Research Progress on Lactic Acid Bacteria Alleviating Intestinal Oxidative Stress (Postprint)

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Abstract

In mammals, increased generation of free radicals or diminished capacity for their scavenging results in substantial accumulation of free radicals within the organism. Excessive free radicals inflict damage upon biomacromolecules including DNA, lipids, and proteins in intestinal tissues, thereby causing oxidative stress injury to the intestine and impairing nutrient absorption, utilization, and animal growth and development. Consequently, implementing nutritional regulatory strategies to mitigate intestinal damage induced by oxidative stress holds significant importance for maintaining intestinal and overall animal health. Lactic acid bacteria, as an integral component of the innate immune system, function to preserve intestinal mucosal barrier integrity and microecological equilibrium, enhance systemic immunity, promote animal growth, and sustain intestinal and overall health. This review examines the alleviative effects of lactic acid bacteria on intestinal oxidative stress and the potential mechanisms underlying their probiotic functions through antioxidant activity, from the perspective of small intestinal mucosal barrier function, thereby providing a reference for the comprehensive understanding and scientific application of the antioxidant effects of lactic acid bacteria.

Full Text

Research Progress in the Relieving Effects of Lactobacillus on Intestinal Oxidative Stress

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Abstract: Excessive accumulation of free radicals in mammals occurs when their production increases or scavenging capacity decreases. These surplus free radicals damage biological macromolecules such as DNA, lipids, and proteins in intestinal tissues, causing intestinal oxidative stress injury that affects nutrient absorption, utilization, and animal growth. Therefore, nutritional interventions to reduce intestinal damage from oxidative stress are crucial for maintaining animal intestinal and overall health. As an essential component of the innate immune system, lactic acid bacteria (LAB) maintain intestinal mucosal barrier function and microecological balance while enhancing immunity, promoting animal growth, and preserving intestinal and overall health. This review examines the alleviating effects of LAB on intestinal oxidative stress and the potential mechanisms underlying their antioxidant functions from the perspective of intestinal barrier function, providing insights for deeper understanding and scientific application of LAB antioxidant activity.

Keywords: lactic acid bacteria; oxidative stress; intestinal barrier; antioxidant mechanism

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Lactic acid bacteria (LAB) are a group of bacteria that ferment carbohydrates to produce lactic acid. Widely distributed in the intestines of humans and animals, as well as in food and the environment, LAB have attracted considerable attention due to their unique biological functions. Research demonstrates that LAB can inhibit the growth and metabolism of pathogenic bacteria in the gastrointestinal tract, maintain intestinal microecological balance, reduce endotoxin and serum cholesterol levels, improve nutrient digestibility, modulate immune responses, prevent diarrhea and other intestinal diseases, and mitigate intestinal oxidative stress damage.

Oxidative stress is a biological process resulting from an imbalance between oxidation and antioxidant systems, leading to elevated free radical levels that damage macromolecules such as DNA, lipids, and proteins [1-2]. Free radicals in organisms include superoxide radical ($O_2^{-}\cdot$), hydrogen peroxide (H_2O_2), hydroxyl radical ($\cdot OH$), singlet oxygen ($^1O_2$), hydroperoxyl radical ($HOO\cdot$), alkyl hydroperoxide (ROOH), alkyl peroxyl radical ($ROO\cdot$), alkoxyl radical ($RO\cdot$), hypochlorite ion (ClO^-), ferryl ion ($Fe^{4+}O$), perferryl ion ($Fe^{5+}O$), nitric oxide (NO), and others. Excessive free radicals can induce various intestinal diseases including enteritis, intestinal motility disorders, and increased mucosal permeability, thereby affecting growth and development [3-6].

1.1 Factors Inducing Oxidative Stress in the Intestine

As the primary site for nutrient absorption and an important immune and endocrine organ, the intestine is highly susceptible to oxidative stress induced by

environmental changes. Evidence indicates that altered nutrient composition (such as high-protein diets), antigens, pathogenic bacteria, mycotoxin contamination, and heavy metal pollution can all trigger intestinal oxidative stress. Reactive oxygen species (ROS) damage depends on both free radical content and antioxidant capacity. Under normal conditions, the antioxidant system rapidly scavenges ROS (Figure 1 [Figure 1: see original paper]), maintaining homeostasis without causing oxidative damage. However, when ROS production exceeds scavenging capacity, the dynamic balance is disrupted, leading to ROS accumulation and intestinal oxidative stress injury. Numerous studies have identified mitochondrial respiration byproducts, degradation products of macromolecules like lipids and proteins, metabolites produced during macrophage phagocytosis of viruses, and the cytochrome P450 system as major sources of ROS in the intestine [7-9]. These findings demonstrate that multiple factors can induce intestinal oxidative stress, with these factors varying according to changes in intestinal contents.

Abbreviations: NADPH (reduced nicotinamide adenine dinucleotide phosphate); $NADP^+$ (nicotinamide adenine dinucleotide phosphate); Cyt C (cytochrome C); SOD (superoxide dismutase); GSH (glutathione); GSH-T (total glutathione); GSH-R (R-glutamyl cysteine); GS-SG (oxidized glutathione); GSNO (S-nitrosoglutathione); HbO_2 (oxyhemoglobin).

Figure 1 Diagram of partial free radical scavenging pathways in mammalian cells

1.2.1 Damage to the Intestinal Mucosal Mechanical Barrier

The intestinal mucosa represents the largest interface between the body and external environment, making it vulnerable to attack by pathogenic microorganisms, pro-inflammatory factors, and antigens. The mechanical barrier prevents external damage to the small intestine and reduces mucosal permeability to maintain intestinal health [10-11]. This barrier consists primarily of tight junction proteins, a monolayer of epithelial cells, and the mucus layer. The small intestine contains four cell types: absorptive cells, goblet cells, enteroendocrine cells, and Paneth cells. Absorptive cells comprise 90% of intestinal epithelial cells and are responsible for nutrient absorption. Goblet cells secrete mucins and peptides that are crucial for barrier maintenance and repair. Enteroendocrine cells coordinate nutrient uptake, digestion, and absorption as part of the diffuse neuroendocrine system. Paneth cells produce lysozyme that degrades and digests intestinal bacteria, endotoxins, and antigens, thereby protecting the intestine. Epithelial cell junctions include tight junctions, gap junctions, adherens junctions, and desmosomes. Tight junctions seal the spaces between adjacent epithelial cells, preventing luminal substances from entering the internal environment via paracellular routes. The mucus layer forms a protective film on the epithelial surface that lubricates and nourishes intestinal epithelial cells while blocking direct attack by digestive enzymes, bacteria, and endotoxins. Carrasco [12] demonstrated that decreased tight junction proteins increase intestinal bar-

rier permeability. Davies [13] detected ROS in duodenal ulcer patients. Shaikh et al. [14] found that free radical scavengers promote intestinal wound healing. Barbadoro et al. [15] reported increased ROS during human small intestinal mucosal damage and inflammation, suggesting oxidative stress involvement in intestinal injury and inflammatory responses. Yu [16] observed that oxidants damage intestinal barrier function and affect absorption. These studies indicate that excessive ROS disrupts tight junction protein expression and intestinal barrier function, potentially impairing absorption and overall metabolism.

1.2.2 Damage to the Intestinal Immune Barrier

The intestinal immune system prevents invasion by pathogenic microorganisms and maintains epithelial immune function. During oxidative stress, free radicals participate in immune responses and cause intestinal damage. Lamina propria lymphocytes are the primary site for immune response and effector functions, promoting secretory immunoglobulin A (S-IgA) production to exert immune functions [17]. Li et al. [18] found that S-IgA reduces intestinal inflammation and inhibits interferon-γ production to maintain barrier function. Other studies have shown that intestinal oxidative stress decreases IgA, IgG, and IgM levels, reduces T cell numbers, and causes abnormal cellular immunity [17]. Thus, excessive ROS promotes intestinal inflammation and damages the immune barrier.

1.2.3 Damage to the Intestinal Biological Barrier

The intestine harbors 10^{13}–10^{14} commensal microorganisms that secrete bacterial toxins and promote intestinal motility, forming a complex ecosystem. Under normal conditions, the intestinal microecological balance is maintained as beneficial bacteria such as LAB and bifidobacteria adhere to epithelial cells, inhibiting pathogen colonization and invasion. Elevated ROS disrupts this balance, allowing pathogens like *Escherichia coli*, *Streptococcus faecalis*, *Staphylococcus*, and *Pseudomonas aeruginosa* to enter the internal environment through intercellular spaces, triggering inflammatory responses. Inflammatory factors then activate macrophages to produce ROS during phagocytosis, which inhibits LAB colonization and function, further exacerbating biological barrier damage [18]. Hayashi et al. [19] found that 5-fluorouracil disrupts intestinal flora, alters permeability, and damages barrier function. These findings suggest that ROS affects not only microbial adhesion to epithelial cells but also nutrient metabolism between microbes and the host, ultimately damaging the biological barrier.

2.1 Effects on Intestinal Mucosal Barrier Function

Lactobacillus promotes intestinal health by regulating microbial balance. Under normal conditions, LAB and other flora maintain a stable intestinal microecosystem through mutual restriction and dependence. During oxidative stress, this homeostasis is disrupted, leading to pathogen overgrowth, increased permeability, and impaired nutrient absorption. Yoda et al. [20] reported that LAB inhibits pathogenic damage to intestinal epithelial cells and improves volatile

fatty acid profiles. Wang [21] found that LAB reduces oxidative damage in human colon epithelial HT29 cells. Smits et al. [22] demonstrated that LAB inhibits apoptosis induced by 4-nitroquinoline-1-oxide. Nuobariene et al. [23] showed that LAB promotes intestinal motility and maintains barrier function. These findings indicate that LAB possesses antimicrobial activity, can inhibit ROS damage to epithelial cells, suppress apoptosis, and maintain mucosal barrier function.

2.2 Effects on Intestinal Immune Function

Intestinal flora influences the development of a more stable mucosal immune system. Studies show that germ-free animals have fewer lymphocytes and underdeveloped lymphoid tissues, indicating that intestinal flora promotes immune system development and maturation. Lactobacillus can induce anti-inflammatory factors in epithelial cells, reducing inflammation and protecting the intestine from damage. Xu et al. [24] demonstrated in newborn piglets that LAB increases T cell numbers and enhances mucosal immunity. Chen et al. [25] reported that LAB promotes mucus secretion by goblet cells, maintains tight junction protein structure and function, and inhibits pathogenic damage to epithelial cells. Therefore, intestinal LAB plays a crucial role in host immune system development by stimulating both specific and non-specific immune responses for disease resistance, though the underlying mechanisms require further investigation.

2.3 Effects on Intestinal Flora

Beneficial and pathogenic bacteria in the animal intestine interact to maintain internal stability. When beneficial bacteria predominate, animal health is promoted; conversely, disease susceptibility increases. As one of the most extensively studied probiotics, Lactobacillus produces short-chain fatty acids that lower intestinal pH and inhibit pathogen growth. Additionally, LAB metabolites can kill or inhibit various Gram-positive bacteria, promote nutrient absorption, and exert antioxidant effects. As a dominant flora in the animal digestive tract, LAB prevents pathogen invasion and reduces production of fecal odor compounds such as 3-methylindole (skatole), indole, ammonia, and cresol, thereby reducing environmental pollution [3]. The protective effects of LAB are closely related to their colonization and adhesion to epithelial cells [26]. Chen et al. [27] found that *Lactobacillus salivarius* effectively reduces *Salmonella* colonization in chicken intestines, decreasing intestinal damage. Xiao et al. [28] reported that *Lactobacillus acidophilus* DHA adheres to porcine ileal, colonic, and cecal epithelial cells with higher adhesion rates than *Lactobacillus bulgaricus* and *Streptococcus thermophilus*. Bao et al. [29] showed that *Lactobacillus plantarum* inhibits *Helicobacter pylori* adhesion to Caco-2 cells, preventing oxidative stress injury. Wang et al. [30] found that *Lactobacillus bulgaricus* significantly inhibits pathogenic *E. coli* adhesion to chicken embryo intestinal epithelial cells. These findings demonstrate that LAB plays a vital role in improving gastrointestinal function and maintaining microbial balance. Although numerous studies high-

light the importance of inhibiting pathogen colonization for intestinal health, the molecular mechanisms underlying LAB-mediated inhibition require further investigation.

3.1 Reactive Oxygen and Free Radical Scavenging Systems

Reactive oxygen and free radical scavenging systems prevent or eliminate cellular damage. Murtaza et al. [31] showed that LAB-fermented soy milk effectively scavenges free radicals. Xin et al. [32] demonstrated that LAB reduces non-alcoholic fatty liver disease in obese mice by attenuating inflammation and mitochondrial injury. Hu et al. [33] found that LAB scavenges oxidative effects of divalent metal ions. Chen et al. [34] isolated two LAB strains from fermented foods and confirmed their antioxidant activity, showing that LAB enhances free radical scavenging capacity and prevents oxidative damage. These studies indicate that intestinal LAB participates in the host antioxidant system to scavenge ROS and reduce oxidative injury.

3.2 Redox Regulation Systems

The redox regulation system in LAB comprises three components: the thioredoxin system, NADH oxidase/NADH peroxidase system, and glutathione reduction system. In the intestine, LAB are partially lysed, releasing intracellular contents. Most LAB contain superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) that exert antioxidant effects when released. Ebel et al. [35] studied *Lactobacillus fermentum* ME-3 isolated from healthy infants and found it contains reduced glutathione and manganese-SOD (Mn-SOD) that eliminate hydroxyl radicals, demonstrating its effectiveness as a cellular antioxidant. Itoh et al. [36] showed that *Lactococcus lactis* possesses SOD activity that reduces oxygen radical-induced cellular damage. Liu et al. [2] demonstrated that bifidobacteria scavenge free radicals by increasing SOD activity and reduced glutathione content while blocking the conversion of ROS to hydroxyl radicals. These reports indicate that active substances produced during normal LAB metabolism synergistically prevent ROS-induced cellular damage, with mutual protective effects among system components. Deficiency in any member can cause irreversible cellular damage and disease.

3.3 Oxidative Damage Repair Systems

Oxidative damage repair systems directly or indirectly repair damaged cellular components, primarily by regulating expression of repair-related proteins at the DNA level. Guo et al. [37] suggested that intermediate or final metabolites of probiotics may possess antioxidant activity, representing one potential mechanism. Fu [38] showed that dietary LAB supplementation reduces oxidative stress-induced DNA damage, potentially preventing cancer. Zhang [39] demonstrated that LAB handles oxygen radicals using high manganese concentrations and SOD. Tian [40] found that LAB reduces toxicity of oxygen radicals and

H_2O_2 through reduced glutathione. These findings confirm that LAB possess strong antioxidant capacity, though this varies among strains.

Normal intestinal function depends on the integrity of the mechanical, immune, and biological barriers. Oxidative stress damages barrier function, reduces immune capacity, and disrupts the dynamic balance between probiotics and pathogens, leading to intestinal dysfunction and secondary injury. Numerous studies confirm that LAB exhibit various antioxidant activities and modulate specific and non-specific immune responses. These effects are associated with LAB' s influence on intestinal flora structure, epithelial permeability, and tight junctions, thereby maintaining intestinal health and normal immune function. Recent advances in metabolomics, genomics, and proteomics provide new insights and research tools for exploring the absorption and antioxidant mechanisms of LAB-derived antioxidant substances in vivo.

References

[1] YANG Yuhong, BAI Ming. Preliminary discussion on antioxidant mechanism of lactic acid bacteria[J]. China Dairy, 2011(7): 68-73.

[2] LIU Jing, HUANG Shanshan, ZHAO Zheng. Research progress on antioxidant capacity of lactic acid bacteria[J]. China Dairy Industry, 2010(5): 38-41.

[3] ZHANG Yang, YUAN Jieli. Effects of intestinal probiotics on immune function[J]. Chinese Journal of Microecology, 2003, 15(4): 246-248.

[4] ZHANG W G W, SUN Z, SUN T, et al. PCR Screening and sequence analysis of iol clusters in *Lactobacillus casei* strains isolated from koumiss[J]. Folia Microbiologica, 2010, 55(6).

[5] SUN Z H, LIU W J, ZHANG J C, et al. Identification and characterization of the dominant lactic acid bacteria isolated from traditional fermented milk in Mongolia[J]. Folia Microbiologica, 2010, 55(3): 270-276.

[6] ZHANG Y, DU R T, WANG L F, et al. The antioxidative effects of probiotic *Lactobacillus casei* Zhang on the hyperlipidemic rats[J]. European Food Research and Technology, 2010, 231(1): 151-158.

[7] LAFFLEUR F, PSENNER J, SUCHAOIN W. Permeation enhancement via Thiolation: in vitro and ex vivo Evaluation of hyaluronic acid-cysteine ethyl ester[J]. Journal of Pharmaceutical Sciences, 2015, 104(7): 2153-2160.

[8] ZHENG Xuefang, LIU Bo, LAN Jianglin, et al. Study on the biocontrol effect of microbial fermentation bed on pathogenic *Escherichia coli* in pig houses[J]. Scientia Agricultura Sinica, 2011, 44(22): 4728-4739.

[9] MA Aiguo, COLLINS A R, DUTHIE S J. Analysis of oxidative DNA damage and repair capacity in different cells[J]. Carcinogenesis, Teratogenesis & Mutagenesis, 1997, 9(3): 138-142.

[10] HUANG Canghai, QIAO Shiyan, LI Defa, et al. Study on probiotic *Lactobacillus* inhibiting *Escherichia coli*[J]. Chinese Journal of Animal Science, 2003, 39(6): 27-28.

[11] CHEN Lu, SU Mingxing, LIU Bo, et al. Optimization of culture conditions for probiotic LPF-2 inhibiting *Escherichia coli*[J]. Chinese Agricultural Science Bulletin, 2009, 25(16): 13-16.

[12] CARRASCO D. Uttered from the heart: guilty rhetoric among the Aztecs[J]. History of Religions, 1999, 39(1): 1-31.

[13] DAVIES N M. An clearer portrait observational studies?[J]. Epidemiology, 2015, 26(4): 505-508.

[14] SHAIKH M, RAJAN K, FORSYTH C B, et al. Simultaneous gas-chromatographic urinary measurement of sugar probes to assess intestinal permeability: use of time course analysis to optimize its use to assess regional gut permeability[J]. Clinica Chimica Acta, 2015, 442: 24-32.

[15] BARBADORO P, ANNINO I, PONZIO E, et al. Fish oil supplementation reduces cortisol basal levels and perceived stress: a randomized, placebo-controlled trial in abstinent alcoholics[J]. Molecular Nutrition & Food Research, 2013, 57(6): 1110-1114.

[16] YU Qinghua. Study on the regulation of intestinal epithelial cell tight junctions[D]. PhD Thesis. Nanjing: Nanjing Agricultural University, 2009: 78-90.

[17] ZHANG Qijin, SUN Tiansong, MENGHE Bilige, et al. Study on the effects of lactic acid bacteria preparation on IgG in mouse serum and SIgA in intestine[J]. Heilongjiang Animal Science and Veterinary Medicine, 2006(9): 7-9.

[18] LI Chao, WANG Chunfeng, YANG Guilian. Research progress on intestinal adhesion and immunomodulatory effects of exopolysaccharides from lactic acid bacteria[J]. Food Science, 2014, 35(11): 314-318.

[19] HAYASHI A, KIMURA M, NAKAMURA Y, et al. Anti-atopic dermatitis effects and the mechanism of lactic acid bacteria isolated from Mongolian fermented milk[J]. Journal of Dairy Research, 2009, 76(2): 158-164.

[20] YODA K, SUN X, KAWASE M, et al. A combination of probiotics and whey proteins enhances anti-obesity effects of calcium and dairy products during nutritional energy restriction in aP2-agouti transgenic mice[J]. British Journal of Nutrition, 2015, 113(11): 1689-1696.

[21] WANG Shumei. Screening of anti-colon cancer functional probiotics and study on their mechanism of inducing HT-29 cell apoptosis[D]. PhD Thesis. Harbin: Harbin Institute of Technology, 2014: 90-110.

[22] SMITS H H, ENGERING A, VAN DER KLEIJ D, et al. Selective probiotic bacteria induce IL-10-producing regulatory T cells in vitro by modulating den-

dritic cell function through dendritic cell-specific intercellular adhesion molecule 3-grabbing nonintegrin[J]. Journal of Allergy and Clinical Immunology, 2005, 115(6): 1260-1267.

[23] NUOBARIENE L, CIZEIKIENE D, GRADZEVICIUTE E, et al. Phytase-active lactic acid bacteria sourdoughs: isolation and identification[J]. LWT-Food Science and Technology, 2015, 63(1): 766-772.

[24] XU Yigang, CUI Lichun, GE Junwei, et al. Co-expression of CSFV T cell epitope E290 peptide and PPV VP2 protein in *Lactobacillus casei* and detection of specific antibodies in immunized mice[J]. Acta Microbiologica Sinica, 2007, 47(4): 667-672.

[25] CHEN Delong, ZHU Hongliang, XU Guangyong, et al. Effects of lactic acid bacteria on focal adhesion kinase phosphorylation and tight junction protein occludin expression in IPEC-J2 cells[J]. Acta Veterinaria et Zootechnica Sinica, 2013, 44(2): 283-288.

[26] LI Pengcheng. Study on the mechanism of *Lactobacillus acidophilus* S-layer protein antagonizing intestinal pathogen adhesion or invasion to host cells[D]. PhD Thesis. Nanjing: Nanjing Agricultural University, 2011: 89-100.

[27] CHEN Liyuan, SHI Jinzhou, LI Yan, et al. Identification of a lactic acid bacterium against *Salmonella pullorum*[J]. Journal of Anhui Agricultural Sciences, 2005, 33(11): 2072-2073.

[28] XIAO Rong, WANG Yuanliang, LI Zongjun. Research progress on adhesion of probiotic lactic acid bacteria[J]. Food and Fermentation Industries, 2008, 34(5): 134-137.

[29] BAO Weichen, CHEN Xia, SHAO Yuyu, et al. Research progress on symbiotic mechanism between *Lactobacillus bulgaricus* and *Streptococcus thermophilus*[J]. China Dairy Industry, 2011, 39(12): 20-23.

[30] WANG Ting, WANG Renjun, ZHOU Jing, et al. Isolation and screening of lactic acid bacteria inhibiting *Escherichia coli* from chicken intestine[J]. China Animal Husbandry & Veterinary Medicine, 2012(1): 121-124.

[31] MURTAZA N, BABOOTA R K, JAGTAP S, et al. Finger millet bran supplementation alleviates obesity-induced oxidative stress, inflammation and gut microbial derangements in high-fat diet-fed mice[J]. British Journal of Nutrition, 2014, 112(9): 1447-1458.

[32] XIN J G, ZENG D, WANG H S, et al. Preventing non-alcoholic fatty liver disease through *Lactobacillus johnsonii* BS15 by attenuating inflammation and mitochondrial injury and improving gut environment in obese mice[J]. Applied Microbiology and Biotechnology, 2014, 98(15): 6817-6829.

[33] HU Xiaoli, SUN Jin, LE Guowei, et al. Study on hydroxyl radical scavenging by antioxidant lactic acid bacteria in vitro colon environment[J]. Chinese Journal of Microecology, 2009, 21(6): 488-492, 496.

[34] CHEN Wei, TIAN Fengwei, ZHAO Xin, et al. Research progress on lactic acid bacteria intervention in oxidative stress[J]. Journal of Chinese Institute of Food Science and Technology, 2012, 12(11): 1-7.

[35] EBEL M E, AWE O, KAPLAN M H, et al. Diverse inflammatory cytokines induce selectin ligand expression on murine CD4 T cells via p38 α MAPK[J]. The Journal of Immunology, 2015, 194(12): 5781-5788.

[36] ITOH M, SASAKI H, FURUSE M, et al. Junctional adhesion molecule (JAM) binds to PAR-3: a possible mechanism for the recruitment of PAR-3 to tight junctions[J]. The Journal of Cell Biology, 2001, 154(3): 491-498.

[37] GUO Jinling, ZHENG Qiuhong, LIU Xiang, et al. Antibacterial test of lactic acid bacteria and its culture against pathogenic *Escherichia coli* in chickens[J]. China Feed, 2008(8): 34-36.

[38] FU Longyun. Study on oxidative stress resistance and aerobic growth of lactic acid bacteria[D]. PhD Thesis. Weihai: Shandong University, 2013: 230-245.

[39] ZHANG Juan. Study on the regulatory mechanism of glutathione on stress resistance of lactic acid bacteria[D]. PhD Thesis. Wuxi: Jiangnan University, 2008: 369-378.

[40] TIAN Fengwei. Study on screening, characterization and functional evaluation of lactic acid bacteria for alleviating oxidative stress[D]. PhD Thesis. Wuxi: Jiangnan University, 2012: 456-469.

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