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Original Article

Yeast hydrolysate as a functional anti-obesity ingredient: appetite suppressive effects of yeast hydrolysate in food-deprived mice

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Summary. The aim of this study was to investigate the appetite suppression effects of yeast hydrolysate (YH). Male ICR mice (8 weeks old) were randomly divided into three groups (n=6) as follows: Saline group, which was treated with saline (control); YH0.5 group, which was treated with YH 0.5 g/kg BW; and YH1 group, which was treated with YH 1 g/kg BW. At the beginning of the experiment, the mice were intraperitoneally (IP) injected with either saline or YH. The results showed that YH caused a significant attenuation of food intake in fasted mice ($p<0.05$) and significantly lowered serum ghrelin levels (YH0.5, 2002.22 pg/mL; YH1, 2337.65 pg/mL vs. Saline, 3363.61 pg/mL, $p<0.05$). This study indicates that the appetite suppression effects of YH is likely explained by the attenuation of ghrelin.

Key words: Yeast hydrolysate, ghrelin, appetite, obesity

Introduction

Yeast naturally rich in minerals, vitamins, amino acids and proteins, plays a fundamental role in human food and nutrition. Yeast extract has high potential as a source of biologically active molecules and functional food ingredients (1). The small proportion of yeast extract (mainly containing peptides below 10 kD) and yeast hydrolysate (YH), which could be related to satiety, has been industrially purified from *Saccharomyces cerevisiae* by protein hydrolysis (2). Recently, YH has formed a large and growing dietary market as a useful anti-obesity supplement (3). It was reported that YH could suppress weight gain in obese animal models and assist in weight loss in obese humans (3-5). To elucidate the mechanisms of YH in fighting obesity, a distribution of neurotransmitters was investigated in the hypothalamus of rats treated with YH using histochemical methods (6, 7). YH was found to alter appetite-related neurotransmitters in the central nervous system (CNS). Although the exact mechanisms for the anti-obesity effects of YH are not fully understood, these studies support the idea that YH might induce weight loss through appetite control via the CNS. To confirm the hypothesis that YH suppresses appetite, we observed food intake by animal subjects for several hours after YH treatment in food-deprived mice. We also investigated whether YH treatment might affect serum appetite-related hormone levels.

Materials and methods

Preparation of yeast hydrolysate (YH)

Saccharomyces cerevisiae IFO 2346 was incubated in medium containing 2% molasses, 0.6% $(NH_4)_2SO_4$, 0.1% $MgSO_4 \cdot 7H_2O$, 0.2% KH_2PO_4, 0.03% K_2HPO_4,

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and 0.1% NaCl for 3 days at 30°C. After incubation, the culture was centrifuged at 10,000 × *g* for 20 min. The cells were suspended with 20 mM phosphate buffer (pH 7.0) and hydrolysed with bromelain at 30°C for 4 hr. The hydrolysate was subsequently centrifuged at 10,000×*g* for 20 min. The supernatant was then passed through a 10 kDa molecular weight screening membrane (Sartocon cassette, Sartorius, Germany). The fractions with peptides smaller than 10 kDa were freeze-dried.

Appetite suppression study

The experimental protocol was reviewed and approved by the Korea University Animal Care Committee. Male ICR mice were obtained at 8 weeks of age from Daehan Biolink (Cheongju, Korea). The mice were randomly divided into three groups (n=6) as follows: Saline group, which was treated with saline (control); YH0.5 group, which was treated with YH 0.5 g/kg BW; and YH1 group, which was treated with YH 1 g/kg BW. Food was withdrawn for 12 hr before the experiment. At the beginning of the experiment mice were intraperitoneally (IP) injected with either saline or YH. Venous blood was collected 30 min post-injection from the tail vein. Serum ghrelin and leptin levels were measured by enzyme immunoassay using a Bio-Plex Pro Mouse Diabetes kit (Bio-Rad Co., CA, USA). Pre-weighed food jars were then introduced into the cages and a sheet of clean white paper was placed under each cage to collect any spillage. The amounts of food consumed after 1, 2, and 4 hr post-injection were determined. The experiment began at approximately 10:00 a.m. so that all weighing was done during the light period of the day-night cycle (the lights were on between 8.00 a.m. and 6.00 p.m.).

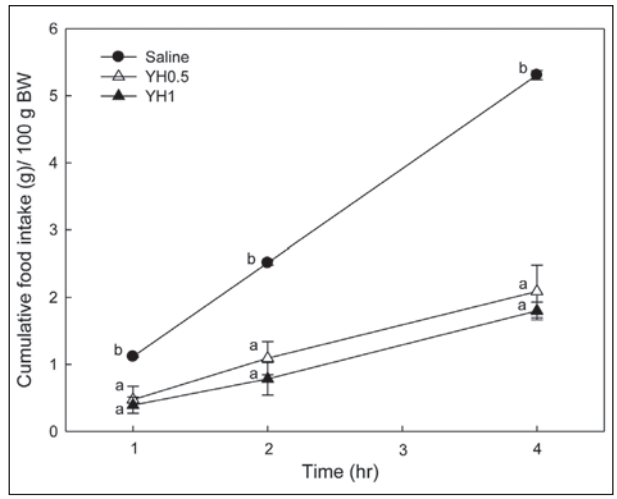

Figure 1. Cumulative food intake of ICR mice for 4 hr following intraperitoneal injection of yeast hydrolysate (YH) after 12 hr of food deprivation. The values are the means±SD for 6 mice. Means with different superscript letters are significantly different at p<0.05 according to Tukey's multiple range tests. Saline, group treated with saline (control); YH0.5, group treated with YH0.5 g/kg BW; YH1, group treated with YH 1 g/kg BW.

Statistical analyses

All statistical analyses were performed with the Statistical Package for Social Sciences ver. 12.0 (SPSS, IL, USA). The differences between groups were statistically evaluated by one-way analysis of variance (ANOVA) and Tukey's multiple tests. All data were two-sided with a 5% significance level and were reported as mean ± standard deviation (SD).

Results and discussion

Figure 1 shows the cumulative food intake during a 4 hr period after YH treatment following 12 hr of

Table 1. Serum leptin and ghrelin levels of ICR mice following the intraperitoneal injection (IP) of yeast hydrolysate after 12 hr of food deprivation

Parameters	Serum hormone level (pg/mL)		
	Saline	YH0.5	YH1
Leptin	371.50±28.33	402.64±21.23	476.66±42.05^{NS}
Ghrelin	3363.60±394.85^{b}	2002.22±724.58^{a}	2337.65±269.21^{a}

The values are the means±SD for 6 mice. Means with different superscript letters are significantly different at p<0.05 according to Tukey's multiple range tests. Saline, group treated with saline (control); YH0.5, group treated with YH 0.5 g/kg BW; YH1, group treated with YH 1 g/kg BW. NS, not significant.

food deprivation. YH caused a significant attenuation of food intake in fasted mice; when compared to the vehicle control (Saline group), YH significantly reduced food intake at all time points ($p<0.05$). However, dose-dependent results were not observed; there was no significant difference between YH0.5 and YH1. Serum leptin and ghrelin levels were measured at 30 min after YH injection (Table 1). YH tended to evaluate serum leptin level without a significant difference. YH significantly lowered serum ghrelin levels (YH0.5, 2002.22 pg/mL; YH1; 2337.65 pg/mL vs. Saline, 3363.61 pg/mL, $p<0.05$). However, there were no dose-dependent differences between YH0.5 and YH1 groups in terms of serum appetite-related hormone levels.

The results of this study on food intake agreed with a recent clinical study showing that the reduction of food intake in YH group was significantly greater than in the vehicle group (5). Faipoux et al. (2) investigated the suppression effects of yeast protein on food intake. They found that rats fed a high yeast protein load reduced their next meal and daily food intake more than rats fed any other well-balanced, amino acid, high protein load or wheat starch diet. They also reported on a preliminary study of gastric emptying in rats receiving yeast protein loads showing that yeast protein was emptied more rapidly through the pylorus than total milk protein during a meal, which may induce satiety. They concluded that yeast proteins enhance satiety more than other proteins. Catiau et al. (1) reported the satiating potential of yeast extract, which is rich in hydrolysed yeast proteins, particularly in terms of food intake. They suggested that the yeast extract had the satiety activity that could be attributable to hormone secretion, which would imply a decrease in food intake and a decrease in weight. As shown in Table 1, YH significantly inhibited the ghrelin secretion relative to that of the vehicle. The ghrelin is produced mainly by the stomach before the meal and acts on the hypothalamus to trigger the sensation of hunger (8). The satietogenic mechanism of YH could also be related to the reduction in ghrelin, which is known as an orexigenic hormone that stimulates appetite.

In conclusion, this study indicates that the appetite suppression effects of YH is likely to be explained by the ghrelin attenuation; it was believed that YH would diminish the sensation of hunger by reducing the secretion of orexigenic factors such as ghrelin that send satiety signals to the brain, terminating food intake in the short term. These observations suggest that YH may be useful in controlling food intake. However, additional investigations are required to determine the chemical identity of the bioactive functional constituents of YH on appetite suppression (2-6).

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