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Study of Seed Dormancy Mechanisms; Causes and Control

Syeda Nasreen, M. Yousaf, Akbar S. Mohmand and M. Ashraf Mailk
Oilseed Research Programme, National Agricultural Research Center, Park Road, Islamabad, Pakistan

Abstract: Dormancy mechanisms in cereals and oilseed crops were reviewed. Objective was to determine the causes of seed dormancy. The methods of control of seed dormancy were also reviewed and the following results were obtained. During seed dormancy, a mature embryo remains inactive but viable. An understanding of seed dormancy mechanisms can be helpful in optimizing the distribution of seed germination in time or space. Seed coats, cotyledons and growth hormones play an important role in maintaining seed dormancy. Seed dormancy can also be controlled by environment and genetic factors.

Key words: Seed dormancy, inhibitors, hormonal theory, imbibition, dormancy mechanisms

Introduction

Sometimes seeds are sown that do not have the capacity of germination even in the presence of favorable environmental conditions. Either they are non-viable, empty or dormant. The seed germinating capacity can be determined by performing a germination test (ISTA-Rules, 1985). When fresh ungerminated seeds remain at the end of the test period, they are suspected to be dormant. Such seeds do not germinate until some particular requirement (either endogenous or exogenous) is satisfied (Butler). Dormancy is a state in which there is a block to germination due to some inherent inadequacy of the mature embryo, which may be manifested only when the embryo suffers some external constraints such as that imposed by the seed coat or other enclosing tissues.

Categories of dormancy: According to their manner of seed origin, different categories of seed dormancy can be recognized.

Primary dormancy: Dormancy inherent in the seed at the end of its development on the mother plant - Dormancy arises in the developing and maturing seeds.

Secondary dormancy: Seeds whose germination has been inhibited fail to recover even when the inhibitory factor is removed. These seeds are said to enter in a state of dormancy called secondary or induced dormancy.

Biological significance of seed dormancy: Seed dormancy is a device for optimizing the distribution of germination in time or space and its importance is therefore best seen in ecological context. Distribution in time can be achieved by spreading germination over an extended period. This is because seeds of many species show variability in depth of dormancy. (Bewlyey, 1982). Basic patterns with respect to the temporal distribution of germination were recognized by Salisbury (1961) which are:

a Quasi-simultaneous, when germination of all the seeds occurs over a relative brief period.
b Intermittent, irregular germination over long time periods, showing essentially multi modal distribution
c Continuous, in which members of the population germinate over an extended time period, with no clear peaks.
d Periodic germination, which is multi modal but shows more regular periodicity.

The above mentioned patterns result from the dormancy characteristics and also from the interplay between these and the control of germination itself by the various environmental factors such as temperature.

The dormancy mechanism can also operate to secure a suitable place for germination. For example, those seeds whose dormancy is broken by light are clearly unable to germinate when buried deeply in soil. Germination is thus limited to the top few millimeters of soil. This can be an advantage to a small seedling carrying food reserves which support growth only for a relatively short time since it can quickly establish itself as a photosynthesizing autotrophic plant.

Dormancy mechanisms: There are basically two types of dormancy which involve different mechanisms.

Embryo dormancy: where the control of dormancy resides within the embryo itself.

Coat imposed dormancy: in which the dormancy is maintained by the structures enclosing the embryo, i.e. the seed coat.

Control mechanisms in embryo dormancy: The embryo dormancy can be recognized by the failure of the viable, mature embryo to germinate even when it is isolated from the seed or dispersal unit. Such naked embryo when placed on a wet substratum, remains dormant even though the conditions are suitable for germination itself. If the intact seed has previously been subjected to a dormancy breaking treatment, the embryo does germinate showing that conditions really are favorable.

The evidence suggests that the control of embryo dormancy involves:

Cotyledons: In many cases the cotyledons are responsible for inhibiting the growth of the axis in dormant embryo. Part of this evidence comes from experiments in which one or both cotyledons have been removed by cutting off just one cotyledon of *Euonymus europeus* (Bulard, 1960). This procedure is also effective in haze (Jarvis, 1975). Amputation of both cotyledons is needed to cause germination of Fraxinus excelsior embryos (Bulard, 1963). Embryonic axes of barley cultivars with embryo dormancy can be stimulated to germinate by excision of the scutulum (which is regarded as modified cotyledon). (Grahl, 1970). These are the findings which strongly suggest that dormancy of the axis in the intact embryo is maintained by some action of cotyledons. The physiological and biochemical basis for the action of cotyledons in dormancy is unknown. There is little evidence that abscisic acid (*Coryllus avellana*) derived from the testa is present in the cotyledons (Jarvis, 1975).

Role of germination-inhibitors: An important inhibitor found in most of the dormant embryo is Abscisic acid (ABA). This is suggested by the finding that enlargement and greening is prevented in cotyledons wetted with a solution of ABA (Durand, 1975). It can be postulated that the inhibitor especially those in the cotyledons control the growth of the axis.

The experimental evidence for this hypothesis comes from:

a Inhibitors are found in embryos of many species possessing embryo dormancy.
b Leaching out of the inhibitors is found to promote germination in isolated dormant embryo.
c Treatments which break dormancy in some cases cause a drop in inhibitor levels in the embryo.

Inhibitors have been isolated from dormant embryos of several species. The most prominent member is abscisic acid (ABA). It is found in the embryos and also in the covering tissues (Sondheimer, 1966). Here correlations can be seen between the depth of embryo dormancy and the concentration of Abscisic acid. For example, the more deeply dormant embryos of the lovell cultivar of peach (*Prunus persica*) contains approximately 1.5g ABA/g dry weight, whereas the less dormant embryos of the Tetela cultivar have only 0.2 microgram ABA/g dry weight (Martin, 1972). Prolong washing (leaching) can induce germination in dormant embryo of Sorbus (Flemion, 1959). Leaching out of inhibitors (possibly coumarin) from dormant embryos of Elaegnus umbellate provokes this germination (Hamilton, 1976).

Coat-imposed dormancy: Seed dormancy in the majority of species is imposed by the structures surrounding the embryo. These are often referred to as the seed coat. The seed of many grasses consists of a caryopsis which are in turn surrounded by soft papery glumes. The glumes are easy to remove but lemma and palea are not. The plumule can emerge only by pushing the lemma and palea apart. If lemma and palea are firmly locked together, the expansion on the plumule may be stopped. This may be the reason why some grasses, e.g., *Brachiara* spp. respond to acid scarification (Howells, 1971). The machanism by which the seed coat imposes dormancy is poorly understood but the evidence points to a number of possibilities. The covering structures may prevent embryo germination because they;

a Interfere with the water uptake. (Ballard, 1973.
b Interfere with the gaseous exchange (Brown, 1940)
c Contain chemical inhibitors (Torrey, 1976).
d Act against a barrier against the escape of inhibitors from embryo (Wareing, 1957).
e Modify the light reaching the embryo (Karssen, 1970)
f Exert a mechanical restraint (Egley, 1974).

One or more of the above mentioned factors may be responsible for the maintenance of dormancy.

Control of dormancy: The entry into dormancy is controlled by several factors.

Genetic factor: The dispersal units generally consist of three genetically different tissues:

a A diploid embryo produced by fertilization of the ovum,
b A triploid endosperm containing one set of paternal genes and two sets of maternal genes and
c The diploid testa, pericarp, glumes, palaes and lemmas all of maternal genetic constitution.

Dormancy can be inherent within the embryo or can be imposed by these extra embryonic tissues.

Environmental factor: Vegis has developed a theory of dormancy inception which invokes a combined effect of relatively high temperature(s) and the limited oxygen supply to the embryo imposed by the covering tissues. Dormancy is progressive (Vegis, 1964) consisting of three phases. These phases are characterized by the narrowing in pre-dormancy and widening in post dormancy of the temperature range which allows germination. In pre-dormancy, the temperature range over which the seed is dormant gradually become wider, until true dormancy is achieved; the gradual termination of dormancy subsequently occurs as the temperature range for germination then widens. Seed dormancy in many species varies with the provenance. For example, seeds of Tsuga canadensis are more dormant when produces in the Southerly latitudes of Tennessee. Pinus strobus also produces more dormant seeds at more Southerly locations. But Liquidamber styraciflua seeds, when coming from the Northerly New Jersey are more deeply dormant than those derived from Louisiana in South (Baskin, 1973). Variation in depth of dormancy also occurs from year to year. The seeds of certain hybrids of Rosa vary greatly in their dormancy characteristics according to the years in which the crosses were made (Von, 1966).

The thermoperiod (i.e. day and night temperature) also influences dormancy. For example, seeds of Anagallis arrenis have a very low dormancy when produced on a regime of 30-25°C (Day/night) and extreme dormancy at 20-15°C (day/night).

Hormones: Dormancy is controlled by interacting promotor and inhibitors. The inhibitor ABA is an important inducer of dormancy. The inhibitor, Abscisic acid (ABA) appears in the grains during their development and is retained into maturity by the dormant varieties but not by those which are non-dormant. Inhibitors extracted from dispersal units form variety of chemical compounds like coumarin, oxalate and vanillic acid. Abscisic acid can inhibit RNA and protein synthesis in vivo (Chen, 1970).

The development of Hard coat: The hard-coated condition is inherited (Barton, 1965) and may be induced by environmental factors. Nutritional factors are additionally important, for example, relatively high levels of calcium promote hard-coateness in crimson clover. The time of flowering and harvest, locality and climate are influential (Barton, 1965) of particular importance are the rate and degree of drying of the seed, the coats becoming increasingly hard and impermeable as the moisture content of the seed falls (Barton, 1965). It is generally agreed that impermeability is brought about by the dehydration of the seed. Andrew (1957) working with *Medicago tribuloides*, found that the precursors for impermeability were present in the seed coat 21 days after flower fertilization. The impermeable layer completely forms at 9.5% seed moisture (Andrew, 1975). Generally, if the seed moisture content exceed 12-14%, the seed coat is permeable and if the seed moisture content is less than 3-4%, the seed coat is irrevrsely impermeable. At intermediate seed moisture content, the seed coat is impermeable but may be made permeable by manipulating the external relative humidity over a period of several months.

Discussion

Failure of the seed to germinate under optimum conditions does not conclusively prove that the seed is not viable. The other possibility remains namely, it may be dormant. The most reliable, simple and quick test for viability and dormancy is the tetrazolium test. In this method, the seed is soaked in a solution of tetrazolium chloride. The principal behind this is that, in the viable seed, the presence of certain enzymes like dehydrogenase oxidase oxidize the colorless dye which turns deep red (International Seed Testing Association, 1985). The basic course of dormancy is the inability of the embryonic axis to overcome the constraints against growth which are acting upon it, constraints which reside within the embryo itself (embryo dormancy) or belong to the enclosing structures (coat-imposed dormancy). As many events occur during germination, it seems possible that a block on any one could account for the failure of the embryonic axis to grow.

According to the "Hormonal Theory", dormancy depends on the interaction between inhibitory and promotive "hormones", such as abscisic acid and gibberellin. To a large extent, the theory owes its origin to the effects of exogenous growth regulators on germination and dormancy, in which dormancy can be maintained, imposed or released by these chemicals applied singly or in combination (Amen, 1968). Such effects are considered to reflect the situation in the seed where it is held; inhibitors impose and maintain dormancy and promotor relieves it. All the various natural releasing factors such as light, chilling, alternating temperatures and after ripening may operate by causing changes in blancs of these inhibitors and promoters. The important events that occur during the germination of seed are: Imbibition, Hydration of the protoplasm, Activation and synthesis of Enzymes, Increase in respiration, increased synthesis of nucleic acids and proteins, Synthesis and release of hormones from the embryo, Increased cell enlargement and cell division, Hydrolysis of reserve food substances present in the endosperm and cotyledons, Utilization of soluble organic substances by the developing embryo, Growth of the radicle into the root and the plumule into the shoot.

If the seed is not dormant and if it is viable, it soon starts germinating when placed under favorable conditions. In the dry seed, the rate of metabolism low. After imbibition, the enzyme activity in seed is increased. This increase may be because of two reasons: Either the existing enzyme molecules which are inactive in the dehydrated condition become activated upon hydration. New enzyme molecules may also be freshly synthesized. The first detectable change in the metabolism of germinating seed is the rise in respiratory rate which begins 1-2 hrs. after imbibition. Depending upon the type of reserve food in the seed, the ratio of the volume of oxygen taken to the volume of carbon dioxide given out, i.e., RQ is also different. It is less than 1 in Sun flower and other oilseed where fatty acids are the respiratory substrate. It is close to 1 in cereals which contain carbohydrates. During germination, there is an active synthesis of both DNA and RNA in the embryo. This is followed by rapid mitosis and emergence of the radicle. This is the first visible sign of germination. Further growth of seedling is at the expense of reserve food supplied by the cotyledons in oilseed crops such as in sunflower and by the endosperm in cereals. During germination, hormones are released by the embryo and are transferred to endosperm or cotyledons. Under the influence of hormones, hydrolyzing enzymes are synthesized. In castor, peanut, sunflower and other oilseeds, the enzyme lipase hydrolyses fats into fatty acids and glycerol. In general, there are several naturally occurring compounds which inhibit germination of seeds. Seeds of many species are known to contain several phenolic compounds such as ferulic acid, parasorbic acid, coumarin and other. These compounds are present either in the embryo

or in the seed coats and inhibit germination. In most of the oil seed crops like sunflower, presence of an inhibit hormone in the seed namely abscisic acid prevents germination (Page-Degivry, 1993).

References

Amen, R. D., 1968. A model of seed dormancy. Bot. Rev., 34: 1-31.

Andrew, 1957. Interaction of the moisture and nutritional regimes of hard seed production in Barrel medic, Medicago tribuloides. Desr. Aust. Plant Nutr. Conf. Proc., 1: 79-91.

Ballard, L. A. T., 1973. Physical barrier to germination. Seed Sci. Technol., 1: 285-303.

Barton, L. V., 1965. Seed Dormancy, In: Encyclopedia of Plant Physiology, Ruhland, W. (Ed.) Berlin, springer, pp: 909-924.

Baskin, J. M., 1973. In Physiology and Biochemistry of seed. Am. Midl. Nat., 90: 493-497.

Bewlyey, J. D., 1982. Viability. Dormancy and Environmental Control. In: Physiology and Biochemistry of Seed, pp: 62-65.

Brown, R., 1940. In: Physiology and Biochemistry of seeds Ann. Bot. (London) 4: 379-395.

Bulard, C., 1960. In Physiology and Biochemistry of seeds J. D. Bewlyey. C. R. Acad Sci., 250: 2922-2924.

Bulard, C., 1963. In Physiology and Biochemistry of seeds, J. D. Bewlyey. Phyton (Buenos Aires), 20: 115-125.

Butler, J. E. (year not known) Seed Dormancy and Hard seededness; Standards Branch. Dept. of Primary Industries Queensland (personnal communication).

Durand, M., 1975. In Physiology and Biochemistry of seeds, J. D. Bewlyey Physiol. Veg., 13: 603-610.

Egley, G. H., 1974. In Physiology and Biochemistry of seeds Weed Sci., 22: 535-540.

Flemion, F., 1959. In Physiology and Biochemistry of seeds Contrib-Boyce. Thompson Inst., 20: 57-70

Grahl, A., 1970. In Physiology and Biochemistry of seeds. Proc. Int. Seed Test Association, 35: 427-438.

Hamilton, D. F., 1976. In Physiology and Biochemistry of seeds. Can J. Bot., 54: 1068-1073.

Howlls, K. J., 1971. Saw Scarifier for large-grained legumes. J. Aust. Inst. Agric. Sci., 37: 58-160

International Seed Testing Association, 1985, Seed Sci. and Technol., 13: 299-355, Zurich, Switzerland.

Jarvis, B. C., 1975., In Physiology and Biochemistry of seeds. New Phytol., 75: 491-494.

Karssen, C. M., 1970. In Physiology and Biochemistry of Seed. Acta Bot., Neerl., 19: 297-312.

Martin, M. H., 1972. In Physiology and Biochemistry of Seeds. New Phytol., 71: 99-104.

Page-Degivry, L. E., 1993. Involvement of ABA in hormonal regulation of sunflower embryo dormancy-proceedings of the fourth International workshop on Seeds; Basic and applied aspects of seed Biology. Angers, Francs., 20-24 July, 1992. 615-623; 19 ref.

Salisbury, E., 1961. In Physiology and Biochemistry of Seeds. Weeds and Aliens, London: Collins.

Sondheimer, E., 1966. In Physiology and Biochemistry of Seeds Pl. Physiol., 43: 1443-1447.

Torrey, J. G., 1976. In Physiology and Biochemistry of Seeds. J. G. Bot. Gaz., 137: 262-268.

Vegis, A, 1964. Dormancy in higher plants. Ann. Rev. Plant Physiol., 15: 185-224.

Visser, T., 1956. In Physiology and Biochemistry of Seeds. Proc. K. Ned. Akad. Wed. C., 59: 314-324.

Von, A., 1956. In Physiology and Biochemistry of seed. Am. J. Bot. 43, 7-12.

Wareing, P. F., 1957. In Physiology and Biochemistry of Seeds. Physiol. Plant, 10: 266-280.