1. INTRODUCTION

*Escherichia coli* is a member of the genus *Escherichia* within the family *Enterobacteriaceae*. It is a motile, Gram-negative rod-shaped bacterium which is commonly found in the lower intestine of warm-blooded animals. *E. coli* was discovered by German paediatrician and bacteriologist Theodor Escherich in 1885. It serves as a major commensal throughout its life as a harmless saprophyte but Larulle (1889) was the first to suggest the possible role of *E.coli* as a pathogenic organism. Pathogenic *E.coli* is one of the most important groups of bacteria causing diarrhoea and extra intestinal infection in humans and animals.

Diarrhoeagenic *E.coli* can be broadly classified into 5 main classes: 1) Enterotoxigenic *E. coli* (ETEC), 2) Enteropathogenic *E. coli* (EPEC), 3) Enteroinvasive *E. coli* (EIEC), 4) Enteroaggregative *E. coli* (EAEC) and 5) Enterohaemorrhagic *E. coli* (EHEC). However, overlap among the categories exists. For example, both STEC and EAEC express toxins that contribute to disease and some isolates of EAEC and ETEC exhibit invasiveness (Khan and Steiner, 2002).

Enterotoxigenic *E. coli* (ETEC) strains are noninvasive, and they do not leave the intestinal lumen. ETEC is the leading bacterial cause of diarrhoea in children in the developing world, as well as the most common cause of traveler's diarrhoea. Hosts are humans, pigs, sheep, goats, cattle, dogs, and horses. ETEC uses fimbrial adhesins (projections from the bacterial cell surface) to bind enterocyte cells in the small intestine. ETEC can produce two proteinaceous enterotoxins. (1) The larger of the two proteins, heat labile (LT) enterotoxin, is similar to cholera toxin in structure and function. (2) The smaller protein, heat stable (ST) enterotoxin causes cGMP accumulation in the target cells and a subsequent secretion of fluid and electrolytes into the intestinal lumen.

Enteropathogenic *E. coli* (EPEC) is the causative agent of diarrhoea in humans, rabbits, dogs, cats and horses. Like ETEC, EPEC also causes diarrhoea, but the molecular mechanisms of colonization and aetiology are different. EPEC strains lack fimbriae, ST and LT toxins, but they utilize an adhesin known as intimin to bind host intestinal cells.
This virotype has an array of virulence factors that are similar to those found in *Shigella*, and may possess a shiga toxin. Adherence to the intestinal mucosa causes a rearrangement of actin in the host cell, causing significant deformation. A change in intestinal cell ultrastructure due to “attachment and effacement” is likely the prime cause of diarrhoea in those afflicted with EPEC.

Enteroinvasive *E. coli (EIEC)* is found only in humans and causes a syndrome that is identical to Shigellosis, with profuse diarrhoea and high fever.

Enteraggregative *E. coli (EAEC)* is found only in humans. EAEC strains are so named because they have fimbriae which aggregate tissue culture cells. They bind to the intestinal mucosa to cause watery diarrhoea without fever. They are non-invasive. They produce a hemolysin and a ST enterotoxin similar to that of ETEC.

Enterohemorrhagic *E.coli (EHEC)*: EHEC produces toxins, known as verotoxins (VT) or Shiga-like toxins(ST) because of their similarity to the toxins produced by *Shigella dysenteriae*. Verotoxigenic *E.coli (VTEC)* was identified by Konowalchuk and his coworkers (1977) as a distinct group of *E.coli* named as verotoxic *E.coli (VTEC)*, which had the ability to produce toxins with profound and irreversible action on Vero cells. Symptoms of the diseases caused by EHEC include abdominal cramps and diarrhoea that may in some cases progress to bloody diarrhoea (haemorrhagic colitis). The most famous member of this serotype is strain O157:H7. This particular strain is linked to the 2006 United States *E. coli* outbreak due to fresh spinach. Non-O157 EHEC also causes illness and outbreaks. More than 37,000 illnesses are attributed to non-O157 EHEC serotypes. The incidence of non-0157 EHEC is increasing; there are at least 200 serotypes of *E.coli* that are capable of producing shiga toxin, despite limited laboratory testing (Zach Mallove, 2010).

STEC infections are reported from different parts of the world, being found in 75 to 100 per cent of episodes of sporadic Hemolytic Uremic Syndrome(HUS) in Europe, North America, Canada, and Latin America especially in Argentina(Griffin and Tauxe,
1991; Lopez et al., 1989; Rowe et al., 1993). It is prevalent in all 12 provinces and territories of Canada and more common in northern than southern states of the United States (Spika et al., 1998). In continental Europe, human STEC infection has been reported from 16 countries where these strains have become an important public health problem (Caprioli and Tozzi, 1998). In the United Kingdom, especially in England, Wales, Scotland and Northern Ireland, there has been a significant increase in the isolation of O157 STEC over the last decade (Smith et al., 1998). However, in Australia, O111:H7 is currently the most frequent cause of human disease while the incidence of O157:H7 has been particularly low compared to other industrialized countries (Robins Browne et al., 1998). In most of the Asian countries, STEC is not yet a major health problem, except in Japan, where 29 outbreaks have been reported between 1991 to 1995 (Michino et al., 1998). Few reports are available on isolation of STEC from Hong Kong (Leung et al., 2001), Thailand (Brown et al., 1989), Malaysia (Radu et al., 1998), India (Khan et al., 2002) and Sri Lanka (Tokhi et al., 1993).

In India the total death rate of newborn due to diarrhoea alone is estimated to be 10.2 percent in 1986 but STEC has not been identified as an etiological agent of diarrhoea in these cases. There is a lack of surveillance for this organism due to the difficulty in isolating STEC. However, STEC strains belonging to the serotype O157 have been reportedly isolated in India from sporadic cases of diarrhoea, but the isolated strains have not been well characterized and the origin of these strains is uncertain. At present, reports on the occurrence of STEC in Indian cattle are lacking, though extensive efforts have been made to isolate STEC in cattle and meat. Khan et al. (2002a) showed that the prevalence of STEC was 18 percent in cow stool samples, 50 percent in raw beef samples in Calcutta. Recently Bandyopadhyay et al. (2011) had reported 47% of STEC in lambs from Andhra Pradesh. Thus STEC strains are clearly present in the food chain in India. However there are only a few reports of isolation of STEC (Priya Rajendran et al., 2009) from Tamil Nadu. Thus keeping in view, the above facts, a proposed study was planned to detect and characterize the field isolates of STEC from cattle, human and poultry in Coimbatore by cultural, biochemical and molecular methods and also to find a bioactive phytochemical as an anti shiga toxin agent.