

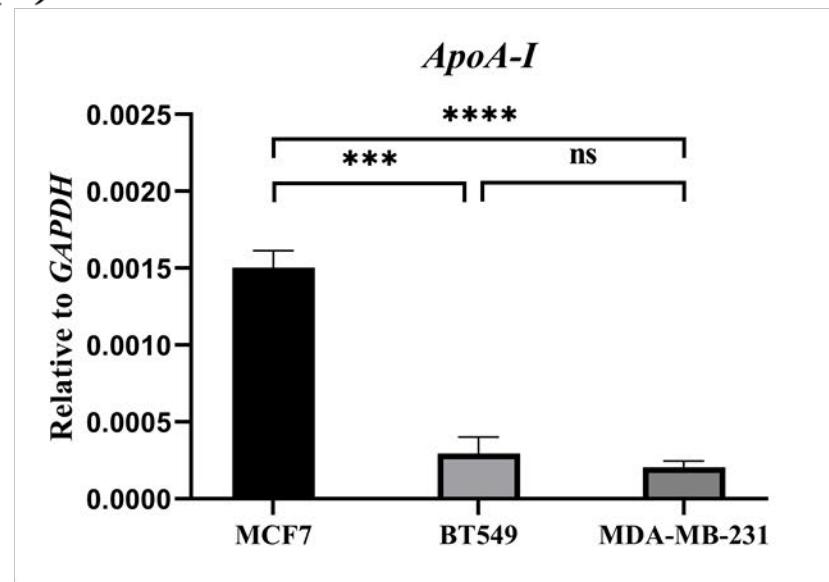
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# ER $\alpha$ promotes transcription of tumor suppressor gene *ApoA-I* by establishing H3K27ac-enriched chromatin microenvironment in breast cancer cells

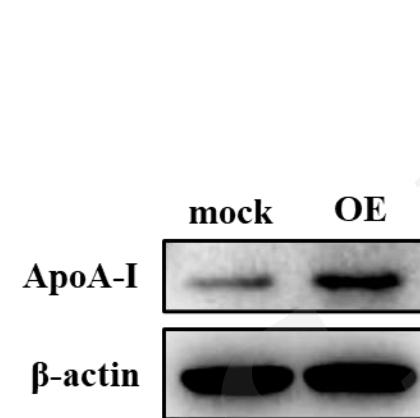
**Key words:** ApoA-I; ER $\alpha$ ; H3K27ac; p300; breast cancer

**Fig. 1 ApoA-I inhibits the proliferation and migration of MDA-MB-231 cells**

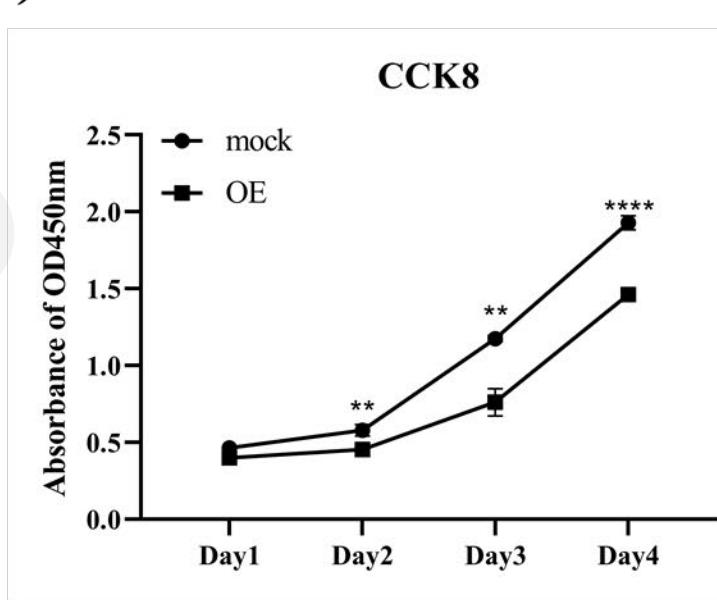
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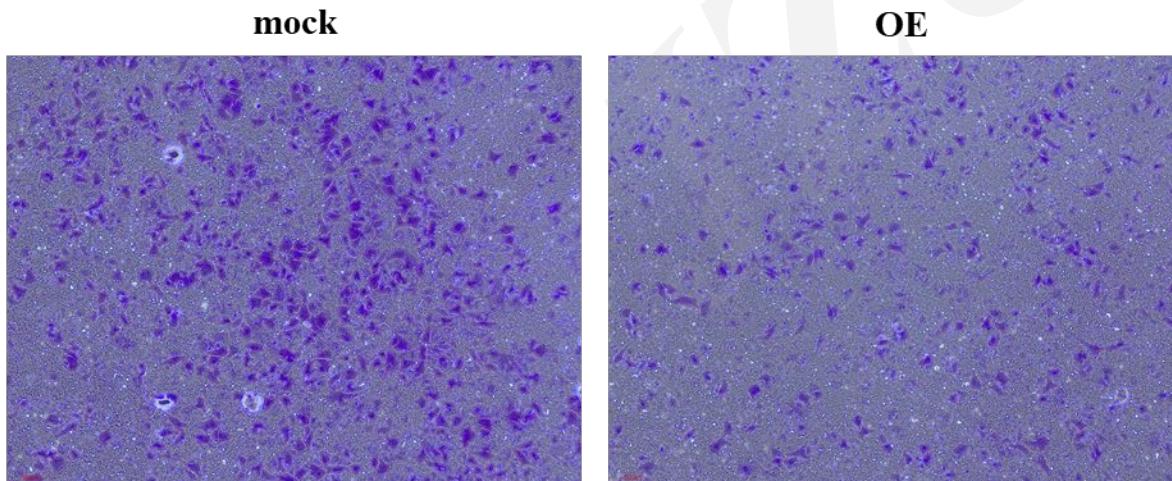
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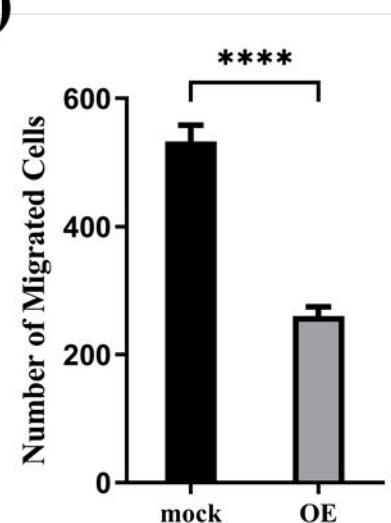
**(c)**



**(d)**

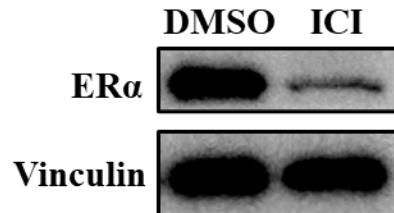


**(e)**

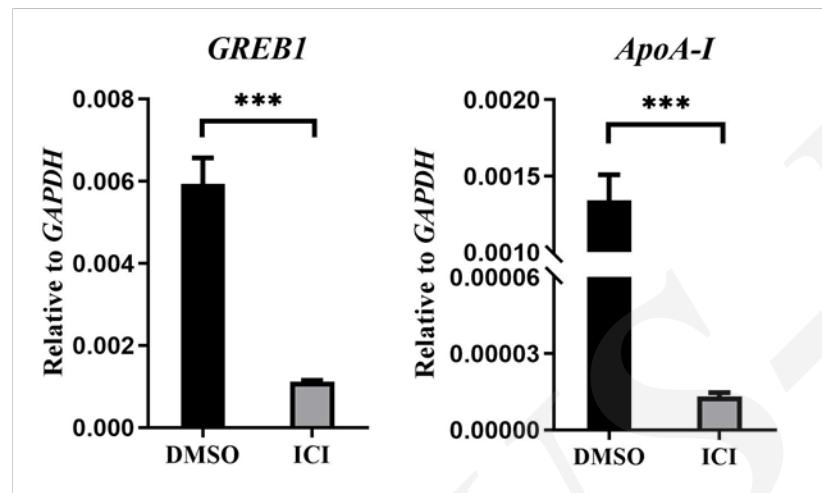


**Fig. 2 E2/ER $\alpha$  signaling induces *ApoA-I* transcription in breast cancer cells.**

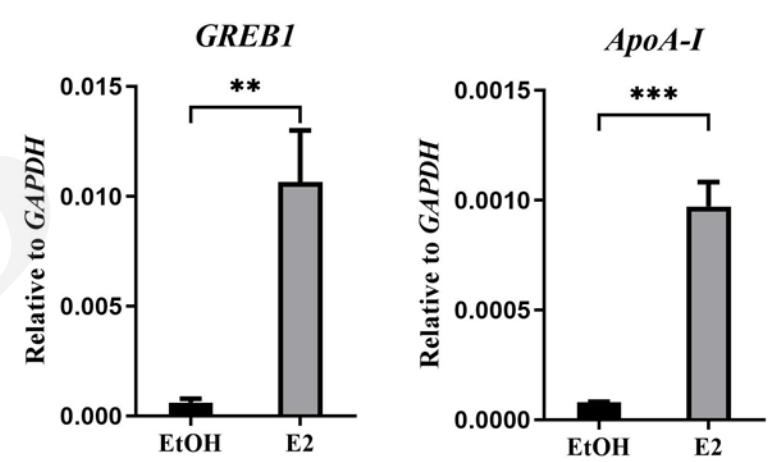
**(a)**



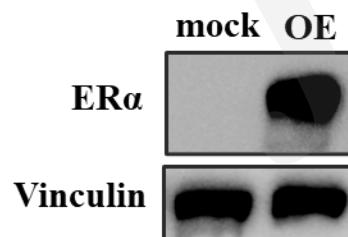
**(b)**



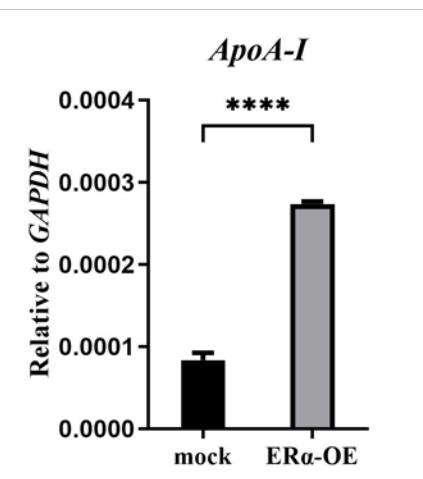
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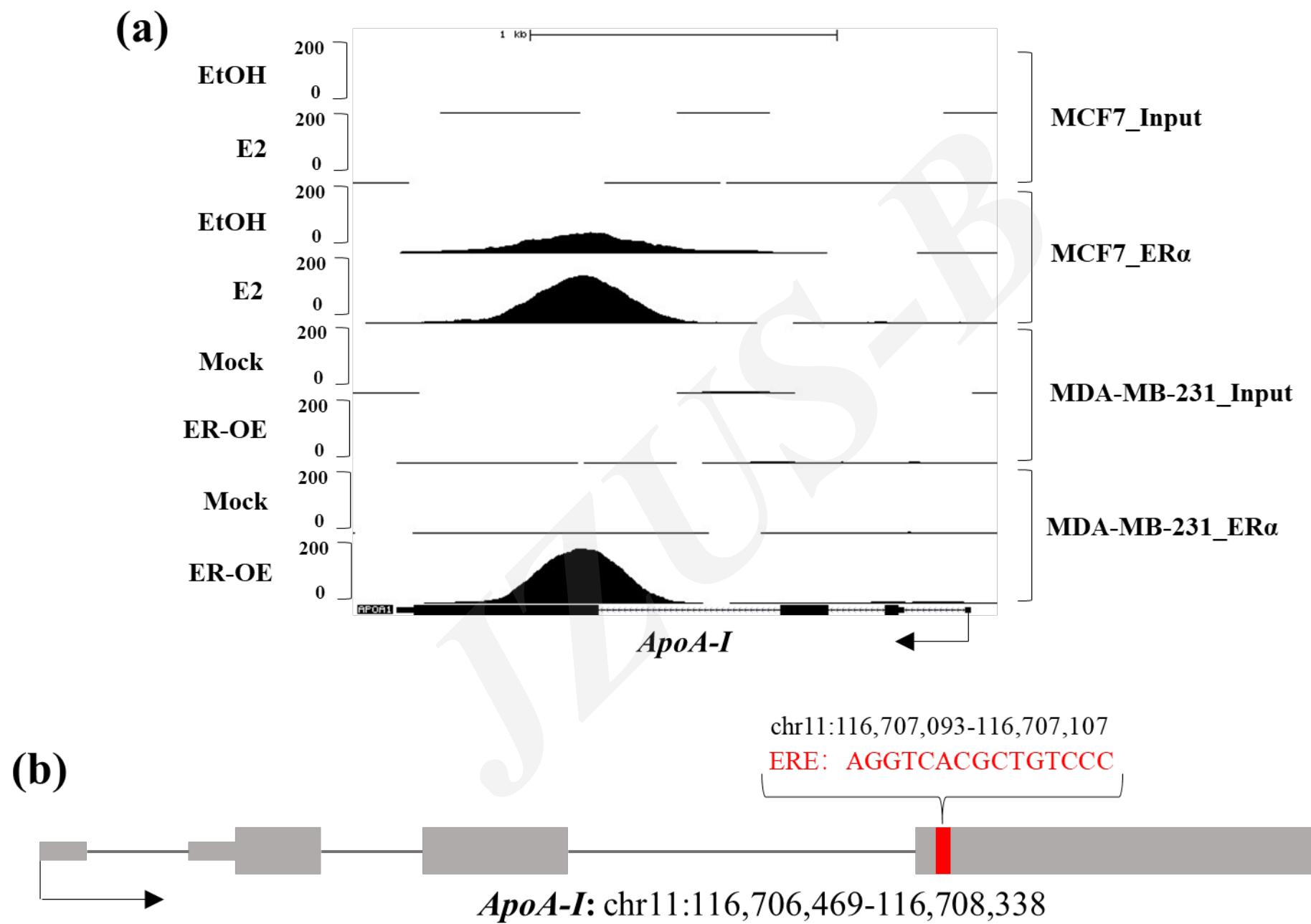
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**(e)**

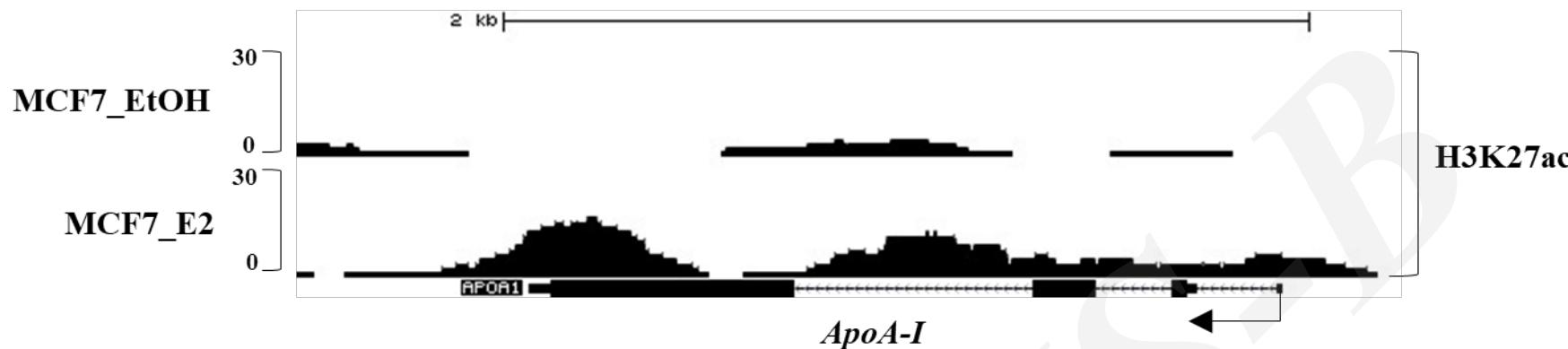


**Fig. 3 ER $\alpha$  directly binds to last exon of ApoA-I gene**

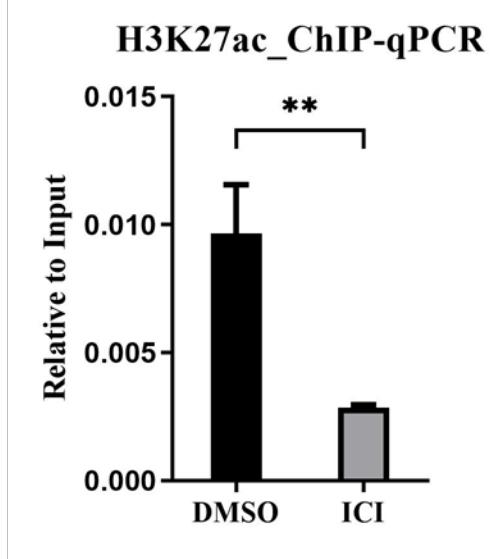


**Fig. 4 p300-mediated H3K27 acetylation is involved in the transcriptional regulation of *ApoA-I* by ER $\alpha$**

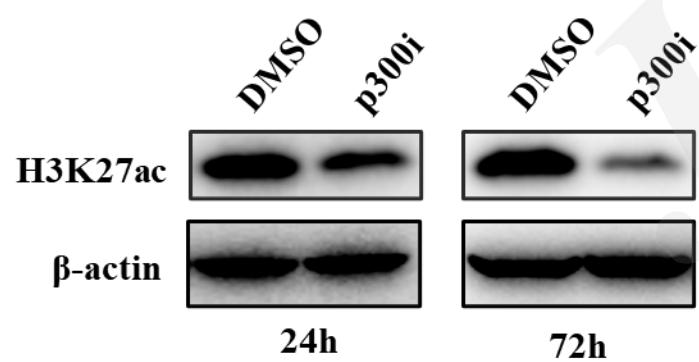
**(a)**



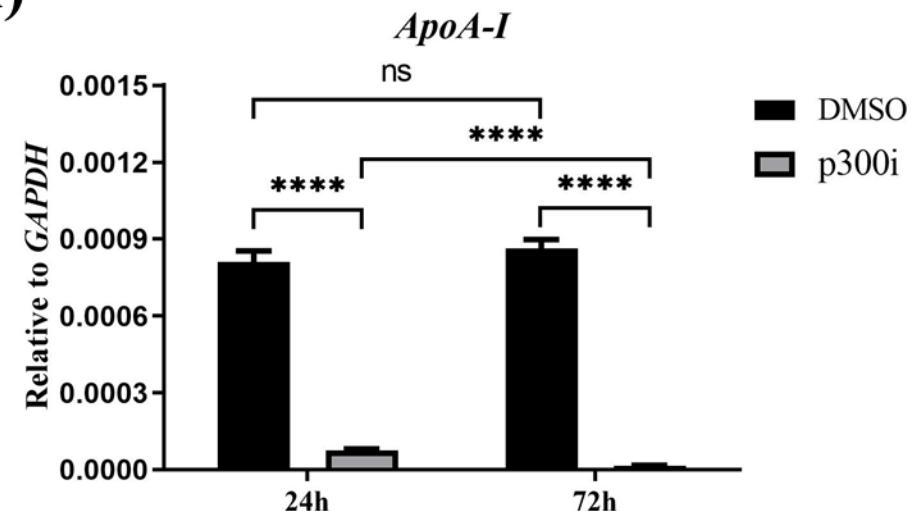
**(b)**



**(c)**



**(d)**



**Fig. 5** The mRNA expression levels of ER $\alpha$  and ApoA-I show a positive correlation in breast cancer

