. Images were acquired using an inverted phase-contrast microscope at x200 magnification. The images shown are representative results from three independent experiments. Scale bar: 50 μm. Con, vehicle control; DMSO, dimethyl sulfoxide; PTFAb, *Paulownia tomentosa* flower absolute.

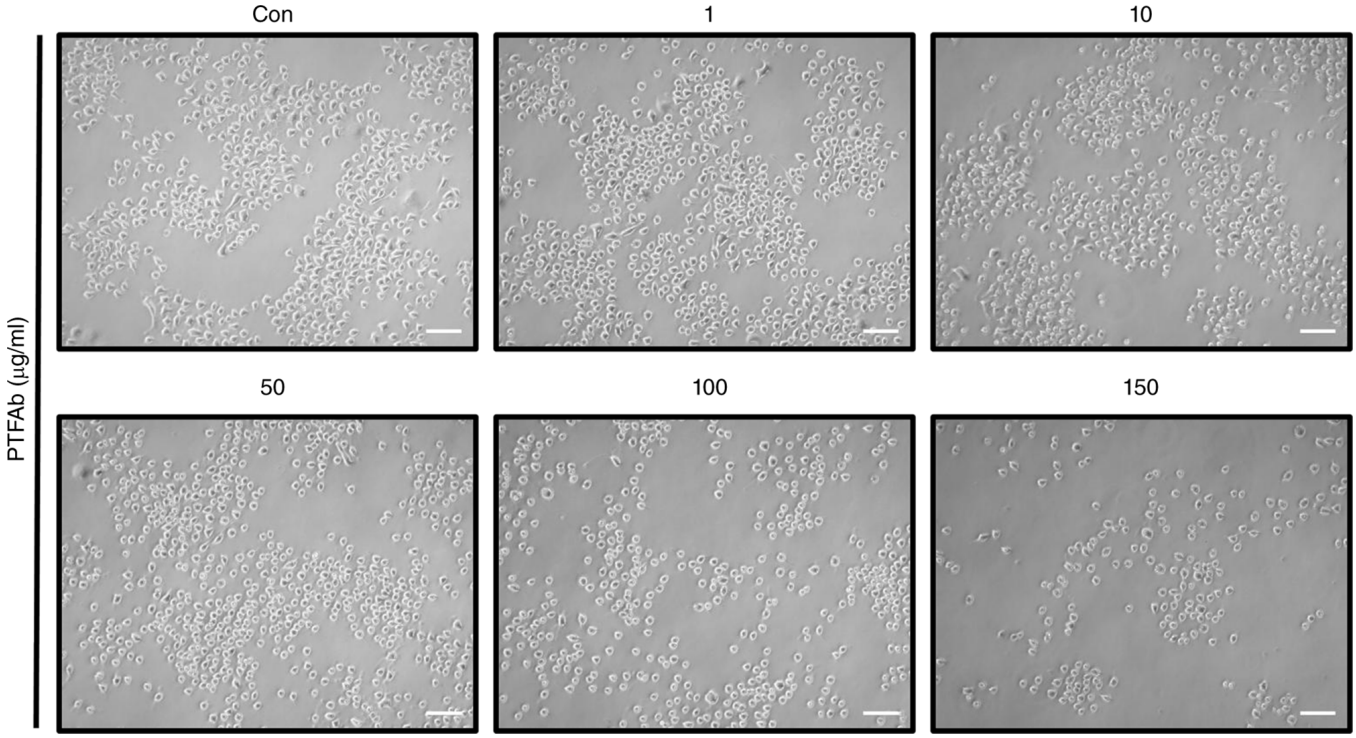

Figure S2. Effects of PTFAb on the expression of t-SNARE proteins in RBL-2H3 cells. (A) Representative images. RBL-2H3 cells were incubated for 48 h in the presence or absence of PTFAb (0.1-100 μg/ml). The final DMSO concentration was maintained at 0.1% in all experimental groups, and control cells were treated with 0.1% DMSO as the vehicle control. The cell lysates were immunoblotted with the indicated antibodies. (B-E) The graphs show the expression levels of the (B) syntaxin 1a, (C) syntaxin 4, (D) SNAP 23 and (E) SNAP 25 proteins shown in panel A. The expression levels of these proteins were quantified by normalization to β-actin (loading control) and presented as a percentage relative to the vehicle control group. DMSO, dimethyl sulfoxide; PTFAb, *Paulownia tomentosa* flower absolute; SNAP, synaptosomal-associated protein; t-SNARE, target-soluble N-ethylmaleimide-sensitive factor attachment protein receptor.

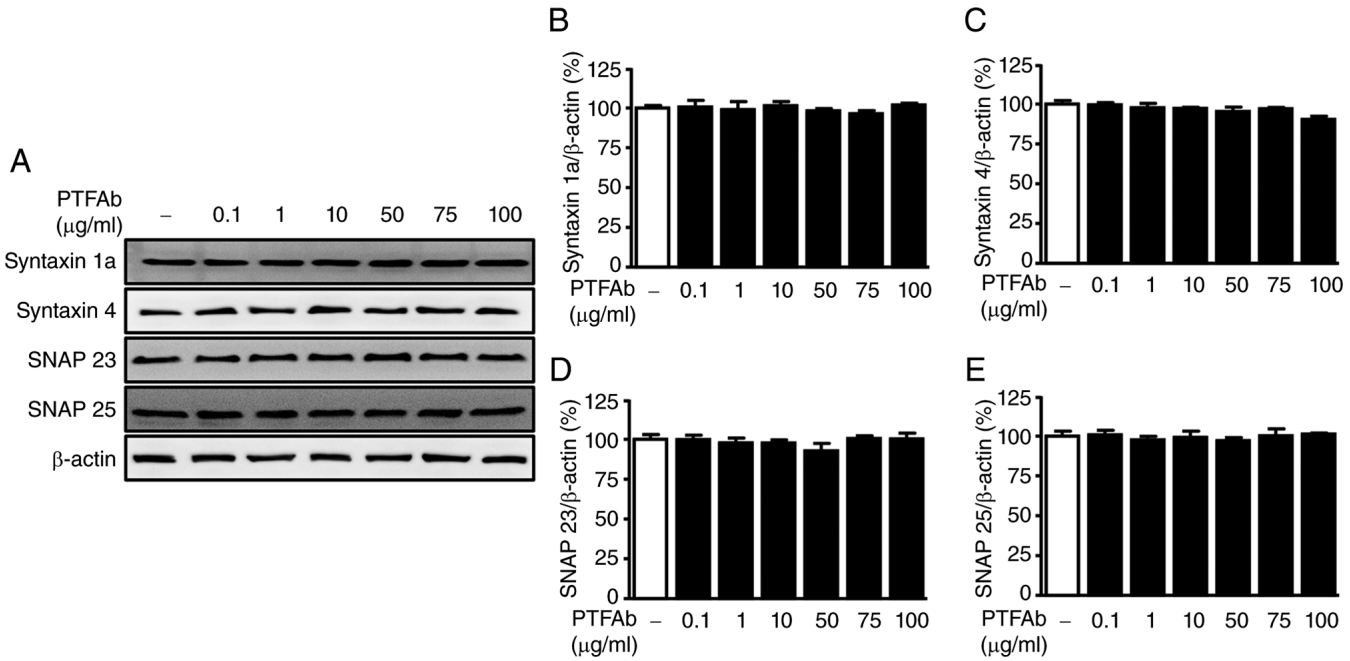

Figure S3. Quantitative analysis of total Syk, PI3K, and AKT levels in RBL-2H3 cells shown in Fig. 4. The final DMSO concentration was maintained at 0.1% in all experimental groups. Total (A) Syk, (B) PI3K and (C) AKT protein levels were quantified and normalized to β-actin as a loading control to confirm equal protein loading across samples. The values are presented as a percentage relative to the anti-DNP IgE-sensitized cells treated with 0.1% DMSO alone. Data are expressed as the means ± SEMs (n=3 for each protein). AKT, protein kinase B; anti-DNP IgE, anti-dinitrophenyl immunoglobulin E; DMSO, dimethyl sulfoxide; DNP-BSA, 2,4-dinitrophenyl-labeled bovine serum albumin; PI3K, phosphoinositide 3-kinase; SEMs, standard errors of the means; Syk, spleen tyrosine kinase.

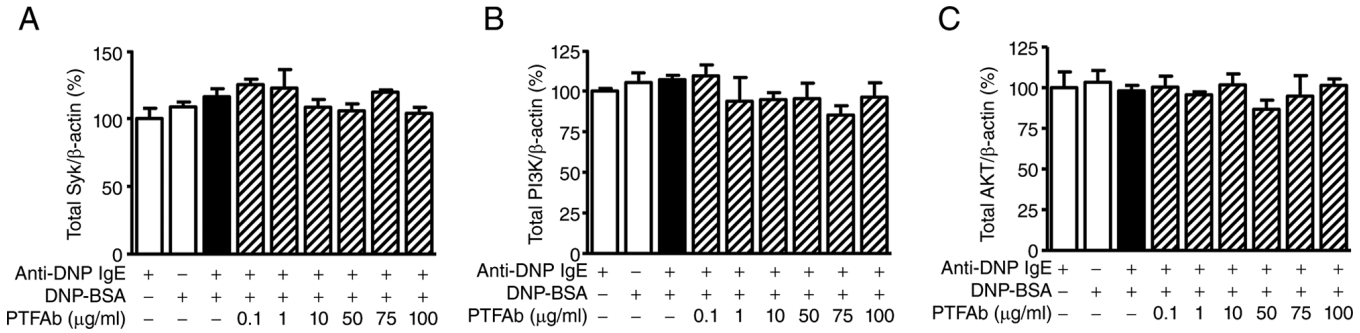

Figure S4. Effects of PTFAb on the phosphorylation of MAPKs in RBL-2H3 cells. (A) RBL-2H3 cells were sensitized with anti-DNP IgE and then stimulated with DNP-BSA. The cells were treated with PTFAb (0.1-100 μg/ml), and the expression levels of p-ERK1/2), p-p38), and p-JNK were analyzed by Western blotting (n=3 for each protein). The final DMSO concentration was maintained at 0.1% in all experimental groups. For each MAPK, the phosphorylated form, total form, and corresponding β-actin were obtained from the same gel and membrane. Different MAPKs were analyzed on separate gels and are presented as assembled images. (B-D) Quantification of the (B) p-ERK1/2, (C) p-p38 and (D) p-JNK levels normalized to the respective total protein levels (total-ERK1/2, total p38, and total JNK), and presented as percentages relative to anti-DNP IgE-sensitized cells treated with 0.1% DMSO alone. (E-G) Quantification of (E) total ERK1/2, (F) total p38 and (G) total JNK levels normalized to β-actin. Values are presented as percentages relative to the anti-DNP IgE control. Anti-DNP IgE, anti-dinitrophenyl immunoglobulin E; DMSO, dimethyl sulfoxide; DNP-BSA, 2,4-dinitrophenyl-labeled bovine serum albumin; MAPK; mitogen-activated protein kinase; p-ERK1/2, phosphorylated extracellular signal-regulated kinase; p-JNK, phosphorylated c-Jun N-terminal kinase; p-p38, phosphorylated p38 mitogen-activated protein kinase; PTFAb, *Paulownia tomentosa* flower absolute.

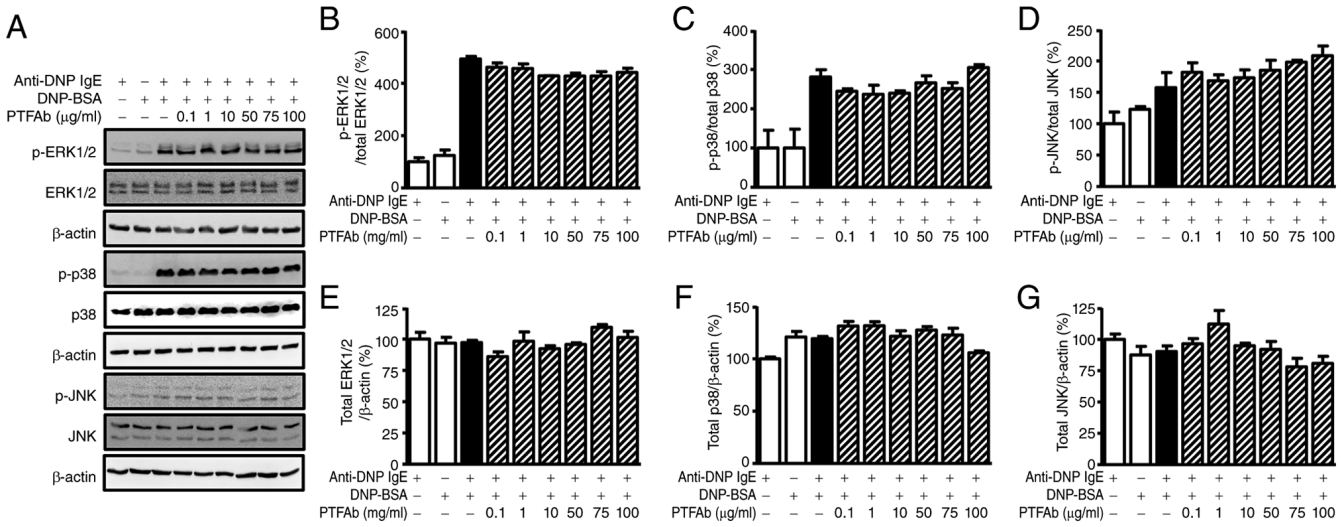

Figure S5. Effects of signaling pathway inhibitors on the inhibitory effect of PTFAb on IgE-mediated degranulation in RBL-2H3 cells. RBL-2H3 cells were incubated for 48 h with or without the indicated signaling inhibitors, including piceatannol (30 μM), LY294002 (0.1 μM), SB203580 (30 μM), and PD98059 (10 μM) in the presence or absence of PTFAb (50 μg/ml). The cells were sensitized with anti-dinitrophenyl immunoglobulin E (anti-DNP IgE; 200 ng/ml) for 10 h and subsequently stimulated with 2,4-dinitrophenyl-labeled BSA (DNP-BSA; 20 ng/ml) for 1 h. β-Hexosaminidase activity in the conditioned medium was measured using a colorimetric substrate assay as described in the Materials and methods. (A) Effects of individual signaling pathway inhibitors on β-hexosaminidase release (n=3). (B) Effects of co-treatment with signaling pathway inhibitors and PTFAb on β-hexosaminidase release (n=3). The final DMSO concentration was maintained at 0.1% in all experimental groups. The β-hexosaminidase release from anti-DNP-IgE-sensitized cells treated with 0.1% DMSO alone was set as 100%. $^{*}P<0.05$ vs. anti-DNP IgE/DNP-BSA-stimulated cells in the presence of 0.1% DMSO alone. Anti-DNP IgE, anti-dinitrophenyl immunoglobulin E; DMSO, dimethyl sulfoxide; DNP-BSA, 2,4-dinitrophenyl-labeled bovine serum albumin; PTFAb, *Paulownia tomentosa* flower absolute.