Seed Dormancy: Give Your Seeds a Wake-Up Call! ©

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INTRODUCTION

Seed dormancy is a condition where seeds will not germinate even when the environmental conditions (water, temperature, light, and aeration) are permissive for germination (Hartmann et al., 2011). Not only does seed dormancy prevent immediate germination, it also regulates the time, conditions, and location where germination will occur. In nature, different kinds of dormancy have evolved to aid the survival of a species by programming germination for particularly favorable times in the annual seasonal cycles (Baskin and Baskin, 1998).

The major seed dormancy categories include:

1) Primary dormancy:
   a. exogenous dormancy (physical).
   b. endogenous dormancy (physiological, morphological, and morphophysiological).
   c. combination dormancy (physical plus physiological).
2) Secondary dormancy.

The focus of this paper will be to describe the characteristics associated with the major dormancy types.

PHYSICAL DORMANCY

Exogenous physical dormancy is imposed upon the seed from factors external to the embryo including the outer seed coat or parts of the fruit coverings (Hartmann et al., 2011). The main reason seeds with physical dormancy fail to germinate is because the seed are impermeable to water. Physical dormancy is found in at least 17 plant families, including horticulturally important families like the *Fabaceae*, *Malvaceae*, *Cannaceae*, *Geraniaceae*, and *Convolvulaceae* (Baskin et al., 2000). For horticultural crop production, seeds are scarified to mechanically abrade the seed coverings or seeds are treated with concentrated sulfuric acid to alleviate physical dormancy (Hartmann et al., 2011). In nature, exposure to high temperature or fluctuating temperatures is the most likely cause of dormancy release (Geneve, 2003).

Two features characterize seeds with physical dormancy. Physically dormant seeds have an outer seed or fruit cell layer comprised of macrosclereid cells and there is also a surface feature within the outer seed layers that functions as a water gap to allow water imbibition.

Macrosclereid Cell Layer

Macrosclereid cells form the outer cell layer in the seed coat or fruit wall in physically dormant seeds and are responsible for preventing water uptake (Fig. 1). These cells belong to a plant cell type called sclereids. Sclereids are characterized by extensive secondary wall formation and are usually non-living at maturity. During the later stages of seed development, the macrosclereid cells become impermeable to water and seal the seed making the seed (or fruit covering) impervious to water (Rolston, 1978).

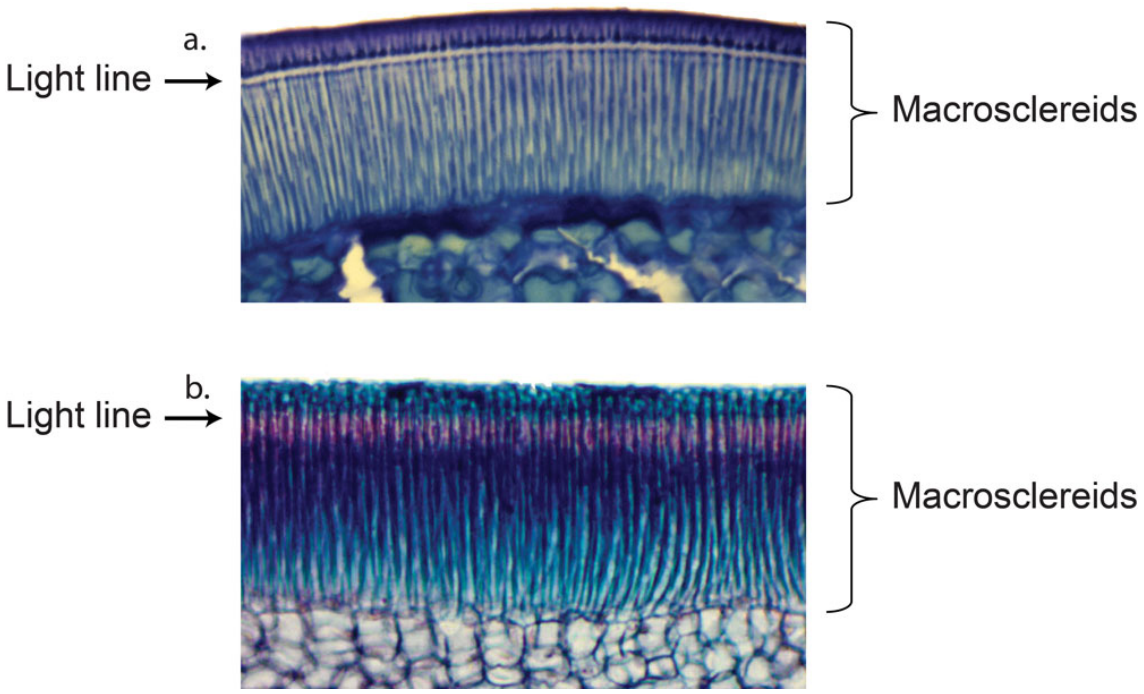

Fig. 1. Macrosclereid layer and light line in (a) eastern redbud (*Cercis canadensis*) and (b) canna (*Canna indica*).

The Water Gap

There is usually a single area of the seed coat that acts as a water gap to relieve physical dormancy and initiate imbibition. It is thought that water gap structures act as environmental sensors to detect appropriate times for germination based primarily on temperature (Baskin et al., 2000). Water gap structures are usually associated with areas of the seed coat where there were natural openings in the ovule during seed development such as the hilum, micropyle, and chalaza.

For many seeds, temperature is the environmental signal that relieves physical dormancy (Hartmann et al., 2011). High temperature or temperature fluctuations physically alters the water gap, which opens to permits water uptake (Baskin et al., 2000). For example, treating *Canna* seeds with moist heat for 24 h causes a temperature-sensitive water gap structure (the imbibitonal lid) to erupt and separate from the seed allowing water entry into the seed (Fig. 2).

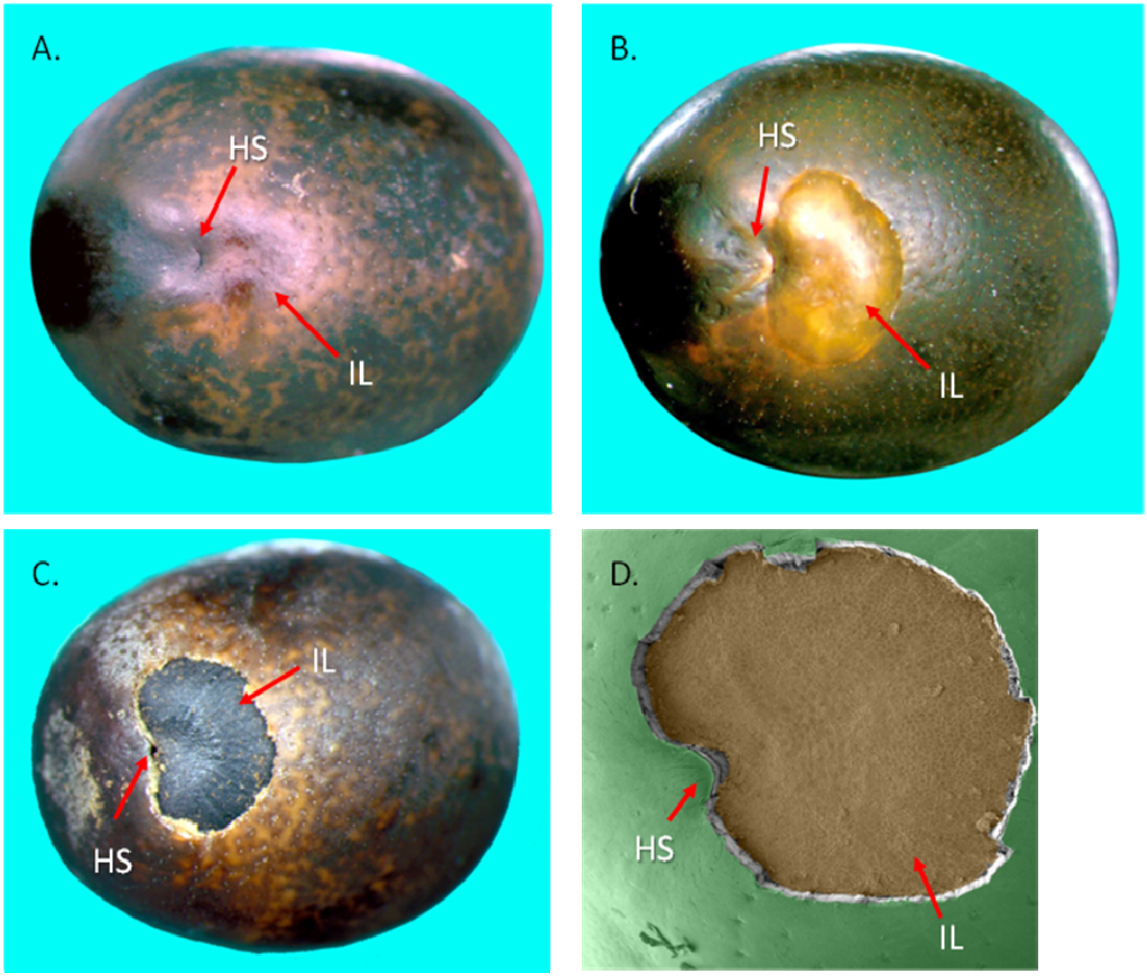

Fig. 2. Dormancy release in canna (*Canna indica*) before (A) and after (B) treating the seed in boiling water for one minute. (C) and (D) Seed after removal of imbibitional lid.

PRIMARY ENDOGENOUS DORMANCY

Seeds with endogenous dormancy fail to germinate primarily because of factors within the embryo and endosperm. These factors can be either physiological or morphological or a combination of the two.

The most common mechanism for delaying germination is physiological dormancy. The basic model for maintenance of physiological dormancy is that the embryo lacks the growth potential to allow the radicle to escape the restraint of the seed coverings (Fig. 3). Growth potential is the force used by the radicle to penetrate seed coverings. Many species with physiological dormancy have seeds that germinate normally if the seed coverings over the radicle are removed (the exception is deep physiological dormancy). The physical strength of the endosperm and seed coverings has been shown to restrict germination in both herbaceous (lettuce, pepper, and tomato) and woody (redbud and lilac) plants. Dormancy in these species is overcome by weakening seed coverings, by increasing growth potential in the embryo, or by a combination of seed covering and embryo effects.

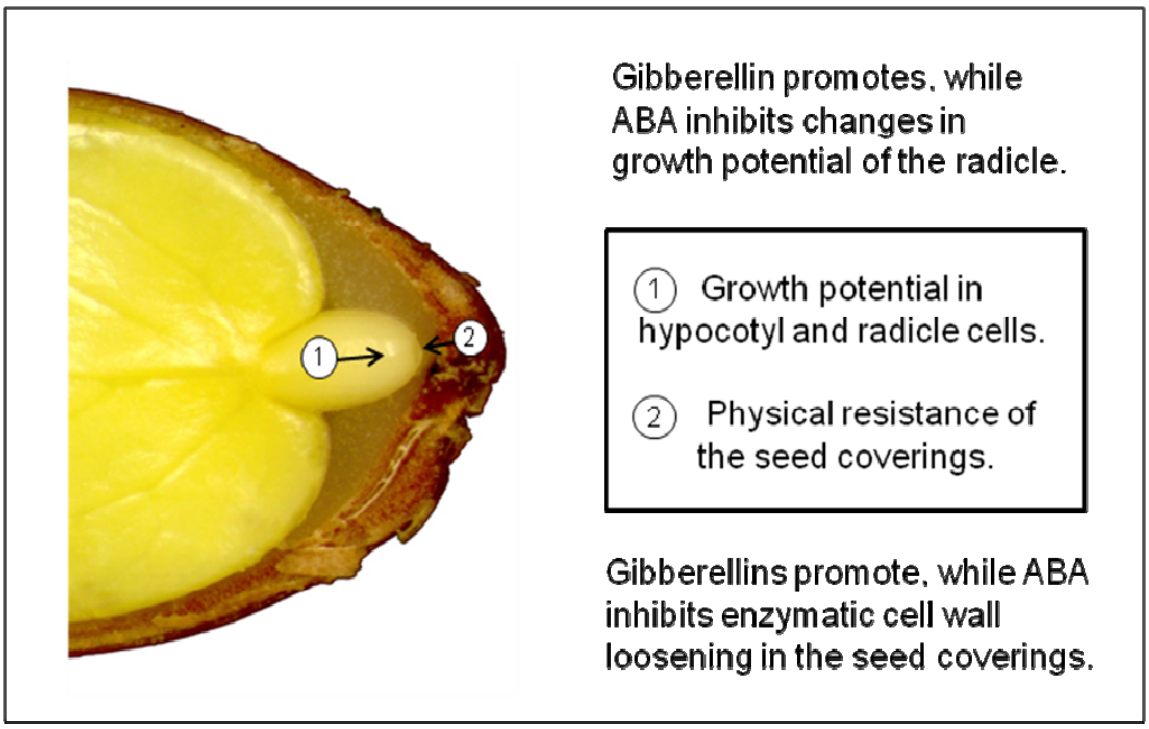

Fig. 3. The basic model for physiological dormancy involving the contrasting forces of growth potential from the embryo versus the restricting forces of the covering materials.

The current model for hormone interactions related to dormancy release in seeds involves the major hormones abscisic acid (ABA) and gibberellin (GA) (Finkelstein et al., 2008). Abscisic acid controls the establishment and maintenance for dormancy, while GA appears to control initiation and completion of germination. The ratio of ABA to GA induced signal transduction is as important as the active hormone levels for dormancy release (Finch-Savage and Leubner-Metzger, 2006; Kucera et al., 2005). Other hormones, especially ethylene, have a modifying impact on this relationship (Fig. 4). It has become well established that there is an antagonistic relationship between ABA and ethylene for a number of plant responses including seed dormancy and germination (Beaudoin et al., 2000). Therefore, one common mechanism for stratification and ethylene-induced dormancy release in plant species such as *Echinacea* could be through a change in ABA sensitivity (Woods and Geneve, 2008).

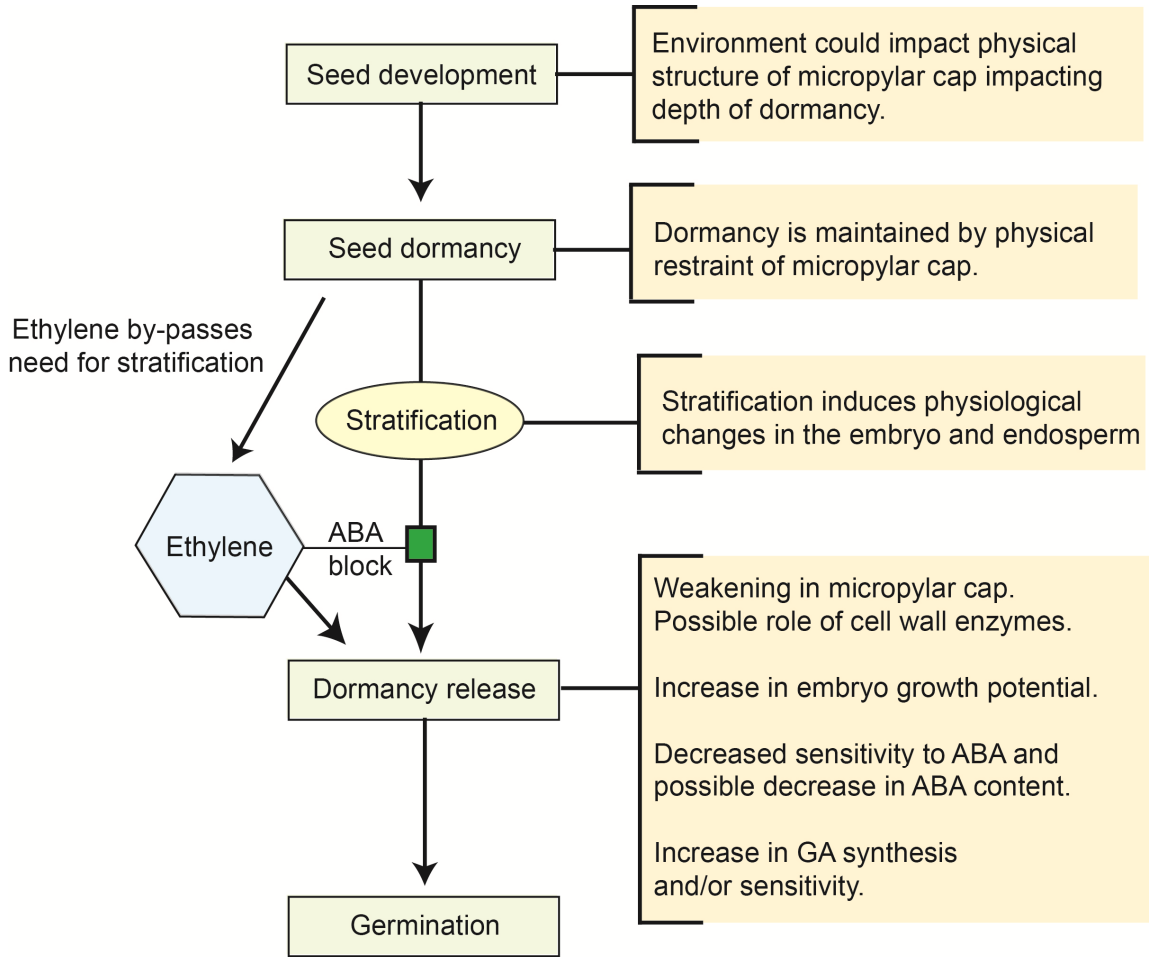

Fig. 4. A general model for dormancy maintenance and release in seeds with physiological dormancy.

MORPHOLOGICAL AND MORPHOPHYSIOLOGICAL DORMANCY

Morphological dormancy occurs in some seeds where the embryo is not fully developed at the time of seed dissemination. Seeds have morphological dormancy if they require more than 30 days to germinate, have an embryo that fills less than one-half of the mature seed, and have an embryo that must grow inside the seed before the radicle can emerge (Baskin and Baskin, 1998). The process of embryo growth is usually promoted by a period of warm temperature, but can also take place during chilling temperatures.

Seeds with morphophysiological dormancy have an underdeveloped embryo that also displays physiological dormancy. In some cases, the morphological dormancy must be satisfied before physiological dormancy release. These seeds take longer than 30 days to germinate. The simplest form of morphophysiological dormancy is found in species such as *Heptacodium* that requires only warm temperatures to relieve dormancy (Fig. 5). In other cases there is more of an obvious two step process where warm stratification induces the embryo to grow to a critical size within the seed followed by moist chilling to satisfy physiological dormancy, which finally permits germination.

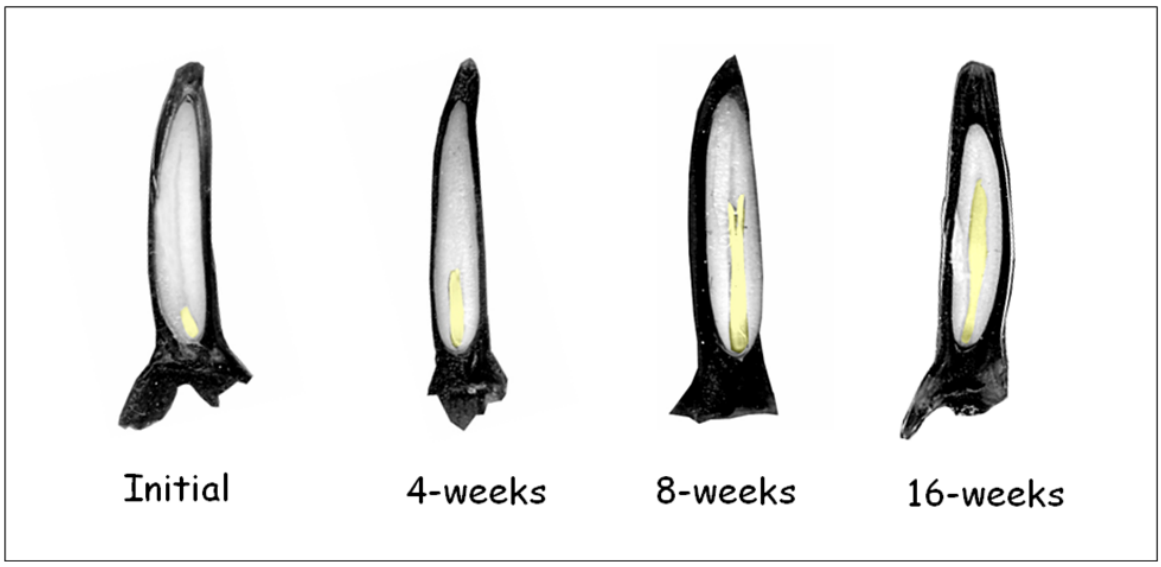

Fig. 5. *Hepticodium* displays simple morphophysiological dormancy. Embryo growth within the seed during dormancy release in *Hepticodium* proceeds for 12 weeks of warm temperature.

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