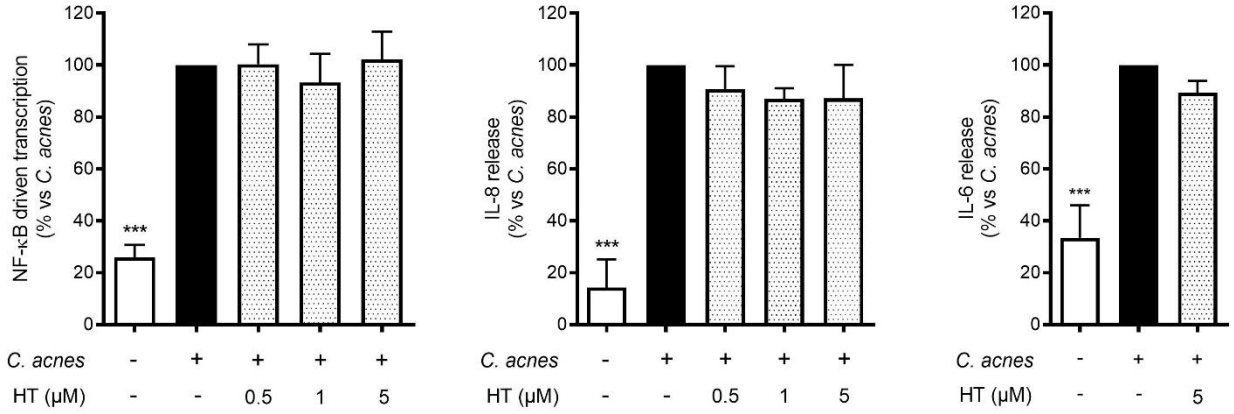
Supplementary material



(a) (b) (c)

Figure S1. Effect of HT treatment on the NF-κB-driven transcription (0.5-5 μM) (a) for 6 h, IL-8 release (0.5-5 μM) (b), and IL-6 release (5 μM) (c) in HaCaT cells infected with *C. acnes* (O.D.=0.1) for 24 h. Apigenin (20 μM), representing the reference inhibitor, abrogated the NF-κB-driven transcription (-100%) and inhibited the release of IL-8 (-42%) and IL-6 (-97%). The amount of IL-8 in the stimulated condition was 426.41 ± 96.97 pg/mL. *** $p < 0.001$ vs. stimulus.

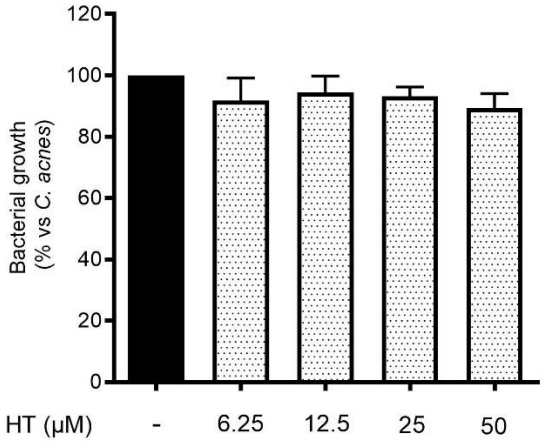


Figure S2. Effect of HT treatment (6.25-50 μM) on the growth of *C. acnes* (O.D.=0.1) for 24 h. Erythromycin (0.2 μg/mL) was used as antibiotic control (-80% of growth vs. control).

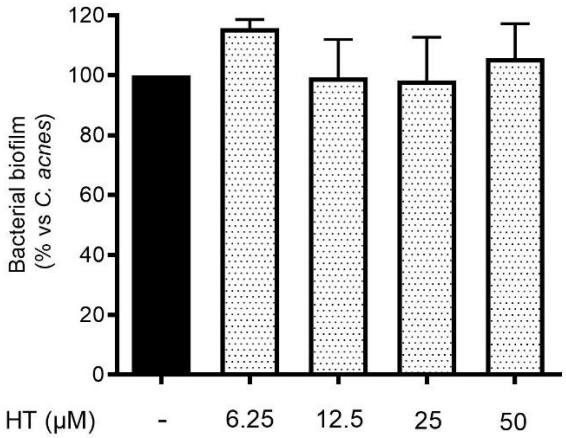


Figure S3. Effect of HT treatment (6.25-50 μM) on the biofilm formation of *C. acnes* (O.D.=0.1) for 24 h. Erythromycin (0.2 μg/mL) was used as antibiotic control (-90% of growth vs. control).

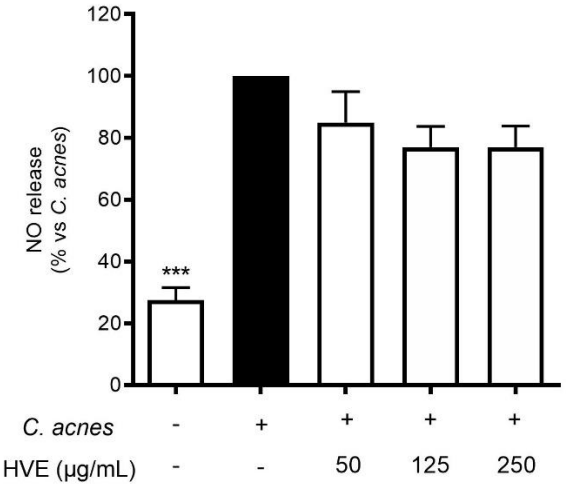
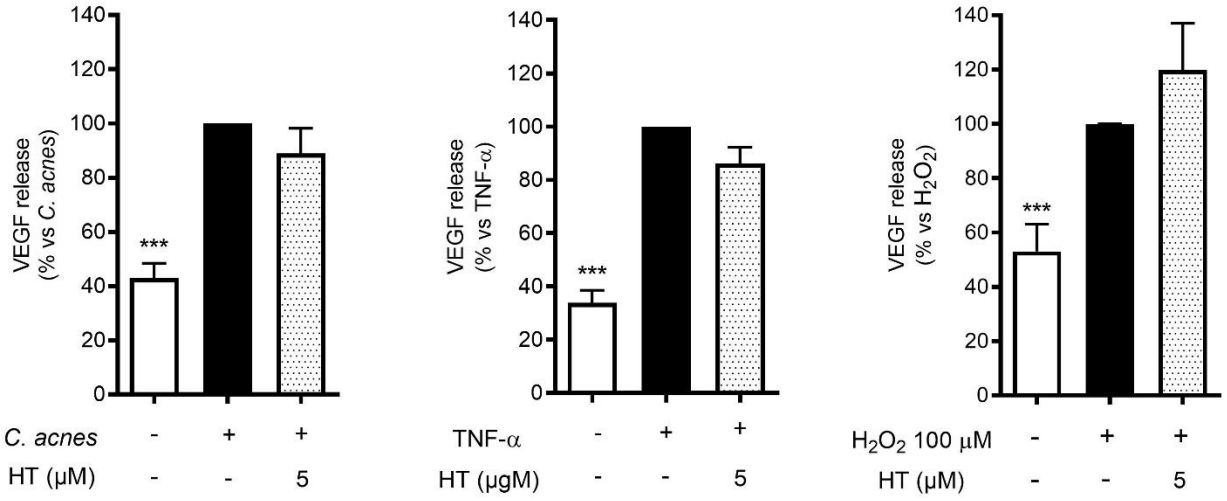


Figure S4. Effect of HVE treatment (50-250 μg/mL) on the NO release (b) in HaCaT cells infected with *C. acnes* (O.D.=0.1) for 24 h. EGCG (40 μM), representing the reference inhibitor, inhibited the release of NO (-57%). *** $p < 0.001$ vs. stimulus.



(a) (b) (c)

Figure S5. Effect of HT treatment (5 μM) on VEGF release induced by *C. acnes* (a), TNF-α (10 ng/mL) (b), or H_2O_2 (100 μM) in HaCaT cells after 24 h. Apigenin (20 μM), representing the reference inhibitor, inhibited VEGF release induced by H_2O_2 (-57%), TNF-α (-43%) and *C. acnes* (-33%). The amount of VEGF in the presence of TNF-α or *C. acnes* were 407,48 ± 3.98 pg/mL, and 47,29 ± 2,27 pg/mL in the presence of H2O2. *** $p < 0.001$ vs. stimulus.