Chapter One

Introduction
1. INTRODUCTION

The recent years have witnessed a sharp increase in the global incidence of several infectious diseases. Many of the well known illnesses thought to be under control are reemerging and exotic infections not previously known are reappearing and becoming causes of serious global concern. Among the various infectious diseases, diarrhoea is one of the common health hazards and the prime cause of morbidity and mortality in children and adults in developing countries throughout the world with an estimated 3.7 to 4.6 million deaths annually (Glass et al. 1991). One organism contributing to this global fear of reemerging infectious diseases is *Vibrio cholerae*, a motile, Gram negative, curved *Vibrio* with a single polar flagellum which is the causative agent of the life threatening disease cholera. The disease is manifested by severe dehydration as a consequence of the rapid and extreme loss of fluid and electrolytes during the course of infection with the passage of white "rice watery" stools along with vomiting resulting in hypovolemic shock which may lead to death in absence of prompt and appropriate treatment.

Based on the presence of heat stable somatic (O) antigens, *V cholerae* has been broadly classified into two groups - *V.cholerae* O1 and *V.cholerae* non-O1. On the basis of variation in the antigenic forms and variation in certain traits, the O1 serogroup is further differentiated into two different serotypes namely Inaba and Ogawa and each serotype into two biotypes namely classical and ElTor. Cholera toxin, a potent protein enterotoxin, elaborated by the O1 serogroup of *V.cholerae* is the major mediator of the disease. Some other ancillary secretogenic factors, besides cholera toxin, are also reportedly responsible for the precipitation of the disease though the entire cascade of mechanism of action of these factors still remains obscure.

Until recently, only strains belonging to the O1 serogroup of *V.cholerae* caused
epidemic and pandemic cholera while those belonging to the other serogroups (collectively known as *V.cholerae* non-O1) were not associated with large epidemics but were the causative agents of sporadic diarrhoea and occasionally caused extraintestinal infections. But in the year 1992, this existing scenario of *V.cholerae* changed. An unprecedented event was witnessed in October 1992 in the Indian subcontinent with the emergence of a novel toxigenic strain of *V.cholerae* which did not agglutinate with the O1 polyvalent antiserum but possessed the potential to cause explosive outbreaks of cholera-like infections which assumed epidemic proportions in a short span of time, a happening that has never occurred in the recorded history of the disease cholera (Ramamurthy *et al.* 1993; Albert *et al.*, 1993). Shimada *et al.* (1993) assigned these strains to a new serogroup namely O139 and appended a synonym ‘Bengal’ to symbolize the first outbreaks caused by this strain in areas along the coast of the Bay of Bengal.

The sudden emergence of *V.cholerae* O139, its capability of causing full-blown cholera and life threatening dehydration and its relentless proclivity to spread has led to a unique situation, demanding quick answers about its origin and clonality and also necessitating a review of the prevailing strategies and beliefs in combating and controlling cholera.