

News & Comments

Artemisinin on Mastitis Caused by *Escherichia coli* in Mice and Bovine Mammary Epithelial Cells*Yawei Song*

One of the most prevalent illnesses on dairy farms is bovine mastitis, an inflammatory disease of the mammary tissue brought on by mechanical irritation, pathogenic bacteria, and chemical and physical damage. Mammary gland defence mechanisms can be divided into nonspecific immunity and specific immunity. In the initial stages of infection, nonspecific immunity, also known as innate immunity, serves as the primary defence mechanism. *E. coli* is common in the natural environment and does not often infect people or animals, although it can do so when those people or animals have weakened immune systems. In addition, a recent study hypothesized that the pathogenicity of *E. coli* in bovine mammary glands is linked to the development of a brand-new pathogenic phenotype called mammary pathogenic *Escherichia coli* (MPEC).

Solarbio provided Artemisinin (Beijing, China). We purchased DMEM/high-glucose medium from Servicebio in Wuhan, China. Inoculated *E. coli* (ATCC25922) was placed on LB agar and incubated at 37 °C. Because it is simpler to manipulate and less expensive than other experimental animals, the mouse mastitis model is thought to be a good choice for studying bovine mastitis. It also offers important insights into the pathogenic mechanisms of bovine mastitis. The female mice treated with 50 mg/kg of artemisinin by oral gavage once daily for three days after the onset of mastitis was induced in the mice were the same as the mice treated with *E. coli*.

After being fixed with 4% formalin for 72 hours, the fresh tissues were dehydrated by being soaked in gradient alcohol. After neutral resin had been applied, the slices were examined under an optical microscope. *E. coli* is extensively present in the environment and frequently infects dairy cow udder tissues, resulting in inflammation. It is regarded as one of the main mastitis-causing agents. The extensive use of antibiotics has led to a gradual rise in *E. coli* resistance. We were able to better grasp whether artemisinin could replace antibiotics for the prevention and treatment of mastitis thanks to the authors' investigation into the impact of the drug on *E. coli*-induced mastitis and their examination of the associated inflammatory signalling pathways.

Artemisinin and its derivatives have demonstrated strong effectiveness against malaria, but as research advances, reports of its anti-inflammatory properties are increasingly emerging. Previous research has demonstrated that artemisinin increased survival when mice were attacked by heat-inactivated *Staphylococcus aureus* by decreasing TLR2 expression and activating NF- κ B and did so in



a dose-dependent way. Furthermore, it was shown that *E. coli* infection activated the inflammasomes NLRP3 and NLRC4 in bovine mammary epithelial cells, mediating the inflammatory response. The interaction between LPS and TLR4 activated NLRP3 and caused the NLRP3 inflammasome to develop with ASC proteins. Caspase-1 shearing, and the creation of active IL-1 and IL-18 were caused by the inflammatory enzyme.

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KEYWORDS

Mastitis; *E. coli*; artemisinin; MAC-T cells; mice

