Ageing is universal, progressive, deleterious accumulation of changes over the time and so far irreversible that increases the probability of death. Ageing in seeds can best be defined as an increased probability of death as symptoms of seed ageing are reduction rate and uniformity of germination during time as age increases. The exact cause of loss of seed viability is still not known. One broad-based hypothesis is the “free-radical theory of ageing” suggesting that endogenous ROS if accumulate in excess amounts to continuous damage at cellular and molecular level, including DNA. Such deterioration increases with ageing, where ROS exceed beyond the antioxidant system balance resulting in age-related deterioration. The telomere shortening has been described as a causative factor in determining the longevity of the organism. Oxidative stress accelerates telomere loss, while antioxidants decelerate it. Thus oxidative stress is a critical modulator of telomere loss and telomere-driven senescence. A recent technique, ‘osmopriming’ has been developed to enhance the repair process in the cell, thus averting the oxidative stress/ageing-induced cellular and nuclear damage.

In this thesis, an attempt has been made to understand the seed physiology (Chapter 2), ROS metabolism (Chapter 3) and DNA quality, telomere length and telomerase activity (Chapter 4) during natural and accelerated ageing and their priming. The Chapter 5 includes the summary and conclusion based on results obtained. The findings of the thesis will contribute significantly to our understanding seed of senescence and its management.