Camel whey protein improves oxidative stress and -histopathological alterations in lymphoid organs through Bcl XL/Bax expression in a streptozotocin-induced type 1 diabetic mouse model

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Abstract:

Type I diabetes (T1D) is a characterized by the inflammation of pancreatic islets and destruction of cells. Long and persistent uncontrolled diabetes tends to degenerate the immune system and increase the incidence of infections in diabetic individuals. Most serious diabetic complications are mediated by the free radicals, which damage multiple cellular components through direct effects of the cell cycle regulatory proteins. Camel whey protein (CWP) has antioxidant activity and decreases the effects of free radicals. However, the effects of CWP on lymphoid organs have not been studied in the context of diabetes. Therefore, the present study was designed to investigate the dietary influence of CWP supplementation on the lymphoid organs in streptozotocin (STZ)-induced type 1 diabetic mouse model. Three experimental groups were used: non diabetic control mice, diabetic mice, and diabetic mice treated with CWP. Induction of diabetes was associated with a marked reduction in glutathione (GSH) levels; decreased activities of GSH peroxidase (GSH Px), manganese superoxide dismutase (MnSOD) and catalase; increased reactive oxygen species (ROS) levels and iNOS activity in plasma and lymphoid organs. Furthermore, diabetic mice exhibited alterations in the expression of Bax and Bcl-XL, and subsequently pathological alterations in the architecture of the bone marrow, pancreas, thymus, and spleen. Interestingly, treatment of diabetic mice with CWP robustly restored glucose, insulin, GSH, and ROS levels and the activities of GSH Px, MnSOD, catalase and iNOS. Additionally, supplementation of diabetic mice with CWP improvement in the architecture of lymphoid tissues and rescued from apoptosis through direct effects on the Bax and Bcl-XL proteins. These data revealed the therapeutic potential of CWP against diabetic complications mediated damages of lymphoid organs.

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