1. INTRODUCTION
Cysticercosis is caused by *Cysticercus cellulosae*, the larval form of *Taenia solium*. It occurs in human when it becomes an unintended intermediate host in the life cycle of the parasite. In humans the most common routes of infection is ingestion of *T. solium* eggs from contaminated food and also less frequently by fecal-oral autoinfection from patients harboring the adult parasite in their intestines. While the cysts develop in any human tissue, they have a predilection for the central nervous system (CNS), skeletal muscle, subcutaneous tissue, and eyes (Ehrenfried et al., 2001). When cysticerci develop in brain and spinal cord, the condition that arises is called as neurocysticercosis (NCC), similarly that of eye is called as ophthalmic cysticercosis. Ophthalmic cysticercosis may be ocular or extra ocular cysticercosis depending upon the location of the parasite in the eye or its adnexa. Both NCC and ophthalmic cysticercosis are pleomorphic in their presentation due to individual differences in the number, size, and location of the parasite, as well as differences in the severity of the host's immune reaction to the parasite (Yancey et al., 2005).

Cysticercosis has a wide geographic distribution particularly in areas of prevalence with low socio-economic status, poor hygiene and where pig farming is common. It is endemic in Mexico, Central and South Africa, Central and South America, India, and some areas of Far East (Del Brutto and Sotelo, 1988). In an earlier WHO estimate, approximately 50,000 deaths occur due to NCC annually throughout the world, with no less than 20,000 million people infected with cysticerci (Schantz et al., 1983). Cases of NCC are also increasingly reported from developed countries because of increased migration and travel (Garcia and Del Brutto, 2005).

### 1.1. The parasite

The life cycle of *T. solium* occurs between man and pig; man acting as the definitive host harboring the adult tapeworm whereas pig as an intermediate host. Leucart was the first to describe the life cycle of the parasite in 1856. He demonstrated that man acquires infection by ingestion of undercooked pork harboring the cysticerci. A four-cup-shaped suckers and double row of hooklets attaches its scolex to the intestinal wall and develops into sexually mature adult worms during a period of 62 days to 72 days.
The adult *T solium* inhabits the upper jejunum of small intestine in human. The adult *Taenia* measures 2-3 meters in length and has a scolex, neck and segments. The scolex is muscular, provided with hooklets, which causes attachment of the scolex to the intestinal mucosa and hence characteristically known as “armed tapeworm”. It is round, measuring 1mm in diameter and has four suckers and is armed with a conspicuous rostellum. The latter consists of two rows of small and large hooks, alternating with each other. The neck is short about 5mm in length. The strobila consists of 800-1000 segments or proglottids. The mature segments are wider than they are long and contain one set of male and female reproductive organs. Whereas, the gravid proglottids are longer than they are broad (12mm by 6mm). The gravid segments of *T. solium* consist of 150-200 testes, an ovary situated in the posterior side without an accessory lobe, and a vaginal opening which lacks a muscular sphincter. The common genital opening is present laterally in the middle of each segment alternating between the right and left side. The gravid segment consists of a median longitudinal stem showing branched structures on each side of the segment. The uterus gets completely filled with eggs and each gravid segment contains nearly 2-5 X 10^4 eggs (Parija, 2004). The gravid segments, after being detached from the intestine and the eggs, both are excreted out in the faeces.

The eggs are brown colored; round shaped and measure 41μm to 43μm in diameter. The eggshell encloses the hexacanth larva, which is infective stage to both pig and human. The eggs remain viable up to 8 weeks during which they remain infectious for pig and man.

Pig, as the natural intermediate host, swallows the eggs along with the excreta where the ova develop into oncospheres (hexacanth larvae) in the intestine and get released within 24 hours to 72 hours. The released hexacanth larvae are carried by circulation *via* the blood stream to different tissues and organs of the body. At these sites, the embryo remains embedded in the musculature and develops to the metacestode or bladder worm, *C. cellulosae* that requires 9 weeks to 10 weeks for complete development.

The larva of *T. solium* is a small, semitransparent, opalescent white bladder like structure and elongate oval in shape measuring 5 to 18μm in breadth and 0.6 to 1.8 cm in length. The
bladder is filled with fluid rich in albumin and salts. The metacestode contains a small-invaginated head, the scolex, which has a ring of suckers and also hooks.

Occasionally man becomes infected in the same way as pig by eating food or drinking water contaminated with eggs (Figure-1). The eggs hatch in the intestine of human and subsequently are carried by the circulatory system. Finally, the development of *Cysticercus* larva takes place in various tissues and organs of man leading to a serious disease known as cysticercosis. The commonest target organ is the CNS; the cerebral grey matter, the meninges and the ventricles are being the common sites, but the spinal cord is a rare site. Muscles followed by subcuticular tissues, eyes and more rarely the heart, lungs, kidney and liver are the other tissues affected in cysticercosis (Parija, 2004).

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**Figure-1** Life cycle of *Taenia solium*, the pork tapeworm [source- http://www.dpd.cdc.gov/dpdx]
1.2. Transmission of infection to human

A lack of an effective universal public sanitation system, poor personal hygiene and unsupervised pig-farming industry are important factors that contribute to the continued presence of this otherwise eradicable disease in developing countries (Schantz et al., 1993). Humans acquire *T. solium* intestinal taeniasis by ingestion of raw or undercooked pork containing *C. cellulosae* but acquire cysticercosis by:

1. Ingestion of food and water contaminated by the eggs present in the infectious faeces of a *Taenia* carrier. Infection is also acquired through fecal contamination of water or soil where vegetables are grown and eaten improperly washed. Thus, amongst many Indians who are vegetarians and do not consume pork, this appears to be the common mode of infection.

2. Anus-hand-mouth transfer of the eggs by contaminated hands of persons with poor personal hygiene (*exogenous auto infection*).

3. More rarely when the proglottids or ova regurgitate into the stomach by reverse peristalsis i.e., *Taenia* eggs released from *T. solium* gravid segments are thrown back to the duodenum, where they hatch and cause tissue infection (*endogenous auto infection*) (Schmidt and Roberts, 2001).

1.3. The disease

1.3.1. Neurocysticercosis

NCC has traditionally been treated as a single disease entity. However, the clinical presentation, pathogenesis and optimal treatment of NCC vary depending on the number and location of the lesions within the brain and the nature of the host inflammatory response. The parasite burden in an individual may vary from one to hundreds of cysts of differing ages (Yancey et al., 2005).
In brain, the oncospheres of the parasite may burrow into the brain parenchyma, meninges, ependyma and choroid plexus. If the oncosphere lodges in the meninges or choroid plexus, it may enter the subarachnoid space or ventricles and may assume a different larval form, i.e. racemose variety, in which the cyst without a protoscolex resembles a bunch of grapes (Del Brutto, 1992). The parasites can be free floating in ventricles (Yancey et al., 2005). Rarely, occlusion of the basal arteries may cause cerebral infarction due to secondary inflammation (Del Brutto, 1992).

The spinal cord involvement is rare but cysticerci can be located in the meninges or within the spinal cord. In the absence of scolex visualization, cysticercotic involvement of the spinal cord or spinal subarachnoid space has a nonspecific appearance on MR imaging (Leite et al., 1997). Spinal cord involvement may vary from 16% to 20% in relation to the brain involvement (Colli et al., 1994). In case of spinal cord infections the morphological features are not different from that of brain, but the inflammation is more severe (Del Brutto and Sotelo, 1988).

**Clinical signs and symptoms of NCC**

Clinical manifestations of NCC depend on the number, size, and location of the cysticerci, host immune responses and sequelae of previous infestations. Seizure is the most common symptom. The other clinical symptoms are: symptoms of increased intracranial pressure, chronic meningitis, abnormal psychiatric manifestations, encephalitis and radiculomyelopathy involving the spinal cord and its covering membranes.

**Seizure:** Human NCC is a major cause of epileptic seizures and other neurologic morbidity worldwide (Garcia et al., 2005). Recurrent seizures may occur at any stage of the disease. The seizures may be generalized or focal, and the focus identified by visual, olfactory, hallucinatory, amnesic or other varieties of aura. Recurrent seizure may be the only symptom (McCormick et al., 1982).

**Raised intracranial pressure:** The hallmark of extraparenchymal NCC is increased intracranial pressure (Yancey et al., 2005). Patients with cysticerci in the cerebral ventricles...
typically present with obstructive hydrocephalous that may be due to a valve effect obstructing flow of CSF. Patients with cysticerci in the subarachnoid space, the basilar cisterns, or fissures may present with seizures from coexisting parenchymal disease or obstructive hydrocephalous from ventricular disease. They may also present with mass effect, communicating hydrocephalous, or vasculitis and strokes (Yancey et al., 2005).

**Psychiatric manifestation:** Psychiatric manifestation is a rare presentation of NCC, which may be seen up to 5% of patients (Mahajan et al., 2004). Progressive loss of intellect and memory, change in behavior, confusion and disorientation are the common psychiatric symptoms. Manic depression, psychoneurosis and psychopathic behavior may lead to misdiagnosis of NCC. Although it is regarded as a rare cause of dementia, mild cognitive impairment may be a much more prevalent neuropsychological feature of patients with NCC. The extent to which organic mechanisms related to brain lesions may underlie the mental changes is yet unclear, although the similar sex distribution of patients with or without depression, as well as the above mentioned correlations, provides further evidence of the part played by organic factors in the cause of these syndromes (Forlenza et al., 1997). A high prevalence of cysticercosis in the psychiatric patients, compared with healthy individuals has been observed in a psychiatric institution in the state of Tachira (Venezuela) wherein the authors have postulated that cysticercosis could be the origin of the psychiatric disorders of these patients (Meza et al., 2005).

**Focal signs:** Focal signs are more commonly seen in case of involvement in the cerebral hemisphere than the hindbrain. Hemi-paresis, dysphasia, visual disturbance and cognitive impairments are more frequently detected. The basal meninges are less commonly involved. Various cranial nerve palsies including optic atrophy, hypothalamic and pituitary disorders and an acute neurological deficit such as hemiplegia is seen in cerebral infarction. Paraplegia and other signs are seen in spinal cord involvement due to extra or intra-medullary compression (Mathuriya et al., 2001).

**Cysticercal meningitis:** Chronic meningitis is a rare manifestation of NCC with the symptoms of headache, vertigo, vomiting, and papilloedema, an altered level of consciousness and gait disturbances (Sotelo et al., 1985). However, in one study,
approximately 50% of patients were found presenting cysticerci-meningitis (Rogel-Ortiz and Vera-Pedro, 1997).

Encephalitis: Encephalitis is another clinical manifestation of the parenchymal form of the disease. It may be impossible to differentiate from viral and other forms of encephalitis. This was previously thought to occur in children, but now also described in adults, particularly in young females (Rangel et al., 1987).

Cerebral infarction: Occlusion of small terminal arteries due to vasculitis may lead to multiple parenchymal infarctions. Large infarcts secondary to middle cerebral artery occlusion also have been described (Wraige et al., 2003).

1.3.2. Ophthalmic cysticercosis

Ocular cysticercosis is commonly seen in the Indian subcontinent. Sub-conjunctival space is the most common site of infection in intraocular cysticercosis (Malik et al., 1968). Lacrimal gland and eyelids are less commonly involved in cysticercosis (Sen, 1980). Anterior chamber cysticercosis is unusual (Das et al., 2002). Extraocular muscle (EOM) form is the commonest type of adnexal cysticercosis in the orbit. Subconjunctival space is the common site, followed by the eyelid, optic nerve, retro-orbital space and lacrimal gland (Pushker et al., 2002).

The subretinal space is considered to be another common site of intraocular cysticercosis where the parasite enters through the posterior ciliary arteries (Bartholomew, 1975). This reaction is probably initiated by the release of toxins released from the parasite and is usually accentuated on its death. 91% of the cysts were found to be localized in subretinal space or in the vitreous, in a study reported by Topilow et al., (1981). Intravitreous or subretinal cysts usually lead to blindness within three years to five years unless the parasite is surgically removed from the eye (Junior, 1949). The mature larva in the vitreous becomes encapsulated and may remain alive for years (Cano, 2001). Viable Cysticercus induces a mild to moderate inflammatory response, whereas, dead parasite induces a severe inflammatory reaction (Hutton et al., 1976).
Very few cases of optic nerve cysticercosis are reported in the literature and most of these cases being reported from India (Menon et al., 2000; Gulliani et al., 2001; Verma et al., 2002; Bajaj and Pushker, 2002). Association of orbital cysticercosis with systemic cysticercosis is quite uncommon (Bajaj and Pushker, 2002).

**Clinical signs and symptoms of ophthalmic cysticercosis**

Ophthalmic cysticercosis is initially asymptomatic but in the later stage symptoms are apparent due to increase in cyst size or destruction of eyeball by the chronic inflammatory reaction. Wide varieties of manifestations are seen in ocular cysticercosis. These include localized edema, pain, blepharitis, blepharoptosis, conjunctivitis, subconjunctival abscess, extraocular muscle myositis, restricted ocular motility, proptosis, uviitis, optic neuritis or atrophy, papilloedema, retinal breaks, chorioretinal scars, exudative or rhegmatogenous retinal detachment (Cano, 2001; Sharma et al., 2003)

The clinical manifestations of orbital or adnexal cysticercosis are entirely different and depend on the location, size, relation to adjacent structures and stage of evolution of the cyst. Some of the parasitic factors are involved in ocular cysticercosis i.e. modulation of the immune response of the host, mediated by larval products (a soluble RNA-peptide, some metacestode surface sphingoglycolipids) seems to occur in vivo (Andriantsimahavandy et al., 1996).

**1.4. Immunity**

There is no evidence of any acquired immunity to the adult stage of *T. solium* and *T. saginata*. However, immunity does develop to the larval stages and it is possible to induce immunity to these stages with crude or defined antigens. Immunity is stage specific and onchosphere antigens induce protection against the earliest larval stages but, if these survive, there is no protection against the later larval stages. Conversely, antigens derived from cysticerci induce immune responses against late stages but not the early ones (Chavarria et al., 2003).
The immune responses elicited by cysticerci in naturally infected pigs are classified into four separate stages based on a histological examination of metacestodes in different stages of viability or degeneration (Alvarez et al., 2002). In stage I the parasites are surrounded by a thin layer of collagen type I, and in the stage II there is a sparse inflammatory infiltrate. In stage III, granuloma formation is evident, and in the stage IV the parasite is surrounded by an eosinophil-rich infiltrate and degenerated vesicular membrane. The final stage, IV, is detected mainly in the heart but not in the brain. The granulomatous reaction in swine resembles to that in human patients, but differs by the abundance of eosinophils, the relative paucity of plasma cells, and the discrete deposition of collagen (Alvarez et al., 2002).

The mechanisms of immunity are not at all clear but there is evidence that both eosinophils and antibodies are involved thereby suggesting antibody dependent cell cytotoxicity (ADCC) (Chavarria et al., 2003). The cytokine profile indicates the involvement of T helper type 1 (Th1) like response in Indian patients with NCC (Grewal et al., 2000). A rural community study in Mexico showed distinct immunological profiles in equally exposed individuals differing in outcome of the infection. NCC is found to be associated with a T helper type 2 cells (Th2) response (IgG4, IL-4, IL-5, IL-13) and in endemic area, higher levels of specific antibodies (IgG1, IgG2, IgG4, IgE) and specific cell proliferation was observed in comparison to subjects from an area with low exposure. This was the first study reporting the immunological profile associated with the asymptomatic form of NCC (Chavarria et al., 2003). The immune response against *T. solium* cysticerci appears to have components of both Th1 and Th2, although the underlying mechanisms are yet to be clarified. The parasite is probably killed by eosinophils, which are attracted to the site by lymphoid cells. It is assumed that this specific response is mediated by Th2 cytokines (Chavarria et al., 2003; Bueno et al., 2004; Chavarria et al., 2005).

The active form of NCC is associated with specific serum immunoglobulins (IgG, IgM, IgE, and IgA) in decreasing order, with the highest values being detected among the cases with intraventricular cysts, or with inflammation signs in CSF or in those with multiple clinical manifestations (Odashima et al., 2002).
The eye has a privileged situation with regard to immunity. It is because of the avascularity of the cornea and the lens, and the physiological selectivity of the blood-aqueous barrier as well as absence of lymphatic channels within the globe itself. In general, the helminth infection of the retina elicits a delayed type hypersensitivity reaction. The absence of reaction in the anterior chamber is due to the free mobile nature of the cyst, as well as the lack of immune response to the antigens of *Cysticercus* at this stage (Sachdeva et al., 1995).

1.5. Epidemiology

1.5.1. Epidemiology of neurocysticercosis

The epidemiological data on the prevalence of taeniasis/cysticercosis are limited and an accurate estimation is difficult due to the varied presentation, large number of asymptomatic cases and non-availability of confirmatory diagnosis (Garcia et al., 1991). But on overall, more than 2 million people are estimated to have adult *Taenia* infection world wide, and many more are infected with *Cysticercus* (Garcia et al., 1991). There is a proposal to declare NCC as an international reportable disease (Roman et al., 2000). In 2002, the US Centers For Disease Control and Prevention (CDC) developed a revised strategy for consolidating, enhancing, and improving the effectiveness of CDC's efforts to prevent and control infectious diseases on a global scale. *T. solium* is one example of an imported infectious disease, which has made an impact on the health of the US population but requires international coordinated efforts to prevent or limit transmission from the developing countries endemic for taeniasis/cysticercosis (Schantz and Tsang, 2003).

*Geographical distributions*

*World:* Cysticercosis is endemic in Mexico, Central and South Africa, Central and South America, India, and some areas of Far East (Del Brutto and Sotelo, 1988). Cysticercosis is comparatively rare in Chile, Argentina, and Uruguay although, it is prevalent throughout Latin America (*Figure-2*). Of the 75 million people in Latin America at risk of the disease, 10% develop symptoms (Garcia et al., 2004).
AREAS WHERE CYSTICEROSIS IS ENDEMIC


Seroprevalence studies indicate high rates of exposure to the parasite in several countries. In an epidemiological study in Northern Ecuador, 4.99% cases were found serum antigen positive by a monoclonal antibody based Ag-ELISA, whereas coprological examination was positive in 1.55% cases (Rodriguez-Hidalgo et al., 2003). Human seroprevalence in village ranged from 7.1% to 26.9%. Seroprevalence was higher among individuals with a history of seizures but not in those reporting a history of headache or intestinal taeniasis. Prevalence of taeniasis ranged from 0% to 6.7% in Peru (Garcia et al., 2003a).

NCC is a major cause of epilepsy in Bali (Indonesia), Vietnam, Korea and possibly China and Nepal with rates ranging from 0.02% to 12.6% (Rajshekhar et al., 2003). Rates of taeniasis, as determined by stool examination, have also been reported to range between 0.1% to 6% in certain communities in India, Vietnam, China, and Bali (Indonesia). An astonishingly high rate of taeniasis (50%) has been reported from an area in Nepal populated by pig rearing farmers (Rajshekhar et al., 2003).
A nationwide survey of human parasites in China was conducted during 1988-1992, with coverage of 30 provinces/autonomous regions/municipalities. The overall infection rate of intestinal parasites was 62.6% whereas at provincial level, the highest infection rate (94.7%) was reported in Hainan, and the lowest (17.5%) in Heilongjiang. In Europe, cysticercosis is endemic in Spain, Portugal, and some Eastern European countries, but it is rare in most other countries (Schanz et al., 1992).

Cameroon (Africa) is one of the few countries where the taeniasis-cysticercosis complex has been examined in detail (Zoli et al., 2003). In the Western province of Cameroon, cysticercosis is present in 0.4% to 3% of the local population and it is a major cause of epilepsy with figures as high as 44.6% (Nguekam et al., 2003). In some regions of Nigeria, the prevalence of porcine cysticercosis and human taeniasis is quite high (20.5% and 8.6%, respectively) (Zoli et al., 2003).

*T. solium* cysticercosis is an emerging infectious disease in Papua (Irian Jaya) and Indonesia (Wandra et al., 2003). A preliminary study was carried out using stool examinations for the detection of copro-antigens and adult proglottids after chemotherapy, which was confirmed by mitochondrial DNA analysis using expelled proglottids and metacestodes developed in NOD/Shi-scid mice from eggs of expelled proglottids. Approximately 8.6% of the local population in Kama (5/58), 1 km from the local capital city center, Wamena, were confirmed to harbor adult *T. solium* using these techniques (Margono et al., 2003).

In a 15 year study at WHO/FAO Collaborating Center for Parasitic Zoonoses, Danish Center for Experimental Parasitology, Frederiksberg, Denmark, hospital surveys indicated that cysticercosis is emerging as a serious health problem in the country although most of the information was from the Hanoi area. Surveys for human taeniasis in central and northern provinces indicated a prevalence of 0.2% to 7.2%. In addition to *T. solium*, *T. saginata* and *T. asiatica* are also known to be present in Vietnam (Willingham et al., 2003).

This epidemiological study of cranial CT scans in an endemic rural community in Mexico found that 9.1% of apparently healthy persons had calcified lesions and were completely
asymptomatic. Silent NCC cases did not correlate with the exposure factors tested but showed family aggregation and higher rates of positive serology. Thus, NCC prevalence may be higher than currently considered and host-related factors appear to be involved in infection and pathogenesis (Fleury et al., 2003).

Because of the increasing immigration from endemic areas, such as Mexico, Central and South America, Africa, Asia, Spain, and Portugal, NCC is now considered to be an important disease in the United States (Garcia et al., 2004). Recent studies show prevalence of cysticercosis even in countries where pork consumption is proscribed by religious laws (Al Shahrani et al., 2003; Hira et al., 2004). Cysticercosis, once rare in Australia, is now more frequently diagnosed. This change reflects the countries of origin of new immigrants and the destinations of Australians traveling (Walker et al., 1991). Cysticercosis had been exceedingly rare in Malaysia with only 3 cases of NCC documented one each in 1934, 2001 and 2003 (Abdullah and Nor, 2005). Since NCC is not yet an international reportable disease, the total number of cases diagnosed in the world is not exactly known, yet, the number of reports is increasing each year. In Nepal, the infection is comparatively recently diagnosed. As major hospitals started neurosurgery units, NCC has gradually come to be reported and now it is considered as one of the major food borne parasitic zoonosis in Nepal (Joshi et al., 2003).

India: Several reports of cysticercosis from many countries in Asia such as India, China, Indonesia, Thailand, Korea, Taiwan and Nepal are a clear-cut indicator of the wide prevalence of *T. solium* cysticercosis and taeniasis in these and other Asian countries. However, epidemiological data from community-based studies are sparse and available only for a few countries in Asia (Rajshekhar et al., 2003).

Human cysticercosis is endemic in many areas of India, with predominance in pig rearing areas and lower socio-economic and poor hygiene conditions. In India, NCC is regarded as the second most important cause of intra-cranial space occupying lesions (ICSOL) following tuberculosis (Mahajan, 1982) and is the most common cause of epilepsy (Sawhney et al., 1996). Cysticercosis is observed to be an important cause of epilepsy in up to 50% of Indian patients presenting with partial seizures (Rajashkekar, 2003). Dinakar et al. (1970) reported
epilepsy as the main presenting feature of NCC in South India. Mahajan et al. (1982) reported cysticercosis as a causative agent of seizures in North India. The authors have demonstrated the seropositivity to be higher in patients with focal epilepsy (29.2%) than those with generalized epilepsy (24.2%). A total of 147 cases of NCC were documented from Agassaim village (Goa) by Estibeiro et al., (2000).

NCC is the most frequent and most widely disseminated human neuroparasitosis (Jose, 1997). Clinicians, neurosurgeons and neurologists have reported a number of case studies of NCC mostly from urban neuro centers (Khurana and Jain, 1999; Chandramukhi, 2000; Malla and Mahajan, 2001; Singh et al., 2000).

The prevalence of intestinal taeniasis have been reported to be 0.36% in Pondicherry (Parija and Rao, 1987), 0.9% in Lucknow (Srivastava and Pandey, 1986), 0.8% in Chandigarh (Ramesh et al., 1991) and 0.5% to 15% in northern India (Malla et al., 1992). A stool examination study in Goa showed a prevalence of T. solium infection upto 12% where the residents handle and consume pork (Estibeiro et al., 2000). In a recent prevalence study from rural North India, 38% of the human subjects had intestinal taeniasis and 9.7% had reported seizures (Prasad et al., 2002). In a study in Uttar Pradesh, 2% of 600 human subjects revealed infection by adult T. solium on screening fecal samples (Pathak and Gaur, 1989). However, the comparison of prevalence data from different places is very difficult because no single study protocol is followed in the above studies and also there is no continuous monitoring of the prevalence in any of the places to assess the increase or decrease in the prevalence across years.

Serological studies have shown the presence of Cysticercus antibodies in 29.2% sera from patients of epilepsy and intra cranial space occupying lesions (ICSOL) in Chandigarh (Malla, 2000). In similar studies in Bangalore, presence of Cysticercus antibodies are detected in 18.75% of epilepsy patients, in 13.33% of patients with increased intracranial tension and in 5.47% of children with subacute and chronic meningitis (Chandramukhi, 2000).
In a study from Ludhiana city, Punjab, serum samples from seventeen children (85%) and 14 family contacts (27%) were positive for Cysticercus antibodies by enzyme-linked immunoelectrotransfer blotting (EITB) test using lentil lectin purified glycoprotein (LLGP) antigens. A tendency towards clustering of EITB positive cases within individual families was observed. It was suggested that it might be worthwhile to screen household family contacts of children with small enhancing lesions (SEL) for taeniasis-cysticercosis (Singh et al., 2000).

**Pondicherry:** Pondicherry is a Union Territory of India located on the southern coast of the sea (Bay of Bengal).

The cysticercosis is documented from Pondicherry with an increased frequency. A histopathological study carried out during a period of 10 years (1975-1985) in JIPMER, Pondicherry showed a total of 38 cases of human cysticercosis (Veliath et al., 1985). In another study, a total of 21 childhood NCC cases were reported over five years from 1985 to 1989 (Thakur and Anand, 1991). The information from the Medical Record Department, JIPMER Hospital also shows an increase in the number of patients attending epilepsy clinic. In a recent report, 6.48% of healthy blood donors were detected positive for either anti-Cysticercus antibodies or antigens (Parija et al., 2005).

**1.5.2. Epidemiology of ophthalmic cysticercosis**

Helminth infections of the eye are common in different parts of the world, but some such as cysticercosis, onchocerciasis and ocular toxocariasis are usually encountered only in developing countries (Sabrosa and Zajdenweber, 2002).

In 1829, in Germany, Schoot and Soemmering reported the first case of ocular cysticercosis. In 1864, in Brazil, Dr. Pedraghia described the first case of human C. cellulosae in the eyeball to the 'Academia Nacional de Medicina'. The statistical prevalence of the ocular form in the beginning of the century was mainly due to the emergence of surgical therapy and the possibility of an easy diagnosis than to the scarceness of other forms. Because the ocular cases determine acute and important ophthalmic or painful clinical manifestation, it
was possible to diagnose the processes early and rapidly (Wittig, 2001). Some European statistics, when cysticercosis was epidemiologically frequent in that continent, showed a predominance of ocular form (Vosgien, 1911 and Volovatz, 1902 as cited by Wittig, 2001) whereas Brazilian and American studies in general report the encephalitic localization as the predominant one (Bruck et al., 1994).

Increased research and awareness of various systemic infections places a greater emphasis on the ophthalmologist's knowledge of ocular manifestations of these diseases (Cano et al., 2001). New advances in the diagnosis and treatment, as well as studies of the pathogenesis and histological features of different infectious processes are continually being reported (Wittig, 2001; Rastogi and Jain, 2001; Nash, 2003; Agrawal et al., 2004; Pushker et al., 2004; Sodhi et al., 2004; Sundaram et al., 2004; Mohan et al., 2005; Pushker et al., 2005; Sudan et al., 2005).

Geographical distributions

World: Parasitic infection of the eye is a major health problem in tropical countries. Increasing tourism and the influx of populations from Southeast Asia demand a greater awareness of ocular parasitology (Teekhasaenee et al., 1986). Cysticercosis of the eye has been reported predominantly from *T. solium* endemic areas of the tropics with a high prevalence in India. Recent findings and treatment options regarding ocular manifestations of cysticercosis have increased the awareness of atypical presentations and understanding the pathogenesis of the cysticercosis of the eye (Gabay and Mayers, 1997).

Ophthalmic cysticercosis has been reported from Korea (Seo et al., 1996), Mexico (Cardenas et al., 1992), Thiland (Lerdvitayasakul and Lawtiantong, 1991), Zimbabwe (Mason et al., 1991) and Canada (DiLoreto et al., 1990). Cysticercosis of eye has been reported from Madagascar (Andriantsimahavandy et al., 1996) where, the authors have suggested a modulation of the immune response of the host, mediated by larval products (a soluble RNA-peptide, some metacestode surface sphingoglycolipids). A case of intravitreal cysticercosis causing left uniocular cataract and eventual left visual loss in a healthy female has also been reported from Nigeria (Adegbehingbe et al., 2003).
**INTRODUCTION**

*India:* Ophthalmic cysticercosis is increasingly reported from India and contributes to preventable blindness (David and Mathai, 2000). There are several reports of ophthalmic cysticercosis documented from almost all the parts of India. Cases have been reported from Pondicherry (Rao et al., 1987; Veliath et al., 1985), Chennai (Sharma et al. 2003), New Delhi (Sachdeva et al., 1995; Bajaj et al., 2002; Nainiwal et al., 2002; Pushker et al., 2002; Raina et al. 2002; Verma et al., 2002), Hyderabad (Sekhar and Lemke, 1997; Sekhar and Honavar, 1999), Chandigarh (Gupta et al., 1998), Vellore (David and Mathai, 2000), Alligarh (Agarwal and Amitava, 2000), Rohtak (Gupta et al., 2000), and Mumbai (Ursekar et al., 1998; Natarajan et al., 1999).

In a study from Vellore (South India), retrospective analysis of records of patients presenting to the ophthalmology clinics, with cysticercosis, during years 1990-98 showed a total of 25 patients those who had intraocular cysticercosis (David and Mathai, 2000). The results of a 5-year study of 33 cases of ophthalmic cysticercosis in New Delhi showed the male to female ratio of 2:1 and maximum number of patients (45%) belonged to the age group of 31-40 years. Seventy percent of those patients were of low socio-economic status and 70% were strictly vegetarians. The most common location of cysticerci was in the vitreous (50% of all cases); orbital cysts were present in 5% and subconjunctival cyst in 3%. Most common extraocular site for associated cysticercosis was the brain (18%). There has been a gradual change in the socio-demographic trends of ocular/adnexal cysticercosis (Atul et al., 1995).

*Pondicherry:* Seventeen out of the 38 histopathologically conformed cysticercosis cases were reported to be confined to eyes within a period of 10 years in JIPMER Hospital (Veliath et al., 1985). The first report of acquired inflammatory Brown's syndrome caused by cysticercosis was reported from Pondicherry (Rao et al., 1987). A 24-year-old lady presented with an inflammatory swelling in the supra-nasal quadrant of the left orbit and vertical diplopia. The motility disorder was of typical acquired Brown's syndrome. Histopathological examination of the lesion revealed *C. cellulosae*, a parasitic cyst known to produce a severe inflammatory reaction. Recently, 11 cases of ocular cysticercosis are reported from Pondicherry (Kaliaperummal et al., *In Press*).
1.6. Diagnosis

1.6.1. Diagnosis of NCC

The protean manifestations of NCC make its diagnosis difficult on clinical grounds alone. However, an accurate diagnosis is possible only after suspicion on epidemiologic grounds, proper interpretation of clinical data, and analysis of findings on neuroimaging studies, and specific immunological tests on the cerebrospinal fluid (CSF) and serum. The diagnosis of NCC by any single parameter thus continues to remain difficult.

Study of NCC is usually limited by the difficulty in clearly establishing the specific etiological diagnosis. Hence, Del Brutto has proposed a diagnostic criterion based on clinical, radiological, serological, and epidemiological factors dividing them into absolute, major, minor and epidemiological criteria (Del Brutto et al., 1996). However, the criteria relied heavily on the immunoblot assay as a major criterion, which did not hold the test of time. It has been demonstrated to be sensitive in cases with multiple cysts (94%) but less sensitive with single cysts or calcified lesions (28%) (Wilson et al., 1991). Others criticized it for complexity and difficulties in clinical and epidemiological application in developing countries (Carpio, 1999). However, in the absence of a gold standard for diagnosis of NCC, the criteria of Del Brutto is followed since it is helpful in categorizing the cases of NCC into definitive and or probable groups.

On imaging, identification of a scolex is considered diagnostic of cysticercosis, however, in its absence, other features are non-specific (Jayakumar et al., 2003). A calcified scolex observed by CT scan is diagnostic, but are reported very rarely. Visualization of the eccentrically located invaginated scolex is best possible by MRI. However, first use of MRI may cause the calcific stage to be missed, which is better seen on CT scanning. Moreover, the differential diagnosis with other infectious or neoplastic diseases of the CNS is difficult on the basis of imaging alone (Del Bruto et al., 2001). Nevertheless, one might anticipate certain difficulties in assessing the viability of parenchymal cysticercal lesions by purely morphologic means (Zee et al., 2000).
Thus, a neuroimaging technique of the brain although useful is not considered as a gold standard for diagnosis of NCC (Dorney et al., 2003). The gold standard for diagnosing NCC is pathological confirmation through biopsy or autopsy; unfortunately these procedures have their obvious limitations (Caprio et al., 1998). In pigs results of autopsy and enumeration of the cysts in the carcass may provide a tool for validation of the immunodiagnostic tests, which is impractical in case of human. Hence, serological diagnosis based on antibody or antigen detection may be of immense value to confirm the clinical findings as well as to supplement the diagnosis of radiologically suspected cases of NCC.

Demonstration of specific antibody in serum and CSF by EITB or ELISA continues to be the mainstay in serodiagnosis of cysticercosis. The EITB using purified *T. solium* glycoprotein antigens is used as the immunodiagnostic test of choice and is recommended in routine diagnosis of cysticercosis by WHO and the Pan American Health Organization (PAHO) (Aguilar-Rebolledo et al., 2002; Proano-Narvaez et al., 2002). However, complexity and difficulties in clinical and epidemiological application of the EITB in developing countries is the noted limitation of the test (Wilson et al., 1991; Carpio, 1999). Evaluation of several commercially available ELISA kits are also reported to be of poor specificity (Hira et al., 2004).

The antibody detection has an important drawback in clinical settings: it may indicate exposure to infection and not necessarily the presence of a viable infection, because the serum antibodies may persist long after the parasite has been eliminated by immune mechanisms and/or drug therapy (Harrison et al., 1989; Garcia et al., 2001; Garcia et al., 1997). Hence, detection of parasite specific antigens in the serum or CSF is a useful marker in the diagnosis and prognosis of cysticercosis (Estrada et al., 1989; Tellez-Giron et al., 1989; Choromanski et al 1990; Pardini et al., 2001). And in the seroepidemiological studies of NCC in endemic areas (Parija et al., 2005).

Antigen-capture ELISAs are probably the most reliable and sensitive method for detection of *Cysticercus* antigen in the serum or CSF for of detecting active cases of NCC (Correa et al., 1999). EITB technique although used as a test of choice for detection of *Cysticercus* antibodies, is yet to be evaluated for the detection of *Cysticercus* antigen in the serum or CSF.
for diagnosis of NCC. The EITB technique has been employed earlier in the characterization of secreted parasite antigens in serum and CSF in few studies (Pardini et al., 2001, Epsindola et al., 2002). Hence, in the present study, both ELISA and EITB are being evaluated for detection of *Cysticercus* antigens in various body fluids for diagnosis of NCC.

*Somatic and excretory secretory (ES) antigens*

Traditionally, somatic antigen preparations from various components of the *Cysticercus* cysts have been used for the diagnosis of NCC. Limitation of the use of somatic antigens in the antibody-based diagnostic tests in NCC is that these tests cannot differentiate an active from inactive stage of the parasite. This is due to persistence of serum antibodies for a long period of time, and somatic cysticerci antigens induce immune responses against late or degenerated stages but not the early stages of the cysticerci (Chavarria et al., 2003).

The excretory secretory (ES) antigens are now increasingly used in the pathogenesis as well in immunodiagnosis of helminth infections (Van Kerckhoven et al., 1998; D'Souza and Hafeez, 1999; Espindola et al., 2002; Mollineri et al., 2003). Various serological assays using ES antigens have been evaluated in predicting the parasite burden in human filarial infection (Kaushal et al., 1982; Harnett et al., 1990). ES antigens have also been evaluated with promising results in the diagnosis of porcine cysticercosis (Sciutto et al., 1998; D'Souza and Hafeez, 1999) and bovine cysticercosis (Brandt et al., 1992; Van Kerckhoven et al., 1998). However, studies on *C. cellulosae* ES antigens in the diagnosis of human cysticercosis are few (Ko and Ng, 1998a; Ko and Ng, 1998b; Espindola et al., 2002).

It is suggested that both antibody and antigen detection tests employing the sensitive methods such as ELISA and EITB may be more useful in diagnosis of NCC (Correa et al., 1999). Therefore, in the present study both ELISA and EITB using *C. cellulosae* ES antigens are evaluated, for detection of serum and CSF antibodies; and using polyclonal antibodies raised against the ES antigen is evaluated to detect antigen in the serum and CSF for diagnosis of NCC.
1.6.2. *Urine as a specimen*

The CSF, serum and less commonly saliva are routinely used specimens for immunodiagnosis of NCC. The sensitivity and specificity of the tests using these specimens depend on the antigens used, the techniques used, and type of the lesion(s) (single or multiple; live or degenerated) (Malla, 2001).

The non-invasive specimen such as urine is now being increasingly evaluated as a specimen alternate to serum in the diagnosis of many infectious diseases including the parasitic infections such as, schistosomiasis (Polman et al., 2000), filariasis (Itoh et al., 2001), cystic echinococcosis (Ravinder et al., 2000), onchocerciasis (Vincent et al., 2000), toxoplasmosis (Mahmoud, 2002), and leishmaniasis (Attar et al., 2001; Sarkari et al., 2002; Islam et al., 2004; Vilaplana et al., 2004).

The Co-agglutination (Co-A) was the first serological test developed in this laboratory for detection of urinary *Cysticercus* antigen in the diagnosis of NCC (Parija et al., 2004). In this study, *Cysticercus* antigens in the urine could be demonstrated in 62.5% CT/MRI proven cases of NCC by the CoA test. However, a false positive reaction was observed with 9% of urine from non-cysticercal CNS infection controls. In the present study, an attempt has been made to detect *Cysticercus* antigens in the urine by employing more sensitive immunoassays such as ELISA and EITB for diagnosis of NCC.

1.6.3. *Diagnosis of ophthalmic cysticercosis*

Diagnosis of ophthalmic cysticercosis depends on history of tapeworm infection, ophthalmoscopy, ultrasonography, and biopsy and less commonly on other laboratory tests. Image appearances of ophthalmic cysticercosis depend on the parasitic site and living status of *Cysticercus* in the eye and its adnexa. For diagnosis, ultrasonography is the imaging method of choice followed by MRI for demonstration of the living cysticerci. CT is the method of choice for non-living stage of the ocular cysticercosis (Shi et al., 2000). Differential diagnosis of congenital vitreous cysts from acquired *Cysticercus* cysts requires
careful clinical examination and appropriate laboratory tests (Bayraktar et al., 2004). Ultrasonography alone is not useful to confirm the diagnosis. Ophthalmoscopy may not be helpful in diagnosis of peripherally located parasite (Kruger-Leite et al., 1985) and also in case of severe inflammation (Kapoor and Kapoor, 1978).

Though serodiagnosis is a basic tool used for the clinical management and control of parasitic diseases (Dorny et al., 2003), reports on the use of serological tests in the diagnosis and management of cysticercosis in eye are few (Sachdeva et al., 1995; Grover and Puri, 1996; Sekhar and Lemke, 1997; Menon et al., 2000; Bajaj and Pushker, 2002).

Serodiagnosis by specific antibody ELISA has been evaluated for diagnosis of various other infectious diseases of the eye such as, sparganosis (Yoon et al., 2004) and onchocerciasis (Cho-Ngwa et al., 2003). Though few studies on serodiagnosis of ophthalmic cysticercosis have been reported, information is scanty regarding the technique and antigens used for detection of antibodies in serum in these studies. All these serological tests used in ophthalmic cysticercosis are antibodies-based, and no antigen-based serological tests are yet evaluated in the diagnosis of ophthalmic cysticercosis.

Hence, in the absence of a useful imaging method and a good serological test, laboratory diagnosis of ophthalmic cysticercosis remains far from satisfactory. Therefore, management of both ocular and extra ocular cysticercosis still continues to pose a serious challenge. In the present study, serodiagnosis by demonstration of either *Cysticercus* antigens or antibodies in serum and tear is evaluated for diagnosis and treatment follow-up of ophthalmic cysticercosis.

1.6.4. Use of tear as a specimen for diagnosis of ophthalmic infections

Tear is being evaluated as a specimen alternate to blood for the diagnosis of few parasitic infections such as onchocerciasis, toxoplasmosis and *Acanthamoeba* infections (Lehmann et al., 1998; Ngu et al., 1998; Meek et al., 2000). Secretory IgA (sIgA) is the predominant immunoglobulin present in tears that protects the ocular surface against various antigens (Meek et al., 2000). However, it is not known whether these frequently observed antibody responses are the result of common mucosal immune responses against *T. gondii* or represent
the natural antibody repertoire. Both antigen as well antibodies have been demonstrated in tear for diagnosis of onchocerciasis (Glaze et al., 1984; Ngu et al., 1998; Ayong et al., 2005).

Studies are lacking on the use of tear specimens for diagnosis of ophthalmic cysticercosis. Therefore, in the present study, an attempt is made to evaluate tear as a specimen by demonstration of either specific IgA antibodies or *C. cellulosae* antigens for immunodiagnosis of ophthalmic cysticercosis.

**1.6.5. Diagnosis of NCC in relation to the live vesicular and dead degenerated stages of cysticerci**

Cysticercosis in human is an end-stage infection with a benign natural course. It is observed that cysticerci can modulate the host’s immune response and remains silent due to an active immune tolerance (White, 1997). This inhibits a host response against itself ensuring survival over prolonged periods. Several years may pass between exposure and development of symptoms, which probably occur when these defense mechanisms fall in a degenerating cyst in brain and eye, or one dying from cysticidal therapy. Thus, an antibody response to the somatic antigens of the parasite in unlikely to be elicited in an early stage of the infection (Chavarria et al., 2003).

The living cysticerci may continue releasing metabolic byproducts or ES substances. Hence detection of either the ES substances in body fluids or an antibody response to the ES substances might help in diagnosing a current infection that can easily protect from life threatening consequences of the later stages of the infection. Also asymptomatic carriers can be picked up in a field-based survey in order to estimate the actual prevalence of the disease. Hence, there is a need to establish an early and appropriate diagnosis that can differentiate between the stages of the parasite; it may be an aid to the clinician for deciding an appropriate treatment.

In the present study, somatic antigens and excretory-secretory (ES) antigens (obtained from in vitro culture of *C. cellulosae*) are evaluated to study the varying antibody level in relation to the live vesicular and dead degenerated stages of the parasite in NCC.
1.7. Treatment

1.7.1. Treatment of NCC

Therapeutic measures in cysticercosis include antiparasitic drugs, surgery and symptomatic treatment. Albendazole and praziquantel are effective antiparasitic drugs against the cysticerci. A safe and effective treatment of intestinal tapeworm infection may be achieved with either praziquantel or niclosamide; the well tolerated oral agents that have direct cysticidal effect (Carpio et al., 2002).

Praziquantel (PZQ): It is a broad-spectrum anthelmintic and well-tolerated cysticidal drug. Controlled studies by Sotelo et al., 1985 confirmed its effectiveness. Based on monitoring of PZQ levels in body fluids, an oral dose of 25 mg per kg three times a day and even 50 mg per kg is recommended (Bittencourt et al., 1990). A single day regimen of PZQ has recently been described with similar rate of cyst disappearance in some patients (Del Brutto et al., 1999).

Albendazole (ALB): Albendazole has been found to be the drug of choice for treating cysticercosis though it does not appear to be totally effective for curing cerebral cysts (Willingham et al., 2003). It is an imidazole, recommended for active parenchymal brain and subarachnoid (racemose) cysts, whose cysticidal efficacy was demonstrated by Escobedo et al. (1987) and Sotelo et al. (1988). A dose of 15 mg per kg per day for 1 month is initially recommended, but a shorter course of the same daily dose for 1 week and even three days seems to be equally efficacious (Sotelo et al., 1988). ALB is less expensive and has a better penetration ability into cerebrospinal fluid. ALB has been observed to be clearly superior with a great reduction in the number of cysts in the CT scan after therapy (88% Vs 50%).

Niclosamide: Niclosamide is a poorly absorbed narrow spectrum anthelmintic, available as 500 mg chewable tablets. A single treatment is effective therapy for T. saginata and T. solium tapeworms. Because the drug is poorly absorbed, the typical side effects of niclosamide are mild, occurring at the rate of approximately 10%.

Symptomatic and anti-inflammatory medication: Symptomatic therapy is a critical component of management. For example, antiepileptic drugs are given to treat seizures. Seizures secondary to NCC respond well to first-line antiepileptic drugs. Withdrawal of
antiepileptic drugs can be achieved, although residual calcifications on CT scan mark patients for whom the risk of recurrent seizures is high (Carpio et al., 1989). Corticosteroids are frequently used in patients with cysticercosis to decrease inflammatory reaction due to death of the parasite. The most frequent regimen is dexamethasone at the dose of 4.5 and 12 mg/day. Prednisolone at 1mg/kg/day may replace dexamethasone when long-term steroid therapy is required. Mannitol, at doses of 2mg/kg/day, is also used for acute intracranial hypertension secondary to NCC (Garcia et al., 2002a). Dexamethasone 24-32 mg/day is essential in the treatment of subarachnoid cyst and parenchymal cyst (Del Brutto et al., 1993). Other medications commonly used for symptomatic treatment in NCC are analgesics.

Surgery: Prior to the advent of antiparasitic drugs, surgery was the primary therapy for NCC, mainly open surgery for excision of large cysts or cysts in the ventricles. The role of surgical therapy in the management of NCC has significantly decreased over time and is mainly restricted to placement of ventricular shunts for hydrocephalus secondary to NCC (Madrazo and Flisser, 1992). Recently, less invasive procedures have been described, specifically the use of neuroendoscopic resection for ventricular cysts (King et al., 1999).

1.7.2. Treatment of ophthalmic cysticercosis

There is limited experience on the therapeutic efficacy of the treatment for ophthalmic cysticercosis due to an apparent lack of knowledge regarding the natural course of the infection. But medical management with albendazole merits consideration as seen in cases of extraocular cysticerci (Menon et al., 1999).

Systemic management: The use of systemic corticosteroids and anti-inflammatory drugs temporarily relieves symptoms by suppressing pericystic inflammation. Spontaneous resolution of the cyst may be a part of the natural course of the disease. Treatment with corticosteroids may only be supportive in early resolution of the inflammatory part of the lesion (Sekhar et al., 1999). Medical therapy with praziquantel in intraocular cysticercosis has been a failure with the drug producing a toxic but reversible effect on the parasite, possibly due to insufficient concentration of the drug due to the blood-ocular barrier.
The orbit with its better blood supply may prove to be more amenable to medical therapy. A cent percent success rate has been reported with albendazole therapy in orbital cysticercosis, with a complete disappearance of all the cysts (Menon et al., 1999). By the end of the first week of treatment, there may be a decrease in the overall diameter of the cyst with an increased amount of intracystic echoes and surrounding pericystic infiltration. Subconjunctival cysticerci have been reported to extrude spontaneously within a few days after initiation of albendazole therapy. Caution must be exercised in the use of anthelminthics, because endotoxins produced by dying cysticerci within the globe destroy the eye in 80% of cases in which all cysts have not been removed. Treatment with a combination of oral albendazole and prednisolone is effective in the management of NCC in the orbit (Sekhar and Honavar, 1999). Common regimen for ocular cysticercosis includes albendazole given orally in a dosage of 15 mg/kg body weight for 4 weeks with oral prednisolone in a dosage of 1.5-2 mg/kg body weight.

Surgical management: Surgical removal of the cysts is the most effective means recognized for preserving function in an eye with intravitreous or subretinal cysticerci (Sharma et al., 2003). However, the surgical procedures for removal of the parasite in toto are having varying degrees of success. Destruction of the Cysticercus without removing it from the eye, by methods such as diathermy, photocoagulation, or irradiation, usually results in release of toxins and loss of the eye (Hutton et al., 1976).

1.8. Prevention and control

The 2003 World Health Assembly declared that T. solium taeniasis/cysticercosis is of worldwide public-health importance, and that it is an eradicable parasitic disease (Ito et al., 2003a; Ito et al., 2003b). The importance of education at the community level and indeed community involvement are acknowledged as the key elements in combating this disease. The important measure to prevent and control of T. solium taeniasis/cysticercosis include: a) avoidance of eating raw or insufficiently cooked pork, b) thorough cooking of pork, or freezing to -5°C for 4 days or -24°C for 1 day, which kills cysticerci, c) inspection of the pork for cysticerci, d) implementation of sanitation facilities, e) avoidance of eating raw vegetables grown in fields fertilized with human faeces thus contaminated with eggs of T.
soilnz, and f) treatment of infected persons and improvement of personal hygiene to prevent the risk of intestinal autoinfection.

The taeniasis/cysticercosis disease complex has been recognized as potentially eradicable where, emphasis has been placed on control through mass chemotherapy of human populations to remove tapeworm carriers, but this strategy does not control the source of infections that is cysticercosis in pigs (Garcia et al., 2004). Also, transmission may continue due to incomplete chemotherapy coverage of human carriers or because of immigration of tapeworm carriers into controlled areas (Eddi et al., 2003).

Porcine infection is controlled either by mass anthelmintic treatment or with an effective vaccine (Garcia et al., 2003a; Garcia et al., 2003b). A veterinary benzimidazole, oxfendazole, is more than 95% effective in killing the cysts in the pig when even in a single dose of 30 mg/kg. Partial protection has been achieved by vaccination of healthy pigs with crude and fractionated antigen of scoleces (Flisser et al., 2003); extract of egg antigen and antigen from in vitro culture of onchospheres (Pathak and Gaur, 1990); and T. solium onchospheral antigens (Verastegui et al., 2003). Since little socioeconomic development is expected in the near future, intervention measures for control and eradication of taeniasis/cysticercosis are urgently needed.

The enhanced control of human cysticercosis may be able to contribute to several international initiatives such as WHO's Global Campaign Against Epilepsy-"Out of Shadows", the scaling up of the response to diseases of poverty, the Food Safety Programme and the Partnership for Parasite Control (WHO, 2002).