Aging represents a progression of biochemical events which ultimately lead to the death of an organism. In plants and animals, aging is characterized by a gradual decline in the capacity to synthesize macromolecules (Woolhouse, 1969). Since cellular metabolism and integrity depend on a great variety of enzymes and structural proteins, the ability to synthesize, activate, and preserve various proteins in sufficient quantities seems imperative for survival. Age-associated changes in cotyledon proteins may result from random intracellular damage or mistakes in transcription or translation (Orgel, 1963; Strehler & Freeman, 1980), from activation of genes or gene products for an aging program or from gene repression (Martin & Thimann, 1972; Woolhouse, 1977), or simply from substrate or hormone depletion (Lindoo & Nooden, 1978).

A seed which fails to germinate through aging, when given water and the appropriate environment for reactivation of its biochemical processes, is said to have lost viability (Roberts, 1972). Seeds from low percentage viability stocks have a diminished ability to synthesize RNA and protein (Barker & Bray, 1972; Osborne et al., 1974; Bray & Dasgupta, 1976; Sen & Osborne, 1977), show a failure of respiratory coupling (Ching, 1973), loss of enzyme activities (dehydrogenases [Lakon, 1949], GTP-dependent transferase [Roberts & Osborne, 1973; Bray & Chow, 1976a; Dell’ Aquila et al., 1976]), impairment of ribosomes (Roberts et al., 1973; Bray & Chow, 1976b) and the loss of long-lived poly A-rich RNA (Osborne et al., 1977); all are events associated with slow germination and vigor loss. To date, however, little is known of the biochemical status of the genetic material during loss of seed viability or germination ability.

In 1901, de Vries first questioned that a change in DNA might be the cause of poor germination and the high proportion of morphologically abnormal plants produced from old seeds of evening primrose.