**Objectives:** Echinocandins are indicated as first-line therapy for *Candidozyma auris* (*C. auris*) infections. Nevertheless, an ever-expanding number of breakthrough infections, associated with *FKS1* mutants, have been reported, rendering accurate antifungal susceptibility testing (AFST) crucial to guide therapy. Sensititre YeastOne (SYO) is a widely used commercial colorimetric assay for the AFST of yeasts, owing to its ready-to-use nature and its high concordance with the CLSI broth microdilution methodology. However, it has not yet been validated for echinocandin AFST of *C. auris* isolates with *FKS1* mutations. We therefore evaluated the SYO performance for *C. auris* AFST compared to the CLSI method and its ability to correctly identify *FKS1* mutants.

**Methods:** A total of 115 genetically distinct *C. auris* clinical isolates (62 clade I, 3 clade II, 23 clade III, 22 clade IV and 5 clade V), including a set of 25 clade I isolates harbouring *FKS1* mutations (S639F/P/T/Y, M690V, ΔF635), were tested. The CLSI and SYO AFST was performed according to the M27A4 protocol guidelines and the manufacturer’s recommendations using the YO10 panel, respectively. The SYO MICs of isolates with and without *FKS1* mutations were compared. The categorical agreement (CA) together with major errors (MaEs) and very major errors (VmEs) between CLSI and SYO were determined using the CLSI epidemiological cut-off values (ECVs) 1, 0.5 and 0.5 mg/L and the CDC tentative resistance breakpoints 4, 4 and 2 mg/L for anidulafungin, micafungin and caspofungin, respectively.

**Results:** The echinocandins CLSI and SYO MIC distributions are shown in **Figure**. The CLSI-SYO agreement (±2 twofold dilutions) was very good for anidulafungin (90%) and micafungin (84%), but moderate for caspofungin (67%). Compared to CLSI, the modal MICs with SYO were 1 and 2 dilutions higher for anidulafungin and micafungin, respectively, but 2 dilutions lower for caspofungin. Although *FKS1* mutants had significantly higher anidulafungin/micafungin SYO MICs than isolates without *FKS1* mutations (0.5->8 versus ≤0.25 mg/L), 24/90 (27%) isolates had similar caspofungin SYO MICs (0.5->8 mg/L). Based on the CLSI ECVs, 8/25 (32%), 7/25 (28%) and 3/25 (12%) of *FKS1* mutants were WT to anidulafungin, micafungin and caspofungin, respectively. Adopting a SYO-specific ECV of 0.25 mg/L, all *FKS1* mutants were non-WT to anidulafungin and micafungin, but not to caspofungin with SYO. The CLSI-SYO CA using the CLSI ECVs was 95% (5% MaEs) for anidulafungin, 96% (3% MaEs, 1% VmEs) for micafungin and 86% (9% MaEs, 5% VmEs) for caspofungin. The CLSI-SYO CA using the CDC breakpoints was 96% (4% MaEs) for anidulafungin, 97% (3% MaEs) for micafungin and 81% (18% MaEs, 1% VmEs) for caspofungin.

**Conclusions:** Anidulafungin and micafungin SYO MICs misclassified a significant number of *C. auris* *FKS1* mutants as WT based on the CLSI ECVs.Discrepancies were reduced when a SYO-specific anidulafungin/micafungin ECV of 0.25 mg/L was used. Moreover, SYOcouldaccurately exclude/predict anidulafungin and micafungin resistance in *C. auris*, based on the CDC breakpoints. On the contrary, caspofungin SYO MICs should be interpreted with caution as a marker of *FKS1* non-WT phenotype or echinocandin resistance in *C. auris*.

