

Table S1. Clinical isolates of *Candida* spp. used in this study.

<i>Candida</i> spp.	Source ^a	Strain ^b
<i>C. albicans</i>	ATCC MYA-2876	SC5314
<i>C. albicans</i>	ATCC90028	YLO12
<i>C. albicans</i>	HIV patient	YH050001
<i>C. albicans</i>	HIV patient	YH050005
<i>C. albicans</i>	HIV patient	YH050072
<i>C. glabrata</i>	ATCC9003	YLO8
<i>C. glabrata</i>	HIV patient	YH050105
<i>C. krusei</i>	ATCC6258	YLO6
<i>C. krusei</i>	HIV patient	YH050075
<i>C. tropicalis</i>	ATCC13803	YLO86
<i>C. tropicalis</i>	HIV patient	YH050007
<i>C. tropicalis</i>	HIV patient	YH050013
<i>C. tropicalis</i>	HIV patient	YH050114
<i>C. parapsilosis</i>	ATCC22019	YLO7
<i>C. dubliniensis</i>	HIV patient	YH050092

^a HIV patient, *Candida* strains isolated from HIV-infected patients.

^b These strains were provided by Hsiu-Jung Lo (Yu et al., *Antimicrob Agents Chemother.* 2011, 55:4918-4921).

Table S2. Minimal fungicidal concentrations (MFCs) of hep 25 against *C. albicans* and non-*albicans Candida* clinical isolates.

<i>Candida spp.</i>	Source	Strain	MFCs ($\mu\text{g/ml}$) ^a
<i>C. albicans</i>	ATCC MYA-2876	SC5314	100
<i>C. albicans</i>	ATCC90028	YLO12	>50
<i>C. albicans</i>	HIV patient	YH050001	>50
<i>C. albicans</i>	HIV patient	YH050005	>50
<i>C. albicans</i>	HIV patient	YH050072	>50
<i>C. glabrata</i>	ATCC9003	YLO8	50
<i>C. glabrata</i>	HIV patient	YH050105	50
<i>C. krusei</i>	ATCC6258	YLO6	25
<i>C. krusei</i>	HIV patient	YH050075	25
<i>C. tropicalis</i>	ATCC13803	YLO86	25
<i>C. tropicalis</i>	HIV patient	YH050007	25
<i>C. tropicalis</i>	HIV patient	YH050013	25
<i>C. tropicalis</i>	HIV patient	YH050114	50
<i>C. parapsilosis</i>	ATCC22019	YLO7	25
<i>C. dubliniensis</i>	HIV patient	YH050092	50

^aThe MFCs were defined as the lowest drug concentration that killed $\geq 99.9\%$ of cells and determined according to Basso et al. (Antimicrob Agents Chemother. 2018, 62(6)) with some modifications.

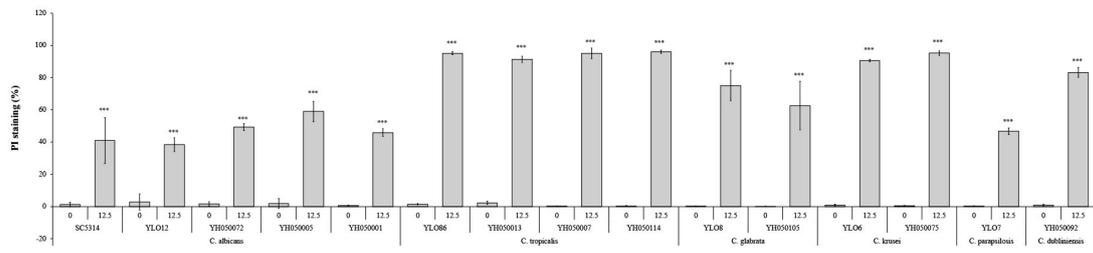


Figure S1. The activity of hep 25 against various clinical isolates of *C. albicans* and non-*albicans* *Candida* spp. The cells killed by hep 25 (PI-positive cells) were quantified by flow cytometry and normalized to the number of control cells (without hep 25 treatment) and reported as a percentage. 0: cells without hep 25 treatment, 12.5: cells with 12.5 µg/ml hep 25 treatment. The results are presented as the mean ± standard deviation of three independent experiments; *** P < 0.001.

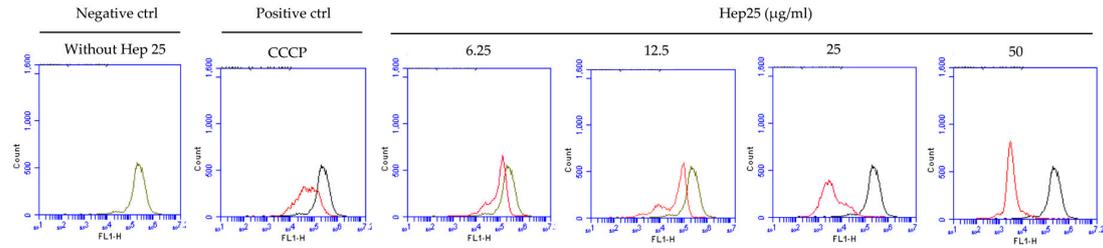


Figure S2. Mitochondrial depolarization detected with DiOC6(3). Cells treated with various concentrations of hep 25 or with 50 μ M of CCCP (red line) had decreased MMP fluorescence intensity compared to that of the untreated cells (black line). The results shown are from one of the three independent experiments.

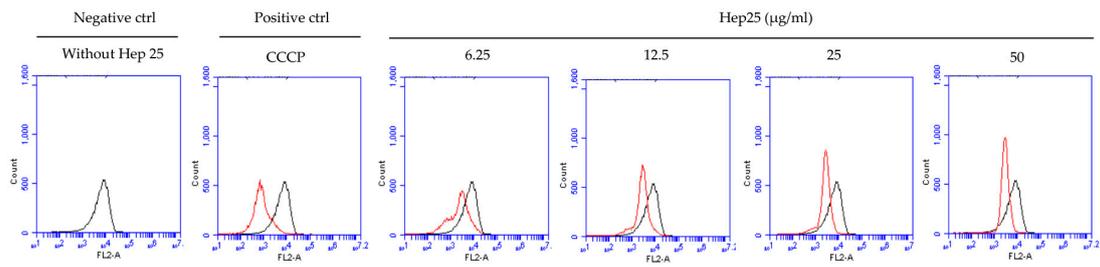


Figure S3. Mitochondrial depolarization detected by TMRM. Cells treated with various concentrations of hep 25 or with 50 μ M CCCP (red line) had a decreased MMP fluorescence intensity compared to that of the untreated cells (black line). The results shown are from one of the three independent experiment.

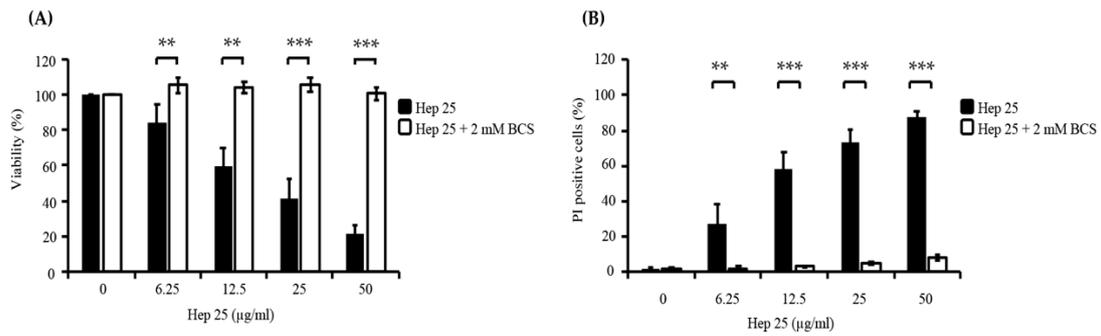


Figure S4. The effect of 2mM BCS on the candidacidal activity of hep 25. (A) Killing of *C. albicans* cells by hep 25 cotreated with or without 2 mM BCS was determined by the number of CFUs and expressed as the percentage of viable cells; **** P < 0.01 and *** P < 0.001.** (B) The killing activity of hep 25 with or without 2 mM BCS was detected by PI staining. The cells killed by hep 25 with or without 2 mM BCS were quantified by flow cytometry and reported as a percentage. The results are presented as the mean \pm standard deviation of three independent experiments; **** P < 0.01 and *** P < 0.001.**