Toxocara canis and Toxocara cati, roundworms of dogs and cats are probably the most common gastrointestinal helminthes of domestic canids and felids. First described in late seventeenth century Toxocara has been found to infect dogs, cats and mice. The larvae of Toxocara canis have been reported in different organs like intestine, liver, lungs, kidneys, skeletal muscles and nervous tissue of rat and mice (Taylor and Holland, 2001). The prevalence of the infection throughout the world shows different infection rates of Toxocara canis in dogs; it is 18.3% in Italy (Legrottaglie et al., 2003), 25.7% in France (France et al., 1997); 18.3% in Indiana (Kazacos, 1978). Saeki et al. (1997) found a prevalence of 79.9% of Toxocara canis in puppies of stray dogs in Ibaraki Prefecture. D’Souza (2002) reported 2.7%-28% of prevalence of Toxocara canis in Bangalore (India). The
prevalence of patent *Toxocara canis* infection is highest in young dogs and much less common in adult animals.

**Mode of transmission**

**Infectious agents**

The life cycles of *Toxocara canis* and *Toxocara cati* are complex. Adult worms in the intestinal tract of infected dogs and cats shed large number of eggs. These eggs find their way into the environment, where their hosts defecate. Once into the field these may be ingested by natural hosts as well as paratenic host (Figure 1.1). The larvae then hatch and migrate through blood vessels all over the body and are referred as visceral larva migrans (VLM). In young animals tracheal migration occurs through the lungs and trachea into the intestine, where these larvae mature. In paratenic hosts and most adult dogs and cats that have some degree of acquired immunity, the larvae undergo somatic migration to various tissues and remain arrested in the tissues of the paratenic host until they do not reach in the intestine of their definitive host. After predation of *Toxocara* infected paratenic hosts by dogs or cats, larvae are released and develop into adult worms in the intestinal tract which after their complete development start shedding a large number of eggs along with their faeces. In the pregnant bitch and queen, dormant tissue larvae are reactivated and migrate across the placenta to infect the foetus. New born puppies and kittens also acquire infection through ingestion of larvae in milk (Overgaauw, 1997).
Fig. 1.1. Life cycle of *Toxocara canis*

- **Eggs ingested**
- **Dogs > 5 weeks (Non-pregnant)**
  - Larvae released in Intestine
  - Larvae migrate to various organs where their development is arrested
  - In pregnant and lactating dogs the larvae can be reactivated and cause
    - Intestinal infection of the mother
    - Infection of the offspring (By transplacental and transmammary transmission)

- **Dogs < 5 weeks**
  - Circulation → lungs → bronchial tree → Esophagus
- **External Environment**
  - Embryonated egg with larvae
  - Eggs passed in faeces

- **Human and other Paratenic host**
  - Larvae released in intestine
  - In heavy infections larvae can be passed in faeces
Contamination of the environment with *Toxocara* eggs

*Toxocara* eggs when passed by way of defecation by dogs and cats are unembryonated and non infectious. These eggs can however develop into infectious stages within a period of 3 weeks to several months depending on soil type and climatic conditions like temperature and humidity. Eggs will develop to an infectious stage that can survive under optimal environmental conditions and have the potential to survive for at least one year (Parsons, 1987). No larval development occurs at temperature below 10°C and larvae may die if the temperature is less than -15°C. The larval stage or somatic larvae in tissues have always been considered to be infective.

Infection of dogs and cats with *Toxocara* eggs

Tracheal migration

When the infective *Toxocara* eggs are ingested by young dogs, tracheal migration of larvae occur through liver, vascular system and the lungs. The larvae break out into the alveoli and migrate through trachea and pharynx into the stomach. After swallowing, they then complete their development in stomach and small intestine. Eggs appear in the faeces after 4 to 5 weeks. In a survey among dogs and cats in the USA (Lightner *et al.*, 1978), the patent *Toxocara* infection was found in 2 week to 2 month old dogs and in 2 to 6 month old cats.
Age resistance

When a puppy attains the age of one to two months, there is very less probability of the newly hatched *Toxocara canis* larvae developing into adult ascarids, the somatic migration however shows progressive increase (Overgaauw, 1997). The failure to produce patent infection in older dogs is termed as age resistance. An inverse relationship between the recovery rate of adult ascarids and the age of the dog is observed. The difficulty in development experienced by the infective stage larvae to the next stage suggests that the resistance is directed against the infective stage of the parasite. Two mechanisms of evasion of the host’s immunity by the larval stages have been suggested- one is hypobiosis of the tissue larvae that renders the parasite less susceptible to interference from the metabolism of the host and the other is immunosuppression of the host immune system. The immunosuppressive effect of pregnancy and lactation may permit tissue larvae, or larvae from a newly acquired infection to undergo tracheal migration and subsequent intestinal development. In general, new infections of the lactating bitch can occur by ingestion of immature fourth stage larvae from vomit or faeces of the puppies. Larvae can develop into adults without tracheal migration. The finding of *Toxocara* eggs in the faeces of a bitch one week after parturition and prior to the detection of eggs in the faeces of her puppies leads to the hypothesis of tracheal migration of activated somatic larvae in the bitch (Overgaauw, 1997).
Somatic migration

Adult dogs and cats can get infection by ingestion of infective *Toxocara* eggs from the environment, mainly from contaminated soil. Larvae hatch in the intestine and invade the mucosal layer. Migration occurs either passively via lymph and blood or actively by penetration of the tissues and invasion of different parts of the body. Gradually somatic larvae accumulate in the tissues (somatic migration), persisting for long periods in a manner similar to that seen in paratenic hosts. More than half of the embryonated eggs appeared to be discharged without hatching in the intestine (Overgauuw, 1997).

Infection of dogs and cats with *Toxocara* larvae

Transplacental migration

Several studies have shown that nearly 100% of puppies are infected in uterus from day 42 of gestation by somatic larvae (Lloyd et al., 1983). This transplacental migration or intrauterine infection is the most important mode of transmission in dogs.

Transmammary transmission

After activation, somatic *Toxocara* larvae of dogs and cats are transmitted via the colostrum and milk referred to as transmammary, lactogenic or milk borne infection. Following ingestion by the offspring the larvae undergo development without tracheal migration. Larvae are found to pass in the bitch’s milk for at least 38 days after parturition (Overgauuuw, 1997).
Introduction

Transmission through paratenic hosts

Paratenesis is a mode of infection ensuring continuous survival of some larval nematodes. Small mammals are suggested to play an important role as paratenic host in rural and urban localities. After ingestion of a paratenic host infected with Toxocara canis larvae, the larvae develop directly in the intestine because they have already migrated in the preceding host and have reached an appropriate stage of maturity such that they can develop into adults in the intestine (Dubinsky et al., 1995). The paratenic route thus seems to play a significant role in transmission of Toxocara canis. The life cycle of Toxocara canis is more complex than that of Ascaris, with one phase involving developmental arrest in intermediate hosts (including humans) as well as a complete phase within its definitive canine host that involves vertical transmission from infected pregnant bitches to their pups. Adult female Toxocara canis worms, which are generally found only in young puppies and lactating bitches, excrete up to 200,000 eggs per day (Liu, 1999).

The present problem

The dog’s role as a definitive host for a number of zoonotic parasites has been widely studied and recognized as being a significant public health problem worldwide. It is estimated that over 31% of USA (Schantz, 1991), 53% Australian and 52% of European households own a dog, cat or bird (Beck, 2000). Most pet dogs are considered an integral part of the owner’s family and are treated accordingly. Pet dogs provide a sense of safety, security, companionship, amusement and have also been shown to benefit
their owner’s health. In countries where the population of stray dogs is well controlled, most of the attention is primarily focused on domesticated pets. Veterinarians are on the ‘frontline’ of preventing transmission of zoonotic diseases from pets to people. The attitudes and economic status of pet owners in these developed countries also ensure that the available veterinary resources are well utilised.

Zoonoses due to canine parasites however are far from confined to the developed world. Although the companion animal industry is not recognised as being as strong in developing countries, uncontrolled populations of stray and semi-domesticated dogs exist in close proximity to increasing densities of human populations, in both rural and urban environments. In the socioeconomically disadvantageous communities, poor levels of hygiene, over crowding, together with a lack of veterinary attention and zoonotic awareness, exacerbates the risks of transmission of disease. There is an estimated 19.2 million stray dogs in India alone and despite efforts of control; the dog population is believed to be rising (World health organization, 1996). The average obstacle identified as a cause for rising stray dog populations include the absence or insufficient enforcement of existing legislation of dog population management measures (human destruction, reproductive control) due to lack of government support and resources (world health organization, 1996, Dutta, 2002). Most cultural attitudes in India treat stray dogs as dirty animals and carriers of disease especially with regard to rabies and welcome the idea of stray dog population control. However a portion of stray dogs in India are
infact semi-domesticated by socioeconomically disadvantaged communities such as street dwellers in urban areas. The proportion of true strays in rural areas that have no reference household is usually low. Stray dogs are commonly adopted as puppies especially by children and provide their human counterparts with companionship and security. However, most owned dogs in these rural areas have no restrictions and often roam freely without supervision. The close human-dog contact shared in these situations coupled with a complete lack of veterinary attention puts their human counterparts at high risk of exposure to zoonotic diseases (Toxocariasis).

Current animal welfare legislation in India carried a program of sterilization, vaccination and rehoming, together with human euthanasia of terminally ill or rabid animals (Central Government India, 2001). Accordingly in district Srinagar, a total number of 93 stray dogs were sterilised by the Veterinary department under the Physical Achievements of Animal and Birth Control (ABC) and immunization programme from the period 29th November to 7th December 2004. Due to restricted resources however, the programme primarily targets major cities with high densities of ownerless animals rather than the rural populations, where interactions between domestic and sylvatic cycles of zoonoses thrive. However if resources were made available to implement dog population control programmes in these rural areas, education and cooperation of locals would be a major factor for the most serious, well recognized and well documented canine zoonoses in India. Yet in countries where significant
mortalities result from other more common and life threatening zoonoses and infectious diseases such as malaria and tuberculosis, it is not surprising that resources are harvested in research and control of these diseases of priority. This pattern of resource allocation may account for the relatively low attention paid to canine parasitic zoonoses in the medical literature of India. Most studies consist of sporadic case reports or seroprevalence studies of the more common parasitic zoonoses such as hydatid disease and Toxocariasis. However, despite the strong evidence to show the endemicity of these serious or zoonotic diseases, documentation and surveillance data concerning to the prevalence and risk factors associated with zoonotic parasites of dogs in India is lacking. There is an urgent need for more recent parasitic data to be obtained. Moreover, human activity (behavioural patterns, migration, and environmental pollution) play an important role in determining the prevalence or emergence of parasitic zoonoses, in the animal population or ecology. A more comprehensive and accurate approach to study zoonoses would therefore necessitate conducting simultaneous parasitic surveys in both humans and animals from the environment in order to correlate data on an epidemiological and molecular level. However, few studies have achieved this level while conducting surveys of zoonotic significance (Hopkins et al., 1997).

Several nematode infections in humans are caused by infective larvae of species that are normally parasitic for other carnivorous mammals. In such larva migrans syndromes, the developmentally arrested nematode larvae do not mature to adult worms within the human body,
but rather migrate through superficial tissues or deep organs, causing local and occasionally severe inflammation. In its most florid form, as classically described by Beaver, the syndrome of visceral larva migrans comprises eosinophilia, fever and hepatomegaly and is most commonly due to zoonotic infection with the dog roundworm *Toxocara canis*. *Toxocara canis* causes milder form of human toxocariasis and a clinically distinct syndrome of ocular larva migrans.

**Mode of transmission to the human**

Toxocariasis is a public health problem. Man acts as an unnatural host in which *Toxocara* larvae will not develop but migrate and survive for a long time. The mode of transmission to humans is by oral ingestion of infective *Toxocara* eggs from contaminated soil (sapro-zoonoses), unwashed hands or consumption of raw vegetables (Glickman *et al.*, 1981). Some infections may occur from ingestion of larvae in undercooked organs and muscle tissues of infected paratenic hosts such as chickens, cattle and sheep. Transmission to the second generation is theoretically possible by infective larvae of canine ascarid nematode *Toxocara canis*, a ubiquitous parasite of dogs in temperate and warm climates.

**Clinical manifestations in humans**

While most people infected by *Toxocara canis* do not develop covert clinical disease, three clinical syndromes have been associated with *Toxocara* infection in humans; visceral larva migrans (VLM), ocular larva migrans (OLM) and covert toxocariasis (Taylor and Holland, 2001). In humans, ingested *Toxocara canis* larvae penetrate the intestinal wall and
disperse to other organs via lymphatic and venous pathways without undergoing further development (Glickman and Schantz, 1981). The body’s immune mechanism usually encapsulates the larvae in granulomas, depending on their location can manifest clinically. The presence of freely migrating larvae within the tissues contributes to the pathology, which is a function of the extent and frequency of infection, the site of infection and the intensity of the inflammatory response.

Visceral larva migrans describes the marked immune response to the migration through the liver and tissues. The mean age of the patients at diagnosis is usually 18 months to 4 years of age (Schantz et al., 1979). Clinical pathology is usually characterized by persistent eosinophilia, leucocytosis, hyperglobulinaemia and a raised *Toxocara* titre and may be accompanied by clinical signs of fever, hepatomegaly and cough due to varying degrees of pulmonary infiltration (Taylor and Holland, 2001). VLM is usually self-resolving, however, in rare instances myocarditis, pleural effusion (Altcheh et al, 2003) and neurological involvement (Vidal et al., 2003) has been reported. Treatment of clinical cases is usually unsatisfactory. To date, treatment with albendazole, thiabendazole (Sturchler et al., 1989) has proven the most efficacious.

The mean age of patients with ocular disease is usually 8 years; however, the condition is often diagnosed in teenagers and adults as well (Schantz et al., 1980). Lower infective doses of *Toxocara* larvae are usually associated with a higher probability of OLM than VLM, which may explain the older age of onset (absence of pica) of OLM and why *Toxocara* serum
antibody titres are generally lower in persons with OLM than VLM (Schantz et al.; 1979, Schantz, 1989). OLM is usually caused by a single larva entering the eye and its vascular channels and is usually unilateral in presentation. Presenting complaints may include decrease or loss of vision, strabismus at leukokoria due to a variety of lesions such as posterior and peripheral retinochoroiditis, optic papillitis and endophthalmitis (Fan et al.; 2004c, Altcheh et al., 2003). Treatment varies according to the type and severity of the lesions and currently includes a combination of medical intervention using albendazole, systemic steroids and vitreoretinal surgery.

The term covert toxocariasis has been used to describe a clinical syndrome in patients with raised Toxocara titres and non-specific signs including abdominal pain, anorexia, vomiting, nausea, sleep, cough, limb pains and fever (Glickman et al.; 1987, Taylor et al., 1987). Other associated illnesses such as social, learning and behavioural abnormalities, epilepsy, asthma and transient myositis have also implicated Toxocara as the cause of this wide range of clinical entities; however, the interpretations to these findings have to be debated (Schantz, 1989; Taylor and Holland, 2001).

**Diagnosis of Toxocariasis**

Recently PCR-based methods have been designed for differentiating the species of Toxocara from larvae in animal tissues. However, reliance nowadays is placed on correlating clinical presentation and appropriate risk factors with antibody titres (Taylor and Holland, 2001). The test that is widely recommended at present is an ELISA, which utilises Toxocara larval excretory-secretary antigens (TES). These ES antigens are derived from in
vitro culture of infective *Toxocara* larvae and has shown excellent sensitivity (Gillespie *et al.*, 1993, Yamasaki *et al.*, 2000, Yokoi *et al.*, 2002)

**Toxocariasis in India and Kashmir valley**

Although there is a lot of literature available on the seroprevalence of Toxocariasis in humans worldwide, but there is a minimal literature available on the seroprevalence of Toxocariasis in humans in India however, there is a gross underestimation of its incidence. The only random survey conducted in a rural area in Haryana, found 6.4%, (out of the 94 individuals tested) to be sero-positive to *Toxocara canis* (Malla *et al.*, 2002). History of pica or contact with dogs could not ascertain risk factors for sero-positive individuals. In two separate studies conducted in Chandigarh and New Delhi, 7 out of 30 and 14 out of 68 suspected ocular cases of toxocariasis were found to be sero-positive for *Toxocara canis* (Malla *et al.*, 2002, Mirdha & Khokar, 2002). Parasite surveys conducted over thirty years ago found *Toxocara canis* to be most common parasite of stray dogs in Miraj (prevalence 55%) and Calcutta (prevalence 82%) (Traub *et al.*, 2005). However, the ages of these dogs were unknown and it is possible that a younger population of dogs was surveyed. In contrast, the prevalence of *Toxocara canis* in adult stray dogs in Madhya Pradesh was found to be 2.7% (Sahasrasbudhe *et al.*, 1969). Forty six percent of public parks & 32% of school grounds were found contaminated with *Toxocara ova* in Andhra Pradesh (Kumar & Hafeez, 1998). In Kashmir valley except the hospital based report by Ahmad *et al.*, 2002 no work has been conducted on Toxocariasis. So keeping in view the aforesaid comments, the present study
was taken into consideration to know prevalence of *Toxocara canis* in dogs, the actual seroprevalence of human Toxocariasis and its probable risk factors.

**JAMMU AND KASHMIR STATE**

Jammu and Kashmir State constitutes the northern most extremity of India and is situated between 32°.15″ and 37°.05° north latitude and 72°.35° and 83°.20° east longitude. The total area of the state is 2, 22,236 sq kms. The state ranks 6th in area and 17th in population among the states and union territories of India (Gupta, 2006).

Jammu and Kashmir has four geographical zones of (1) sub-mountain and semi-mountain plain known as Kandi or dry belt, (2) the Shivalik ranges (3) the high mountain zone constituting the Kashmir valley, Pir panchal range and its offshoots, and (4) the middle run of the Indus River comprising Leh and Kargil. There is a sharp rise of altitude from 1000 feet to 28250 feet above the sea level, within state’s four degree of latitude.

Jammu and Kashmir is northern most state of India comprising three distinct climatic regions viz. Arctic cold desert areas of Ladakh, and Sub-tropical region of Jammu and Temperate Kashmir valley.

The presence of large number of water bodies dominates the life activities in Kashmir valley. It is a true saying that “All that glitters is not gold”, because even to this day, there is a dearth of clean portable piped water supply to many houses in the rural and urban areas of Kashmir valley. People use the river or lake water for washing, cleaning, ablution
and even drinking. The untreated refuse of many localities even in cities drain directly into the river or lakes. The continuous contamination of the water bodies by the dogs due to their habit of defecating in open fields make it unsafe for the above mentioned purposes.

Unfortunately, there is seldom any detailed study of the incidence of human toxocariasis and its other effects in the Kashmir valley. This study is a step towards the same direction. The public health importance attached to this peril of human toxocariasis cannot be belittled by growing awareness about the problem or by easier availability and increasing use of chemotherapeutic agents against parasite. In planning the control programmes for toxocariasis, three approaches generally considered are (1) sanitation, hygiene and access to safe water—aimed at controlling transmission by reducing soil and/or water contamination (2) education and improved standard of housing—aimed at reducing transmission and reinfection by encouraging healthy behaviours (3) chemotherapy—aimed at reducing the infection of Toxocara in dogs and human beings. This will result in an immediate improvement in human health and development.

While sanitation, hygiene, access to safe water, education and improved standard of housing have long term implications and need constant commitment and substantial economic support for implementing control of parasitic infestations, issues like chemotherapy offer short term measures for control, once the burden of parasitism is defined in a high risk group like human and they are targeted.