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Research Article

Selenium-Enriched Yeast Alleviates Oxidative Stress-Induced Intestinal Mucosa Disruption in Weaned Pigs

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To explore the e□ect of selenium-enriched yeast (SeY) on intestinal barrier functions in weaned pigs upon oxidative stress, a 2 × 2 factorial design was utilized and thirty-two pigs were randomly assigned into four groups. Pigs with or without exposure to oxidative stress (diquat challenge) were fed with a basal diet or a SeY-containing diet. The trial lasted for 21 days, and result showed that SeY supplementation attenuated body-weight reduction and significantly decreased the serum concentrations of diamine oxidase (DAO) and D-lactic acid in pigs upon diquat challenge ($P < 0.05$). Diquat challenge decreased the villus height and the ratio of villus height to crypt depth (V/C) in the jejunum and ileum ($P < 0.05$). However, SeY supplementation not only elevated the villus height and the ratio of V/C ($P < 0.05$) but also improved the distribution and abundance of tight-junction protein ZO-1 in the jejunum epithelium. Interestingly, SeY supplementation acutely decreased the total apoptosis rate of intestinal epithelial cells in pigs upon diquat challenge ($P < 0.05$). Moreover, SeY elevated the content of antioxidant molecules such as glutathione peroxidase (GSH-Px) and catalase (CAT) but significantly decreased the content of malondialdehyde (MDA) in the intestinal mucosa ($P < 0.05$). Importantly, SeY elevated the expression levels of critical functional genes such as the nuclear factor erythroid-2-related factor 2 (Nrf2), heme oxygenase-1 (HO-1), sodium/glucose cotransporter 1 (SGLT1), and B-cell lymphoma-2 (BCL-2) in the intestinal mucosa upon diquat challenge ($P < 0.05$). Moreover, the expression of caspase-3 was downregulated by SeY in the duodenum and jejunum mucosa ($P < 0.05$). These results indicated that SeY attenuated oxidative stress-induced intestinal mucosa disruption, which was associated with elevated mucosal antioxidative capacity and improved intestinal barrier functions.

1. Introduction

Intestinal barrier mainly consists of a single layer of enterocytes and intercellular tight junctions of enterocytes. It serves as a selective permeable membrane that is not only responsible for nutrient digestion and absorption but also constitutes the first line of defence to prevent a variety of harmful substances from entering the intestinal mucosa and systemic circulation [1]. Moreover, the gastrointestinal tract also serves as the biggest immune organ for animals [2]. A variety of stimuli such as bacterial infections and stresses may impair the integrity of the intestinal barrier. For instance, overproduction of reactive oxygen species (ROS) not only promoted secretion of inflammatory cytokines but also induced intestinal mucosal damage and dysfunction [3]. Evidence is accumulating to show that overproduction of ROS may result in DNA damage and intestinal cell apoptosis, which increases the intestinal permeability and facilitates translocation of luminal antigens into subepithelial tissues leading to a series of intestinal and systemic inflammatory response [4]. Therefore, the avenue to alleviate the ROS-induced disruption of the intestinal barrier has attracted considerable research interest worldwide.

Selenium (Se) is an essential trace element for animals. Importantly, Se is a critical component of the enzyme glutathione peroxidase (GSH-Px), which detoxifies lipid peroxides and provides protection of cellular and subcellular membranes against ROS damage [5]. As compared to