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Cysticercosis: immunology and immunotherapy

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SUMMARY

Taenia solium cysticercosis is an important cause of human neurological disease in many developing countries. Porcine cysticercosis contributes to economic hardship and completes the parasite life-cycle. In humans and pigs cysticerci usually live within host tissues without causing inflammation or disease. The mechanisms of immune evasion by living cysticerci may include sequestration within immunologically privileged sites; antigenic shifts; molecular mimicry of host-like antigenic determinants; masking of cysticercal antigens by host immunoglobulins; and modulation of host immune responses. However, the degeneration of one or more cysticerci is associated with granulomatous inflammation which in humans may result in transient or progressive symptoms.

Cysticercosis is a disease of poverty. Public health and animal husbandry measures have eradicated the disease from developed countries but are difficult to apply in endemic regions where pigs are usually reared on a subsistence basis. In contrast to preventative measures, an inexpensive *treatment* for infected pigs may be an effective way of controlling the parasite and preventing human disease because it may double the value of cysticercotic pigs, providing an incentive for widespread use. Immunotherapy with cysticercal antigens may cause degeneration of cysticerci, potentially curing porcine cysticercosis. Our blinded randomised placebo-controlled study assessed the efficacy and safety of immunotherapy in 28 naturally parasitised pigs. Four weight-matched groups were inoculated with purified cysticercal antigen, crude cysticercal antigen with Freunds adjuvant, adjuvant alone or saline alone. Immunotherapy was well tolerated but had no effect upon the macroscopic appearance or histology of cysticerci. Most of the pigs given crude antigen plus adjuvant developed new antibody bands on electro-immuno transfer blot and the crude antigen caused a significant increase (from 10% to 34%, $p<0.04$) in the proportion of cysticerci that failed to evaginate and were therefore not viable for causing human infection. Although immunotherapy with cysticercal antigens caused a statistically significant fall in the viability of cysticerci, this immunological reaction was not great enough to be of value in preventing human disease.

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