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Probiotics in human health

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ABSTRACT

Probiotics are live microbial food supplements or components of bacteria, which have been shown to have beneficial effects on human health. Probiotic bacteria are used to treat or prevent a broad range of human diseases. Different species of microorganisms such as lactic acid bacteria or yeasts have been proposed for human use. Probiotics could be used for several conditions such as diarrhea, irritable bowel syndrome, urinary tract infections, immune disorders, cancer, Helicobacter pylori, lactose intolerance, hyper cholesterolaemia, inflammatory bowel disease and allergy. The aim of this review is to consider the current evidence on the effects of probiotics on human health.

Keywords: Probiotic, Antimicrobial activity, human health, intestinal microflora

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INTRODUCTION

Probiotics are defined as the viable microorganisms that exhibit a beneficial effect on the health of the host by improving its intestinal microbial balance. The term probiotics was first coined by Lilly and Stillwell in 1965 in reference to substances produced by protozoa, which stimulated the growth of other organisms [1, 2]. A probiotic organism should be nonpathogenic and non-toxic, and also resistant to low pH and to bile salts to improve its chances of survival in the gastrointestinal tract. The human gut contains 10 times more bacteria than cells elsewhere. The enormous biomass consists of over 400 known bacterial species that generate intense metabolic activity and are of key importance for human health. There is a wide range of species in the gut including *Bacteroides*, *Lactobacillus*, *Clostridium*, *Fusobacterium*, *Bifidobacterium*, *Eubacterium and Peptostreptococcus*. Gut bacteria with identified beneficial properties include mainly *Bifidobacterium* and *Lactobacillus* species. It is critical to human health that a balance is maintained between these species. When the balance is disturbed disease can result [3, 4]. Probiotic consumption is reported to exert a myriad of beneficial effects including: enhanced immune response, balancing of colonic microbiota, vaccine adjuvant effects, reduction of fecal enzymes implicated in cancer initiation, treatment of diarrhea associated with travel and antibiotic therapy, control of rotavirus and *Clostridium difficile*-induced colitis and prevention of ulcers related to *Helicobacter pylori*. Probiotics are also implicated in the reduction of serum cholesterol, the antagonism against food-borne pathogens and tooth decay organisms, the amelioration of lactose malabsorption symptoms as well as candidiasis and urinary tract infections [5, 6]. In this review, we will attempt to present the effects and mechanisms of probiotic action on human health.

PROBIOTICS AND HUMAN HEALTH

Probiotics and *Helicobacter pylori*:

Helicobacter pylori is the first formally recognized bacterial carcinogen and is one of the most successful human pathogens, as over half of the world's population is colonized with this gram-negative bacterium. The genus *Helicobacter* belongs to the subdivision of the *Proteobacteria*, order *Campylobacterales*, family *Helicobacteraceae*. This family also includes the genera *Wolinella*, *Flexispira*, *Sulfurimonas*, *Thiomicrospira*, and *Thiovulum* [7, 8]. *H. pylori* seroprevalence is increased with increasing age, lower socioeconomic status and crowdedness in the household, which are probably interrelated factors [9]. In recent years, the development of alternative anti-*H. pylori* treatments has been actively pursued, and investigations have been carried out to define components that could be used either as monotherapy or synergistically in combination with antimicrobials thus resulting in more effective anti-*H. pylori* therapy or alternative ways of controlling *H. pylori* infection. These novel treatments could potentially reduce the costs related to the treatment of *H. pylori* associated diseases. The effect of probiotics on *Helicobacter pylori* has been studied. Most evaluations have been done either in laboratory assays or in animal models. Results in animal models demonstrate that some lactobacilli inhibit *H. pylori* attachment and prevent colonization. Results in humans show that milk fermented by a *Lactobacillus johnsonii* strain can help control *H. pylori* gastric infections, but cannot eradicate *H. pylori* from the stomach [10, 11]. In study Aiba et al, among the lactobacillus species examined in vitro, *Lactobacillus salivarius* but not *L. casei* or *L. acidophilus* proved to be capable of producing a high amount of lactic acid and thus completely inhibiting the growth of *H. pylori* in a mixed culture [12]. In study Sýkora et al, Supplementation with fermented milk, containing live special probiotic *L. casei* DN-114 001, confers an enhanced therapeutic benefit on *H. pylori* eradication in children with gastritis on triple therapy [13]. Myllyluoma et al, found, probiotic supplementation did not diminish significantly the frequency of new or aggravated symptoms during *H. pylori* eradication. However, our data suggest an improved tolerance to the eradication treatment when total symptom severity was taken into account. Furthermore, the results show that probiotic bacteria are able to survive in the gastrointestinal tract despite the intensive antimicrobial therapy [14]. In study Ryan et al, Growth inhibition of *H. pylori* by *L. salivarius* is strain-dependent and is not linked to any particular environmental niche or geographic location. Strains of *L. salivarius* showing highest anti-*H. pylori* activity may be useful as an adjunct in the treatment of strains that are resistant to conventional antibiotics [15].

Probiotics and Urinary tract infections:

Urinary tract infections are common clinical entities occurring in a variety of pediatric patient groups. The causative organisms are known to include *Escherichia coli*, *Proteus* spp., *Klebsiella* spp., *Staphylococcus* spp., mainly intestinal uropathogens [16]. *Lactobacilli* are the dominant bacteria of the vaginal flora where they prevent infection by suppressing pathogenic bacteria. Recurrent urinary tract infections are associated with decreases in vaginal *Lactobacilli* and an overgrowth of pathogenic bacteria. Thus probiotics represent a potential strategy for preventing recurrent urinary tract infections in women [17]. In a mouse model, *Lactobacillus fermentum* was effective in the treatment of *E. coli* urinary tract infection when orally administered with low doses of ampicillin [18].

Probiotics and Traveler's, Rotavirus and Antibiotic-associated diarrhea:

Traveler's diarrhea

Traveller's diarrhea is estimated to affect more than 60% of travellers to developing countries and, in terms of frequency and economic impact, is the number one health problem for international travel. Travel is a risk factor for infectious gastroenteritis. A recent meta-analysis revealed evidence of a protective effect by *S. boulardi* and by mixture of *Lactobacillus acidophilus* and *Bifidobacterium bifidum* [19].

Rotavirus diarrhea

Rotavirus is a common cause of diarrhea amongst children in developing countries. Several Lactobacillus species have been tested and an effect shown against *Rotavirus* associated diarrhea. Rotavirus often causes gastro enteritis in hospitalised children but can also cause milder diarrhea in children who are cared for in day care centres or similar institutions. Researchers in Denmark studied the effect of *Lactobacillus rhamnosus* and *Lactobacillus reuteri* on acute diarrhea in children in day care centres. They found that on average those receiving the probiotic had diarrhea which lasted 40 hours less than those who received the placebo [20].

Antibiotic-associated diarrhea

Antibiotic-associated diarrhea (AAD) occurs in about 5–25% of adult patients and 11–40% of children upon administration of broad-spectrum antibiotics. *Clostridium difficile* is a major agent, although diarrhea may be related to general changes in intestinal microflora. Biller et al reported a series of four children with at least three

recurrences of *C. difficile* successfully treated with *Lactobacillus* Spp. [21]. *Lactobacillus* GG reduces the recurrence rate of *Clostridium difficile*, and patients report disappearance of abdominal cramps and diarrhea [22].

Probiotics and Cancer:

Cancer is one of the most important deaths causing in the world and many factors as chemicals, rays, viruses and genetic factors may influence it. There are many studies that have been suggested using of probiotic products as cancer risk reducer. Hosono et al, were the first to report that milk fermented with *L. delbrueckii* subsp. *bulgaricus*, *Lactococcus lactis* subsp. *lactis* or *Enterococcus faecalis* exhibited an antimutagenic activity against NQO [23]. Lankaputhra and Shah, proved that *Lactobacillus spp.* has good activity in decreasing mutagenic substances [24]. Zobel et al, showed that, *L. acidophilus* and its culture extract prevented from DNA damage by MNNG [25]. Heui-dong and Chang- Ho showed that, *L. plantarum* KLAB 21 was isolated from Kimchi can inhibit four mutagenic and carcinogenic agents effects; Aflatoxin B1, NQO, MNNG and NPD. He used two salmonella strains TA100 and TA98. Results showed that the bacterial culture supernatant inhibited mutagenic effects of MNNG (98.4%) in presence of TA100 and NQO (57.3%) in presence of TA98 [26]. Kazemi et al, showed that, *Lactobacillus* species (*L. Plantarum*, *L. casei*, *L. brevis*) could inhibit mutagenic agents activity until 40%, that is very good antimutagenic activity [2]. Chalova et al, evaluated the ability of some probiotic bacterial supernatants to decrease the effects of two mutagenic substances benzo[a]pyrene and sodium azide in different growth phases and *Bifidobacterium adolecenti* ATCC 15703 had 48.7% inhibitory in Log phase duration, *L. plantarum* ATCC 8014 showed 59.37% inhibitory function on mutagenic substance benzo[a]pyrene, and *L. plantarum* ATCC 8014 had 54.64% inhibitory on mutagenic substance sodium azide in lag phase duration [27].

Mechanisms of Anti-carcinogenicity:

- Binding of Carcinogens
- Effects on Bacterial Enzymes
- Production of anti-tumorigenic or anti-mutagenic compounds
- Enhancement of the host's immune response

Probiotics and Irritable Bowel Syndrome (IBS):

IBS is a chronic condition that is characterised by intermittent abdominal pain, altered bowel habits (diarrhea and/or constipation) and other gastrointestinal symptoms including flatulence and bloating in the absence of structural abnormalities in the intestine. Drouault-Holowacz et al, found an increase in patients with satisfactory relief of overall IBS symptoms and of abdominal discomfort/pain with time in those receiving a probiotic mix (*B. longum* LA 101, *L. acidophilus* LA 102, *Lactococcus lactis* LA 103, *St. thermophilus* LA 104) [28]. A study by Kajander et al, found an effect on IBS symptoms of a mixture of probiotic strains consumed as a milk drink. The decrease in IBS symptom score was significantly more pronounced in the group receiving probiotics compared to the placebo group [29]. In a study from Cork, Ireland, assessing individual bacterial species, improvement in symptoms with bifidobacteria was associated with changes in the relative production of anti-inflammatory interleukin (IL)-10 to proinflammatory (IL-12) cytokines [30]. O'Sullivan and O'Morain, found that *L. rhamnosus* GG had little effect on IBS symptoms whereas *L. plantarum* 299V had a measurably beneficial effect [31, 32].

Probiotics and Inflammatory bowel disease (IBD):

Ingestion of probiotic bacteria has the potential to stabilize the immunological barrier in the gut mucosa by reducing the generation of local proinflammatory cytokines. The term inflammatory bowel disease (IBD) is an umbrella term comprising different conditions of the gut, of which the two main types are ulcerative colitis (UC) and Crohn's disease (CD). IBD refers to a group of disorders of unknown aetiology that are characterized by chronic or recurrent mucosal inflammation. The well-defined, nonpathogenic strain *E. coli* Nissle 1917 has proven more effective in preventing relapse in Crohn's disease patients compared with a placebo [33]. *S. boulardii* has shown some success in relieving the symptoms of active Crohn's disease and in reducing the risk of relapse [34]. Campieri et al, showed that VSL#3 significantly reduced the risk of relapse in Crohn's disease patients postoperatively compared with the control group who were given mesalazine [35].

Probiotics and Reduction in serum cholesterol:

Ischemic heart disease is a major cause of morbidity and mortality that is often associated with elevated cholesterol levels and primary prevention with lipid lowering drugs or dietary modification can reduce the incidence and mortality of ischemic heart disease in healthy individuals. There is a high correlation between dietary saturated fat or cholesterol intake and serum cholesterol level. Probiotic bacteria are reported to de-conjugate bile salts:

deconjugated bile acid does not absorb lipid as readily as its conjugated counterpart, leading to a reduction in cholesterol level. A wide variety of probiotic products have been used in clinical trials of serum lipid modulation [36]. The oral administration of *L. johnsonii* and *L. reuterii* in pigs resulted in a decrease in serum cholesterol [37]. Serum cholesterol reduction has been also demonstrated in rats with the administration of a probiotic mixture [38].

CONCLUSION

Probiotics are found in dairy products, plants, meat products, sewage, manure humans and animals. These kinds of bacteria have positive effects on immune system by inhibition of pathogen attachment to epithelial cells, changing the receptor of bacterial toxins, producing antimicrobial substances such as acid, bacteriocins, fatty acid and aromatic compounds and competition for food. At presence, with increasing of the antibiotic resistance and side effects of chemical drugs, it seems, we need to use alternative remedies. Probiotics and their produced metabolites can have therapeutic application in future.

REFERENCES

[1] Lilly, D.M., Stillwell, R.H. *Science*, **1965**, 147,747-748.
[2] Kazemi Darsanaki, R., Issazadeh, K. Khoshkholgh Pahlaviani, M.R.M. Azizollahi Aliabadi, M. *J Pure Appl Microbio*, **2012**, 6, 1677-1682.
[3] Suvarna, V.C. Boby, V.G. *Current science*, **2005**, 88, 1744-1748.
[4] Collado, M.C., Isolauri, E. Salminen, S. Sanz, Y. *Curr Drug Metab*, **2009**, 10, 68-78.
[5] Zerehpoosh, S., Kazemi Darsanaki, R. *Journal of Biology and today's world*, **2013**, 2: 41-52.
[6] Kazemi Darsanaki, R., Ghaemi, N. Mirpour, M. Mirdavoudi, F. *Qom Univ Med Sci J*, **2012**, 6, 37-44.
[7] Goodwin, C.S., Armstrong, J.A. Chilvers, T. Peters, M. Colins, M.D. Sly, L. McConnell, W. Harper, W.E.S. *Int. J. Syst. Bacteriol*, **1989**, 39, 397- 405.
[8] Fox, J.G. **Gut**, 2002, 50, 273-283.
[9] Kenneth, W.T., Shiu-kum, L. *Journal of Gastroenterology and Hepatology*, **1999**, 14, 844–850.
[10] Gotteland, M., Brunser, O. Cruchet, S. *Aliment Pharmacol Ther*, **2006**, 23, 1077-1086.
[11] Hamilton-Miller, J.M. *Int J Antimicrob Agents*, **2003**, 22,360-366.
[12] Aiba, Y., Suzuki, N. Kabir, A.M. Takagi, A. Koga, Y. *Am J Gastroenterol*, **1998**, 93, 2097-2101.
[13] Sýkora, J., Valecková, K. Amlerová, J. Siala, K. Dedek, P. Watkins, S. Varvarovská, J. Stozický, F., Pazdiora, P. Schwarz, J. *J Clin Gastroenterol*, **2005**, 39,692-698.
[14] Myllyluoma, E., Veijola, L. Ahlroos, T. Tynkkynen, S. Kankuri, E. Vapaatalo, H. Rautelin, H., Korpela, R. *Aliment Pharmacol Ther*, **2005**, 21,1263-1272.
[15] Ryan, K.A., Daly, P. Li, Y. Hooton, C. O'Toole, P.W. *J Antimicrob Chemother*, **2008**, 61, 831-834.
[16] Lim, I.S, Lee. H.S. Kim, W.Y. *J Korean Med Sc*i, **2009**, 24, 57-62.
[17] Barrons, R., Tassone, D. *Clin Ther*, **2008**, 30,453-68.
[18] Silva de Ruiz, C., Lopez de Bocanera, M.E. Nader de Macias, M.E. Pesce de Ruiz Holgado, A.A. *Biol Pharm Bull*, **1996**,19,88–93.
[19] Guarino, A., Vecchio, A.L. Canani, R.B. *Current Opinion in Gastroenterology*, **2008**, 25, 18–23.
[20] Rosenfeldt, V., Michaelsen, K.F. Jakobsen, M., Larsen, C.N. Moller, P.L. Tvede, M. Weyrehter, H. Valerius, N.H. Paerregaard, A. *Pediatr Infect Dis J*, **2002**, 21,417-419.
[21] Biller, J.A., Katz, A.J. Flores, A.F. Buie, T.M. Gorbach, S.L. *J Pediatr Gastroenterol Nutr*, **1995**, 21, 224-226.
[22] Pochapin, M. *Am. J. Gastroenterol*, **2000**, 95, 11–13.
[23] Hosono, A., Sagae, S. Tokita, F. *Milchwissenschaft*, **1986**, 41, 142.
[24] Lankaputhra W.E.V., Shah, N.P. *Mutat*, **1998**, 397,169–182.
[25] Zoble B.L., Neudeker, C. Domizlaff, J.I.S. Schillinger, U. Rummney C. Morreti, M. *Nutr. Cancer*, **1996**, 26, 365 - 80.
[26] Park, H.D., Rhee, C.H. *Biotechnology Letters*, **2001**, 23, 1583–1589.
[27] Chalvo V.I., Lingbeck, J.M. Kwon, Y.M. Ricke, S.C. *Journal of Environmental Science and Health Part B*, **2008**, 43,193 -198.
[28] Drouault-Holowacz, S., Bieuvelet, S. Burckel, A. *Gastroenerologie Clinique et Biologique*, **2008**, 32, 147–52.
[29] Kajander, K., Myllyluoma, E. Rajilic-Stojanovic, M. *Alimentary Pharmacology& Therapeutics*, **2008**, 27, 48–57.
[30] Camilleri, M. *J Clin Gastroenterol*, **2006**, 40, 264- 269.
[31] O'Sullivan, M.A., O'Morain, C.A. *Liver Dis*, **2000**, 32, 294–301.

[32] Niedzielin, K. Eur. *J. Gastroenterol*. *Hepatol*, **2001**, 13, 1143–1147.
[33] Rembacken, B.J., *Lancet*, **1999**, 354, 635–639.
[34] Guslandi, M. *Dis. Sci*, **2000**, 45, 1462–1464.
[35] Campieri, M. *Gastroenterology*, **2000**,118, 4179.
[36] Naruszewicz, M., Johansson, M.L, Zapolska-Downar, D, Bukowska, H. *Am J Clin Nutr*, **2002**, 76, 1249–55.
[37] Du Toit, M., Franz, C.M, Dicks, L.M. *Int J Food Micr*, **1998**, 40:93–104.
[38] Fukushima, M., Yamada, A., Endo, T. *Nutrition*, **1999**, 15, 373–8.