THE IMMUNOLOGY OF THE HOST-PARASITE RELATIONSHIP IN *Taenia solium* CYSTICERCOSIS: IMPLICATIONS FOR PREVENTION AND THERAPY

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INTRODUCTION

Cysticercosis is a parasitic disease that results from ingestion of the microscopic eggs of the *Taenia solium* tapeworm. These eggs contaminate the environment in endemic areas and pigs which ingest them become intermediate hosts for the parasite by developing pea sized *T. solium* larvae in their tissues, a condition called porcine cysticercosis. Pigs usually remain healthy despite this parasitosis, but their meat is greatly reduced in value. In the Andean region of Peru, up to 50% of pigs have cysticercosis, contributing to economic hardship and malnutrition.[1] When humans eat infected pork, the *T. solium* larvae hatch out in the bowel where they develop into adult tapeworms which can grow up to 8 m in length. These intestinal tapeworms produce eggs which are released into the environment in the feces, completing the parasite life-cycle. Humans are, therefore, the natural definitive host to the adult tapeworm.

Infection of pig tissues with *T. solium* larvae and of the human bowel with adult tapeworms constitute the natural life-cycle which is essential for the continuing existence of the parasite in communities. This propagation depends upon evasion or modulation of host immunity because the larval and adult parasites must survive without killing their hosts and without being destroyed by host immune responses. The evolution of the parasite would be expected to select parasites which most efficiently parasitize pig tissues and human intestines and which cause minimal disease in these hosts.

The principal significance of the *T. solium* parasite is, however, its ability to infect human tissues as an 'accidental' intermediate host; when humans in-

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gest microscopic *T. solium* eggs, they develop cysticercosis. Human cysticercosis is, therefore, caught from humans with tapeworms and not from pigs. The presence of *T. solium* larvae in human tissues is an important cause of neurological disability and mortality in many developing countries, including 0.5 to 4% of people in Peru[2] and Mexico.[3] This human cysticercosis is a 'dead end' for the parasite and there has, therefore, been no evolutionary pressure for the *T. solium* larvae to evade immune recognition in human tissues. The human host does not have persistent tolerance for the larval stage of the parasite and the resultant immune recognition and inflammation may contribute greatly to the pathogenesis of human cysticercosis.

THE IMMUNOLOGY OF ASYMPTOMATIC CYSTICERCOSIS

The pig is the natural intermediate host for *T. solium* larvae and multiple cysticerci may survive within pig tissues. In some endemic regions, examination of pigs tongues for palpable living larvae before purchase has been routine practice since biblical times because this reliably diagnoses cysticercosis.[1] The usual absence of apparent illness in infected pigs is remarkable considering that thousands of cysticerci are often found at autopsy, scattered throughout neurological and other tissues.

In humans, autopsies of victims of warfare and road traffic accidents have revealed that a large proportion (typically 80%) of neurocysticercosis infections are asymptomatic, discovered incidentally at necropsy (reviewed by Gemmell *et al*, 1988[4]). Living cysticerci may occasionally cause disease through local pressure effects or by obstructing the flow of cerebrospinal fluid, despite the absence of a host inflammatory response. In contrast, symptomatic cysticercosis is usually associated with inflammation around one or more degenerating cysticerci.

The immune response to cysticerci has been studied mainly because of the need for a diagnostic blood test. Although the literature is confused by numerous serologic tests evaluated in different ways, it is clear that virtually all cases of symptomatic cysticercosis are associated with a detectable humoral immune response, the exception being a minority of single-cyst infections. Imaging studies with radio-labeled antibodies have also demonstrated that these circulating anticysticercus antibodies have access to the surface of cysticerci.[5] Furthermore, sero-epidemiological studies in endemic regions have revealed a similar rate of antibody positivity in healthy people to the prevalence of asymptomatic cysticercosis suggested by autopsy series.[2]