World Journal of Dairy & Food Sciences 5 (2): 160-163, 2010
ISSN 1817-308X

Potentials of Wild Strain S*accharomyces cerevisiae* as Probiotic in Gastroentritis

[1]S.E. Abah, [1]S.U. Oranusi, [2]G. Abah and [1]O.S. Taiwo

[1]Department of Biological Sciences, Microbiology Programme,
College of Science and Technology, Covenant University, Canaanland, Ota, Ogun state, Nigeria
[2]Animal Science Department, University of Uyo, Akwa-Ibom State, Nigeria

Abstract: Wild strain of *Saccharomyces cerevisiae* has been found to be an effective probiotic in rats induced with diarrhea by oral administration of enterotoxigenic *Escherichia coli* (ETEC). Two groups of rats (A and B) induced with ETEC were examined for clinical sign (diarrhea, without red blood cells in stool) after 10hours of oral administration of ETEC. Group A was administered with filtered corn drink fortified with *S. cerevisiae* as probiotic while group B received only filtered corn drink as placebo. There was a significant decrease ($p<0.05$) in gastrointestinal pH, moisture content of stool and colony forming units of ETEC recovered per gram of stool in group A within 5 days of oral administration of probiotic compared with group B that received the placebo. Continuous increase in weight was observed in group A while decrease in weight was observed in group B. This wild strain of *S.cerevisiae* was found to be effective as probiotic in gastroenteritis caused by ETEC in rats, an implication for its use in humans, by lowering the gastrointestinal pH below the optimum pH required for colonization and growth of ETEC.

Key words: S. cerevisiae · Probiotics · Diarrhea, ETEC

INTRODUCTION

Diarrhea is one of the clinical sign of gastrointestinal disorders mainly caused by ETEC. [1-3] Diarrhea is characterized with the frequent loss of watery stool (unformed stool) for about 3 or more times a day [4]. 20-40% of diarrhea is caused by ETEC [5]. Diarrhea disease is the second leading cause of death; about 25 million enteric infections occur each year. This infection cause significant morbidity and death, particularly in elderly people and children younger than 5 years of age. It has been estimated that 4-6 million children die each year of diarrheal disease, particularly in developing countries in Asia and Africa. Even in developed countries, significant morbidity occurred as a result of diarrheal illness [6-10]. Onset of diarrhea result due to changes in delicate balance of water and electrolyte in the bowels leading to massive fluid secretion. In many cases the process is mediated by enterotoxin [11-13] the enterotoxin acts on adenylate cyclase enzymes which catalyzes the formation of ATP (Adenosine triphosphate) to cyclic adenosinemonophosphate (CAMP). The increased level of CAMP stimulates the secretion of ion into the lumen from the intravascular fluid store, leading to diarrheal condition.

Most diarrheas can be treated and prevented by populating the intestine with a skillful array of beneficial microflora by the administration of probiotics. These good bacteria and yeast protects by performing certain vital function through several mechanism. Probiotics are best gotten from fermented food and drinks that contains them. Probiotics have been shown to be of some benefit in acute infectious diarrhea in children [14-15], prevention of necrotizing enterocolitis in very low birth-weight infants, prevention of allergic atopic dermatitis in children [16] and prevention of relapse of ulcerative colitis [17]. Increasing number of multidrug resistant pathogen in hospital is a major driving force into the use of probiotic. Probiotics offers numerous benefits including low cost preparation, long shelf life and ease of administration.

The use of wild strain of *S. cerevisiae* as probiotics for diarrhea in rats is a novel research indicating its safety/use in humans, toward providing affordable therapy to diarrhea claiming millions of life in Africa who has no access to medical care.

Corresponding Author: Abah Samuel Eneojo, Department of Biological Sciences, Microbiology Programme, College of Science and Technology, Covenant University, P.M.B 1023, Canaanland, Ota, Ogun State, Nigeria, E-mail: samuelabah@gmail.com.

MATERIALS AND METHODS

Experimental rats of about 5 months old of the same sex were weighed and grouped as A and B, each group consist of five rats. Both groups were induced with diarrhea by oral administration of ETEC ATCC11900 which was collected from National Medical Research Institute (NIMR) in Lagos, Nigeria. The ETEC was grown in a nutrient broth. The bacterial suspension that correspond with 0.5 McFarland standard of 1.5×10^{8}CFU/Ml was serially diluted, 10^{-6} of the dilution was orally administered to both group. The rats were observed to show clinical symptom of watery stool (diarrhea) [18, 19] without fecal leucocyte at the 10^{th} hour of oral administration.

Corn drink (gruel) was filtered using membrane filter apparatus with 0.45micrometer bacteriologic filter and was then fortified with wild strain of *S.cerevisiae* isolated from rotten Irish potato purchased from Maiduguri market, Borno State, Nigeria and identified by its morphological and physiological characteristics as described by Yarrow [20] and Yabaya and Jatau [21] with slight modification. The corn drink fortified with *S.cerevisiae* was then administered to group A as probiotics and only filtered corn drink was administered to group B as placebo. The administration of probiotic and placebo to both groups was within 5 days.

Observable measurable parameters for the period of 5days include; pH of stool as an index of intestinal pH, changes in weight of rats (subsequent weight subtracted from the original initial weight), moisture content of stool (total weight of stool minus dry weight divided by total weight multiplied by 100), bacterial count of ETEC in colony forming units recovered from per gram of stool (CFU/g of stool) by methods described in Afred E. Brown [22] using MacConkey agar. ETEC was identified on agar as the only lactose fermenting, flat, dry, pink colonies with surrounding darkerpink area of precipitated bile salt [23].

RESULTS

The pH of stools in group A showed a gradual decrease, decrease in water content of stool, decrease in the number of CFU/g of stool and changes in weight also decrease with administration of probiotics (Table 1). Group B receiving placebo showed no tangible change in pH, water content of stool increases slightly alongside with changes in the weight of rats and a higher number CFU/g of stool (Table 1). pH, water content of stool, CFU/g of stool and changes in weight decreases significantly ($P<0.05$) in Group A compared to Group B.

DISCUSSION

The continuous decrease in pH to about 2.9 was hypothesized to be responsible for reduction in the CFU recovered per gram of stool, decrease in water content of stool and decrease in change in weight which implies that the rats were actually gaining weight following the administration of probiotics as observed in Group A. *S. cerevisiae* in the corn drink had significantly reduced the intestinal pH making it unfavourable for the growth of ETEC. Little or no changes in intestinal pH in Group B was due to the absence of *S. cerevisiae* in the corn drink leading to higher number of CFU/g of stool, increase in water content of stool and increase in weight change. Wild strain of *S. cerevisiae* can be said to be an effective probiotic for ETEC induced diarrhoea through the mechanism of lowering the intestinal pH. *S. cerevisiae* had been used to prevent traveller's diarrhea in about 1231 people traveling all over the world, it had significantly lowered infection rate in groups administered with it [22]. It had also been documented that food borne *S. cerevisiae* can survive a pH as low as 2.5 after 4h of incubation, yeast generally has demonstrated survival at such low pH [23], making it to be very effective in eliminating intestinal bacteria like ETEC by inducing pH inbalance.

Table 1: Mean values of the measured parameters in group A and B

PH		water content (%)		Changes in weight		CFU/g	
A	B	A	B	A	B	A	B
6.9	7.0	80.0	80.0	24.0	21.0	27×10^{6}	26×10^{6}
5.4	6.9	75.0	81.0	23.0	21.5	17×10^{6}	25×10^{6}
4.1	7.2	60.0	82.0	22.5	22.0	7×10^{5}	26×10^{6}
3.3	6.8	40.0	81.7	22.0	21.0	4×10^{5}	24×10^{6}
2.9	6.8	30.0	83.0	20.2	22.5	2×10^{5}	27×10^{6}

It was concluded that *S. cerevisiae* functions as probiotic in ETEC induced gastroenteritis by lowering of intestinal pH below the optimum pH required for the colonization and growth of the ETEC.

ACKNOWLEGEMENT

I acknowledge Mr. Taiwo of Microbiology Laboratory, National Medical Research Institute (NIMR) Lagos for providing me with *E. coli* strain that I used in my work.

REFERENCES

1. James, P.N. and B.K. James, 1998. Diarrheagenic *E. coli*; Clin. Microbiol. Rev., 11(1): 142-201
2. Taylor, J., M.P. Wilkins and J.M. Payne, 1961. Relations of rabbit gut reaction to enteropathogenic Escherichia coli. Br. J. Exp. Pathol., 42: 43-52.
3. Dupont, C., *et al.,* 1971. Pathogenesis of *E. coli* diarrhea. N. Engl. J. Med., 285: 1-9.
4. Khin, H., S.Y. Khin, T. Marlar and V. Yvan, 2008. Effect of Saccharomyces boulardii in the treatment of acute watery diarrhea in myanmer children: A randomized controlled study. Am. J. Trop. Med. Hyg., 78(2): 214-216.
5. Arduino, R.C. and H.L. Dupont, 1993. Traveller's diarrhea, baillieres Clin. Gastroenterol., 7: 365-385
6. Albert, M.J., S.M. Faruque, A.S. Faruque, P.K. Neogi, M. Ansaruzzaman, N.A. Bhuiyan, K. Alam and M.S. Akbar, 1995. Controlled study of Escherichia coli diarrheal infections in Bangladeshi children. J. Clin. Microbiol., 33: 973-977.
7. Flores-Abuxapqui, J.J., G.J. Suarez-Itoil, M.R. Heredia-Navarrete, M.A. Puc-Franco and J. Franco-Monsreal, 1994. Frequency of enterotoxigenic Escherichia coli in infants during the first three months of life. Arch Med. Res., 25: 303-307.
8. Hoque, S.S., A.S. Faruque, D. Mahalanabis and A. Hasnat, 1994. Infectious agents causing acute watery diarrhoea in infants and young children in Bangladesh and their public health implications. J. Trop. Pediatr., 40: 351-354.
9. Mangia, A.H., A.N. Duarte, R. Duarte, L.A. Silva, V.L. Bravo and M.C. Leal, 1993. Aetiology of acute diarrhea in hospitalized Children in Rio de Janeiro city, Brazil J. Trop. Pediatr., 30: 365-367.
10. Hirayama, T., 1995. Heat-stable enterotoxin of E. coli in: Moss J, B Iglewski, M Vaughan, AT Tu, editors. Bacterial toxin and virulence factor in disease. New York, N.Y: Marcel Dekker, Inc., pp: 281-296.
11. Hirst, T.R., 1995. Biogenesis of Cholera toxin and related Oligomeric enterotoxins in: J. Moss, B. Iglewski, M. Vaughan, A.T. Tu, editors. Bacterial toxins and virulence factors in disease. New York, N.Y: Marcel Dekker, Inc., pp: 123-184.
12. Hol, W.G.J., T.K. Sixma and E.A. Merritt, 1995. Structure and Function of E.coli heat labile enterotoxin and cholera toxins B Pentamer. In: J. Moss, B. Iglewski, M. Vaughan, A.T. Tu, editors. Bacterial toxins and virulence factors in disease. New York, N.Y: Marcel Dekker, Inc., pp: 185-223.
13. Isolauri, E., M. Juntunen and T. Rautanen, 1991. A human lactobacillus strain (lactobacillus GG) promotes recovery from acute diarrhea in children. Pediatrics, 88: 90-97.
14. Majamaa, A., E. Isolauri, M. Saxelin, *et al*., 1995. Lactic acid bacteria in the treatment of acute rotavirus gastroenteritis. J. Pediatr Gastroenterol. Nutr., 20: 333-338.
15. Kalliomaki, M., S. Salminen, H. Arvilommi, *et al*., 2001. Probiotics in primary prevention of atopic disease: a randomized placebo controlled trial. Lancet., 357: 1076-1079.
16. Kruis, W., 2004. Antibiotics and Probiotics in inflammatory bowel disease. Aliment Pharmacol. Ther., 20: 575-578.
17. Betty, A.F., F.S. Daniel and A.S. Weissfeld, 2007. BAILEY and SCOTT'S Diagnostic Microbiology. 12th edition, pp: 884.
18. Betty, A.F., F.S. Daniel and A.S. Weissfeld, 2007. BAILEY and SCOTT'S Diagnostic Microbiology. 12th edition, pp: 326.
19. Yarrow, D., 1998. Methods for the isolation, maintenance and identification of yeasts. In: Kurtzman CP, JW Fell, editors'. The yeasts: a taxonomic study. Amsterdam: Elsevier Science BV; pp: 77-100.
20. Yabaya, A. And E.D. Jatau, 2009. Investigating wild east baking potentials. Middle-East J. Scientific Res., pp: 320-322.
22. Betty, A.F., F.S. Daniel and A.S. Weissfeld, 2007. BAILEY and SCOTT'S Diagnostic Microbiology. 12th edition, pp: 328.

23. Alfred, E.B., 2007. Benson's Microbiological Apllication 10th Edition, pp:151-154.
24. Kollaritsch, H.H., P. Kremsner, G. Wiedermann and O. Scheiner, 1989. Prevention of traveller's diarrhea: Comparison of different non-antibiotic preparations. Travel med. Int., pp: 9-17.
25. Alis, V.A., K. Skovgaard and L. Jespersen. 2005. *In vitro* screening of probiotic properties of *S. cerevisiae* var boulardii and food borne *S. cerevisiae* strain. Intl. J. Food Microbiol., pp: 29-39.