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Effects of exosomes derived from *Trichinella spiralis* infective larvae on intestinal epithelial barrier function

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Abstract

Muscle larvae of *Trichinella spiralis* parasitize the host intestinal epithelium. The mechanisms of exosomes participating in the invasion of *T. spiralis* muscle larvae are unclear. Hence, the purpose of this study was to explore the effect of exosomes derived from *T. spiralis* infective larvae (*Ts*Exos) on the barrier function of porcine small intestinal epithelial cells (IPEC-J2). First, *Ts*Exos were successfully obtained, and their ingestion by epithelial cells was validated. Furthermore, the optimal induction condition was determined by the CCK8 kit, and we found that exposure to 150 μg/mL *Ts*Exos for 12/24 h decreased the viability of IPEC-J2 cells by 30%. Based on this outcome, the effects of *Ts*Exos on cell biological processes and tight junctions were studied. After coincubation of *Ts*Exos and IPEC-J2 cells, the results showed a significant increase in the content of FITC-dextran and in the levels of lactate dehydrogenase (LDH) and reactive oxygen species (ROS). The rate of apoptosis increased by 12.57%, and nuclear pyknosis and nuclear rupture were observed. After the cells were induced by *Ts*Exos, the expression of IL-1 was upregulated, but the expression of IL-10, TGF-β, TLR-5, MUC-1 and MUC-2 was downregulated. *Ts*Exo induction also led to a decrease in the levels of ZO-1, CLDN-3, and OCLN. In conclusion, *Ts*Exos are involved in several cellular biological processes, and they function by disrupting physiological and biochemical processes, hyperactivating innate immunity, and damaging tight junctions.

Keywords: Exosomes, *Trichinella spiralis*, barrier function, tight junction

Introduction

Trichinella spiralis is a zoonotic parasitic disease found worldwide [1]. In the intestinal infection stage of *T. spiralis*, it invades multiple cells by sensing appropriate ligands [2]. The process of invasion may subsequently lead to cell membrane rupture and cytoplasmic destruction, thereby inducing apoptosis and causing changes in membrane permeability [3, 4]. Additionally, to resist the invasion of *T. spiralis*, the host activates the autoimmune system; however, its overreaction can be harmful to the host itself [3].

Recent studies have reported that most parasites can secrete exosomes that can deliver their cargo to host cells upon ingestion [5–9]. Exosomes derived from parasites, when transferred to host cells, can create a conducive microenvironment for parasite immune escape by regulating host cell proliferation, cell signalling pathways, and gene expression [10, 11].

Song et al. found that serine proteases and cysteine proteases in the excretory/secretory proteins (ESPs) of *T. spiralis* destroyed gut epithelial integrity by degrading tight junction proteins and played key roles in *T. spiralis* invasion, growth and survival in the host [12]. As an important component of ESPs, exosomes are involved

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