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In this review, we briefly summarize the current understanding of how fungal pathogens can persist antifungal treatment without heritable resistance mutations by forming tolerant persister cells. Fungal infections tolerant to antifungal treatment have become a major medical problem. One mechanism leading to drug recalcitrance is the formation of antifungal persister cells. These cells have wild-type genotype with the ability to survive exposure to antifungal agents due to changed membrane composition, upregulated stress response, and enhanced cell wall integrity. Knowledge of the mechanisms regulating entry and exit of the persister phenotype is limited, but it has recently been shown that the inhibition of the growth regulating TORC1 pathway induces fungal persistence. The phenotypic properties of persister cells and the involvement of the TORC1 pathway indicate that persister cells are quiescent in G0 of the cell cycle. This knowledge leads us to suggest that the identified shared drug-tolerance mechanisms of persister and quiescent cells may serve as a foundation for developing novel treatment strategies that are independent of growth mode against systemic fungal infections.