**PREFACE**

*Escherichia coli* and several other microorganisms possess enzymatic systems to repair damages caused by radiations, carcinogens and mutagens (Howard-Flundren, 1969; Assl, 1974; Litkin, 1976). Several genes are involved in the repair of irradiated *E. coli* and the same set of genes are involved in the repair of lesions inflicted by carcinogens and mutagens (Clark and Ganesan, 1975; Ishi and Kondo, 1975; Auerbach, 1976). These genes have been mapped and the products of some have been identified (Hamawalt and Setlow, 1975; Gudas and Pardue, 1975). Four repair systems have been identified: (1) Photoreactivation, (2) Excision repair, (3) Constitutive recombination repair, and (4) Inducible error-prone repair.

The primitive earth was under constant exposure of hazardous high doses of ionizing and ultraviolet radiations. While the former has high penetrating power and ionizing capabilities, ultraviolet light is selectively absorbed by DNA, the genetic material of most living organisms. Various DNA repair systems are believed to have evolved to cope with offensive exposures of ionizing and non-ionizing radiations. Because of the formation of ozone layer in the upper atmosphere, the ionizing radiations are not found now in natural environment at a value high enough to constitute a hazard to living organisms. It is, therefore, logical to question the role of
these repair systems, since an unwanted system ought to have been discarded during the course of evolution. Several speculations were made around 1970 that the DNA repair systems may be involved in normal metabolism e.g. genetic recombination (Howard-Flanders and Theriot, 1966), to cope with nonphysiological environmental conditions like pH, temperature and ionic strength (Bridges et al., 1969), and of course to alleviate the hazards of environmental mutagens (Ames, 1974; Ishi and Kondo, 1975).

We became interested in the role of DNA repair system under non-physiological conditions. Encouraged by the preliminary report of Bridges et al. (1969) on the correlation between heat and radiation sensitivities, we selected E. coli K-12 and its phage λ, and the effect of non-physiological temperature (52°C) was studied.

In the first chapter of this dissertation, damages induced by radiation and temperature, and DNA repair systems have been described.

The second chapter describes the bacterial and phage strains, composition of media and buffer.

The third chapter describes a new methodology developed for the purification of bacteriophage λ.

Fourth chapter deals with the survival of various radiation sensitive mutants of E. coli K-12 exposed to 52°C,
recovery of heated cells, effects of the metabolic inhibitors on recovery, and heat induced mutagenesis.

Fifth chapter is devoted to the description of biochemical aspects of heat-damage and repair in E.coli. This chapter describes the nature of heat-lesions in DNA, RNA, protein and cell membrane.

Sixth chapter deals with the effect of 52°C on λ.

Seventh chapter is comprised of the general discussion. The purpose of the general discussion is to coordinate very briefly the entire data and propose a model on heat lesions and their repair.

This is followed by abstract and bibliography.