Objectives
Microbes are phagocytosed inside macrophages and degraded in the acidic environment of the lysosomes. However, several pathogens manipulate host cellular processes to their advantage and evade transport to the degradative compartment and survive as intracellular pathogens. It is now well established that *Salmonella* enters macrophages by triggering its own uptake via cytoskeletal rearrangements and subsequently, establishes an intracellular niche by inhibiting its transport to lysosomes. To this effect, *Salmonella* secretes several effectors into the host cytoplasm by a specialized secretion system. A complex interplay between a number of host and pathogen encoded factors is envisaged as part of *Salmonella* survival mechanism.

Recent findings from our laboratory have shown that a *Salmonella* effector, SopE recruits the host transport molecule, Rab5 on the *Salmonella*-containing phagosomes and subverts targeting to the lysosomes (Mukherjee et al., 2001). Moreover, temporal acquisition of another family of transport molecules, SNAREs on *Salmonella*-containing phagosomes speculated the involvement of different effectors in this process. However, the mechanism of recruitment of SNAREs by *Salmonella* on its phagosomes needs to be elucidated. Accordingly, studies were initiated in the present thesis to achieve the following objectives:

1. Identification and characterization of *Salmonella* effector molecules which are involved in the recruitment of SNARE(s) on phagosomes.
2. Determination of the role of the identified effector molecule(s) in *Salmonella* trafficking in macrophages.