



EVALUATION OF BACTERIAL COINFECTION IN PATIENTS WITH OTOMYCOSIS IN A TERTIARY CARE CENTRE

Dr.Naveen Kumar B^{1*}, Dr.Gayathri Devi R², Dr.Dianitta DevapriyaVeronica³, Dr.Santhosh⁴, Dr. Pavithra R⁵

^{1*}Assistant Professor, Department of ENT, ACS Medical College and Hospital, Dr.MGR Educational and Research Institute (Deemed to be university)

²Assistant Professor, Department of ENT, ACS Medical College and Hospital, Dr.MGR Educational and Research Institute (Deemed to be university), gayathri.r1994@gmail.com

³Associate Professor, Department of ENT, ACS Medical College and Hospital, Dr.MGR Educational and Research Institute (Deemed to be university)

⁴Post Graduate, Department of ENT, ACS Medical College and Hospital, Dr.MGR Educational and Research Institute (Deemed to be university)

⁵Assistant Professor, Department of Pathology, ACS Medical College and Hospital, Dr.MGR Educational and Research Institute (Deemed to be university)

Abstract

Background: Otomycosis is a common external ear infection frequently seen in tropical regions. While fungi are the primary pathogens, bacterial coinfections are increasingly recognized, complicating treatment outcomes.

Objectives: To identify the predominant fungal and bacterial species in otomycosis and to evaluate the coexistence of fungal and bacterial pathogens.

Methods: This prospective study included 130 clinically and microbiologically confirmed cases of otomycosis (aged 18–65 years) attending the ENT outpatient department of a tertiary care hospital in Chennai over six months. Fungal and bacterial isolates were identified using standard microbiological techniques.

Results: The majority of patients were aged 26–35 years (30.77%), with a slight female predominance (55.38%). *Aspergillus niger* was the most common fungus isolated (64.62%), followed by *Candida* spp. (21.54%). Bacterial growth was observed in 94.61% of samples, with Methicillin-Resistant *Staphylococcus aureus* (MRSA) (38.46%) and *Pseudomonas aeruginosa* (20%) being predominant. Notably, *A. niger* commonly coexisted with MRSA (23.81%) and *Pseudomonas aeruginosa* (22.62%), while *Candida* spp. were strongly associated with MRSA (71.43%).

Conclusion: The high prevalence of bacterial coinfection in otomycosis, particularly with resistant organisms such as MRSA, highlights the need for combined antifungal and antibacterial therapy. Accurate microbiological diagnosis is essential to guide effective treatment and reduce recurrence.

INTRODUCTION

One of the most prevalent disorders seen in a general otolaryngology clinic is otomycosis, which has been reported to affect around 9%^[1] and 27.2%^[2,3] of patients with otitis externa symptoms and up to 30%^[4,5,6] of patients with discharging ears. The worldwide prevalence statistics of otomycosis indicate that around 9%-30% patients with the signs and symptoms of EAC infection have otomycosis^[7,8,9]

In tropical and subtropical areas with high temperatures and humidity, otomycosis is extremely

prevalent^[10]. Aquatic sports, including swimming and surfing, are particularly associated with otomycosis because of repeated exposure to water resulting in removal of cerumen and drying of the external auditory canal^[16]. Otomycosis is predominantly unilateral and seen across all the age groups, but most of the cases of otomycosis occur in patients aged 21 -30 years with equal male – female distribution^[17,18]

Patients with otomycosis commonly present with complaints of ear pain, ear itching, ear discharge, foreign body sensation in the ear canal ^[12]. In some cases, hypoacusis, tinnitus and/or hearing impairment may also be reported^[13,14]. Sometimes, the fungus could be a secondary invader in instances of otitis externa. Consequently, otomycosis can occur in conjunction with mixed fungal and bacterial infections^[15].

In otomycosis, the squamous epithelium of the external ear canal is usually affected, and the causative fungi most often reside in the medial aspect of the ear canal. This is partially because the inferior tympanic recess allows debris to accumulate there, and partially because this part of the ear canal is warmer and darker than the others, which promotes the growth of fungi^[11]. Despite the ongoing debate on whether fungi are the actual infectious agents in otomycosis or simply colonizers due to weakened local host immunity caused by bacterial infections, the majority of clinical and laboratory data indicates that otomycosis is indeed a legitimate pathological condition^[13]

A.niger or *A. flavus* complex in the genus *Aspergillus* spp., and *Candida albicans*, *Candida parapsilosis* in the genus *Candida* spp. have been reported to be common causative agents of otomycosis^[19,20]. *Aspergillus niger* spores are visualized as fine coal dust that has been scattered inside the ear canal. They might also look like a crumpled newspaper or blotting paper. Conversely, external ear *Candida* infections are characterized by a white, cheesy substance that resembles sebaceous material and, in extreme situations, may even fill the ear canal. In these situations, the ear canal is frequently lined by a pseudomembrane, which, when removed, shows a granular and friable membrane underneath^[21].

Clearly, early diagnosis and treatment of otomycosis depend heavily on identifying the kind of opportunistic fungal infection and whether there is associated bacterial involvement. Currently, the primary clinical methods for identifying opportunistic fungi are isolation, culture, and microscopic analysis based on bacterial and fungal morphology^[22].

Treatment options for otomycosis include local debridement, such as microaspiration, topical or systemic antimicrobial and antifungal medications, and management of underlying predisposing factors and causative pathogen termination^[23].

Materials and Methods

Study Design: Prospective observational study.

Study Setting: ENT outpatient department, ACS Medical College & Hospital, Chennai. Study

Duration: 6 months.

Sample Size: 130 patients.

Inclusion Criteria:

- Patients aged 18–65 years.
- Clinically suspected and microbiologically confirmed cases of otomycosis.

Exclusion Criteria:

- Tympanic membrane perforation.
- Prior ear surgery.
- Chronic systemic illnesses.
- Recent antifungal/antibacterial therapy.
- Associated dermatological conditions of the ear.
-

Data Collection: Clinical history and otoscopic examination were recorded. Otomycotic debris was collected aseptically prior to intervention. Fungal isolates were identified by direct microscopy and culture, while bacterial isolates were identified by gram staining, culture, and antibiotic sensitivity testing as per CLSI guidelines.

RESULTS:

In the study of 130 participants, it was found that n= 40 (30.77%) belonged to the age group of 26 to 35 years, n=34 (26.15%) belonged to the age group of 18 to 25 years and n= 27 (20.77%) belonged to the age group of 56 to 65 years, which contributed to majority of study population.

AGE RANGE	n(%)
18-25	34(26.15)
26-35	40(30.77)
36-45	19(14.62)
46-55	10(7.69)
56-65	27(20.77)
TOTAL	130(100)

Of the total 130 participants in study, n=72 (55.38%) were females and n= 58 (44.61%) were males, showing mild female preponderance. The mean age of female was 37.7 ± 14.03 years and mean age of male was 36.8 ± 14.32 years. The overall mean age of the study population was 37.33 ± 14.10 years.

SEX	n(%)
FEMALE	72(55.38)
MALE	58(44.61)
TOTAL	130(100)

Commonest type of fungi seen in this study were, *Aspergillus niger*, *Aspergillus flavus*, *Candida* species, *Rhizopus*. Out of 130 fungal cultures, n=84 (64.62%) showed *A.niger* which was the most commonest fungus seen in this study. Second most common fungus seen was *Canidida* which accounted to n=28 (21.54%).

FUNGUS	n(%)
<i>A.niger</i>	84(64.62)
<i>Candida</i>	28(21.54)
<i>A.flavus</i>	14(10.77)
<i>Rhizopus</i>	4(3.08)
TOTAL	130(100)

Fungal cultures across male and female from the study population, shown below.

FUNGUS	MALE	FEMALE	n(%)
A.niger	40	44	84(64.62)
Candida	15	13	28(21.54)
A.flavus	3	11	14(10.77)
Rhizopus	0	4	4(3.08)
TOTAL	58	72	130(100)

Out of 130 (100%) samples sent for bacterial cultures, n= 123 (94.61%) showed growth of bacteria from the otomycotic debris. The 123 bacterial cultures obtained were further classified according to their gram positive or negative nature, and gram positive bacteria n= 75 (57.69%) was found to be more than gram negative n=48 (36.92%).

Bacteria	n(%)
Gram -ve	75 (60.97)
Gram+ve	48(39.02)
total	123(100)

The commonest type of bacteria seen in this study was Methicillin Resistant Staphylococcus aureus (MRSA) n=50 (38.46%). Second commonest bacteria seen was Pseudomonas aeruginosa (Pa) n=26 (20.00%). n=7 samples (5.38%) had no bacterial growth.

BACTERIA	n(%)
Methicillin Resistant Staphylococcus aureus (MRSA)	50(38.46)
Pseudomonas aeruginosa (Pa)	26(20.00)
Coagulase-Negative Staphylococci (CoNS)	20(15.38)
Klebsiella pneumoniae (Kp)	16(12.30)
Escherichia coli (E.coli)	6(4.61)
Methicillin-Susceptible Staphylococcus aureus (MSSA)	3(2.31)
Actinobacteria	2(1.53)
No growth	7(5.38)
TOTAL	130(100)

Cultures obtained were classified gram positive or negative accordingly, depending upon the bacterial type.

Gram (-ve) bacteria	MALE	FEMALE	n(%)
<i>Pseudomonas aeruginosa</i> (Pa)	7	19	26 (54.17)
<i>Klebsiella pneumoniae</i> (Kp)	5	11	16(33.33)
<i>Escherichia coli</i> (E.coli)	4	2	6(12.50)
Total Gram (-ve) bacteria	16	32	48(100)

Gram (+ve) bacteria	MALE	FEMALE	n(%)
Methicillin Resistant <i>Staphylococcus aureus</i> (MRSA)	20	30	50(66.67)
Coagulase-Negative <i>Staphylococci</i> (CoNS)	7	13	20(26.67)
Methicillin-Susceptible <i>Staphylococcus aureus</i> (MSSA)	0	3	3(4.00)
Actinobacteria	2	0	2(2.67)
Total Gram (+ve) bacteria	29	46	75(100)

Most common gram positive bacteria seen was Methicillin Resistant *Staphylococcus aureus* (MRSA) n=50 (66.67%) out of 75 Gram positive bacteria. Most common gram negative bacteria seen was *Pseudomonas aeruginosa* (Pa) n=26 (54.17%) out of 48 Gram negative bacteria.

Fungal cultures also exhibiting bacterial growth were recorded and tabulated. Out of n=84 samples with *Aspergillus niger*, n=83 showed bacterial growth

Bacteria with <i>Aspergillus niger</i>	n(%)
Methicillin Resistant <i>Staphylococcus aureus</i> (MRSA)	20(23.81)
<i>Pseudomonas aeruginosa</i> (Pa)	19(22.62)
Coagulase-Negative <i>Staphylococci</i> (CoNS)	18(21.43)
<i>Klebsiella pneumoniae</i> (Kp)	13(15.48)
<i>Escherichia coli</i> (E.coli)	6(7.14)
Actinobacteria	3(3.57)
Methicillin-Susceptible <i>Staphylococcus aureus</i> (MSSA)	3(3.57)

No growth	1(1.19)
Total	84(100)

MRSA, n=50 (23.81%), *Pseudomonas aeruginosa* n=26 (22.62%), CoNS n=20 (21.43%) were found to be commonly present in otomycosis samples with *Aspergillus niger*. MRSA was found to be the most common bacteria co-existing with *Aspergillus niger*.

Out of n=28 samples with *Candida* growth, n= 27 showed bacterial growth.

Bacteria with Candida	n(%)
Methicillin Resistant <i>Staphylococcus aureus</i> (MRSA)	20(71.43)
<i>Pseudomonas aeruginosa</i> (Pa)	7(25.00)
No growth	1(3.57)
Total	28 (100)

MRSA, n=20 (71.43%) was the most common bacteria co-existing with *Candida*.

Aspergillus and *Candidal* species in otomycosis always showed coexistence with MRSA and Pa which accounts for about 66 out of 130 patients with otomycosis (50.76%)

DISCUSSION

In this study conducted in a tertiary care center involving n=130 patients, n=40 (30.77%) patients were found to be in the age group of 26 to 35 years. Otomycosis is commonly found to be affecting the early middle age group. This population group is known for frequent travel and sport activities. Tropical and sub-tropical regions experience climatic variations including hot summers and rainy weather. These conditions, along with coastal humidity, contribute to increased moisture in the external auditory canal, thereby raising the incidence of otomycosis [24].

This study showed an increased incidence of otomycosis in the female population. Practice of washing, drying and setting of hairs by women also increases the humidity in the external auditory canal encouraging otomycosis [25].

The study population was always presented with complaints of otalgia, ear itching, discharge, discomfort, ear fullness and/or ear blocking sensation [12]. Otomycotic debris was universally seen in all patients and the same was collected for microbiological examination before any intervention such as aural toileting or topical agents which also excluded the chances of contamination and no growth in culture.

Commonest fungi seen causing otomycosis was *Aspergillus niger* (64.62%), and second common fungi was *Candida* (21.54%). This correlates with other studies showing that *Aspergillus* species and *Candida* species are the commonest organisms causing otomycosis [26]. Otomycotic debris, a mixture of fungal spores, serous discharge, granulations, and purulent material always showed the presence of bacteria in culture [27].

The bacterial cultures classified by gram staining showed predominantly a gram positive group (60.97%). Commonest gram positive bacteria was MRSA and commonest gram negative bacteria was *Pseudomonas aeruginosa*. The bacteria seen along with otomycosis were usually found

to be skin commensals^[28]. The commonest bacteria seen was Methicillin Resistant *Staphylococcus aureus* (MRSA) 38.46% and the second most common bacteria seen was *Pseudomonas aeruginosa* (Pa) 26% followed by Coagulase-Negative *Staphylococci* (CoNS) 15.38%.

Mutualism, commensalism, parasitism are three main phases of fungal interaction with the host which decides fungal infectivity when right conditions are met^[29]. Numerous studies done previously postulate various mechanisms showing interactions between fungi and bacteria at molecular levels^[30].

Fungus coexisting with bacteria in otomycosis, which was well appreciated previously in various studies, never showed any species level correlation between specific fungus and specific bacteria^[31]. This study showed that *Aspergillus niger* always had a bacterial growth commonly with MRSA, *Pseudomonas aeruginosa*, Coagulase Negative *Staphylococci* (CoNS).

Aspergillus species, especially *Aspergillus niger* which has been studied extensively in various fields showing anti-microbial activity against various bacteria especially MRSA and *Pseudomonas aeruginosa*, owing to its biofilm disruption and competitive nutrient dependent properties^[32,33,34]. From this study it was seen that *Aspergillus niger* has been showing co-existence with MRSA and *Pseudomonas aeruginosa*, which leads to the fact that under different regional scenario in human body especially in external auditory canal *Aspergillus niger* plays a different mechanism aiding to a symbiotic relationship with *staphylococcus* species especially MRSA and *Pseudomonas aeruginosa*.

This study also showed that *Candida* species always had a bacterial growth commonly with MRSA and *Pseudomonas aeruginosa*. *Candida* increases the extracellular pH, which stimulates the production of a major cytotoxic agent (alpha toxin) by *Staphylococcus aureus*. Moreover, the *Candida* increases the production of efflux pumps in *Staphylococcus aureus*, increasing the resistance of bacteria to antimicrobial agents and hinders wound healing. This infectious synergism is dependent on the expression of staphylococcal alpha-toxin, and secretion of this potent virulence factor is actually augmented by *Candida albicans*^[35]. High prevalence of MRSA is a matter of concern especially considering its resistance to beta lactam antibiotics and its association with chronicity and recurrence of infections.

Various virulence factors like morphogenesis, hyper mutability and secreted factors,

including lipid mediators affect and damage hosts to facilitate rapid and aggressive colonization and infection. When combined *Candida* and *Pseudomonas aeruginosa*, secrete large amounts of radicals that can elicit oxidative damage to each other, as well as the host. *Pseudomonas aeruginosa* known for its biofilm capability and its resistance mechanism, further complicates otomycosis, particularly in immunocompromised individuals or those with prolonged antibiotic use.

Both of these opportunistic pathogens are able to form resistant biofilms. *Candida albicans* and *Pseudomonas aeruginosa* are both able to utilize arachidonic acid (AA), liberated from the host cells during infection, to form eicosanoids. The production of these eicosanoids, such as Prostaglandin E2, by the host and the pathogens may affect the dynamics of polymicrobial infection and the outcome of infections^[36].

Aspergillus and candidal species in otomycosis always showed coexistence with MRSA and *Pseudomonas aeruginosa*.

All these patients with otomycosis received treatment accordingly by thorough regular aural toileting, topical antifungal medications, topical or systemic antibiotic medications as per antibiotic sensitivity. Aural pain/ fullness recovered in 2-3 days and complete recovery of ear canal resulted in 10-14 days of treatment with no complications in all patients. This study also highlights the importance of treating an otomycotic patient with antifungal as well as antimicrobial medications.

CONCLUSION

Bacterial presence in otomycosis, well appreciated from microbiological studies, now warrants the need for a combined approach treatment. The coinfection pattern observed in this study underscores the importance of accurate microbiological diagnosis in otomycosis cases. Empirical anti-fungal therapy alone may be inadequate in the presence of bacterial coinfection, and combined antimicrobial therapy may be warranted to achieve optimal clinical outcomes.

Topical antibiotic preparations or systemic antibiotics were given to address the bacterial infection

and topical antifungal treatments were sufficient enough to address fungal infection. Bacterial cultures seen along with fungus in otomycosis raises the suspicion of a symbiotic relationship between fungus and bacteria. Further studies are needed to evaluate antimicrobial resistance patterns in these coinfecting bacteria to optimize treatment protocols and reduce recurrence.

REFERENCES

1. Mugliston T, O'Donoghue G. Otomycosis—a continuing problem. *Journal of Laryngology and Otology*. 1985;99(4):327–333.
2. Pontes ZB VDS, Silva ADF, Lima EDO, et al. Otomycosis: a retrospective study. *Brazilian Journal of Otorhinolaryngology*. 2009;75(3):367–370.
3. Fasunla J, Ibekwe T, Onakoya P. Otomycosis in western Nigeria. *Mycoses*. 2008;51(1):67–70.
4. Kurnatowski P, Filipiak A. Otomycosis: prevalence, clinical symptoms, therapeutic procedure. *Mycoses*. 2001;44(11-12):472–479.
5. Pradhan B, Ratna Tuladhar N, Man Amatya R. Prevalence of otomycosis in outpatient department of otolaryngology in Tribhuvan University Teaching Hospital, Kathmandu, Nepal. *Annals of Otology, Rhinology and Laryngology*. 2003;112(4):384–387.
6. Munguia R, Daniel SJ. Otological antifungals and otomycosis: a review. *International Journal of Pediatric Otorhinolaryngology*. 2008;72(4):453–459. doi: 10.1016/j.ijporl.2007.12.005. [DOI] [PubMed] [Google Scholar]
7. Fasunla J, Ibekwe T, Onakoya P. Otomycosis in western Nigeria. *Mycoses*. 2008;51(1):67–70.
8. Agarwal P, Devi LS. Otomycosis in a Rural Community Attending a Tertiary Care Hospital: Assessment of Risk Factors and Identification of Fungal and Bacterial Agents. *J Clin Diagn Res*. 2017 Jun;11(6):DC14-DC18.
9. Mofatteh MR, Naseripour Yazdi Z, Yousefi M, Namaei MH. Comparison of the recovery rate of otomycosis using betadine and clotrimazole topical treatment. *Braz J Otorhinolaryngol*. 2018 Jul-Aug;84(4):404-409.
10. Alarid-Coronel J, Celis-Aguilar E, Escobar-Aispuro L, Muñoz-Estrada V. Otomycosis in immunocompetent patients: Clinical and mycological features. Our experience with 40 cases. *Clin Otolaryngol*. 2018 Feb;43(1):373-377
11. Zaror L, Fischman O, Suzuki FA, Felipe RG (1991) Otomycosis in Sao Paulo. *Rev Inst Med Trop Sao Paulo* 33: 169-173.
12. Pradhan B, Tuladhar NR, Amatya RM (2003) Prevalence of otomycosis in outpatient department of otolaryngology in Tribhuvan University Teaching Hospital, Kathmandu, Nepal. *Ann Otol Rhinol Laryngol* 112: 384-387.
13. Ho T, Vrabec JT, Yoo D, Coker NJ (2006) Otomycosis: Clinical features and treatment implications. *Otolaryngol Head Neck Surg* 135: 787-791.
14. Paulose KO, Al Khalifa S, Shenoy P, Sharma RK (1989) Mycotic infection of the ear (otomycosis): A prospective study. *J Laryngol Otol* 103: 30-35.
15. Pontes ZB, Silva AD, Lima Ede O, Guerra Mde H, Oliveira NM, Carvalho Mde F, et al. Otomycosis: a retrospective study. *Braz J Otorhinolaryngol*. 2009;75(3):367–70
16. Kujundzic M, Braut T, Manestar D, et al; Water related otitis externa. *Coll Antropol*. 2012;36(3):893-7
17. Kaur R, Mittal N, Kakkar M, Agarwal AK, Mathur MD. Otomycosis-A clinico-mycological study. *Ear Nose Throat J* 2000;79(8):606-609.
18. Deshmukh J, Surpam R, Band A. Mycological study of aspergillus infections in otomycosis in eastern part of Maharashtra. *Int J Health Sci Res*. 2014;4(10):77-82.
19. Tasić-Otašević S, Golubović M, Đenić S, et al. Species distribution patterns and epidemiological characteristics of otomycosis in Southeastern Serbia. *Journal de Mycologie Medicale*. 2020 Sep;30(3):101011. DOI: 10.1016/j.mycmed.2020.101011
20. Sukumar B, Premamalini T, Shree SN, Kindo AJ. P022 luliconazole—a novel potent imidazole activity against *Aspergillus niger* and *Aspergillus flavus* causing otomycosis. *Med Mycol*. (2022) 60:22.
21. Collee JG, Miles RS, Watt B (1996) Tests for identification of bacteria. Mackie and McCartney

- practical medical microbiology 14: 131-149.
22. de Souza Costa P, Prado A, Bagon NP, Negri M, Svidzinski TIE. Mixed fungal biofilms: from mycobiota to devices, a new challenge on clinical practice. *Microorganisms*. (2022) 10:1721.
23. Hurst WB (2001) Outcome of 22 cases of perforated tympanic membrane caused by otomycosis. *J Laryngol Otol* 115: 879- 880.
24. Gharaghani, M., Seifi, Z. & Zarei Mahmoudabadi, A. Otomycosis in Iran: A Review. *Mycopathologia* 179, 415–424 (2015).
25. Brobby GW. The Discharging Ear in the Tropics: A Guide to Diagnosis and Management in the District Hospital. *Tropical Doctor*. 1992;22(1):10-13.
26. Bojanović M, Stalević M, Arsić-Arsenijević V, Ignjatović A, Randelović M, Golubović M, Živković-Marinkov E, Koraćević G, Stamenković B, Otašević S. Etiology, Predisposing Factors, Clinical Features and Diagnostic Procedure of Otomycosis: A Literature Review. *J Fungi (Basel)*. 2023 Jun 13;9(6):662.
27. Görür K, İsmi O, Özcan C, Vayisoğlu Y. Treatment of Otomycosis in Ears with Tympanic Membrane Perforation is Easier with Paper Patch. *Turk Arch Otorhinolaryngol*. 2019 Dec;57(4):182-186
28. Chiller K, Selkin BA, Murakawa GJ. Skin microflora and bacterial infections of the skin. *J Investig Dermatol Symp Proc*. 2001 Dec;6(3):170-4. doi: 10.1046/j.0022-202x.2001.00043.x.
29. Hall RA, Noverr MC. Fungal interactions with the human host: exploring the spectrum of symbiosis. *Curr Opin Microbiol*. 2017 Dec;40:58-64.
30. Pawlowska TE. Symbioses between fungi and bacteria: from mechanisms to impacts on biodiversity. *Curr Opin Microbiol*. 2024 Aug;80:102496.
31. Wang, F., Xin, C., Liu, J. et al. Interactions between invasive fungi and symbiotic bacteria. *World J Microbiol Biotechnol* 36, 137 (2020).
32. Gomaa AA, Abou-Zied HA, Farhan SM, Bedaiwi RI, Alanazi MA, Glaeser SP, Kämpfer P, Abdelmohsen UR, Mokhtar FA, Abdelaleem ER. *Apium graveolens*-associated *Aspergillus* sp.: metabolomic profiling and anti-MRSA potential supported by in silico studies. *Microb Cell Fact*. 2025 Mar 8;24(1):57.
33. Abdellatif, H., Sehim, A.E., Emam, A.M. et al. Antimicrobial, antibiofilm and antioxidant activities of bioactive secondary metabolites of marine *Scarus ghobban* gut-associated *Aspergillus niger*: In-vitro and in-silico studies
34. Zhao J, Yu W. Interaction between *Pseudomonas aeruginosa* and *Aspergillus fumigatus* in cystic fibrosis. *PeerJ*. 2018 Nov 9;6:e5931
35. Todd OA, Fidel PL Jr, Harro JM, Hilliard JJ, Tkaczyk C, Sellman BR, Noverr MC, Peters BM. *Candida albicans* Augments *Staphylococcus aureus* Virulence by Engaging the Staphylococcal agr Quorum Sensing System. *mBio*. 2019 Jun 4;10(3):e00910- 19.
36. Fourie R, Ells R, Swart CW, Sebolai OM, Albertyn J, Pohl CH. *Candida albicans* and *Pseudomonas aeruginosa* Interaction, with Focus on the Role of Eicosanoids. *Front Physiol*. 2016 Feb 26;7:64.