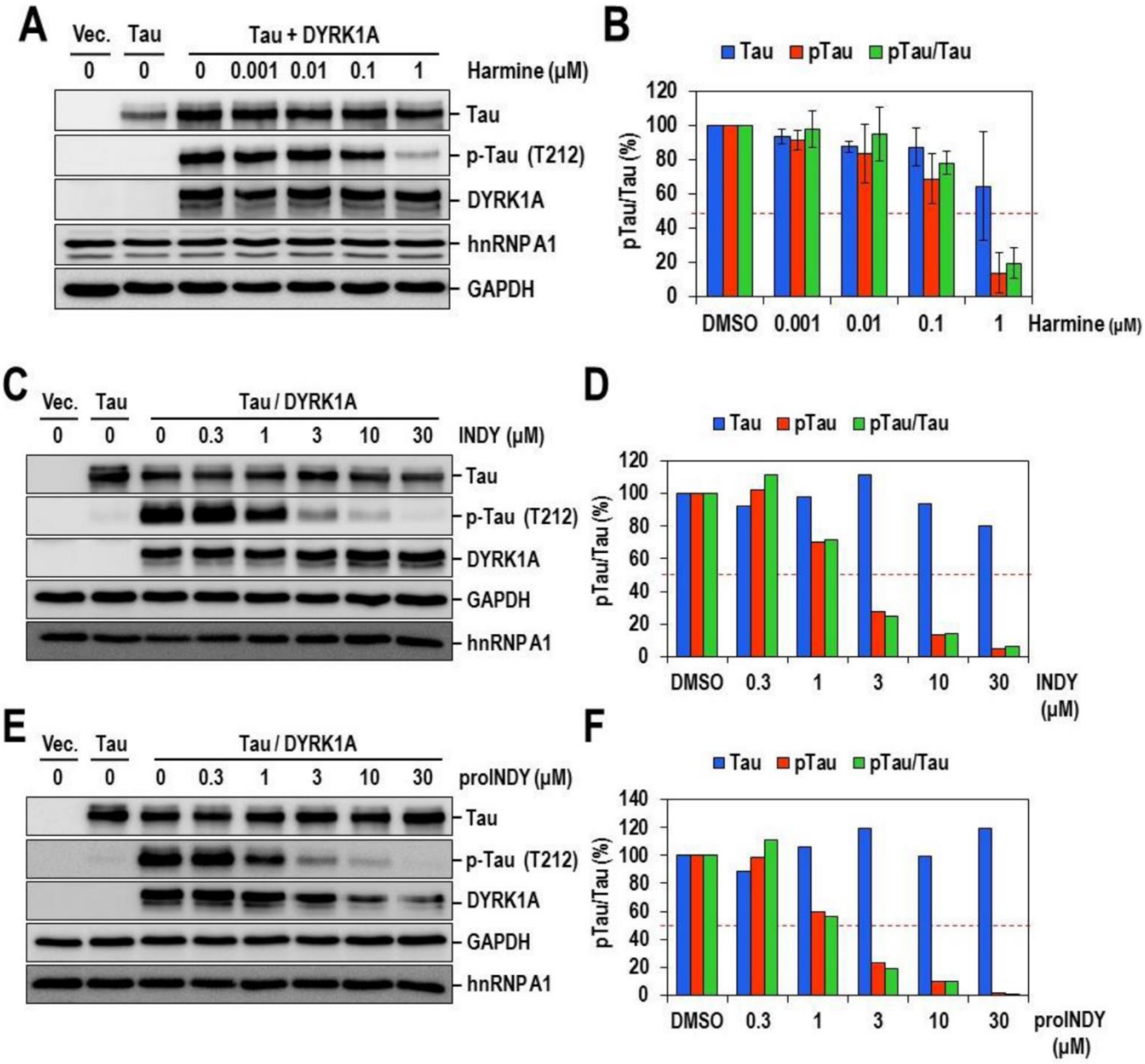
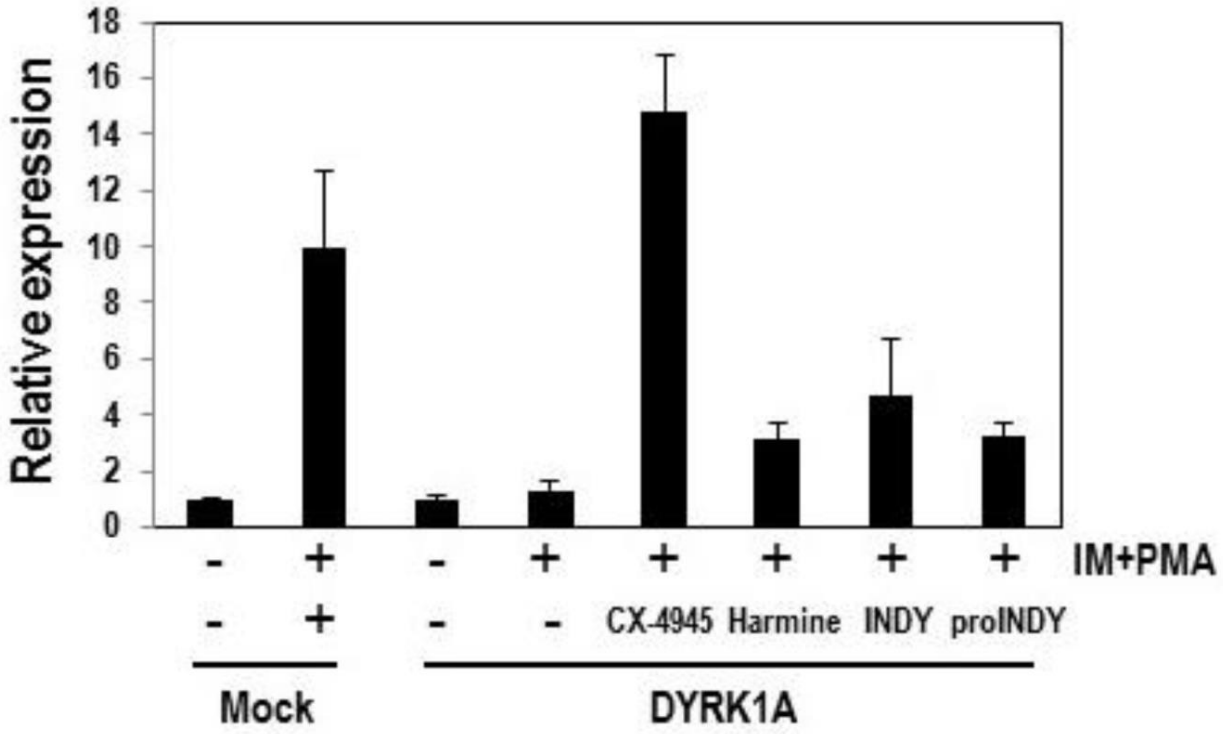


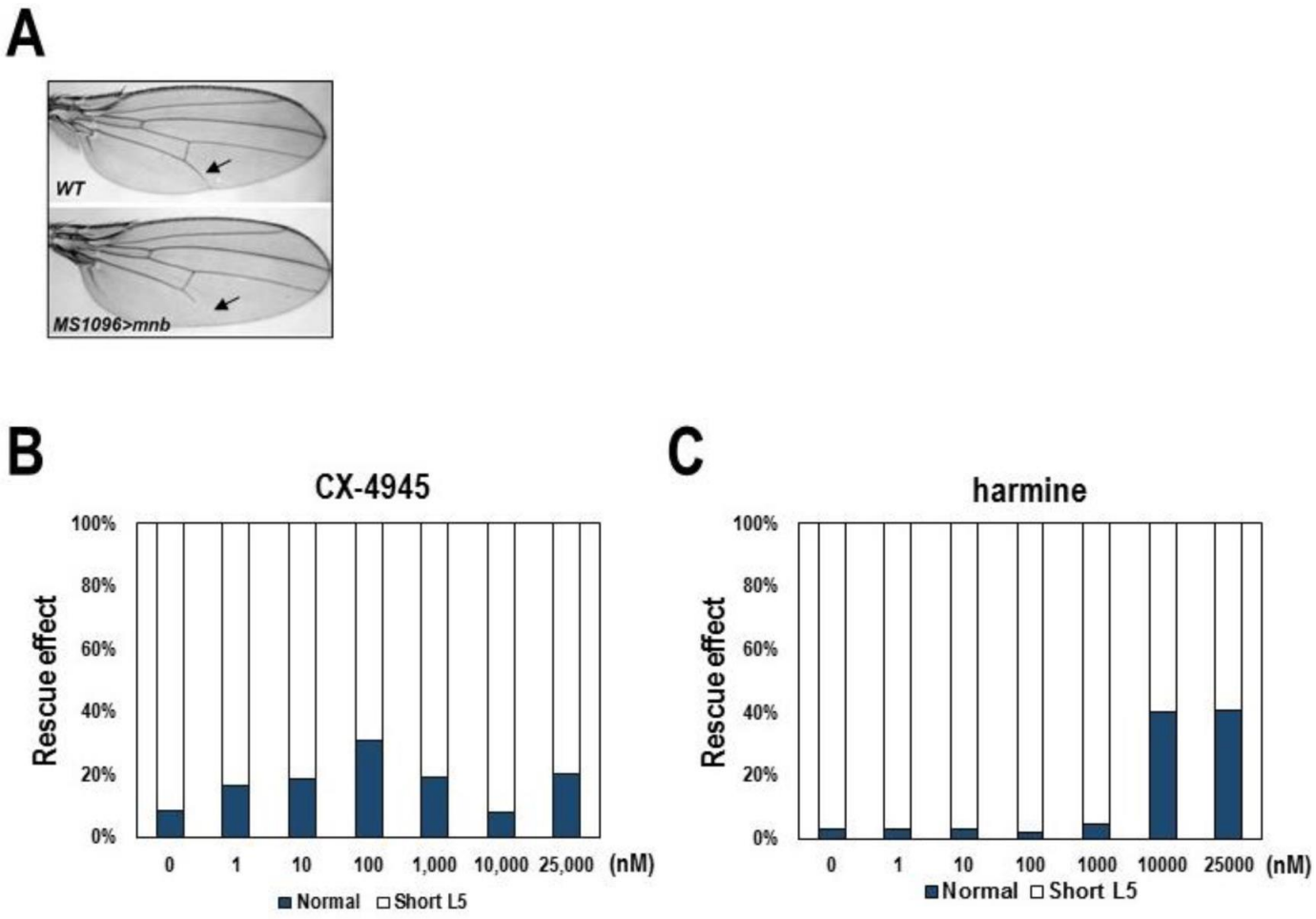
Supplementary Figure 1

293T cells were transfected with the indicated plasmids expressing Tau or DYRK1A. Transfected cells were treated with the indicated doses of harmine (A), INDY (C), and proINDY (E) for 6 hours, and total cell extracts were then subjected to Western blot analysis. The phosphorylated and total Tau proteins in panels A, C, and E were quantified, and the amounts of protein relative to those of DMSO-treated samples are shown in B, D, and F, respectively.



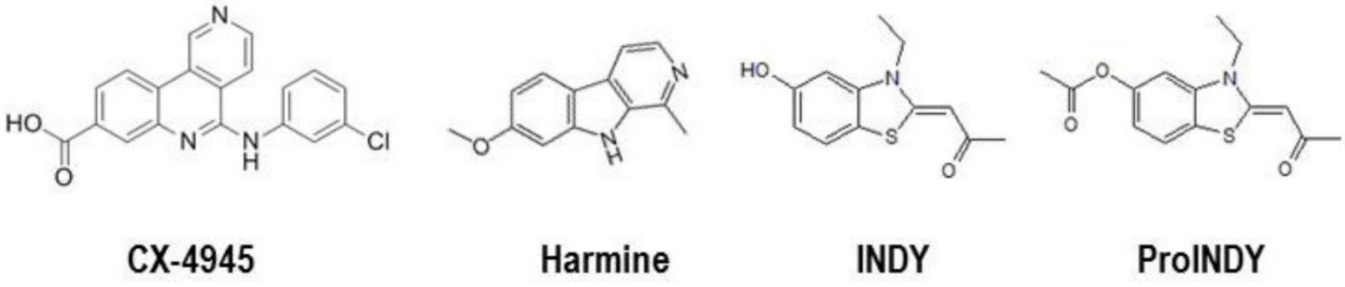
Supplementary Figure 2

The effect of CX-4945, harmine, INDY, and proINDY on NFATc1-mediated transcriptional activation. 293T cells were transfected with NRE-Luc reporter plasmid with or without plasmid expressing DYRK1A and then treated with IM (5 μM) and phorbol 12-myristate 13-acetate (PMA; 10 nM) along with 10 μM of each compounds. Firefly luciferase activities were measured using One-Glo reagents. Luciferase activity in the sample with reporter plasmid alone was set to 1, and the relative luciferase activities were calculated. Averages and SDs were determined from two independent experiments.



Supplementary Figure 3

Rescue of the wing defect phenotype of *mnb*-overexpressing flies by harmine and CX-4945. (A) The wing area from adult flies expressing *UAS–mnb* under the control of *MS1096–gal4*. Expression of *mnb* throughout the wing led to distal truncation of the L5 wing vein (black arrow). Dose-dependent analysis of harmine (B) or CX-4945 (C) on the L5 wing vein defect of *MS1096>mnb* flies. The rescue effect of each treatment was quantified from three independent experiments (n=150 for each).



Supplementary Figure 4

Chemical structures of CX-4945, harmine, INDY, and proINDY.