Alcohol-Poisoning Symptoms

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Lactic Acid Bacteria (LAB) are recognized as “probiotics.” The word “probiotics” is defined as “the live microorganisms conferring a health benefit on the host when administered in adequate amounts”, and probiotic LAB strains traditionally have been used to manufacture fermented foods. It has been reported that some LAB cells and fermented foods containing the bacteria have potent health benefits, such as promoting intestinal homeostasis, possessing anti-allergic properties, and preventing and improving obesity.

Keywords: alcohol-poisoning symptoms ; Lactobacillus plantarum ; live lactic acid bacteria

1. Introduction

Lactic acid bacteria (LAB) are recognized as “probiotics.” The word “probiotics” is defined as “the live microorganisms conferring a health benefit on the host when administered in adequate amounts”[1], and probiotic LAB strains traditionally have been used to manufacture fermented foods. It has been reported that some LAB cells and fermented foods containing the bacteria have potent health benefits, such as promoting intestinal homeostasis, possessing anti-allergic properties, and preventing and improving obesity[2][3][4][5][6][7][8][9].

We have found that some strains stored in our plant-derived LAB library may be useful for preventive medicine, such as immune modulation, reduction of obesity, and anti-allergy[10][11][12][13]. Interestingly, it has been demonstrated through the clinical study carried out by our group that the yogurt manufactured with *Lactobacillus plantarum* SN13T stored in the library reduces significantly the serum γ-glutamyl transpeptidase (γ-GTP) value[14].

During the animal study using mice fed with ethanol, it has been observed that when live SN13T cells were orally administered to mice fed with a diet containing ethanol, death caused by the intake of ethanol was completely avoided. The result demonstrates that recovery from alcohol-poisoning symptoms in mice was observed only with oral administration of the live cells.

Figure 1 shows the survival curves of mice reared using an ethanol-containing diet supplemented with or without SN13T cells. When compared with a group without the intake of ethanol, the cumulative survival rate of the ethanol-intake group without the added live SN13T cells was significantly decreased ($p < 0.001$, Figure 1a), and the mice died within 17 days. However, with simultaneous administration of live SN13T cells, the survival rate of the mice did not decrease when compared with a group receiving no alcohol (Figure 1b), strictly, although only one mouse died. In contrast, the survival rate ($p = 0.362$, Figure 1c) was not improved under supplementation with the heat-killed cells, indicating that there is an obvious difference between the live and heat-killed cell groups ($p < 0.001$, Figure 1d). Thus, the administration of live SN13T cells is essential for improving alcohol-poisoning symptoms.

Figure 1. The Kaplan–Meier survival curves of C57BL/6J mice fed an ethanol-containing diet with or without the addition of live SN13T cells. The group fed without ethanol was also compared. The *p*-values were calculated using the log-rank test. Group I, which was fed only an L10015 diet without the administration of ethanol; group II, which was fed an L10016 diet with ethanol; groups III and IV, which were fed with the simultaneous administration of ethanol and the live or heat-killed SN13T cells, respectively, in the L10016 diet. Each graph shows the comparison of survival rate (%) between Groups I and II (a), II and III (b), II and IV (c), and III and IV (d), respectively.

We also analyzed the intestinal microbiota of mice fed an ethanol-containing diet supplemented with or without live SN13T cells. The intestinal microbiota from mice fed with the ethanol-containing diet without bacterial cells was obviously different from that of mice fed with a diet supplemented without both ethanol and the bacterial cells. In the cecum, the ratios of *Akkermansia*, *Allobaculum*, and Paraprevotellaceae were remarkably decreased by the administration of ethanol, but these phenomena were disappeared with the simultaneous administration with live SN13T cells. On the other hand, the ratio of an RF32 order was notable increased in ethanol-fed group. Interestingly, when live SN13T cells were administered simultaneously with ethanol, the population of RF32 was clearly decreased.

2. Discussion

The metabolite profiles in the cecum were clearly different between ethanol-fed and nonethanol-fed groups. Change in the cecum components, which has a buffering effect against alteration of the intestinal microbiota, may affect the homeostasis. The metabolite analysis showed that putrefactive amines, such as tyramine and cadaverine, were increased in the ethanol-fed group. In addition to putrefactive amines, a rise in the volume of isovaleric acid and valeric acid content was also observed. These compounds are generated by the hydrolysis of protein in the putrefaction process of tissues and organs and cause an offensive odor. These results reveal that the production of compounds related to putrefaction was promoted during ethanol abuse. Therefore, that putrefaction and the observed mouse death are considered to be due to ethanol abuse. The undesirable effects were repressed by the intake of live SN13T cells. Since it is not evaluated at this moment what species and reactions are involved in the putrefaction with ethanol, further studies on the function of indigenous bacteria such as the RF32 order to intestinal microbiota, is necessary to confirm the mechanisms. As a clue, there is an obvious relationship among colonic damage, inflammation, and abundance of the RF32 order[15]. The research supports our hypothesis that the intake of live SN13T cells restores the inflammation caused by ethanol abuse *via* changing the intestinal microbiota.

We have demonstrated that alcohol-poisoning symptoms are improved by the oral administration of live SN13T cells but not by the administration of heat-killed cells. Based on these results, it is presumed that live SN13T cells or their collaboration with other intestinal bacteria may generate bioactive compounds. The bioactive compounds may

cooperatively inhibit the synthesis of harmful products in the intestinal tract. Some research has shown that a change in the intestinal microbiota is involved in lifestyle and mental diseases[16][17]. It is demonstrated that live SN13T cells are significant for maintaining intestinal microbiota balance and restoring from the symptoms of alcoholism.

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