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Commentary Article

Candida Auris Can Now be Immediately Life Threatening. A Commentary on: Fluoxetine is Antimicrobial and Modulates the Resistance Status of Bacteria

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In her 2023 article published in the Journal of Cellular Immunology, Alison Mackay provides a comprehensive summary of the initial reports of penicillin-resistant *S. pyogenes* (1947) and a decade of subsequent infections within a single hospital [1]. The article underlines the significance of toll-like receptors in the identification and elimination of pathogens through the release of cytokines, interferons, and antimicrobial peptides. Additionally, it includes a definition of minimum inhibitory concentration (MIC) and directs readers to the websites of the European Committee on Antimicrobial Susceptibility Testing (EUCAST) and the World Health Organization (WHO), offering access to current MICs of specific microbe/drug combinations and pathogen risk statuses. Furthermore, the article elucidates the mechanisms by which adjunctive Fluoxetine treatment in humans inhibits cellular innate efflux pumps, thereby maintaining higher intracellular concentrations of antibiotics for extended periods.

The article concluded that 0.26 mg/nl of fluoxetine, in conjunction with sub-inhibitory levels of antibiotics, effectively controlled *E. coli*, *P. aeruginosa*, and *S. aureus*. The objective being that smaller doses of antibiotics may slow down, halt, or even reverse resistance. Furthermore, fluoxetine alone proved effective in treating *P. mirabilis* in catheter-associated urinary tract infections (CAUTI), preventing the subsequent development of biofilms, which are notoriously difficult to treat. However, in a laboratory study, similar doses of fluoxetine did not inhibit the growth of *A. baumannii*; in fact, it's resistance to treatment is accelerated.

It is known that carbapenem resistance among *A. baumannii*, *P. aeruginosa*, and enterobacteria were identified as critical priorities by the WHO, and the reduced effectiveness of vancomycin and methicillin in *S. aureus* was a high priority [2]. This remains the case, although it is important to note the inclusion of five other pathogens on the 'high priority' list: *E. faecium, H. pylori, N. gonorrhoeae, Campylobacter* sp., and *Salmonella* sp. Last year's WHO AMR surveillance report [3] documented improvements in treatment susceptibility for *S. aureus*, *N. gonorrhoeae*, and *S. pneumoniae*. However, increased resistance to treatment has been observed in *Salmonella* spp. (ciprofloxacin) and four additional pathogens: *Shigella* spp. (ciprofloxacin), *K. pneumoniae* (imipenem), *E. coli* (cephalosporins), and *Acinetobacter* spp. (imipenem).

Table 1: WHO Fungal treatment priorities.

Fungi			
Critical Priority	**Clinical Manifestations**		
c.neoformans	Meningitis	Pulmonary infection	
c.auris	Sepsis	Wound Infection	Ear Infection
	Urinary Tract Infection		
c.albicans	Genital Infection	Skin and Nail Infection	
	Urinary Tract Infection		
a.fumigatus	Pulmonary Infection		
	Allergic broncopulmonary aspergillosis		
High Priority			
c. glabrata	Urinary Tract Infection		Genital Infection
histoplasma spp	Respiratory Infection	Acute Respiratory Distress Syndrome	
	Mucarales	Murcomycosis	
fusarium spp	Keratitis	Onychomycosis	Mycotoxicosis
c.tropicalis	Vulvovaginitis	Oral thrush	Pneumonia
	Renal Infection		
c. parapsilosis,	Blood Infection	Organ Infection	Biofilms
--	Eumycetoma		

The article under consideration [1] did not extend its discussion to microbes other than bacteria. Nevertheless, the author recently published a review indicating that fungal urinary tract infections (UTIs) are less detectable than bacterial UTIs using the commonly employed urine dipstick [4]. Furthermore, *C. auris* is increasingly resistant to treatment resulting in a significant risk. Similar to Fluoxetine, Fungi can modulate treatment resistance in bacteria. In combination with the challenges in detecting and treating fungi, this can increase the risk presented by bacteria in some cases [5].

Among the ten established criteria for prioritizing pathogens, the World Health Organization (WHO) assigns the greatest importance to their resistance to treatment [6]. Table 1 delineates the critical and high-priority fungi, along with the diseases each is capable of causing. Even among the critical priorities, *C. Auris* stands out as a cause of immediately life-threatening sepsis. This may reflect the effects of *candida* spp. on bacterial virulence, viability and susceptibility to antibiotics as the author has described [5].

A lab study demonstrated that fluoxetine was able to kill fluconazole-resistant *candida* spp. with a MIC of 20-160 µg/nl after 24 hours by damaging its plasma and mitochondrial membranes and triggering apoptosis [7]. The antidepressant was also able to reduce biofilms of both fluconazole-susceptible and resistant isolates by the same mechanism. [idem]. When used in combination with a series of 'azoles', fluoxetine significantly increased the susceptibility of *candida* spp to azoles including during the first 24-hours of biofilm formation [8]. These findings could be important in the future treatment of fungal infection and diagnostically via the consideration of incidental polypharmacy [9].

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