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Structural Mechanism of Seed Deterioration

(*Bhanu Verma, Ashok Kumar Mahawer and Jagdish Yadav)
ICAR- Indian Agricultural Research Institute, New Delhi
*bhanu3904@gmail.com

The process of seed deterioration is difficult to complex, therefore, the conclusions from seed deterioration studies are often evaluate. There are mainly two reasons for this.

1. Most seed deterioration studies have focused on while seed germination or considering the fact that seed deterioration probably does not occur uniformly through enzyme the analysis seed. Thus, without it should be recognized that the seed parts vary in their chemistry and susceptibility to seed deterioration. Studies examining this differential susceptibility of seed parts which collectively influence the whole seed response should be useful in understanding the mechanisms of seed deterioration.
2. All seed lots are composed of individual seeds, each possessing its own unique capability to perform in the field.

Thus, total population studies of seed deterioration based on the performance of the sub sample do not represent what is occurring at the individual seed level. Despite these difficulties, the most visible symptoms of seed deterioration are observed first at the whole seed morphological level and then during germination and seedling growth. However, these are preceded by numerous uitra structural and physiological changes whose symptoms are not as rapidly apparent but can be detected by sophisticated monitoring techniques that attempt to identify changes in the deteriorating seed at the physiological level

Seed Symptoms

a. Morphological changes : e.g. seed coat colour
b. Ultra structural changes : e.g. coalescence of lipid bodies withdrawal of plasma lemma
c. Cell membrane integrity : enhanced seed leachate
d. Loss of enzyme activity : ROS enzymes, respiratory enzymes
e. Reduced respiration
f. Increase in free fatty acids

Performance symptoms: Low germination, delayed seeding emergence, slower rate of seedling growth etc.

Possible Causes of Seed Deterioration

- **Lipid peroxidation**: Of all the models represented to explain seed deterioration, the lipoid peroxidation model has simulated the greatest interest (Wison and McDonald 1986b; Bewley 1986). A free radicle is an atom or a group of atoms with unpaired electron. They can be produced either through autoxidation or enzymatically by lipooxygenase which is present in many seeds. The auto oxidation mechanism is often

initiated by oxygen around unsaturated or polysaturated fatty acids such as oleic and linoliec acids which are most common on seed membranes. The results the release of the free radical often hydrogen from the methylene group of the fatty acid tht is adjacent to a double bond. In other cases, the free radical hydrogen may combine with other free radicals from carboxylic groups leaving a peroxy fee radical. Once these free radicals are initiated, they create profound damage to the membranes, particularly those where electron transport is frequent, and continue to propagate other free radicals until they combine with free radicals which terminate the reaction. The result is the loss of membrane integrated in the case of phospholipids.

It has been noted that lipid auto oxidation occurs in all cells, but in fully imbibed cells, water act as a buffer between the reactive compounds and macro molecules, those preventing enzyme inactivation. Lipid Autoxidation is accelerated at high temperature and increased concentration .Harrington considered this to be a cause of seed deterioration only at moisture content below 6%, since moisture contents from 6% to 12% maintain seed viability, and above 12% other factors for deterioration.

Lipoxygenase enzymes also generate free radicals. However, their activity is greatest when the seed moisture content exceeds 14%, while auto oxidation is believed to occur primarily at low seed moisture contents. Thus, the mechanism of lipid peroxidation may be differing under accelerated ageing (lipoxygenese).Compared to long term ageing (autoxidation) conditions. It should also be noted that oxygen is deleterious to seed which storage based on this proposal, is consistent with the success of hermetic seed storage and that lipid-peroxidation causes loss of membrane integrated. Free radicals also attack compounds other than fatty acids. Changes in protein structure of seeds have also been observed and attributed to free radicals. Free radicals of lipid peroxidase damage cytochromesC by changing its physical and catalytic properties. Sulfhydryl levels decreases in wheat flour with increasing oxygen content. The "sick wheat svndrome" which results in a discoloration of the embryo with increasing storage time has been attributed to condensation reaction between lysine a oe methonine protein residues and reducing sugars. Many of this mutations can he detected as chromosomal aberrations which delay the onset of mitosis for germination while this chromosornal aberrations delay seedling growth, continued development of the seedling results in fewer cells with chromosornal irregularities, Presumely because abnormal cells are not able to compete the normal ones. As, a result, it is argued that mitotic lesions are unlikely to affect the genetic integrity of stored Germplasm.

Degradation of functional structures: As seed deterioration progress, cellular membranes lose their selective permeability. Permitting the cytoplasmic metabolites to leach into the intercellular spaces. Mitochondrial degradation and functional changes appear to play a major role in seed deterioration. Such changes decrease in number as deterioration procedes; mitochondria becomes pemanently swollen and lose their natural swelling-contracting ability, later they become pigmented and fragmented. Degradation of mitochondrial membrane also occurs, leading to the loss of function and eventual fragmentation. Two important aspects of mitochondrial deterioration are an increase in ATPase and a decline in oxidative phosphorylation ability nécessary to complete its respiratory function which leads to low level of ATP fomation. Sinoe ATPase catalyses the breakdown of ATP and ADP, it depletes energy available in the mitochondria.

Inabilty of ribosomes to dissociate: Associated with the degradation of function in deteriorating seeds is the dissociation of ribosomes. Recent evidences indicate that the dissociation of polyribosomes must occur before attatchment of preformed mRNA occurs,

leading to protein synthesis in germinating seedlings. In nonviable seeds, the ribosomes fail to dissociate and protein synthesis is retarded.

Enzyme degradation and inactivation: Decreased activity of enzymes such as catalase, dehydrogenase and glutamic acid decorboxylase in deteriorating seeds is well documented. The general decrease in enzyme activity in the seed lowers its respiratory potential, which in tum lowers both the energy (ATP) and food supply to the germinating seed. Several changes in the enzyme macromolecular structure may contribute to their lowered effectiveness. They may undergo compositional changes by losing or gaining certain functional groups. The enzymes may also undergo rational changes such as

1. Partial folding or unfolding of ultrastructure,

2. Condensation to form polymers

3. Degradation of subunits.

Breakdown in mechanisms for triggering gemination: Harrington (1973) has made a strong case for the idea that the breakdown of various triggering mechanisms also causes seed deterioration. Role of gibberellins and cytokinins in triggering the enzyme acivity leading to gerrmination is well known. Further evidence for this theory is the improved germination in ageing seeds ater exposing to growth hormones.

Other Causes Are

- Formaion and Activation of Hydrolyüc enzymes
- Genetic degradaion
- Depletion of Food Reserves
- Starvaion of Meristenatic of Cells
- Accumulation Toxic Compounds