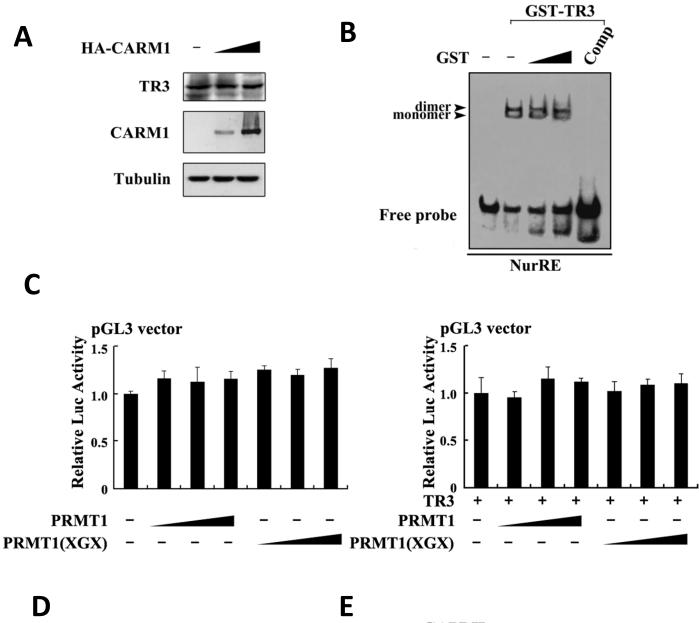
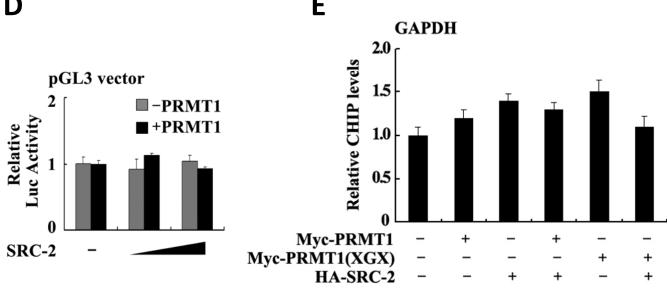
## **Supplementary Figures**

(A) CARM1 could not regulate the expression levels of TR3. 293T cells were transfected with increasing amount of CARM1. After transfection, cells were lysated and then subjected to western blotting. (B) GST did not influence the complex between GST-TR3 and NurRE. GST-TR3 was incubated with different amount of GST, and then subjected to the EMSA assay with NurRE as a probe. (C-D) PRMT1, PRMT1(XGX) and SRC-2 had no effect on the activity of pGL3-promoter vector. Different plasmids as indicated were transfected with pGL3-promoter vector into 293T cells. After transfection, the Luc activity assays were performed. (E) PRMT1, PRMT1(XGX) and SRC-2 had no effect on the binding of TR3 to the promoter of GAPDH. As described in Fig. 3B, the samples of ChIP were quantified by qPCR. (F) si-PRMT1 did not influence the transcriptional activity of Gal-TAD. 293T cells were transfected with increasing amount of si-PRMT1, together with GAL4-TAD in the absence or presence of SRC-2 as indicated. The luciferase activity was then determined. (G) TR3 had no effect on methylation of the substrate of CARM1. CARM1 together with TR3 was transfected into 293T cells. After transfection, cell lysates were collected and subjected to western blotting using antibodies as indicated. (H-J) STAT3 (H), Sam68 (I) and H4 (J) interacted with PRMT1. Different GST-PRMT1 truncation mutants were separately incubated with Sam68 or STAT3 protein immunoprecipitated from 293T cells to detect their interactions as described in Fig. 4E.

## **Supplementary Figures**





F G

