1. INTRODUCTION
1.1 Comparative Genomics

In scenario of an enduring improvements in high-throughput genomic sequencing and the ever-expanding sequence databases, new advances in software programs for post-sequencing functional analysis are being demanded by the applied scientific community. Whole genome comparisons have been heralded as the next logical step toward solving genomic puzzles, such as determining regulatory signals, discovering coding regions and deducing the mechanisms of genome evolution and function.

Microbial diversity related to emerging pathogens, which are becoming an increasing threat to public health in general. Complete genomes of several chemo heterotrophic non-proteobacterial species have been complete or on the verge of completion. These results have revealed the remarkable amount of information on the morphology, physiology and evolution of microbial species, and will help to provide a revolutionary impact on the microbial research.

The first complete genome sequences of cellular life forms have become available in just the last several years. In 1995, the genomes of the first two bacteria, Haemophilus influenzae and Mycoplasma genitalium, were reported, sequence data for additional bacterial genomes have been accumulating at an increasingly accelerated pace. Of the present more than 550 completely sequenced bacterial genomes (http://www.ncbi.nlm.nih.gov/genomes/lproks.cgi), in which some are like from Yersenia Spp. Comparative analyses of these genomes provide a huge and unprecedented resource for discovering novel molecular characteristics that are either unique to particular species or are shared by different non-proteobacteria and they can also provide valuable tools for biochemical, diagnostic, taxonomic and evolutionary studies.
1.2 The Pathogenic Bacteria: *Yersinia pestis*

So Nature killed many through corruptions,

*Death came driving after her and dashed all to dust,*

*Kings and knights, emperors and popes;*

*He left no man standing, whether learned or ignorant...*

~William Langland (c. 1330-1387)

*Yersinia pestis* is a rod-shaped facultative anaerobe with bipolar staining (giving it a safety pin appearance). Similar to other *Yersinia* members, it tests negative for urease, lactose fermentation, and indole. The closest relative is the gastrointestinal pathogen *Yersinia pseudotuberculosis*, and more distantly *Yersinia enterocolitica*. Similar to the other pathogenic strains, there are signs of loss of function mutations. The chromosome of strain KIM is 4,600,755 base pairs long; the chromosome of strain *CO92* is 4,653,728 base pairs long. Like its cousins *Y. pseudotuberculosis* and *Y. enterocolitica, Y. pestis* is host to the plasmid pCD1. In addition, it also hosts two other plasmids, pPCP1 (also called pPla or pPst) and pMT1 (also called pFra) that are not carried by the other Yersinia species. pFra codes for a phospholipase D that is important for the ability of *Y. pestis* to be transmitted by fleas. pPla codes for a protease, Pla, that activates plasminogen in human hosts and is a very important virulence factor for pneumonic plague. Together, these plasmids, and a pathogenicity island called HPI, encode several proteins that cause the pathogenesis, for which *Y. pestis* is famous. Among other things, these virulence factors are required for bacterial adhesion and injection of proteins into the host cell, invasion of bacteria in the host cell (via a Type III secretion system), and acquisition and binding of iron that is harvested from red blood cells (via siderophores). *Y. pestis* is thought to be descendant from *Y. pseudotuberculosis*, differing only in the presence of specific virulence plasmids.
Oriental rat flea (Xenopsylla cheopis) infected with the Yersinia pestis bacterium which appears as a dark mass in the gut. The foregut of this flea is blocked by a Y. pestis biofilm; when the flea attempts to feed on an uninfected host Y. pestis is regurgitated into the wound, causing infection.