Figure S1. (A) Cytotoxicity of sh1, sh2 and sh3 targeting nuclear receptor subfamily 2 group E member 3 to MCF7 cells was detected using the MTT assay. (B) Statistical analysis of wound healing rate of the data presented in Fig. 2E. Nc, negative control; ns, not significant; sh, short hairpin RNA. $^{*}P<0.05$ and $^{**}P<0.01$.

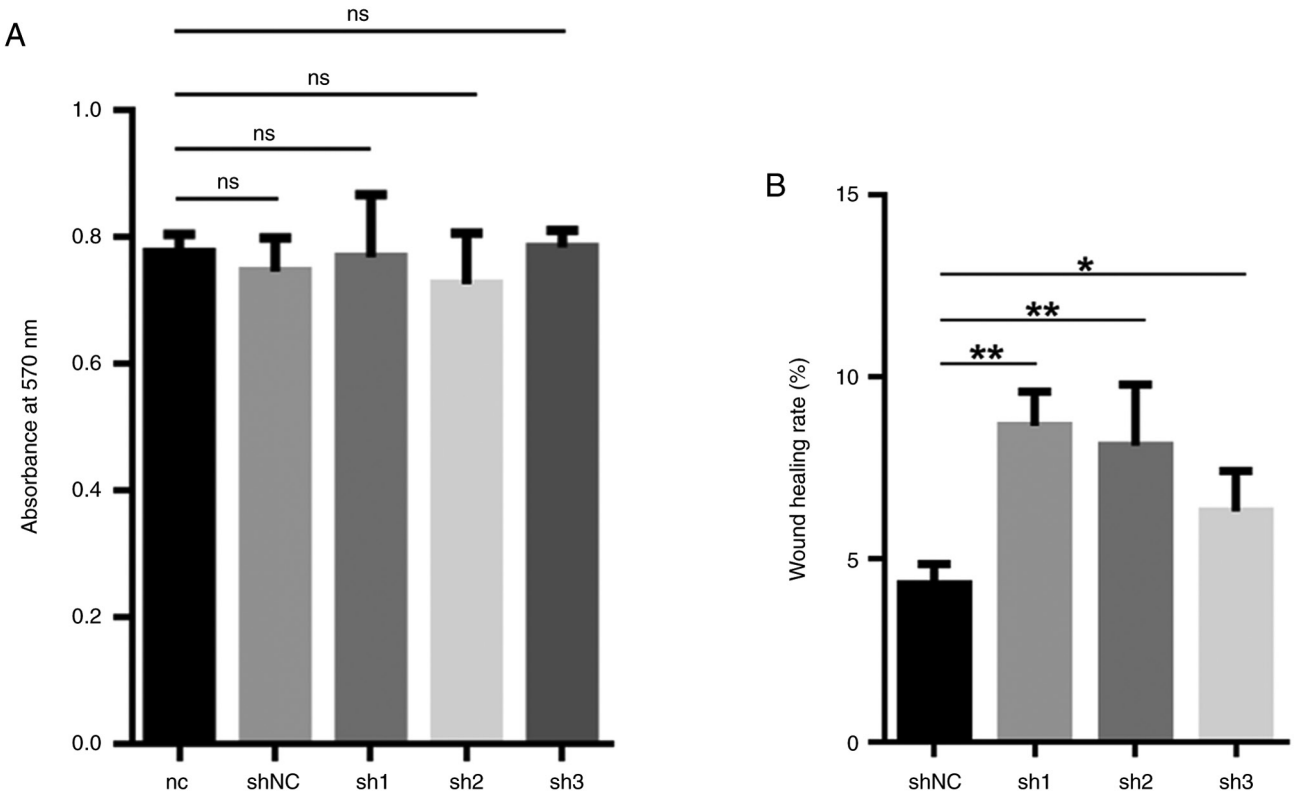


Figure S2. (A) Statistical analysis of colony formation of the data presented in Fig. 2F. (B) Protein expression levels of *nr2e3* were detected in parental and paclitaxel-resistant MCF7 cells by western blotting. GAPDH was used as the endogenous control. NR2E3, nuclear receptor subfamily 2 group E member 3; PR, paclitaxel-resistant; sh, short hairpin RNA; *P<0.05 and **P<0.01.

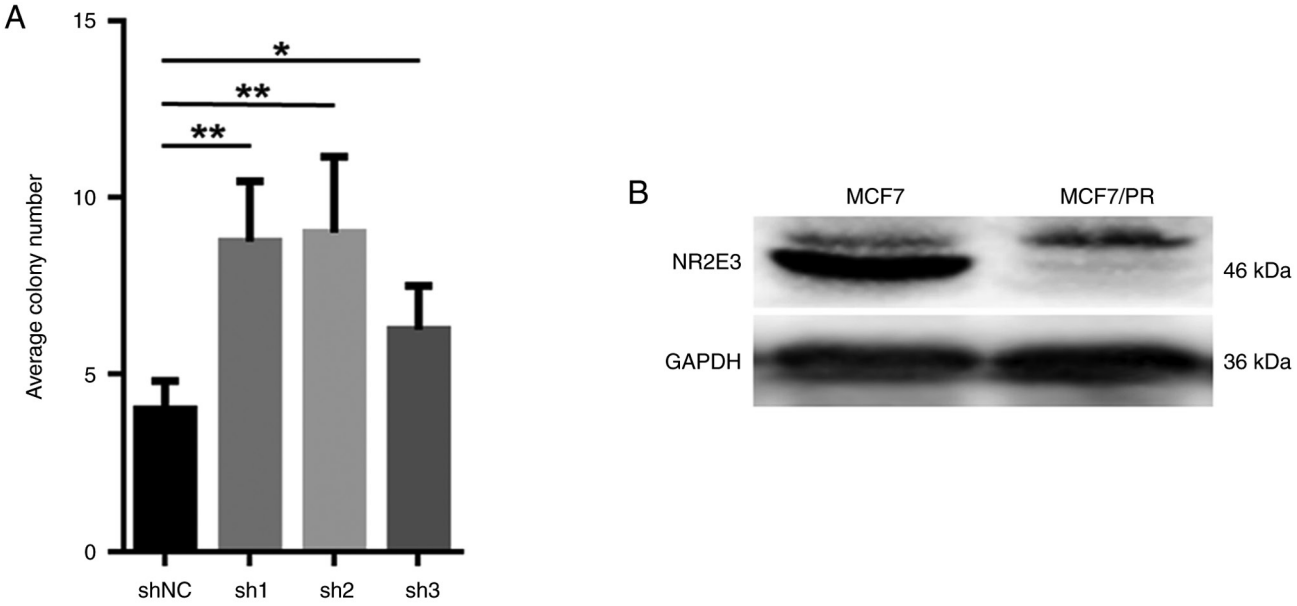


Figure S3. Statistical analysis of the relative protein content of (A) E-cadherin, (B) N-cadherin, (C) VIMENTIN and (D) SLUG in Fig. 3B. *P<0.05, **P<0.01 and ***P<0.001. sh, short hairpin RNA.

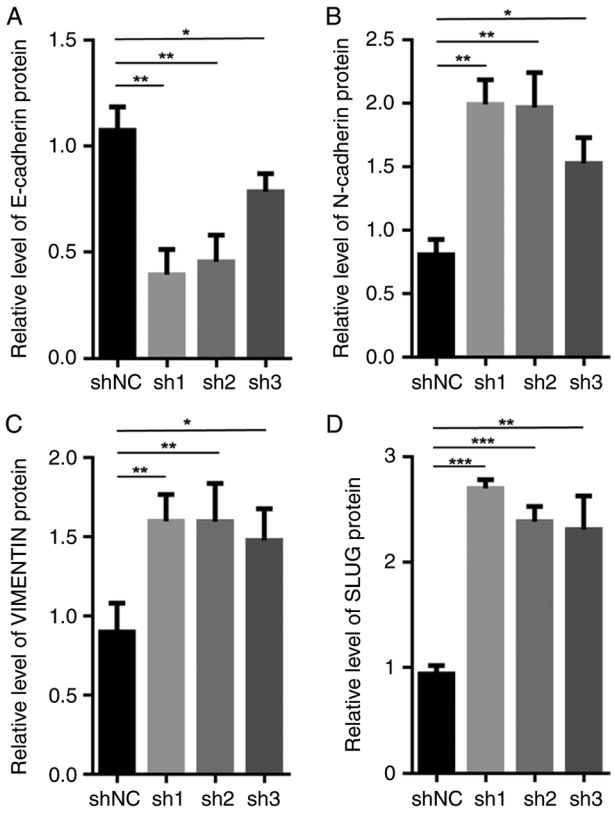


Figure S4. Evaluation of the correlation of *nr2e3* mRNA expression with the mRNA levels of (A) *thra*, (B) *esrra*, (C) *hnf4a* and (D) *ppara* in ER$^+$ breast adenoma samples. Data was provided by The Cancer Genome Atlas database. BC, breast cancer; ER, estrogen receptor; NR2E3, nuclear receptor subfamily 2 group E member 3; THRA, thyroid hormone receptor α; ESRRA, estrogen related receptor α; HNF4A, hepatocyte nuclear factor 4αß; PPARA, peroxisome proliferator activated receptor α.

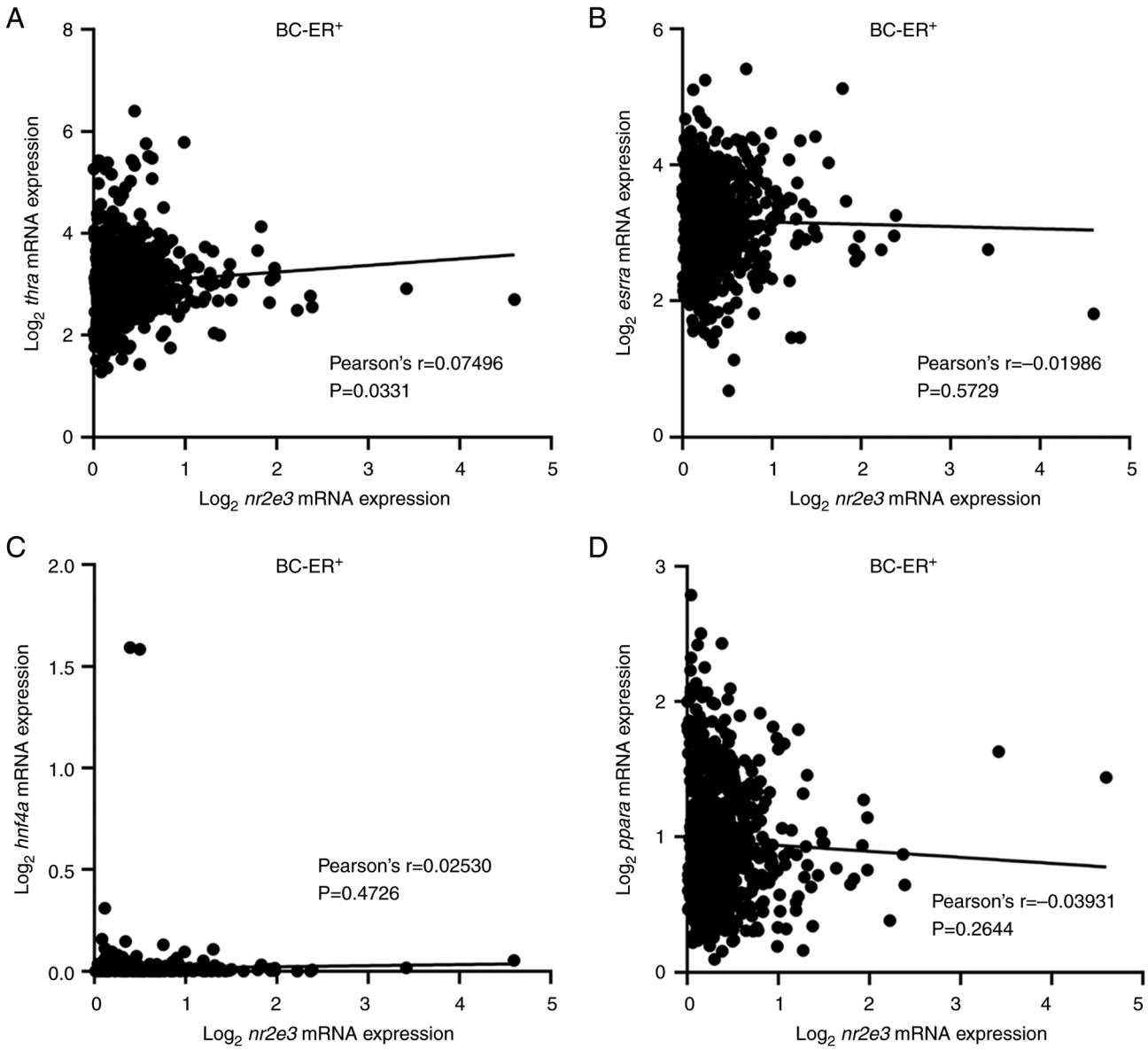
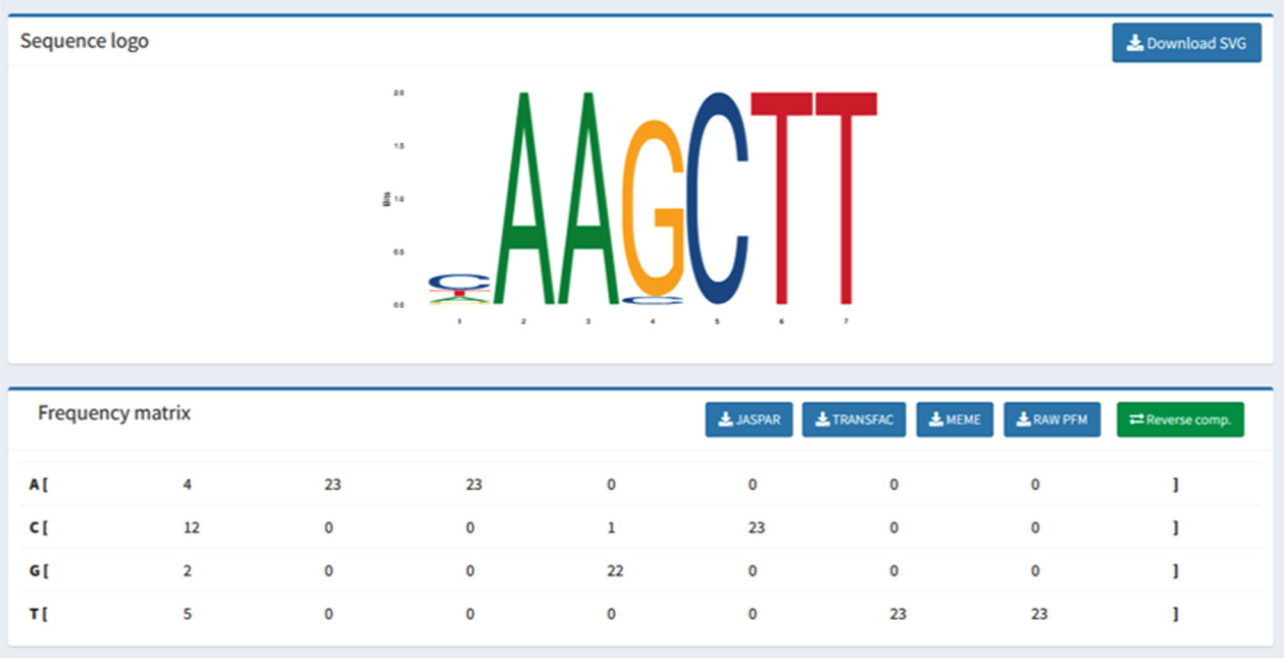


Figure S5. Predicted binding sites of nuclear receptor subfamily 2 group E member 3 on the proximal promoter of the nuclear receptor subfamily 2 group C member 2 gene.



A[	4	23	23	0	0	0	0	]
C[	12	0	0	1	23	0	0	]
G[	2	0	0	22	0	0	0	]
T[	5	0	0	0	0	23	23	]

Figure S6. Protein expression of *nr2c2* was detected in parental and *nr2c2* over-expressed MCF7 cells using a western blotting experiment. GAPDH served as the endogenous control. Untransfected, parental MCF7 cells. NR2C2, nuclear receptor subfamily 2 group C member 2.