8.1.4 Physiological control of dormancy

(a) Hormones as regulators?

Currently we know more about the environmental factors that influence dormancy than about the physiological mechanisms of dormancy. Here we attempt to draw together common features of the diverse types of dormancy in buds and seeds, in particular examining whether inability to grow relates to hormonal factors (Dennis 1994).

Links between genome and physiological processes are illustrated by single-gene seed dormancy mutants, which are either abscisic acid (ABA) deficient (weak dormancy) or gibberellin deficient (extra-deep dormancy) (Karssen and Groot 1987). Induction of seed dormancy is clearly linked to ABA, and gibberellins are required for germination, so in a gross sense these hormones need to be present for normal processes to proceed. Applied hormone experiments lead to similar conclusions: although ABA does not usually prevent break of dormancy, it can inhibit germination and bud growth, often opposing the effects of gibberellins, cytokinins or ethylene. Seeds with various dormancy mechanisms may respond to one or more plant growth regulator (Table 8.3), but there are many reports of germination failure or abnormal seedlings. Light requirement of lettuce and dry storage requirements of barley are overcome by applied gibberellins, but antagonised by applied ABA. Likewise, budburst in peach and apple is promoted by a mix of gibberellin and cytokinin, but inhibited by applied ABA. Cytokinins promote some germination in lettuce but are less effective than gibberellins in most species. Ethylene stimulates germination in celery (Apium graveolens), peanut (Arachis hypogea) and cocklebur (Xanthium strumarium). One conclusion is that a complex balance of inhibitors and promoters regulates entry to and exit from dormancy. Put another way, there are at least two control points and meristem growth may be prevented by either high concentrations of inhibitors or insufficient promoters.
However, data on endogenous plant hormone concentrations do not always support the notion of control by changes in levels of active substances. Quantities of applied plant growth regulators required to cause a response usually vastly exceed normal endogenous content, for example the amount of applied gibberellin required to stimulate barley germination. Rightly, this has led to re-examination of the control mechanisms. Trewavas (1982) argued that tissue ‘sensitivity’ to hormones, that is, capacity to respond, changes with development and environmental stimuli, and that this sensitivity is a major controlling factor. Indeed, phases of sensitivity and insensitivity to applied gibberellins and ABA appear to operate during development, dehydration and dry storage of sunflower seed (Figure 8.4). Other supporting evidence comes from gibberellin- and ABA-insensitive mutants which fail to respond to these hormones regardless of endogenous or applied concentration. Alterations in hormone levels due to mutation are generally much more severe than changes that occur in wild-type plants as a consequence of environmental factors. ABA-deficient tomato (Figure 8.5) and Arabidopsis mutants fail to enter normal dormancy because of a lack of increase in embryo ABA. Surrounding seed tissues absorb most applied ABA without translocating it to the embryo, which may also explain failure of

Figure 8.4 Responsiveness of sunflower embryos to applied gibberellin (GA) is seen only when dormancy has been partially released. Embryos were cultured on 5 µM gibberellic acid (solid symbols) or control medium (open symbols), before (circles) or after (triangles) a 3 d drying treatment which partially broke the endodormancy.

(Based on Le Page-Degivry et al. 1996)

Figure 8.5 Vivipary in wild-type tomato (Sit/Sit, i.e. ABA-synthesising) and ABA-deficient tomato (sit/sit). No seeds germinated within ripe tomato fruits derived from self pollinated Sit/Sit plants. Juice of ripe Sit/Sit fruits contains 0.84 µM ABA and each seed contains 7 pmol ABA. In contrast, vivipary occurred in most sit/sit tomato fruits which have only 0.08 µM ABA and 0.8 pmol ABA per seed. Self-pollinated Sit/sit plants would contain seed of both phenotypes but the mother plants possess the dominant Sit, allowing ANA synthesis. A quarter of the seed (those carrying Sit/Sit and Sit/sit) would not be.

(Based on Groot and Karssen 1992)
seed dormancy induction with applied ABA.

So what is the role of ABA in induction of seed dormancy? In late embryogenesis, ABA concentration increases as water potential decreases. Elsewhere in the plant, responses to altered water potential are also mediated by ABA, typically those associated with water stress (see Section 9.3). ABA alters transcription of a suite of genes, resulting in cessation of synthesis of reserve and other proteins, and modified transcription of some Lea genes (late embryogenesis abundant; see Chapter 10). In cotton, one class of Lea mRNAs increases coincidentally with ABA but another class responds only to drying. Lea genes code for a class of proteins found in many species including cotton, pea and cereals. These proteins are strongly hydrophilic, highly stable and are able to maintain a locally water rich environment at the subcellular level. This may be critical in desiccation tolerance associated with the dormant state.

There is a tenuous association of endogenous inhibitors with release (as distinct from induction) of bud or seed dormancy. Early research suggested a close correlation of progress of dormancy with inhibitors including phenolics such as naringenin in peach and phloridzin in apple, and ABA in several fruit crops. However, endogenous ABA declines in chilled apple buds which burst to produce new shoots, but also in buds never exposed to chilling temperatures which remain dormant. In both chilled and non-chilled apple seeds, ABA levels do not change more than two-fold but only chilled seeds germinate (Figure 8.6). ABA content is similar in dormant and non-dormant wheat but ABA-responsive genes are more abundantly expressed in dormant wheat seeds, implying existence of alternative regulatory factors and perhaps non-transcriptional control of the relevant genes. Embryo endodormancy may therefore be maintained by ABA in only a few species, such as sunflower (Helianthus annuus), where treatment of dormant excised embryos with fluridone, an inhibitor of ABA synthesis, results in growth.
Figure 8.6 Endogenous gibberellin and ABA levels during breaking of dormancy in apple seeds exposed to cold (4-5°C) or warm (20-25°C) temperatures. (a) Germination is dependent on cold treatment. (b) Embryo abscisic acid levels do not decline during cold treatment. (c) Embryo abscisic acid levels do not decline during cold treatment or during germination. (c) Seed gibberellin (GA4+7) levels increase transiently as seed start to germinate.

(Based on Subbaiah and Powell 1992 and Halinska and Lewark 1987; reproduced with permission of Kluwer Academic Publishers)

Can we instead assign control of dormancy break to promotive compounds? Gibberellins are probably the best candidates, based on widespread responses to applications of this class of hormone. In Salix pentandra, where short days induce dormancy and long days release it, a transient increase in active shoot gibberellin (GA1) content is detectable within one day of transferring from short days to long days (Figure 8.7). In hazelnut, endogenous gibberellins are not modified by chilling but GA1 content rises 40-fold after transfer to warm conditions suitable for germination, suggesting a role in growth promotion as distinct from dormancy release. Like-wise, in wild oats (Avena fatua), ‘after ripening’ dry storage releases seed dormancy but has no effect on endogenous gibberellin levels until imbibition, when gibberellin bio-synthesis is substantially enhanced. Light requirements can often be replaced by applied gibberellins, and gibberellin-biosynthesis inhibitors can prevent light-stimulated germination. Endogenous gibberellins increase with chilling and dry storage in Arabidopsis, and with light exposure in lettuce. Gibberellin-deficient Arabidopsis mutants do not germinate unless gibberellin is supplied, and this response is independent of ABA content. However, changes in endogenous gibberellins in wild-type Arabidopsis are less conclusive, suggesting that altered gibberellin sensitivity may
contribute to normal germination control. We are just beginning to understand tissue sensitivity and hormone signal transduction pathways (Section 9.3.1). To conclude, there are some species where there is good evidence for ABA-induced dormancy and gibberellin promotion of meristematic activity but these are not necessarily universal mechanisms. Hormone turnover, conjugation, compartmentation, receptors and signal transduction systems all represent potential control points, and all merit greater attention.

(b) Alternative indicators of dormancy

The hormonal models described above have limitations and some researchers contend that they represent oversimplifications of a complex set of interactive cyclic processes including organogenesis, internode elongation and bud leaf expansion (Crabbe 1994). Biochemical markers such as nucleic acid metabolism and membrane permeability, rather than morphological or physiological characteristics, can also indicate relative depth of dormancy between tissues and organs, and between meristems and submeristems. Adenylate nucleotides are required to maintain basal metabolic activity and even dormant tissues supplied with adenosine increase their adenylate nucleotide (ATP) content. During dormancy break in buds of *Helianthus tuberosus* (Jerusalem artichoke) tubers, levels of both adenylate and non-adenylate nucleotides (NATPs = sum of guanylic (GTP), cytidylic (CTP) and uridylic (UTP) nucleotides) rise as tissues convert ATPs to NATPs, which are essential to sustain growth (Gendraud 1977).

In stems, trunks and developing tubers bearing dormant buds, storage parenchyma acts as a strong sink during metabolite accumulation while nutrient movement into bud meristems may be impeded. Breaking dormancy appears to remove this block and is part of the changes that permit resumption of growth. Water status also influences dormancy. Dormant seeds and sometimes buds have lowered water content which limits metabolism and often assists survival (Vertucci 1989; Faust et al. 1995). Metabolic activities for growth require free water (bulk cellular water) but cannot occur in the bound water associated with macromolecular surfaces. Water content therefore determines the possible types of reactions: at low seed water content (0–8%) only catabolic and non-enzymatic activity occurs, but >25% water content is required for integrated processes such as mitochondrial electron transport and protein synthesis. Water content also determines the ability of seeds to perceive and respond to environmental cues. Apple seeds become sensitive to chilling temperatures only if hydrated to >8% water content, and many seeds such as the weedy coloniser species *Bidens pilosa* acquire light sensitivity only after imbibition.

Water content in bud tissue is generally higher and varies less but may still have a regulatory function. The state of water has been visualised in vegetative buds by using nuclear magnetic resonance imaging. Free and bound water content correlate strongly with bud dormancy release and chilling in low- and high-chill cultivars of apple, Anna (400–700 chill units, typical of subtropical regions) and Northern Spy (2600–3600 chill units, typical of the temperate zone). Very little free water (about 30%) is detectable in bud meristems at the beginning of endodormancy, but this increases to 70–80% after 400h at 4°C in Anna and 3000h in Northern Spy. Seed germination also requires free water, with metabolic activity suppressed in seeds having a water content below 30%. High osmotic potential of tomato fruit tissues may be partly responsible for seed dormancy by keeping seed water content low during late stages of development. With the exception of hard-coated species, most dormant seeds hydrate easily but this does not necessarily lead to immediate germination.

(c) Conclusion

Dormancy remains an intriguing but complex phenomenon. Clearly, plants are well attuned to making use of environmental cues. The ability to enter a period of latent life is remarkable in itself, all the more because plants in effect anticipate adverse conditions before their onset, and thus dormancy can
be established in advance. However, there is no single hypothesis to account for induction, maintenance and breaking of dormancy which is consistent across all species. Interactions of many metabolic and cellular processes with many genes are probably linked to hormonal signals. We need to appreciate more that hormonal control is intrinsically complex, and directly and indirectly influences genome expression, while mediating some environmental cues. Dormancy is a prime example of genotype × environment interaction. Plants use external signals to time entry into a ‘shutdown mode’ (endodormancy, paradormancy), then transition to a ‘standby mode’ (ecodormancy), but have internal controls to prevent inappropriate exit, instead fore-shadowing future favourable conditions. Continuing studies with single-gene mutants and transgenic plants (Chapter 10) should unlock some of dormancy’s secrets.

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