Proceedings of the Nutrition Society (2014), **73 (OCE1)**, E28 doi:10.1017/S0029665114000421

Winter Meeting, 11–12 December 2013, Diet, gut microbiology and human health

Inhibitory effect of plant (poly)phenolics on growth and biofilm formation by *Candida albicans*

M. Shahzad[1], L. Sherry[2], R. Rajendran[2], C. A. Edwards[1], E. Combet[1] and G. Ramage[2]
[1]*Human Nutrition, School of Medicine, College of MVLS, University of Glasgow, Glasgow, G3 8SJ, UK and*
[2]*Glasgow Dental School, Institute of Infection and Immunity, College of MVLS, University of Glasgow, 378 Sauchiehall Street, Glasgow, G2 3JZ, UK*

(Poly)phenolics are plant secondary metabolites, regularly consumed by humans (~1 g/day) in the form of fruits, vegetables and beverages (tea, coffee, wine and juices)[1]. They are thought to be protective against infectious and degenerative diseases via their antioxidant, anti-inflammatory and antimicrobial activities[2]. In recent years, the potential antimicrobial activity of (poly)phenolics has attracted increasing scientific attention, partially due to development of microbial resistance to commonly used antimicrobial agents. *Candida albicans*, an opportunistic fungal pathogen, is one of the major causes of life threatening, invasive candidiasis with estimated mortality rate of 46–75%[3]. The ability of *C. albicans* to grow as a 'biofilm' and its flexibility in adapting to different environmental conditions contributes to unacceptably high rates of morbidity and mortality, of which ineffectual antifungal therapy is a key factor. Therefore, alternative approaches to prevent biofilm formation or destruction and modulation of established biofilms are highly desirable. The aim of the present study was to evaluate the potential role of (poly)phenolics, in the prevention, and possibly management, of chronic oral and systemic fungal infections.

(Poly)phenolics ($n = 14$), belonging to two major groups (flavonoids and non-flavonoids) were examined against *C. albicans* isolates ($n = 20$). Planktonic minimum inhibitory concentrations (PMIC) were determined using the CLSI M-27A broth microdilution method, while sessile MIC was determined by XTT reduction metabolic assay. The most active (poly)phenolic molecules were further tested for their role in preventing *C. albicans* biofilm formation and disruption of mature biofilms (biomass assays); their effect on *C. albicans* morphology and gene expression was assessed through light microscopy and qPCR.

Of 14 (poly)phenolics tested, seven (curcumin, pyrogallol, pyrocatechol, quercetin, gallic acid, caffeic acid, naringenin) were effective inhibitors of planktonic growth of *C. albicans*, while 3,4-dihydroxyphenylacetic acid, epigallocatechin, epigallocatechin gallate, hesperetin, malvidin, pelargonidin, and apigenin had no discernible effect at the highest test concentration. PMIC was lowest for pyrogallol (80 μg/mL) followed by curcumin (100 μg/mL). These two compounds also displayed potent activity against *C. albicans* biofilms ($SMIC_{50} = 40$ & 50 μg/mL, respectively). $SMIC_{50}$ concentration of curcumin and pyrogallol significantly inhibited *C. albicans* biofilm growth when added 0, 1 and 2 h post adhesion ($p < 0.001$). However decrease in biomass was not significant when curcumin and pyrogallol were added at 24 h. In addition, overnight coating of coverslips with $SMIC_{50}$ concentration of curcumin and pyrogallol caused 58% and 15% reduction in initial adhesion of yeast cells to the coverslips. Light microscopy images of curcumin treated cells showed inhibition of hyphal growth, at different treatment points (0, 1, 2, 4 h). qPCR analysis showed differential expression of genes responsible for *C. albicans* adhesion, hyphal growth and elongation following curcumin treatment.

Overall there was a promising antifungal activity and inhibition of biofilm formation by curcumin and pyrogallol. Cellular and *in-vivo* studies are necessary to explore their use in the prevention and management of fungal infections.

1. Augustin S & Gary W (2000) *J. Nutr.* **130**, 2073S–2085Sl.
2. Stefano P & Crispian S (2009) *J. Dent* **37**, 413–423.
3. Brown GD *et al.* (2012) *Sci Transl Med.* **194**(165).

Proceedings of the Nutrition Society

https://doi.org/10.1017/S0029665114000421 Published online by Cambridge University Press