

Antitumor Polysaccharides from Edible Mushrooms and Immunomodulating Action Against Murine Macrophages

Masashi Mizuno,¹ Sachiko Kawakami,¹ Takashi Hashimoto,¹ Hitoshi Ashida,¹ and Kenichiro Minato²

¹Graduate School of Science and Technology, Kobe University, Kobe 657-8501, Japan, and ²Miyagi Agricultural College, Sendai 982-0215, Japan

Epidemiological studies have indicated that almost all cases of cancer are associated with environmental factors including foods. Mushrooms have recently become attractive as health-beneficent foods and as a source material for the development of drugs. Many investigators have isolated and identified antitumor polysaccharides from mushrooms. The antitumor activity of these polysaccharides is caused by the potentiation of the immune response, which involves lymphocyte activation. Thus, antitumor polysaccharides are focused on as immunopotentiators. We have improved the enzyme-linked immunosorbent assay (ELISA) to easily and exactly quantify the antitumor polysaccharides in mushrooms. It has been ascertained that ELISA assay using antibody was available to measure the contents of antitumor polysaccharide. In this study, the contents of an antitumor polysaccharide, lentinan, from *Lentinus edodes* (Berk.) Sing. during storage were measured by ELISA. Moreover, the relationship between the contents and the effects on cytokine production of tumor necrosis factor- α (TNF- α) and nitric oxide (NO) were investigated.

When the mushrooms were stored at low temperature (1°C), the contents of their antitumor polysaccharides and the production of TNF- α and NO showed hardly any changes, but their contents and production decreased markedly at a higher temperature (20°C). These results suggest that low-temperature storage is more effective in maintaining the contents of antitumor polysaccharides and the quality of the mushrooms as health-beneficent foods. The time course of the production of TNF- α and NO from murine macrophages stimulated with lentinan indicated that TNF- α production occurred 8 hr earlier than NO production. When macrophages were simultaneously incubated with lentinan and an anti-TNF- α antibody, NO production was completely inhibited. Neither NOC-18 (NO donor) alone nor L-N-monomethylarginine (NO synthetase inhibitor) with lentinan affected TNF- α production. Taking into consideration these results, it was ascertained that NO production from macrophages was due to the autocrine pathway through TNF- α production.