

Short communication

Are some isolates of candida albicans resistance against silver nanostructures?

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<http://jabs.eu5.org/>

Received: July. 11, 2015

Accepted: July. 22, 2015

Vol. 1, No. 3, 2015, pages 214-220.

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Abstract:

Pathogenic fungi like candida albicans are the cause of life-threatening infections in an increasing number of immunocompromised patients. One important issue in administration of antifungal agents is drug resistance that lead to failure of treatment. Different antimicrobial nanomaterials were studied and no drug resistance have been observed. The best antifungal nanoparticles that were evaluated are silver nanoparticles that can destroy fungal cell by binding protein and membrane. The aim of this research was the assessment of resistance against silver nanostructures on clinical candida isolates. Thirty two clinical candida albicans isolated in Pars hospital lab from candidiasis patients. Firstly approved isolates of candida albicans were grown in Saboraud Dextrose Agar for 2 days at 37 c and secondly, resistance assessment of different silver nanostructures were carried out by disk diffusion method on muler-hinton medium. Nanodots, nanocubes and nanowires of silver were chemically synthesized and confirmed by SEM microscope and then each nanostructure was added to watman paper(1*1 cm) and were hold on medium and incubated at 37 c for 48 hours and finally resistance of each isolate was repored for nanostructures. All isolates were sensitive to silvernanocubes and silvernanowires but two isolates of them (6%) were resistance to silverNanodots. This research showed that nanodots, the most used antimicrobial nanoparticle, are not effective on all isolates of candida albicans.

Keywords:

silver nanostructures-resistance- candida albicans

Introduction:

Pathogenic fungi like candida albicans are the cause of life-threatening infections in an increasing number of immunocompromised patients. One important issue in administration of antifungal agents is drug resistance that lead to failure of treatment. Antimicrobial drug resistance is an important biological phenomenon that has a considerable impact on animal and human health. The prevalence of clinical drug resistance has increased in recent decades with the greater use, and abuse, of otherwise efficacious antimicrobial agents. This has been a significant problem for bacterial pathogens, where resistance to multiple antibiotics severely limits therapeutic options. Antimicrobial resistance is not restricted to bacteria, however, and in the 1990s fluconazole treatment failure emerged due to the development of resistance by the fungal pathogen Candida albicans (1). Antifungal resistance is particularly problematic as initial diagnosis of systemic fungal infection can be delayed and there are few antifungal drugs available. The development of antimicrobial drug resistance is not a new phenomenon – micro-organisms have been responding to toxic environmental stresses for millennia (2). Indeed it is likely that the mechanisms utilized to confer resistance to ‘novel’ synthetic drugs have been selected from an extensive repertoire that has enabled micro-organisms to survive for so long in changing environments.

Exposure to antifungal drugs is, for *C. albicans*, another environmental stress that stimulates responses to mitigate the harmful effects of the drugs and permit continued growth. The antifungal stress is transduced through signalling pathways that induce stress responses and affect the growth and virulence of the organism. Stress-response signalling and its interrelation with *C. albicans* morphogenesis and virulence are complex and beyond the scope of this mini-review. Signalling pathways, and their role in virulence, have been excellently reviewed by [Quinn & Brown \(3\)](#). Although little is known about stress responses in non-albicans Candida species, the stress-response pathways in the model yeasts *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe* have

been well studied (4). Despite extensive congruence in the fungal stress-response networks, there are significant differences in the stress responses of *C. albicans* (5-10). Therefore, although major differences in the stress responses of other fungi will be noted, this review will focus specifically on how *C. albicans* responds to antifungal stress and how the development of resistance is a component of the stress-response network of *C. albicans*. Different antimicrobial nanomaterials were studied and no drug resistance have been observed. The best antifungal nanoparticles that were evaluated are silver nanoparticles that can destroy fungal cell by binding protein and membrane. The aim of this research was the assessment of resistance against silver nanostructures on clinical candida isolates.

Materials and Methods:

Organism and medium

Thirty two clinical candida albicans isolated from candidiasis patients at different origin. Colony isolation was carried out on Saboraud dextrose agar and germ tube test was done for each isolate.

Synthesis of Nanodots, nanocubes and nanowires of silver

Nanodots, nanocubes and nanowires of silver were synthesized by reduction reaction and confirmed by scanning electron microscopy.

Assessment of grow inhibition

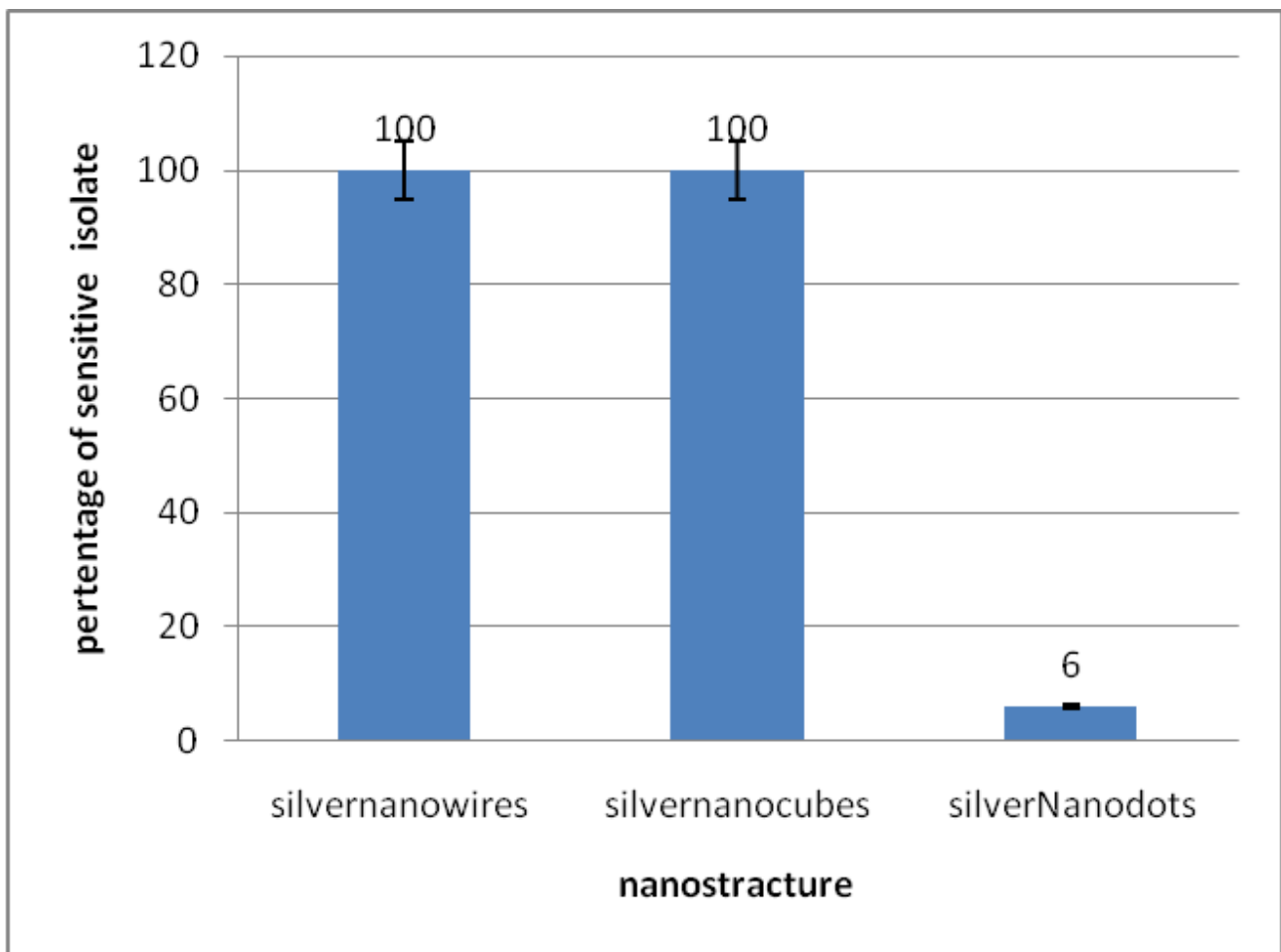
For assessment of grow inhibition of different silver nanostructures, disk diffusion method was done on muler-hinton medium. 10 microliters of each nanostructure was added to watman paper (1*1 cm) and dried at room temperature. Disk were hold on muler-hinton medium and incubated at 37

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degree centigrade for 48 hours and finally inhibition zone diameter of each isolate was recorded for three nanostructures.

Result:

All isolates were sensitive to silvernanocubes and silvernanowires but two isolates of them (6%) were resistance to silverNanodots(Graph 1).



Graph 1. percentage of sensitive isolates for Nanodots,nanocubes and nanowires of silver

Conclusion :

This research showed that nanodots, the most used antimicrobial nanoparticle, are not effective on all isolates of candida albicans.

References:

1. **White, T. C., Marr, K. A. & Bowden, R. A. (1998).** *Clinical, cellular, and molecular factors that contribute to antifungal drug resistance. Clin Microbiol Rev 11, 382–402.*
2. **Wright, G. D. (2007).** *The antibiotic resistome: the nexus of chemical and genetic diversity. Nat Rev Microbiol 5, 175–186.*
3. **Quinn, J. & Brown, A. J. P. (2007).** *Stress responses in Candida albicans. In Candida: Comparative and Functional Genomics, pp. 217–261. Edited by C. d'Enfert & B. Hube. Norwich, UK: Caister Academic Press.*
4. **Roman, E., Arana, D. M., Nombela, C., Alonso-Monge, R. & Pla, J. (2007).** *MAP kinase pathways as regulators of fungal virulence. Trends Microbiol 15, 181–190.*
5. **Enjalbert, B., Smith, D. A., Cornell, M. J., Alam, I., Nicholls, S., Brown, A. J. & Quinn, J. (2006).** *Role of the Hog1 stress-activated protein kinase in the global transcriptional response to stress in the fungal pathogen Candida albicans. Mol Biol Cell 17, 1018–1032.*
6. **Fox, D. S. & Heitman, J. (2002).** *Good fungi gone bad: the corruption of calcineurin. Bioessays 24, 894–903.*
7. **Gregori, C., Schuller, C., Roetzer, A., Schwarzmuller, T., Ammerer, G. & Kuchler, K. (2007).** *The high osmolarity glycerol (HOG) response pathway in the human fungal pathogen Candida glabrata strain ATCC2001 lacks a signaling branch operating in baker's yeast. Eukaryot Cell in press*
8. **Holmes, A. R., Tsao, S., Ong, S. W., Lamping, E., Niimi, K., Monk, B. C., Niimi, M., Kaneko, A., Holland, B. R. & other authors (2006).** *Heterozygosity and functional allelic*

variation in the *Candida albicans* efflux pump genes *CDR1* and *CDR2*. *Mol Microbiol* 62, 170–186.

9. **Jain, P., Akula, I. & Edlind, T. (2003).** *Cyclic AMP signaling pathway modulates susceptibility of Candida species and Saccharomyces cerevisiae to antifungal azoles and other sterol biosynthesis inhibitors. Antimicrob Agents Chemother* 47, 3195–3201.
10. **Karababa, M., Valentino, E., Pardini, G., Coste, A. T., Bille, J. & Sanglard, D. (2006).** *CRZ1, a target of the calcineurin pathway in Candida albicans. Mol Microbiol* 59, 1429–1451.