Supplementary References

- They are responsible for a broad spectrum of microbial infections in the human host [1]
- *Candida albicans* and *Candida parapsilosis* biofilms are relatively resistant to fluconazole, amphotericin B, nystatin, voriconazole and others [2].
- *Aspergillus fumigatus* biofilms are relatively resistant to itraconazole and, to some extent, to caspofungin [3].
- Cryptococcal biofilms are unaffected by fluconazole and voriconazole [4] and biofilms of *Trichosporon asahii* display elevated resistance to amphotericin B, caspofungin, voriconazole, and fluconazole [5].
- Azole and amphotericin B therapies are ineffective against *Pneumocystis carinii* biofilms [6].
- Biofilm-associated resistance mechanisms have been characterized in *C. albicans* and *A. fumigatus* and include drug binding by ECM [7] and production of persister cells [8, 9].
- *C. albicans* transcription factor Efg1, a global regulator of cell surface protein genes and hyphal formation [10] is required for biofilm formation as well [11, 12].
- *C. albicans* cell surface proteins have been reviewed authoritatively [13].
- Increases in ERG gene expression as well as multi-drug resistance transporters has been correlated with increased azole resistance in *Candida albicans* patient isolate samples [14], though their contribution to biofilm-specific azole resistance has not been detected in mature biofilms [15].