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Supplemental information

Narciclasine targets STAT3 via distinct mechanisms in tamoxifen-resistant breast cancer cells

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Table S1 Primers sequences for qPCR analysis

Gene	Forward primer	Reverse primer
STAT3	AGTGACCAGGCAGAAGATGC	CACGTACTCCATCGCTGACA
Cyclin D1	GTGCCACAGATGTGAAGT	GTAGGACAGGAAGTTGTTGG
β-Actin	ATTCCTATGTGGGCGACGAG	CCAGATTTTCTCCATGTCGTCC

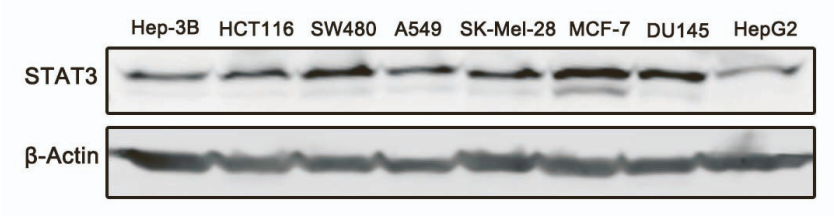

Fig. S1. The expression levels of STAT3 in various cancer cell lines by western blot analysis.

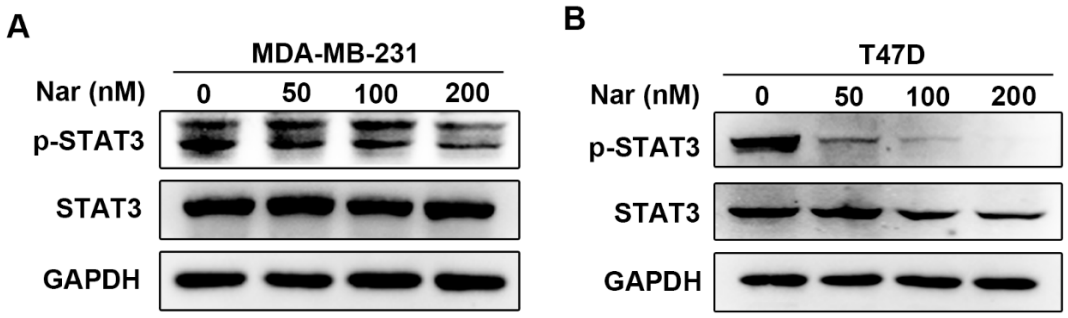

Fig. S2. STAT3 and p-STAT3 levels in MDA-MB-231 and T47D cells treated with different concentrations of Nar. (A, B) The inhibitory effects of different concentrations of Nar on p-STAT3 and total STAT3 in MDA-MB-231 (A) and T47D (B) cells.

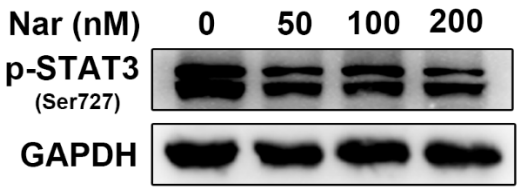

Fig. S3. The effects of different concentrations of Nar on p-STAT3 (Ser727) in MCF-7 cells.

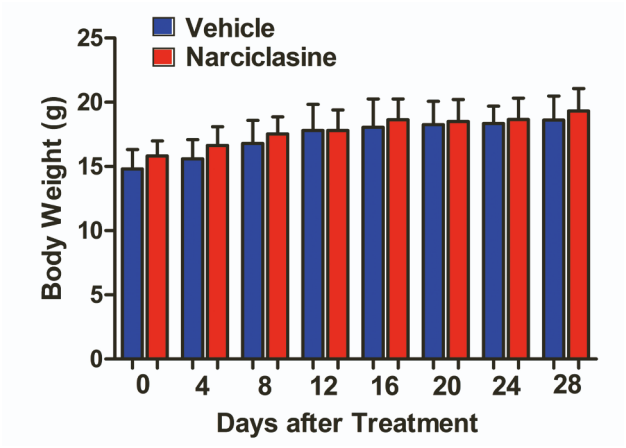

Fig. S4. Body weight during treatment (day 0 represents the day that Nar was administered).

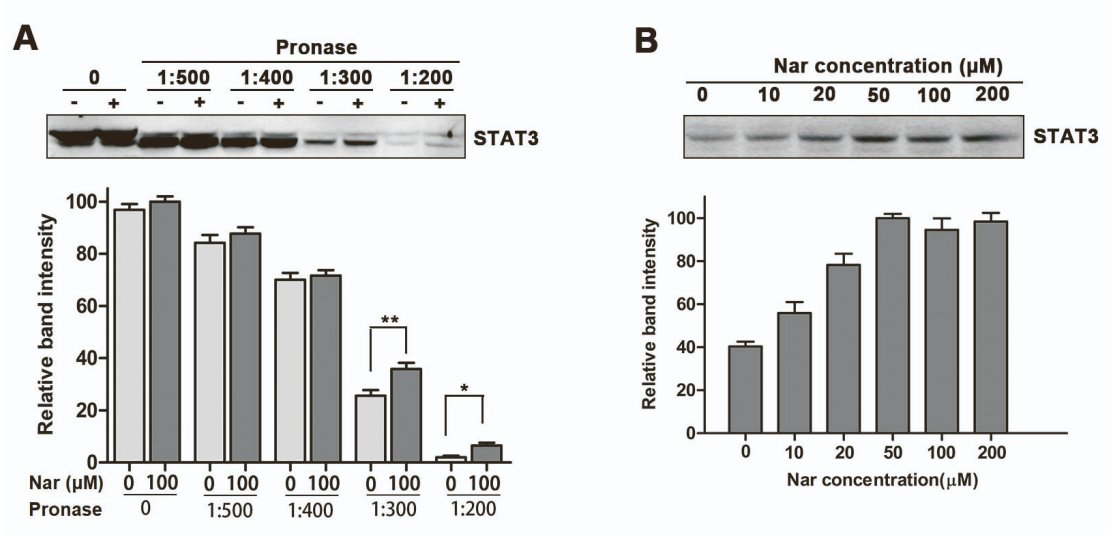

Fig. S5. DARTS analysis was performed to confirm Nar binding to STAT3 target protein.

(A, B) The inhibitory effect of Nar on STAT3 proteolysis at different temperatures and different concentrations were evaluated by Western blotting. Data are shown as mean ± S.D. $^{*}p<0.05$, $^{**}p<0.01$.

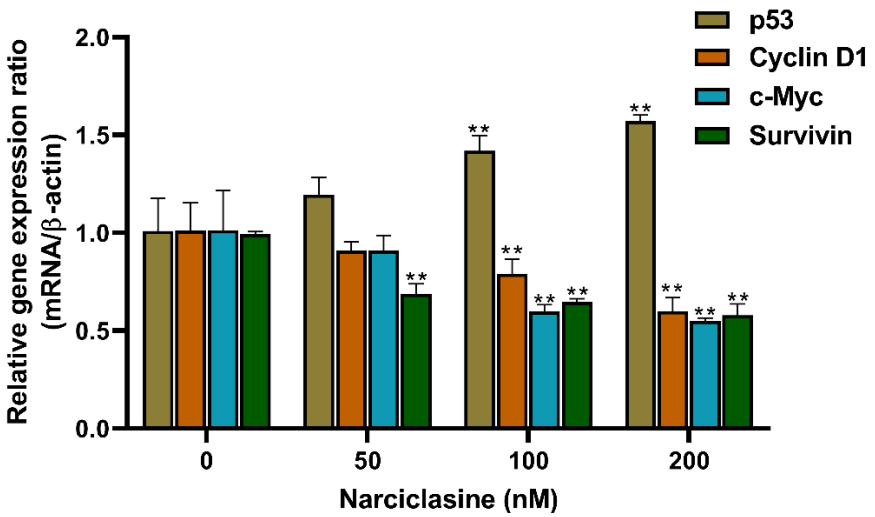

Fig. S6. The mRNA levels of c-Myc, Cyclin D1, Survivin and p53 in MCF-7 cells treated with different concentrations of Nar. Data are shown as mean ± S.D. $^{**}p<0.01$.

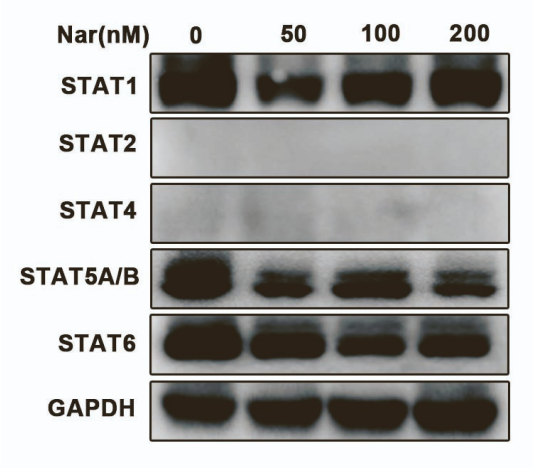

Fig. S7. The expression levels of STAT family members after treatment with different concentrations of Nar for 24 h.

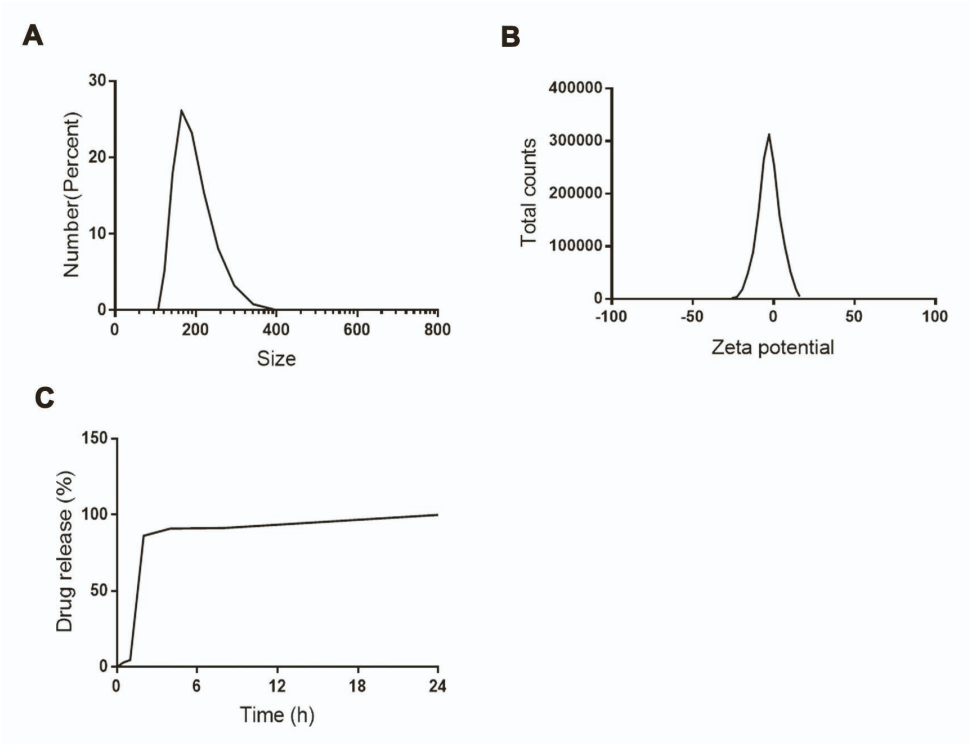

Fig. S8. Characterization of Nar-LPs. (A, B) The size distribution (A) and zeta potential (B) of Nar-LPs. (C) Nar release profiles of nanoparticles.