MAST CELLS IN GASTROINTESTINAL HELMINTH INFECTION

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Trichinellosis is a human disease caused by infection with parasitic nematode *Trichinella spiralis*. It is estimated that more than one billion people are infected by the Trichinellosis. Infection in the intestine by *T. spiralis* is associated with a mastocytosis, an increased number of mast cells. These cells have been shown to be an essential role for successful worm expulsion of gastrointestinal worms through the release of a number of mediators, which provide a central function in host protection against these parasites. The function of mast cells in the expulsion of *Trichinella spiralis* has been investigated using mast cell deficient C-kit mutant models W/W^v. In addition to mast cell deficiency these mice have a number of other abnormalities including anaemia and a lack of interstitial cells of Cajal. Therefore, our aim is to examine if the observations of C-kit models could be replicated in other models of mast cell deficiency.

To investigate the role of mast cells in parasitic infection, W^{sh} mice (C57BL6 background) and Mas-TRECK mice (BALB/c background) were infected with 400 larvae *Trichinella spiralis.* W^{sh} mice are a natural mutant in which has an inversion mutation in regulatory elements upstream of C-kit element that is the receptor for Stem Cell Growth Factor (SCF) and have fewer abnormalities than W/W^v. Mas-TRECK mice are a novel strain in which genetic modification that diphtheria toxin receptor (DTR) is inserted into the intronic enhancer (IE) of *IL4*.

The progression of infection and immune responses generated were examined by counting numbers of worms, analysis of intestinal pathology and cytokine production along with antibody responses. The expulsion of *Trichinella spiralis* from Mas-TRECK mice was observed to be delayed in comparison to the background strain while W^{sh} mice found to be delayed parasite expulsion of *Trichinella spiralis* with significantly higher than those of C57BL/6 strains and was observed to mount a greater immune response against *T. spiralis* than the background strain with increased IgE antibody responses. Analysis of mucosal mast cell number in the small intestine and levels of mMCP-1 in the serum suggested that mast cells may not be completely

ablated in Mas-TRECK mice following treatment with DT may not be completely mast cell-deficient. Therefore, further studies are required to evaluate the benefits of different mast cell deficient strains; particularly estimation of other abnormalities may potentially affect results.

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