THE BOTANICAL REVIEW

Vol. 34 JANUARY-MARCH, 1968 No. 1

A MODEL OF SEED DORMANCY

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ABSTRACT

Seed dormancy is considered as an aspect of growth cessation. It is characterized by partial metabolic arrest with its inception and termination under endogenous hormonal control. Although several distinct types of seed dormancy have been recognized traditionally, an examination of the experimental literature suggests that a common regulatory mechanism exists. Dormancy onset, control, and termination are apparently regulated by a balance of growth inhibitors and promotors. At the onset of dormancy this balance is shifted in favor of the inhibitor component; at its termination it is shifted back in favor of the promotor component. The experimental evidence indicates that the levels of growth-promoting hormones decrease markedly during seed maturation, imposing dormancy. A trigger mechanism stimulates hormone activation and/or synthesis, thus terminating dormancy and culminating in resumption of embryo growth (germination). A model is proposed that embodies a hormonal regulation of four phases of dormancy: an inductive mechanism, a maintenance mechanism, a trigger mechanism, and a germination mechanism. This hormonal

regulation, in conjunction with specific metabolic inhibitors, is considered to mediate the activation and/or synthesis of hydrolytic enzymes. From this model the dormant seed is viewed as a cybernetic system. Particular emphasis is given to the regulatory role of gibberellins in seed dormancy and germination.

INTRODUCTION

The gap between biochemistry and morphogenesis[1] still exists, but it is being bridged slowly and systematically at many points. This review attempts to evaluate the progress being made at one such bridge—seed dormancy. Seed dormancy is an aspect of the phenomenon of growth cessation which has as its crucial point the problem of preserving a potential for growth without loss of biologic integrity. A dormant system has but two possible immediate fates: resumption of growth or death. The central question I aim to explore is by what mechanisms growth is suspended in dormant systems and how this is regulated to preserve the capacity for growth while circumventing death. The available evidence strongly suggests that the temporary suspension of growth implicit in seed dormancy is under endogenous hormonal control and exhibits a cybernetic systems character.

With the development of modern techniques of biochemical analysis and assay, our understanding of the nature and mechanisms of seed dormancy has increased tremendously. The rate of progress has been so rapid that the last five years have provided the bulk of our ontologic knowledge of this phenomenon. The reviews by Amen (1963), Barton (1965a,b), Evenari (1965), Stokes (1965), and Vegis (1964) are essentially descriptive statements concerning the nature of seed dormancy which only allude to the fundamental and common mechanisms. Sufficient experimental evidence has now been amassed to warrant new considerations of the phenomenon of seed dormancy.

A cursory examination of the recent data presented on the mechanisms of seed dormancy tempts one to speculate that these mechanisms may not be so diverse as previously indicated, but rather fall into a common control pattern. Obviously, not all of the experimental facts fit into this pattern, but those which do not may be due, in part, to variations in methodology and definitions of the processes involved. Correspondingly, the apparent mechanisms of dormancy, as they can now be understood, appear to fall into two distinct morphologic categories: those occurring in albuminous seeds, and those in non-albuminous seeds. There seems to be a common regulation of dormancy in all albuminous seeds (and/or achenes), involving a germination inhibitor or hormonal block and a hormonally regulated hydrolysis of starch and protein in the endosperm. In non-albuminous seeds, when dormancy occurs it is usually due to impermeable or hard seed coats (Barton 1965b). The underlying control of both types appears to be similar.

This is not a comprehensive nor systematic review of the experimental literature on seed dormancy. Rather, it is an attempt to integrate and interpret a representative sample of recently reported data. Seed germination per se, that is,

[1]We could say between enzymology and morphology or between molecular genetics and Mendelian genetics.

the resumption of active growth by the embryo, will not be examined here in detail. This aspect has been reviewed extensively by Evenari (1956), Koller *et al.* (1962), Lang (1965), Mayer and Poljakoff-Mayber (1963), Toole and Hendricks (1956), and Wareing (1963). Only the initial biochemical aspects will be examined here.

DEFINITIONS OF DORMANCY

Dormancy has been an elusive phenomenon and difficult to define. Consequently, the term has been applied to a number of unrelated phenomena, resulting in considerable ambiguity (Wareing 1963). In an earlier communication (Amen 1963), I restricted the term dormancy to an endogenously controlled but environmentally imposed temporary suspension of growth, accompanied by reduced metabolic activity, and relatively independent of ambient environmental conditions. Such a state is difficult to ascertain in nature, albeit a useful laboratory definition. A more commonly applied definition of dormancy is a state in which viable seed (or spores or buds) fail to germinate under conditions of moisture, temperature, and oxygen favorable for vegetative growth (Evenari 1956, Wareing 1963, 1965). These interpretations, however, do not adequately distinguish various forms of growth cessation.

Sussman and Halvorson (1966) employed the term dormancy for any partially arrested metabolic state, lying between "normal"[2] metabolism and cryptobiosis. They define dormancy as "... any rest period or reversible interruption of the phenotypic development of an organism." Within this concept they distinguish *constitutive dormancy* (equivalent to true dormancy as employed by Amen 1963 and Vegis 1964), and *exogenous dormancy* (generally equated with quiescence). Their constitutive dormancy delimits "... a condition in which development is delayed due to an innate property of the dormant state such as a barrier to the penetration of nutrients, a metabolic block, or the production of a self-inhibitor." This definition recognizes that dormancy is under endogenous control and is precluded by special pretreatment or relieved by special environmental stimuli. Operationally, it adequately distinguishes dormancy from other forms of growth cessation and provides a heuristic framework for subjecting the phenomenon to rigorous empirical analysis. The inductive and regulatory parameters are readily identified. It is this concept of dormancy that I will employ here, selecting that literature which best illustrates these parameters.

A "seed"[3] will be considered as dormant when and if some inherent condition precludes further growth and development unless a special agent is supplied the dormant system. This inherent condition may involve active or passive inhibition or impermeability resulting in partial metabolic arrest.

TYPES OF SEED DORMANCY

Heretofore, various causes of seed dormancy have been recognized: 1) rudimentary embryos, 2) physiologically immature embryos (inactive enzyme

[2]"Vegetative" metabolism.

[3]Any prime dispersal unit of Angiosperms here considered as a functional system comprising a food storage area, a latent growth axis, and a restrictive covering.

systems), 3) mechanically resistant seed coats, 4) impermeable seed coats, and 5) presence of germination inhibitors (Amen 1963, Bonner & Varner 1965). Examples of seed dormancy due to rudimentary embryos include orchids, ginkgo, and holly. Dormancy due to inactive enzyme systems is exhibited by lettuce, barley, and basswood. Those with mechanically resistant seed coats include juniper, hazelnut, and coconut. Impermeable seed coats are typically exhibited by many legumes. The presence of germination inhibitors is characteristic in seeds of mustard, coffee, tomato, cranberry, apple, cherry, and cocklebur.

Generally, it is those types of seeds with inactive enzyme systems that are photoblastic (i.e., exhibit a light requirement) although, in addition, some of these may have an after-ripening requirement. Concomitantly, some seeds with germination inhibitors may be photoblastic. Such interrelationships indicate that the actual biochemical regulation of seed dormancy in these instances may be similar, with only the triggering factor differing in each. As is often true, one stimulus may substitute for another requirement (Amen 1963).

The postulated existence of common control mechanisms in these types of seed dormancy is also implicit in the standard methods of breaking dormancy (Amen 1963). These include: photoexcitation, scarification (rupture of seed coats), after-ripening, stratification (chilling), leaching, and application of growth regulators. Experimentally, it has been found that fluctuating temperatures and certain growth regulators may compensate for or negate a light requirement (e.g., gibberellin or thiourea may replace it in lettuce seeds). A light requirement may be lost during after-ripening. Or, a seed may be rendered photoblastic by the addition of coumarin. These and numerous other examples (to be elaborated on subsequently) strongly suggest a common if not ubiquitous control mechanism in seed dormancy.

In the following discussion, a representative sampling of the regulatory mechanisms found in seed dormancy will be cited to illustrate the hypothesis that there is probably but one control pattern which may be variously triggered. This control pattern is predicated on the now commonly accepted inhibitor–promotor complex concept.

MECHANISMS OF SEED DORMANCY

It seems reasonable to divide the control of seed dormancy into four relatively distinct developmental phases: 1) *inductive,* characterized by a marked decline in hormone levels; 2) *maintenance,* constituting an "indefinite" period of partial metabolic arrest; 3) *trigger,* representing a period of sensitivity to specific environmental cues; 4) *germination,* marked by increased hormone and enzyme activity consummating in the resumption of growth by the latent embryonic axis. The extent of the literature available on these four aspects of dormancy reflects the degree of our understanding of the processes involved—the early phases being but poorly understood.

Induction Phase

Seed dormancy is an inductive phenomenon, i.e., it is preset during the ontogeny of the seed. Certain events during the maturation of seeds inevitably

lead to the onset of dormancy. These events may be environmentally triggered—e.g., photoinduction, thermoinduction, or chemoinduction.

As in most growth responses, dormancy and germination are regulated by a critical balance of inhibitor–promotor complexes. This idea is first attributed to Hemberg (1949). Both inhibitors and promotors appear to be ubiquitous in growth phenomena (Thimann 1956). During seed maturation this balance may be shifted in favor of the inhibitor component imposing dormancy. This may be accomplished by a curtailment in the synthesis of the promotor component, or by a build up of inhibitory intermediate metabolites, or by a direct antagonism. For example, dormin has been shown to be an *in vivo* antagonist of gibberellin (Thomas, Wareing, & Robinson 1965).

Endogenous growth promotors are in high concentration in the early maturation of mazzard cherry seeds, but these decrease markedly with the cessation of embryo growth and the ripening of fruit (Pillay 1966). On the other hand, dormancy may be due to the formation of impermeable seed coats, as in rice, thus imposing anaerobic conditions on the seed which may result in a build up of metabolic intermediates that may act as growth retardants (Roberts 1964b). With regard to photoinduction and thermoinduction of dormancy, Koller (1962) demonstrated quantitative differences in the response of lettuce seeds to varying light and temperature regimes during seed maturation, illustrating that germinability (or dormancy) is actually preconditioned.

Subsequent research has clearly implicated the gibberellins (and possibly the cytokinins) as promotor components of some inhibitor–promotor complexes (Chrispeels & Varner 1967b). Even as early as 1959, Black and Naylor reported that seed dormancy in oats (*Avena fatua* L.) could be prevented by the application of gibberellic acid (GA_3) to the developing seeds at the free nuclear stage of the endosperm. More recently, Hashimoto and Rappaport (1966a) have indicated that endogenous gibberellins decrease in naturally maturing bean seeds, but at the same time neutral substances, e.g., acidic butanol-soluble substances, markedly increase. In a later report (Hashimoto & Rappaport 1966b), they added that neutral gibberellin-like substances remain unchanged during bean-seed maturation but increase markedly in mature seeds with a coincident decrease in acidic gibberellin-like components. They conclude that this neutral fraction may constitute a reserve form of gibberellin in dry seeds. Parenthetically, it has been shown that a great variety of lipids occur in most seeds (Wolff 1966), some of which may serve as promotors of inhibitor precursors.

From such reports it is becoming increasingly apparent that an active antagonism exists between some growth promotors and inhibitors in many seeds, although in many instances the specific substances have not been identified. Artificial manipulation of the inhibitor–promotor complex, however, is shedding some light on this interaction. For example, Zeevaart (1966) demonstrated that the application of cycocel (2-chloro-ethyl-trimethylammonium chloride) to *Pharbitis* seeds sharply reduces their gibberellin content. In contrast, the promotive effect of exogenous gibberellin may be precluded by specific pretreatments. Pretreatment of peanut seeds with 40°-50° C. temperatures shortens their dormancy from 40 to 15 days (Bailey *et al.* 1958). In work on lettuce

seeds, Haber (1965) concluded that "...heat pretreatment, in contrast to all other inhibitory treatments tried, abolishes subsequent effectiveness of GA_3 at a temperature for which GA_3 is otherwise effective in promoting germination." Changes in endogenous hormone levels (particularly the gibberellins) are clearly implicated at the onset of seed dormancy.

These changes in hormone levels are commensurate with modifications of food reserves in developing seeds. Johri and Maheshwari (1966) have shown that in opium-poppy seeds the accumulation of reserve polysaccharides and protein increases drastically during maturation. They indicate that the reducing-sugar content is high up to the point of the free-nuclear stage of endosperm development; subsequently sucrose becomes most abundant. The activity of the glutamic-alanine transaminase system closely follows the activity of protein increase in these poppy seeds. In addition, maximal RNA[4] content of the developing poppy seeds is reached when the ovules have obtained maximum size, at which time DNA content increases with endosperm growth but then decreases during the maturation of the embryo. These changes are apparently correlated with distinct fluctuations in the amount(s) of nucleic acids in developing seeds in general.

This is clearly shown in the results of Ingle, Beitz, and Hageman (1965) for developing corn caryopses. At the 28-day level of corn endosperm maturation DNA, RNA, and protein content reaches a maximum. Another protein peak is reached at 40 days. In the 30-40-day period the soluble sugar content steadily decreases, as does the endospermal RNA content with a concomitant increase in ribonuclease activity. During the 40-60-day period, all soluble constituents decrease again with DNA and RNA contents also diminishing. Embryo growth is linear during the 46-day maturation period.

In 1962, Drennan and Berrie reported the following observations on germination in oats. Very little amylase activity occurs in dry grains, but it increases considerably during the first few days following germination in non-dormant grains. Dormant grains showed an increase during extended periods of imbibition. Amylase activity occurs primarily in the endosperm but does not increase until after the embryo has resumed growth. Primarily responsible for amylolytic activity is α-amylase, although β-amylase also contributes. They proposed that development of maltase activity in the endosperm is a response to a stimulus from the growing embryo, and is consequently a post-germination change. They conclude that hydrolytic metabolism as a causal factor in oat dormancy should be dismissed. (Subsequent data on other grains dictate that this view be modified—i.e., that hydrolytic activity is actually an early or prephase of germination and is hormone-stimulated.)

The application of exogenous inhibitors of protein synthesis may preclude rather than induce dormancy. In recently reported work, Esashi and Leopold (1967) showed in *Begonia* tubers (usually brought into a dormant state by low-

[4]Abbreviations are: RNA, ribonucleic acid; DNA, deoxyribonucleic acid; TCA, tricarboxylic acid cycle; GA, gibberellic acid; IAA, indoleacetic acid; BAPAase, α-N-benzoyl-DL-arginine-p-nitroanilide enzyme; BAL, 2,3-dimercaptopropanol; mRNA, messenger ribonucleic acid; AMO-1618, 2-isopropyl-4-dimethylamino-5-methylphenyl-1-piperidinecarboxylate methyl chloride.

temperature treatment or red light) that several inhibitors suppressed the induction of dormancy during the period of low temperature. From these results they suggest that DNA- and RNA-dependent protein synthesis is an essential part of the induction of dormancy. Presumably, the metabolic blocks associated with dormancy are related to specific enzymes.

Earlier, Johri and Maheshwari (1965) showed that O_2 uptake is high in developing poppy ovules during pollination, endosperm development, and cotyledonary expansion; then 0_2 uptake falls off markedly. The activity of succinic dehydrogenase is commensurate. They concluded that glycolysis and TCA (Krebs) cycle are operative mainly in early seed maturation.

From these data on the physiologic changes during seed ontogeny, it is evident that specific fluctuations in intermediate metabolites preset subsequent responses—imposing dormancy if the metabolic shift is in favor of growth inhibitors. The significance of these ontogenetic changes will be elucidated subsequently.

Maintenance Phase

The maintenance phase exhibits reduced metabolic activity presumably due to specific metabolic blocks. There is evidence, however, that some catabolic activity occurs in dry dormant seeds. Bradbeer and Colman (1967) showed that cotyledonary and embryonic axes of dormant *Corylus avellana* L. seeds exhibited active acetate ($2\text{-}C^{14}$) metabolism, inferring active enzyme systems of the Krebs TCA cycle and possibly of lipid and protein synthesis. They concluded that seed dormancy is not due to a general metabolic arrest.

The partial and/or specific metabolic blocks associated with the maintenance of seed dormancy are undoubtedly due to the presence of specific endogenous inhibitors. These natural germination inhibitors are too varied and widespread (Evenari 1949) to be enumerated here. Suffice it to say that in principle they are of nearly universal occurrence and often exert a demonstrable functional role in growth and growth cessation. Even in such instances it has been difficult to detect, isolate, and identify endogenous inhibitors; consequently, much research involves the application and interaction of exogenous inhibitors from which endogenous regulatory mechanisms are inferred and rationalized.

Some functional endogenous germination inhibitors, however, have been demonstrated: in *Luzula spicata* (L.)DC., an alpine rush (Amen 1965); in *Echium plantagineum* L., two inhibitors to which the seeds are differentially sensitive (Ballard & Lipp 1959); and in peach seed integuments (Lipe & Crane 1966). In the latter, the inhibitor is presumably "dormin" and appears to be antagonistic with GA_3. Dormin has been shown to be identical to abscisin II (Cornforth, Milborrow, & Ryback 1965). Its action in seeds is undoubtedly similar to that shown by Thomas, Wareing, and Robinson (1965) in birch and maple where bud dormancy is controlled by a relative balance of dormin and gibberellin. That dormin is an *in vivo* antagonist of gibberellin in seed dormancy in barley has been clearly demonstrated by Chrispeels and Varner (1967b). They indicate that the GA_3 enhancement of α-amylase and ribonuclease synthesis in barley aleurone layers is inhibited by abscisin, and conclude that gibberellin expression requires the synthesis of enzyme-specific RNA with abscisin inhibit-

ing this specific RNA. Similarly, a protease (BAPAase) and an antagonistic inhibitor have been localized in the aleurone layer of barley endosperm, the inhibited condition of BAPAase is unrelieved by exogenous GA_3 (Burger & Siegelman 1966). In the potato tuber, however, a functional sprouting inhibitor (β-complex) is readily reversed by exogenous gibberellic acid, tending to support the concept that a relative balance of inhibitors and stimulators participates in controlling rest (Blumenthal-Goldschmidt & Rappaport 1965).

In *Xanthium* seeds, coumarin and xanthatin are naturally occurring germination inhibitors, but exogenous kinetin and red light are effective in reversing this inhibition (Khan & Tolbert 1965). Based upon this finding, Khan and Tolbert (1966a) further elucidated the regulatory mechanism of the inhibitor–promotor complex by inhibiting lettuce-seed germination with exogenous coumarin. The subsequent addition of cycocel reversed this effect although GA_3 and IAA were unable to reverse the coumarin inhibition. They postulated that coumarin and other germination inhibitors participate in the photochemical system. In this instance, cycocel was antagonistic to an inhibitor, whereas in the work on *Pharbitis* seeds Zeevaart (1966) found that cycocel was antagonistic to endogenous gibberellin. He demonstrated that the application of cycocel to the roots carried over into the maturing seeds, thereby limiting the endogenous gibberellin content. Exogenous GA_3 overcame the cycocel inhibition.

An analogous situation is described by Overbeek, Loeffler, and Mason (1967) in the growth of *Lemna minor* L. Here dormin inhibits growth but is reversed by the cytokinin benzyladenine. Apparently dormin and cytokinin constitute an inhibitor–promotor complex for DNA synthesis. For this condition they suggest Monod's allosteric transition model with DNA polymerase as the protein, dormin as the inhibitor, and cytokinin as the activator.

Evidently what constitutes the inhibitor or promotor of a regulatory complex depends on what a particular substance interacts with or antagonizes. The relative concentrations of these substances would seem to play significant roles in whether they are inhibitory or stimulatory to a particular process. In all probability, different inhibitor–promotor complexes regulate specific metabolic pathways, e.g., the catabolism of starch, protein, or lipid reserves.

Khan and Tolbert (1966b) cite that coumarin, cycocel, auxin, and gibberellin interact differently with inhibitors depending on whether antagonism is with the synthesis or the action of a particular antagonist; for example, cycocel is an inhibitor of gibberellin synthesis but not of gibberellin action. This would seem to explain the disparity between the results on cocklebur and lettuce seeds; in the former cycocel interferes with coumarin action whereas in the latter it inhibits gibberellin synthesis. Patulin is similar to coumarin in its effect, and induces light sensitivity in lettuce seeds, and, as with coumarin, gibberellic acid readily negates the patulin effect (Berrie *et al.* 1967).

A vast array of other exogenous inhibitors exhibits similar *in vivo* interaction, particularly arsenicals. McDonough (1967) demonstrated that arsenite alone is inhibitory on lettuce seed, but it is extremely so when in combination with 2,3-dimercaptopropanol (BAL). BAL, however, does not inhibit more effectively when in combination with other thiol reagents. He concluded that this inhibitory action is not related to the light sensitivity of lettuce seeds. The site of

arsenical inhibition is unknown. AMO-1618 has also been shown to effectively inhibit the synthesis of gibberellin (Baldev, Lang, & Agatep 1965).

Barton (1965b) has reviewed the extent to which seed dormancy is imposed by a condition of the seed coats. This may be due to germination inhibitors being localized there, to the impermeability of the coat to gas exchange, or to mechanical restraint being exerted on the embryo by the coat. Wareing (1965) in his review has emphasized that germination inhibitors in the pericarp of both fleshy and dry fruits are common. He concluded that these endogenous inhibitors (non-leachable) often impose a high oxygen requirement on the seed. Generally, testas which are normally impermeable to oxygen must be scarified to overcome the inhibitor block. Moist chilling often renders the inhibitor ineffective, or stimulates promotor action which then overcomes the inhibition. These conditions are illustrated in lettuce and some of the cereal grains.

Ikuma and Thimann (1963b) have demonstrated that kinetin and/or red light promotes pectinase and cellulase activity in lettuce seeds, thereby permitting the embryos to penetrate the restrictive integuments. On the other hand, the seed of rice restricts 0_2 uptake which results in the accumulation of metabolic intermediates, thereby imposing a condition of dormancy (Roberts 1964b). Under anaerobic or sub-atmospheric 0_2 levels, organic oxidants (quinones and peroxides) may be active in dormant seeds such as rye and cucumber (Seigel, Guimarro, & Halpern 1964). These seed coat conditions, however, are usually associated with other parameters which can interact with photocontrol mechanisms.

These observations can be summarized in the idea that functional inhibitors either are directly antagonistic with endogenous promotors or interfere with their synthesis. The hormone promotors apparently serve to activate digestive enzyme systems. In essence, dormancy is due to a lack or inactivity of hydrolytic enzymes in food storage regions and/or testas (Drennan & Berrie 1962). Bonner and Varner (1965) have reviewed the evidence that there is a lack of β-amylase activity in the endosperm of albuminous seeds and that this activity is controlled by the lebel of endogenous gibberellin. Presumably the embryo produces insufficient gibberellin to stimulate amylase activity, resulting in seed dormancy. As demonstrated in many species (particularly barley), the exogenous application of GA_3 elicits germination from seeds exhibiting this and other forms of dormancy. Contrary to this, however, Simpson and Naylor (1962) reported that seeds of *Avena fatua* contain high concentrations of α- and β-amylases, but maltase appeared to be absent. They concluded that dormancy in oats is imposed by a maltase block, but exogenous GA_3 activates or initiates maltase synthesis which provides glucose for germination. However, the same principle is involved in both cases.

It is evident that the inhibitor–promotor complex is the regulatory mechanism in many types of seed dormancy. This is inferred mostly from the exogenous application of substances which demonstrably antagonize certain endogenous substances. What is most apparent, moreover, is that the addition of specific inhibitors will impose dormancy on otherwise germinable seeds, and that the application of a promotor will relieve dormancy. This is evidenced by the fact that most of the five types of dormancy cited above can be broken by

the use of gibberellin, kinetin, or other regulators. A common mechanism appears implicit. In support of this, for example, Curtis and Cantlon (1963) demonstrated that GA_3 substitutes for the after-ripening requirement in *Melampyrum lineare* Desr. The photochemical mechanism in lettuce seed germination can be circumvented by exogenous gibberellin (Khan 1960). GA_3 has been shown to antagonize and overcome the effects of an endogenous germination inhibitor in the seeds of peach (Lipe & Crane 1966) and *Luzula spicata* (Amen 1967). Mittal and Mathur (1965) showed that GA_3 and white light produced additive stimulatory effects in promoting germination in non-photoblastic tomato seeds, and even though long light exposures are inhibitory the exogenous gibberellin overcame some of the restrictive effect of continuous illumination. In photoblastic seeds in general, the photomechanism presumably regulates the level of endogenous gibberellin (Evenari 1965). Stokes (1965) has provided a list of species where GA_3 has been shown to overcome the after-ripening requirement.

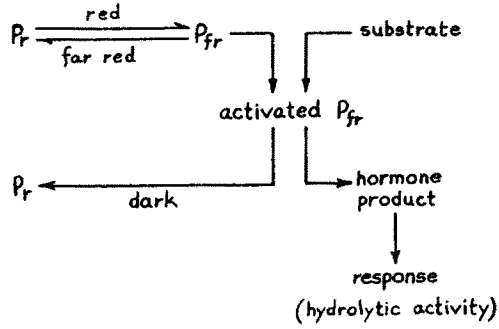
The inhibitor–promotor theme is a simple and attractive model of the regulation of physiologic processes. It postulates a relative balance between a promotor (endogenous gibberellin and cytokinin usually) and an inhibitor (e.g., coumarin or dormin) as the modulator of physiologic responses (Wareing 1963). This explanation is in full agreement with the accepted hormone concept. When the balance of the complex is in favor of the inhibitor, dormancy ensues. We will now direct our attention to those mechanisms which reverse this condition and elicit germination.

Trigger Phase

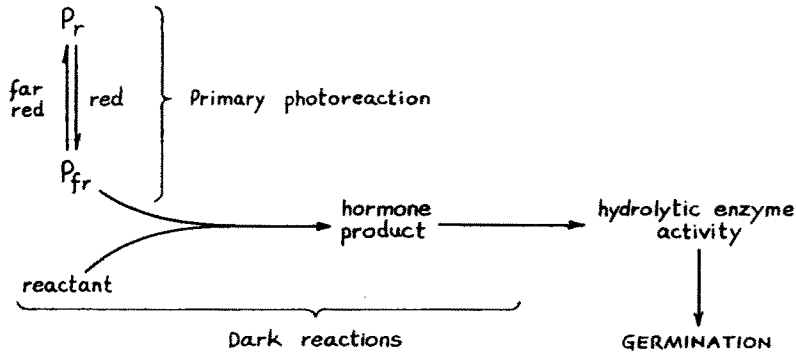
In the germination response of a dormant system there are a "triggering agent" (a factor that elicits germination, but whose continued presence is not essential) and a "germination agent" (a factor whose continued presence is required) (Sussman & Halvorson 1966). The triggering agent may be a photochemical one as in photoblastic seeds, a thermochemical reaction as in after-ripening and stratification, or inhibitor-removal as in scarification and leaching. The germination agent is presumably a hormone. Essentially, the triggering agent shifts the relative balance of an inhibitor–promotor complex to favor the promotor. In principle, all mechanisms of dormancy and germination appear analogous to this, differing only in the chemical nature or concentration of the germination agents involved but with similar functional roles. This can be illustrated in a representative system—lettuce seed.

The phytochrome trigger mechanism in photoblastic seeds apparently requires a dark period after photoconversion. This is diagramed for lettuce seeds by Mancinelli and Borthwick (1964) as follows:[5]

[5] P_r is P_{660} form of phytochrome, P_{fr} is P_{730} form.

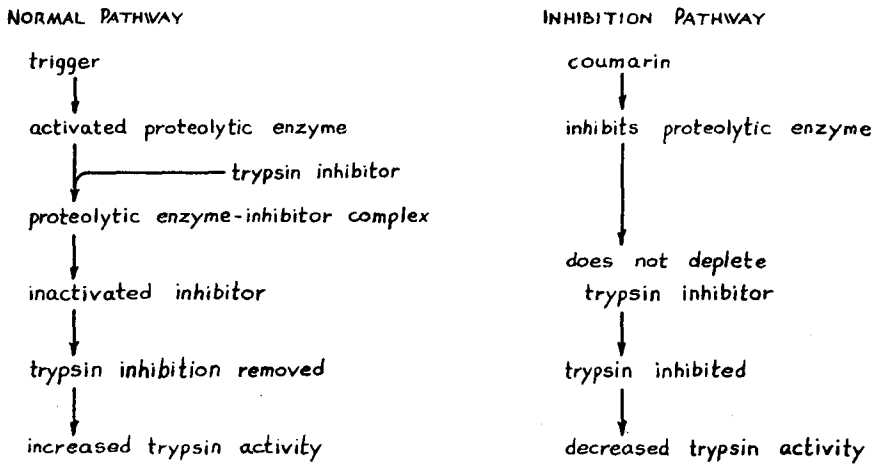


Ikuma and Thimann (1964) have postulated a similar scheme for the germination process in lettuce seeds:



The series of dark reactions occurs close to the embryonic tip.

Shain and Mayer (1965) have elucidated some of the biochemical detail of the hydrolytic phases of the termination of dormancy in lettuce seed. They propose that the trigger mechanism activates an existent proteolytic enzyme which then functions to inactivate a protease inhibitor (identified as trypsin-like), resulting in increased protease activity. They demonstrated that this pathway can be inhibited by exogenous coumarin. The following diagrams summarize these schemes.



In varieties of tomato seeds whose germination is usually inhibited by white light, Mancinelli, Borthwick, and Hendricks (1966) reported that these dark-germinating tomato seeds can be inhibited by exposure to far-red light. In turn, the germination of inhibited seed is promoted by red radiation and reversed by far-red, indicating control by phytochrome. They conclude that the diminution of P_{fr} from minimal exposure to red is accompanied by the disappearance of some endogenous factor (presumably a hormone). These findings illustrate that in some seeds the phytochrome control mechanism can be evoked when otherwise inoperative. This suggests alternate pathways for germination control which may be confluent with different environmental conditions.

These schemes suggest that the photochemical conversion results in the production of an enzyme-releasing hormone which in turn activates an inhibitor-removal enzyme, which ultimately leads to increased enzyme synthesis.

Subsequently, Chrispeels and Varner (1967a) reported that GA_3 stimulates the synthesis of ribonuclease in barley endosperm. They conclude that this ribonuclease is retained in the early stages of germination but is actively secreted subsequently. Their data also show an inhibition of ribonuclease synthesis by actinomycin-D and other metabolic inhibitors. The precise role of the secreted ribonuclease is not clear, but presumably it functions in a manner similar to the amylases and proteases—i.e., to supply specific monomers (e.g., nucleotides) to the embryo. These data clearly show that GA_3 is not a triggering agent, but is continuously required during α-amylase synthesis, implicating gibberellin as a germination agent.

With regard to dormancy in rice seed, Roberts (1963b) demonstrated that GA_3 and kinetin (somewhat) promote germination, IAA and thiourea had a slight promotive effect, but no interactions were apparent. In addition, other auxins, auxin antagonists, sulfhydryl, alanine, and some B vitamins were without effect. These data strongly suggest the high specificity of the metabolic block and the germination agent. On the other hand, Roberts (1963b) found that succinate, fumarate, citrate, and coumarin delayed the breakage of rice dormancy. In a subsequent report (Roberts 1964a), he showed that certain respiratory

inhibitors facilitated the breaking of rice-seed dormancy, thus indicating that a non-respiratory oxidation reaction is involved in the dormancy control mechanism.

Whether or not all of these particular (or analogous) chemical factors are universally distributed or involved in all types of dormant seeds (i.e., lettuce, barley, poppy, etc.) remains to be substantiated by further systematic studies.

Although the precise trigger mechanisms appear clear in some patterns of seed dormancy (particularly those involving photocontrol, after-ripening, and leaching requirements), it is still obscure in other forms—to wit, temperature effects. Capon and van Asdall (1967), for example, showed that in some desert seeds storage at 20° C. for five months was required for germination, whereas with pretreatment at 50° C. only five weeks were needed for germination. In this and similar examples the exact action of extreme temperatures and/or temperature fluctuation is unknown. Such data are difficult to correlate to a trigger mechanism. In addition, it has been reported that extreme temperatures and air drying will cause loss of viability in (wild rice) seed (Simpson 1966). Simpson (1966) also mentions that the usual six-month dormancy period in wild rice can be shortened by after-ripening in low oxygen tension, scarification being relatively ineffective. Pregermination chilling often shortens the period of dormancy. Ballard (1961) has shown that both low oxygen tension and carbon dioxide will break dormancy in subterranean clover. This, however, is also a temperature (22°–25° C.)-dependent process. Subsequently, Ballard and Lipp (1967) demonstrated that 2,4-D alone promoted germination in subterranean clover, but 2,4-D and CO_2 together had no promotive effect on these dormant clover seeds. Toole, Bailey, and Toole (1964) reported similar results for the effect of CO_2 on dormant peanut seeds.

The effects of temperature on seed dormancy and germination are inconsistent and the trigger mechanism is difficult to identify. It may be that various enzyme systems are differentially sensitive to temperature wherein they are activated by a sequence of temperature regimes. Evidence for such a scheme is meager. Generally, dry seeds are insensitive to low temperature but imbibed seeds are highly sensitive. Pollock and Toole (1966) in work on lima-bean seeds conclude that low temperature during imbibition injures the embryonic axis and as an adaptive mechanism the prevention of water uptake by seed coats precludes embryo injury at low temperatures. They indicate that sensitivity to chilling injury is conditioned during seed maturation. The fact remains, however, that the effects of temperature on seed dormancy are the most difficult to reconcile with the model of dormancy proposed herewith.

The situation is much less obscure with regard to scarification and leaching as effective trigger agents, wherein the inhibitor component is physically removed as in *Uniola paniculata* L. (Westra & Loomis 1966), *Luzula spicata* (Amen 1967), and peanut (Toole, Bailey, & Toole 1964). In these examples the inhibitor is either washed out of the coats or a portion of the coats is removed from proximity to the embryo.

Germination Phase

The processes involved in the breaking of seed dormancy and subsequent germination are clearly under hormonal control, and the naturally occurring

hormones (auxins, gibberellins, and cytokinins) appear to function as specific germination agents via inhibitor–promotor complexes. Several such complexes may be involved in the germination response of any one seed species, with each complex being responsible for some specific process (e.g., degradation of seed coats or mobilization of nutrients). Khan (1966) has suggested that the regulatory mechanism involved in breaking dormancy lies in the interaction of endogenous inhibitors and "exogenous hormones" which exert repression and/or derepression of critical DNA sites; for example, kinetin antagonizes an endogenous germination inhibitor in the upper seed of *Xanthium* (cocklebur), thereby breaking its dormancy.

In lettuce seeds, according to Ikuma and Thimann (1963a), kinetin action is presumably in the cotyledons, whereas gibberellic acid action is in the embryo axis. GA_3 contributes the primary growth stimulus (hydrolytic activity) while kinetin stimulates cotyledon expansion (cell division), resulting in seed coat rupture. Although kinetin enhances germination, it is not sufficient in itself, but GA_3 alone induces the germination process. The mechanism proposed by Ikuma and Thimann (1963b) for these actions postulates that kinetin stimulates proteinase, pectinase, and cellulase activity in the cotyledons which in turn reduces the endosperm–integument restrictions; on the other hand, GA_3 promotes amylase activity which supplies the monosaccharides for embryo respiration. In the untreated seeds, the endosperm exerts a restraining influence on the embryo, for radicle elongation in itself is not a limiting factor to germination.

Normally, in photoblastic lettuce seeds, the red light–phytochrome-mediated action promotes these activities thereby permitting the embryo to penetrate the coats (Ikuma & Thimann 1963b). Exogenous kinin and gibberellin circumvent this light requirement, strongly suggesting that phytochrome simply mediates hormone production or activity. This was not confirmed by Leff (1964) who showed that the germination of 'Grand Rapids' lettuce seed was promoted by both kinetin and light, but kinetin alone did not promote dark germination. This photosensitive and gibberellin-producing site in lettuce seeds appears to be in the radicle tip (Scheibe & Lang 1965). In addition, the whole process may be oxygen-dependent, as in rice seeds, where a specific oxidation reaction is requisite to germination (Roberts 1964b). Such a reaction must attain a critical level and the oxidant may be directly involved in the phytochrome reaction.

Thus the site of hormone production is the embryo, but its action on enzyme function occurs principally in the food storage tissues—a feedback system. Paleg (1964) demonstrated that GA_3 stimulates α-amylase activity in barley aleurone cells. Ingle and Hageman (1965) showed that endosperm carbohydrate and protein catabolism is stimulated by exogenous GA_3 in corn, concluding that exogenous gibberellin replaces a component normally supplied by the embryo. Varner (1964), concurrently, demonstrated that "... the α-amylase produced by barley endosperm in response to exogenous gibberellic acid is produced in the aleurone layers by *de novo* synthesis." Later, Mori, Kumazawa, and Mitsui (1965) demonstrated that helminthosporol likewise stimulated the *de novo* synthesis of amylases in rice endosperm. Similarly, the gibberellin-sensitive mechanism in *Melampyrum lineare* does not involve synthesis or activation of starch hydrolytic enzymes by the embryo, indicating that the mechanism is

localized in the endosperm, presumably like that in barley and oats (Curtis & Cantlon 1965). Chrispeels and Varner (1967b) subsequently reported that although GA_3 stimulates α-amylase activity, dormin (abscisin II) inhibits α-amylase synthesis during late germination of barley and actinomycin-D inhibits α-amylase synthesis early in germination. Dormin appears to be, in this instance, a specific anti-gibberellin, inhibiting a specific RNA synthesis.

Akazawa (1965) reviewed the status of these considerations as follows. It is not known for certain that β-amylase hydrolyzes starch *in vivo*. Most α-amylases, however, do hydrolyze raw starch. β-amylase in ungerminated wheat kernels is present in an inactive, latent form, bound chemically to glutenin by disulfide linkage—presumably activated in the germination process by the secretion of a substance capable of releasing (reduction of -S-S-) the enzyme. β-amylase is formed and functions in the endosperm. α-amylase originates in the scutellum and is secreted into the endosperm. The production of α-amylase in the endosperm, mediated by GA_3, arises by *de novo* synthesis. α-amylase accounts for 90 per cent of the amylolytic activity and β-amylase for the remaining 10 per cent in endospermal tissue.

These conclusions suggest two distinct modes of action for the gibberellin hormone: 1) as a reductant for releasing latent hydrolytic enzymes; 2) as an initiator of enzyme synthesis presumably via RNA control. In reviewing the evidence, Overbeek (1966) proposed that gibberellins and kinins both regulate the synthesis of hydrolytic enzymes via RNA control. He suggests, however, that although it is apparent that gibberellins induce the *de novo* formation of α-amylase in aleurone layers, they may also activate other digestive enzymes which degrade cell walls. At any rate, gibberellins may perform a dual role: to provide soluble food for the embryo and to weaken integument restrictions. The precise role of the kinins is less clear, and their action is probably distinct from gibberellin. Ogawa (1967) has found that although exogenous GA_3 increases α-amylase activity in embryo-less rice endosperm, this activity was not influenced by the additions of vitamins, amino acids, organic acids, protease, sucrose, IAA, or kinetin; helminthosporol, however, had a weak additive effect with GA_3.

Other agents have a similar promotive effect on seed germination. Roberts (1963a) showed that rice-seed germination is stimulated by nitrate, nitrite, and hydroxylamine, but not by ammonia or urea. For peanut seeds, Toole, Bailey, and Toole (1964) demonstrated that ethylene gave 100 per cent germination and replaced a weak light requirement. These data are difficult to reconcile with the proposed model; however, it has been reported by Jones (1967) that high concentrations of ethylene inhibit α-amylase production in barley, whereas in moderate concentration and synergistic with GA_3 it markedly increases the amylase level in the endosperm.

These results strongly implicate the DNA–RNA–protein synthesis mechanism in the regulation of seed germination. Although Cherry (1963) reported that RNA increase in the food-storage tissue of the peanut does not precede enzyme activity, he concluded that RNA probably plays a role in hydrolytic enzyme activation and/or synthesis. Later, Tuan and Bonner (1964) demonstrated that dormancy in potato buds is not imposed by inhibition of RNA polymerase, but rather by a direct repression of DNA (presumably through the

stimulation of histone synthesis). Fletcher and Osborne (1966) found that GA_3 stimulates RNA and protein synthesis in dandelion leaves and prevents their senescence, but pretreatment with actinomycin-D precludes GA_3 stimulation. It is known that actinomycin-D inhibits DNA-dependent RNA synthesis. This was demonstrated in bean seeds where the elongation of the embryonic axis is dependent on RNA-protein synthesis, and the application of actinomycin-D inhibits this synthesis, indicating DNA dependence (Walton 1966).

Active RNA synthesis is apparently necessary for cell elongation. In excised soybean hypocotyls, Key (1964) inhibited cell elongation by actinomycin-D, 8-azaguanine, and puromycin. He concluded that the synthesis of specific types of RNA was inhibited, precluding specific enzyme synthesis, thus limiting growth. He also found that the synthesis of this same specific RNA was enhanced by hormones. Later, Cleland (1965) reported that RNA synthesis is not necessary for auxin-induced cell-wall loosening as evidenced by inhibition with actinomycin-D, but RNA synthesis appears to be necessary only for the factor controlling wall enlargement. Wood and Bradbeer (1967) reported that in *Corylus avellana* seeds DNA–RNA synthesis commences only at about 10 hours of imbibition; they concluded that nucleic-acid synthesis probably does not play a part in the early phases of the termination of dormancy. This is consistent with Ikuma and Thimann's (1963a) view of the role of gibberellins and kinins, the latter stimulating over-all nucleic-acid synthesis.

Fluorides are also known to be inhibitory to nucleic-acid synthesis in corn seedling roots (Chang & Thompson 1966). This focuses attention on the role and action of cytokinins. Fox (1966) has postulated that the biochemical action of kinins may be associated with the substituted bases in RNA thereby repressing RNA activity. Thus kinins may be repressing or derepressing agents for specific RNA synthesis, depending on their concentration.

These data, although far from conclusive, do suggest that plant hormones may have a direct effect on nucleic-acid activity. Since gibberellin has been implicated in the releasing (activation) of some hydrolytic enzymes, it may well be similarly involved in the activation of RNA polymerase. The commensurate inhibitory and stimulatory effects of kinetin are more difficult to explain, but its inhibitory role in RNA repression may constitute a feedback mechanism for DNA replication, hence initiating cell division. Moreover, the empirical data remain somewhat inconsistent.

Sussman and Halvorson (1966) have attempted to rationalize the evidence by suggesting that the initial action of gibberellin and kinetin in effecting germination in dormant systems is probably on activating (reducing -S-S- to -SH HS-) enzymes already existent, and later by regulating RNA–protein synthesis in the embryonic axis and elsewhere. They further suggest that the initial stages of germination are a period of degradative reactions, but stages which do not involve protein synthesis. Breakage of -S-S- linkages occurs in wall structures early in germination. This is followed by mRNA synthesis, with one of the primary controls of dormancy and germination being exerted at the information transcription level. This is concluded from the fact that inhibitors (e.g., actinomycin-D) do not affect the initial stages, but completely inhibit protein synthesis later in germination. Their view seems to be a realistic appraisal of

the events associated with the termination of dormancy and subsequent germination.

Whereas the early phases of germination seem to involve primarily enzyme activation and degradative reactions, the later stages are associated with the translocation, mobilization, and assimilation of organic nutrients, i.e., actual growth (cell proliferation). During corn-seed germination, for example, the glyoxylate cycle is operative, resulting in a net increase of sugars being produced from stored lipids (Oaks & Beevers 1964). At this same time in wheat, a marked increase in endogenous gibberellin occurs (Fleming & Johnson 1964). In germinating *Phaseolus* (bean) seeds, the transfer of nitrogen from the cotyledons to the root–shoot axis begins at about 20 hours after imbibition, but actual increases in the root–shoot dry weight are not apparent until about 32 hours (Simon & Meany 1965). Simon and Meany concluded that "...it is unlikely that the timing of these events is controlled by the hydration of root and shoot or by the appearance of amylase in the cotyledons."

In other words, the enzymatic activity occurring in late germination is controlled by factors different from those associated with the trigger mechanism. For example, in Scots-pine germination, light does not exert its control through action on the existent fat metabolism, and the two appear unrelated (Nyman 1966). Proteolytic activity in the cotyledons generally increases during squash-seed germination, but continues to be dependent on the presence of an embryo axis; exogenous cytokinin (e.g., benzyladenine or phenyladenine), however, could replace the effect of the embryo axis (Penner & Ashton 1967).

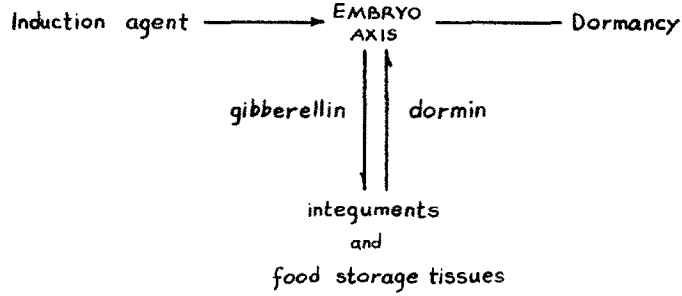
These observations strongly suggest that the gibberellins may be the predominant germination agents early in the germination phase, whereas the cytokinins may exert greater influence later. The available evidence seems to suggest that gibberellin activity is more closely associated with food-reserve degradation, and cytokinin activity with initiation of cell proliferation and expansion. Synergistic effects of these two groups of hormones are apparent, however.

Cybernetic Control

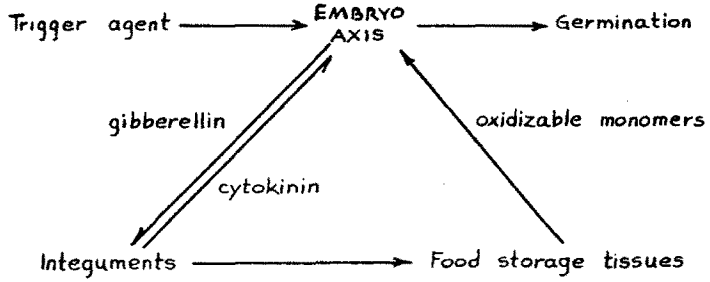
The functional components of a seed system have a juxtaposition such that chemical feedback is potentially operative. During the ontogeny of a dormant seed system, endogenous inhibitors are produced at a level sufficient to counteract the growth hormones, resulting in growth cessation of the embryonic axis. Also, during this maturation process, a trigger mechanism is developed which renders the seed system sensitive to a specific environmental cue. The proper stimulation of the trigger mechanism sets into motion a series of biochemical reactions that terminate in the resumption of growth by the embryonic axis.

The exact sites of inhibitor production are at present vague; however, it would seem rational to speculate that they are produced mainly in the food-storage tissues and/or the integument structures. I suggest that during seed maturation the embryo produces gibberellin hormones which stimulate the development of other seed structures, these in turn producing cytokinin hormones which stimulate embryo growth. Commensurately, non-embryo structures

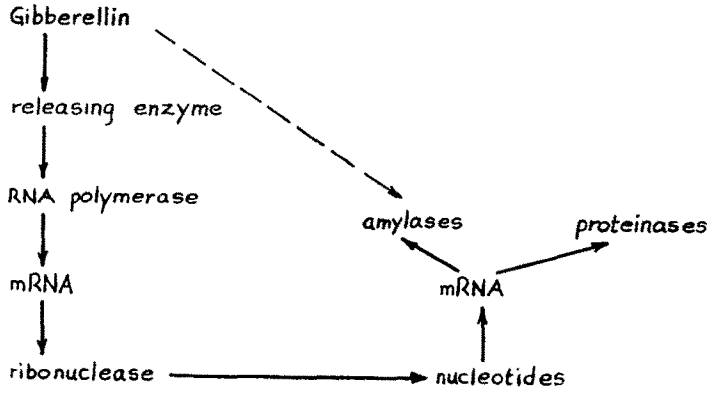
begin synthesizing and accumulating endogenous inhibitors that counteract specific promotors—imposing dormancy. A chemical feedback system is implicit:



Prior to germination, during the trigger phase, the inhibitor effect is reversed, reactivating promotor activity. These biochemical events involve a shift in the relative concentration of the inhibitor and promotor, a condition which turns off and on again specific enzyme systems. In the germination process, the embryonic axis produces a gibberellin hormone that stimulates enzyme-specific mRNA synthesis in the integuments (e.g., aleurone) and/or food-storage tissues. The resulting enzymatic activity is essentially hydrolytic. This may trigger a production in non-embryonic tissues of cytokinin hormones which stimulate enzyme-specific mRNA synthesis in the embryo axis, initiating cell division. This condition also exhibits a cybernetic character:



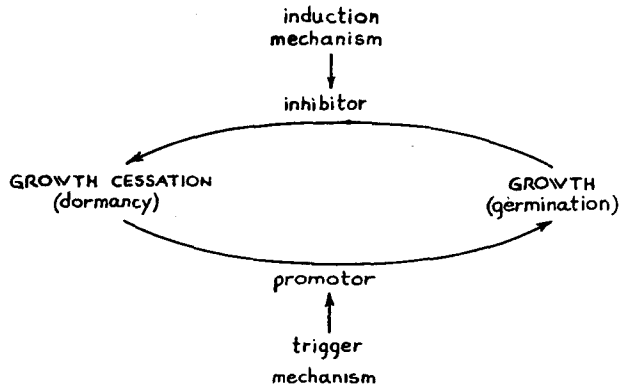
A hypothetical action for the germination-agent gibberellin can be illustrated as follows:



Presumably, most germination inhibitors inhibit specific mRNA synthesis; for example, in the mRNA–amylase system, dormin is the inhibitor and gibberellin the promotor (Chrispeels & Varner 1966, 1967b). In the mRNA–proteinase system, dormin is the inhibitor and cytokinin the promotor (Penner & Ashton 1967, Overbeek, Loeffler, & Mason 1967). For other enzyme systems the inhibitor–promotor complex may be coumarin-gibberellin (Khan & Tolbert 1966a) or coumarin and kinetin (Khan & Heit 1967). In the latter example it was shown that endogenous coumarin and kinin may participate in the repression and derepression of dormancy-controlling sites in rosaceous embryos. As a promotor, kinetin has also been linked with the RNA necessary for tyramine methylpherase production in barley, supporting the conclusion that the cytokinins are more related to protein catabolism than the gibberellins (Steinhart, Mann, & Mudd 1964).

The proteolytic-trypsin–protease system proposed by Shain and Mayer (1965) for the termination of lettuce-seed dormancy may actually function as a derepressor of critical DNA sites. Allfrey and Mirsky (1964) had already indicated that trypsin will hydrolyze histone from DNA, such removal activating the DNA–mRNA system.

Thus, several inhibitor–promotor complexes may regulate the germination response by timing the sequence of specific metabolic events in the ontogeny of a seed. The experimental evidence cited clearly implicates such a mechanism as the naturally occurring regulatory mechanism in seed dormancy. These over-all relationships can be illustrated as follows:



The inhibitor–promotor concept not only contributes to our understanding of growth cessation but may ultimately resolve the problem of uncontrolled growth. Schaeffer and Smith (1963) demonstrated that the hormonal growth response is differentially specific in non-tumor and tumor-mutant strains of tobacco, and a cytokinin may be the endogenous hormonal factor. They concluded that this growth factor in the tumor system appears similar to that in crown-gall cells.

OPERATIONAL MODEL OF SEED DORMANCY

In an earlier communication (Amen 1964) I attempted a crude systems analysis of the parameters in seed dormancy. That scheme envisioned a series of processes in four phases: 1) induction, 2) inactivation (actual growth cessation), 3) termination, and 4) restoration (resumption of growth). Based upon the empirical observations cited above, a modification of this scheme seems appropriate. I will thus propose a hypothetical cybernetic model of seed dormancy and its release.

The four stages above can be reordered as follows:

1. Inductive mechanism—those ontogenetic events which lead to the onset of dormancy, e.g., inhibitor accumulation, integument impermeability.
2. Cryptobiotic control mechanism—the formation and maintenance of metabolic blocks.
3. Trigger mechanism—those transient agents and their products which are sensitive to specific environmental stimuli, and which function as metabolic activators.
4. Germination mechanism—the initiation of cell proliferation which includes:
   a. enzyme activation
   b. degradation of insoluble foods
   c. translocation of soluble foods

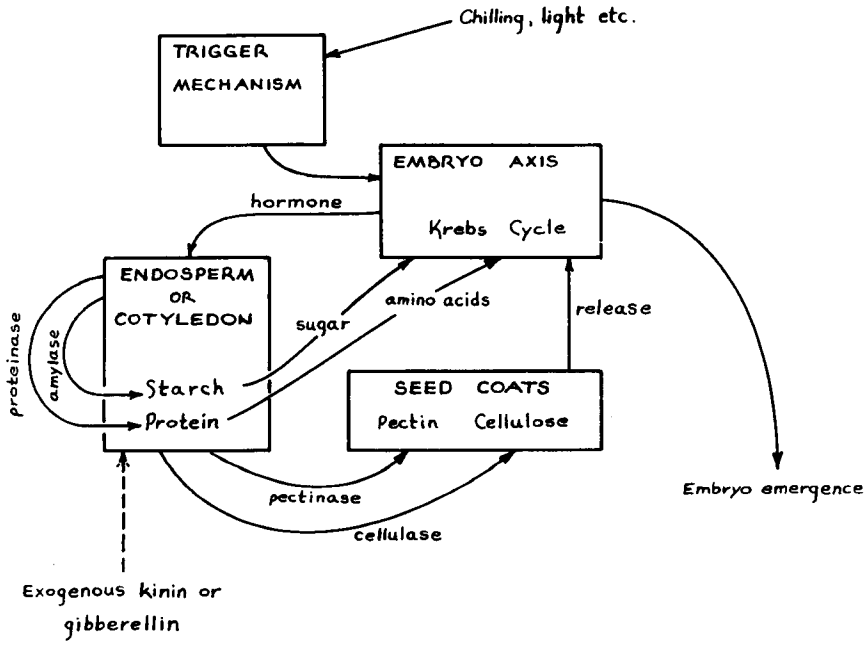


Figure 1. Cybernetic diagram of a seed system.

d. mobilization of nutrients
e. synthetic reactions of growth

Implicit in this scheme is the notion that the seed system involves a food-storage area, a latent-growth axis, and a restrictive covering. The trigger agent receives an environmental stimulus (e.g., light, leaching, chilling, time) and translates this into a chemical message (a hormone) which activates latent enzymes, ultimately restoring full metabolic activity. This scheme seems valid for any type of seed dormancy, although the nature of the trigger agent and the type and site of inhibition may well vary from one species to another. The parameters and structures of the seed system in this scheme are viewed as functional components which may be of variable structural composition. Figure 1 attempts to illustrate this cybernetic character of a dormant seed system.

The exact nature of the trigger mechanism and the point of metabolic inhibition are what makes the types of dormancy appear different. However, the regulatory principles involved are essentially the same and the functional parameters analogous.

In artificially breaking seed dormancy, the application of an "exogenous hormone" simply circumvents the natural trigger mechanism. This is illustrated in Figure 2 for an albuminous seed such as barley. A more detailed model of the

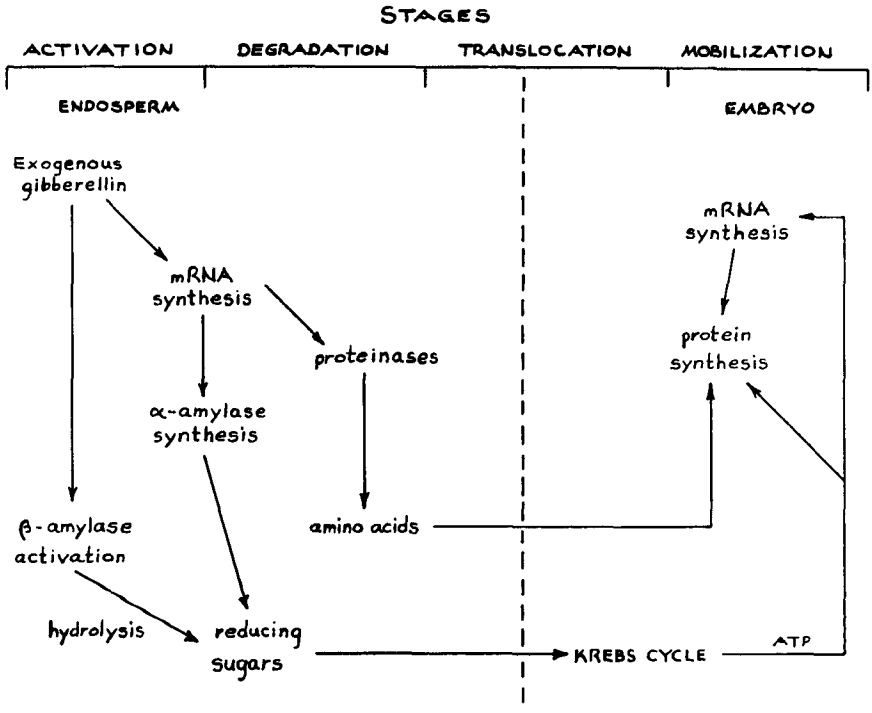


Figure 2. Diagram of some partial processes in seed germination.

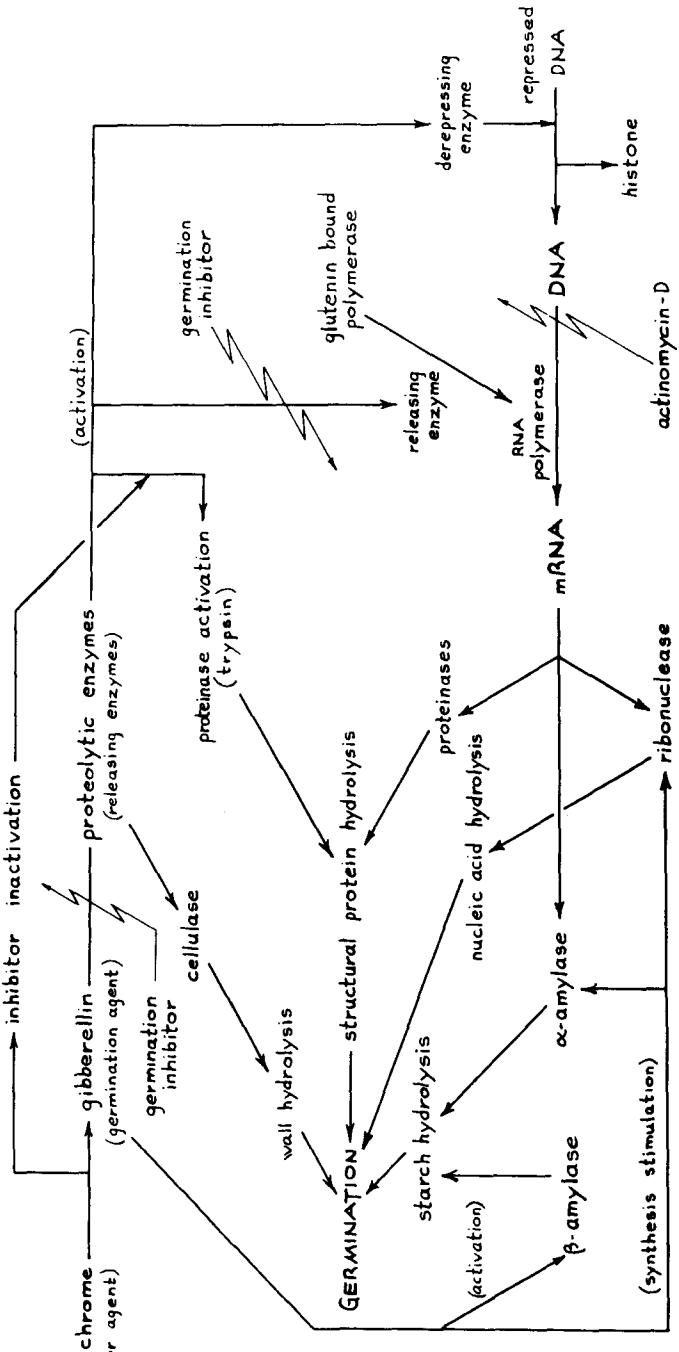


Figure 3. Possible biochemical pathways in the termination of seed dormancy.

biochemical pathways in a photoblastic seed (lettuce) is proposed in Figure 3.

The over-all control of seed dormancy, particularly in albuminous seeds, seems to involve a reduction in the growth-promoting hormone content during maturation, i.e., dormancy onset. Under suitable environmental conditions a trigger factor is activated which increases the hormone content. The hormones (germination agents) exert a dual action: 1) to activate preexistent hydrolytic enzymes, some of which are releasing-enzymes, and 2) to derepress DNA or otherwise stimulate the synthesis of additional hydrolytic enzymes. These degradative reactions supply appropriate monomers for the respiratory activity of the embryo, resulting in germination.

In summary, the onset of dormancy (inductive aspect) is characterized by marked reduction in growth-hormone levels in the food-storage tissues. The termination of dormancy is characterized by a triggering of hormone synthesis and/or activation. This is followed by a series of degradative and translocation steps. The products of the hydrolytic reactions are mobilized by the embryo axis in its respiratory and assimilatory processes, resulting in the resumption of embryo growth.

This type of model would seem to explain the various mechanisms of seed dormancy cited, including seeds with restrictive or impermeable coats, those exhibiting a light requirement, those with a chilling requirement, and those with determinate inhibitors. The trigger agent merely modulates the inhibitor–promotor complex which may function in several ways. A direct biochemical antagonism may occur between the promotor and inhibitor (binding action), thereby precluding stimulation; or both the inhibitor and promotor may affect a common substrate, resulting in inhibition should the promotor be in an inactive form. Endogenous gibberellin in mature dormant seeds may actually exist in a "reserve form."

In this view of dormancy, all metabolic activity is not arrested; some terminal oxidative activity presumably persists albeit at a slow rate. In this way biologic integrity is preserved in the dormant system (i.e., a static steady-state equilibrium). The substrates for the minimal oxidative metabolism may be lipids since they often occur in abundance in dry seeds. Information on respiratory quotients, role, and transformations of lipids in dormant seeds is scanty.

CONCLUSIONS

If the observations presented here have any correspondence to naturally occurring processes, then it seems reasonable to conclude with Overbeek (1966) that endogenous gibberellin is probably a universal component of an inhibitor–promotor complex which constitutes the naturally occurring mechanism in the regulation of seed dormancy and germination. This complex appears to be a modulator of enzyme activity as mediated through the information transcription apparatus of the nucleic acid–protein system. The central role of hormones in growth and growth cessation is implicit; however, the exact biochemical mechanisms of hormone action still need to be elucidated.

There remain unanswered questions and unresolved problems with regard to growth cessation phenomena:

1. What is the role of hormones in disrupting the conjugation of enzymatic and structural proteins? Do they serve as reductive coenzymes?
2. Is the negative hormonal control of growth cessation unique to multicellular systems?
3. What is the extent and chemical nature of endogenous inhibitor–promotor complexes in specific seed systems?
4. What is the ecologic significance of synchronized or differentially timed cycles of growth and growth cessation? Are they always adaptive?
5. Are the mechanism and duration of growth cessation genetically determined, or is it simply a physiologic state confluent with the spatial and temporal relations of maturation?

The resolution of the problems of growth cessation will, in my estimation, play a significant role in formulating a general theory of growth regulation. In this vein, I am confident that investigators of seed dormancy, spore dormancy, insect diapause, mammalian hibernation, and even of cysts and tumors share a common problem.

The phenomenon of dormancy represents a biologic state essentially the converse of malignancy—in the former, growth is virtually suppressed, in the latter, uncontrolled. Presumably the mechanisms of controlled growth are temporarily curtailed but potentially operative in the dormant state, whereas in malignancy they are out of phase. In either situation inhibitors and hormones appear to play an active role (Garb 1966). An elucidation of the ontologic mechanisms of dormancy should therefore, shed considerable light on the nature of malignancy, and should contribute immensely to a general theory of growth regulation.

Previously (Amen 1963) I envisioned growth as a natural and continuous proliferation of order in the continuum of life. If this is the characteristic mode of living systems (i.e., to generate, preserve, and proliferate biologic order), then the suspension of growth constitutes an exception to this natural biologic activity. This may reflect an adaptive condition to those oft occurring situations which do not favor the proliferation of life's basic stuff.

What these considerations and schemes of growth cessation have to do with the reality of life is open to debate, i.e., do the postulated models have any correspondence with reality. They do offer, however, an explanatory model that makes some sense out of the empirical observations. To the scientist who feels that this is all science amounts to anyway[7] it should be intellectually satisfying, but to the scientist bent on fully understanding objective reality and ultimate causes these models probably will be disquieting. I am convinced that there is a need and justification for periodic rationalization of empirical data, otherwise we may lose sight of our search for *meaning*.

ACKNOWLEDGMENT

I am grateful to Dr. Robert L. Sullivan for his critical perusal of the manuscript, and to Dr. John E. Davis, Jr., for his technical assistance.

[7] A theoretical rationalization of data into inherently consistent models.

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ADDENDUM

Evidence continues to mount that the hydrolytic systems of dormant seeds are under endogenous hormonal control. The precise relationship between the promotive and inhibitory factors, however, is not so certain. The hypothesis advanced in the Introduction that the regulatory mechanism of seed dormancy involves both growth promotors and growth inhibitors is further substantiated, however, by pertinent data which suggest that many of these growth regulators act primarily at the transcription and translation stages of enzyme synthesis. This phenomenon appears to be well established for growth processes in other systems. Monroy (1967) in reviewing work on animal morphogenesis concludes that cell maturation is mediated by translation of mRNA, the inhibition or negation of cell maturation occurring at the transcription or translation stage. He proposes that one general control mechanism of development may involve the "shutting-off" of protein synthesis at the ribosomal level via hormonal effectors.

One question pertinent to the acceptance of this scheme should be noted and is summarized by Scarano and Augusti-Tocco (1967, p. 67):

> "It has often been suggested that hormones might act at the gene level by regulating DNA transcription. Naturally, the question asked for histones should also be answered for hormones. What would regulate hormones in their function of regulating gene action? It is more likely that both histones and hormones are components of a more complex mechanism which regulates differential transcription in development."

Although Paleg and Cohen (1967) feel confident that "a gibberellin-like substance is undoubtedly the endosperm mobilizing hormone," other evidence (Wiley & Ashton 1967) strongly suggests that the embryo axis may send several distinct hormonal signals which, in turn, affect the translation and/or transcription of different enzyme systems. This may, as mentioned earlier, involve a cytokinin for protease synthesis and a gibberellin for amylase synthesis. As to the specificity of hormone induced protein synthesis, Fan and Maclachlan (1967) demonstrated in pea epicotyls that IAA readily stimulates protein synthesis in general and cellulase synthesis in particular. Their data reinforce the hypothesis that hormones (in specific concentration or in combination) promote the synthesis of specific enzymes by unmasking appropriate mRNA or de-repressing a coordinate part of the genome. These data when applied to seed dormancy and germination substantiate the view that specific inhibitor–promotor complexes regulate the synthesis of specific enzymes by mediating a portion of the genetic translation or transcription mechanism. Varner (1967), however, presents a convincing argument that gibberellin (GA_3) alone increases the production of amylase, ribonuclease, and protease. That gibberellin alone controls

the production and activity of several hydrolases may appear firm, but should remain open to question.

As to what regulates the hormones, the mode of action of several exogenous inhibitors provides a clue. Khan and Faust (1967) suggest that cycocel and AMO-1618 act as germination inhibitors by interfering with the synthesis of gibberellin-like hormones. Yung and Mann (1967) suggest that gibberellin action may be to activate bound or stored α-amylase-specific mRNA, and that the arylcarbamate and phenylurethane inhibitors presumably interfere with this action. Some inhibitors (e.g., cycocel and AMO-1618) thus inhibit hormone biosynthesis, whereas others like dormin directly inhibit protein synthesis, at either the translation or transcription stage.

This may account for the fact that the application of excess "exogenous hormones" reverses the inhibitory effect of some inhibitors but not of others. Moore (1967) has shown that cycocel and AMO-1618 inhibition can be counteracted by exogenous GA_3 and IAA (in cucumber hypocotyls).

The precise role of endogenous inhibitors raises at least one significant question, the resolution of which should lead to some interesting and fruitful biochemical data. That is, what is the ultimate metabolic fate of endogenous germination inhibitors? Their two major modes of action—inhibition of hormone biosynthesis and direct competition for active sites with hormones—suggest several mechanisms as to how their inhibitory action is reversed, i.e., are they catabolized, denatured, or secreted. Also, since their chemical composition is highly variable, this suggests that their metabolic fate is equally variable. Such data should prove illuminating in our understanding of regulatory mechanisms.

The adaptive significance of these observations to the natural control of seed dormancy is not inherently apparent, but some rational inferences can be made. For one, I seriously doubt that the endogenous production of growth inhibitors is closely linked to or integrated with the gene-specified hormone–enzyme systems in question. That is, I suspect that some correlation exists between the specification of these hydrolytic enzymes and their respective hormonal effectors, or, as Scarano and Augusti-Tocco (1967) suggest, they may be part of a more general control mechanism. I submit, however, that the role and action of growth inhibitors is but coincidental to this, being, in all probability, a byproduct of unrelated metabolic activities but which have, when interacting with specific hormone–enzyme systems, simply conferred (in the form of seed dormancy) some selective advantage to certain temperate species of plants.

The list of specific endogenous inhibitors, compared to the number of known hormones, appears too long to be other than coincidence. This is not to imply that their role in the regulation of growth and growth cessation is any less significant. But a causal or deterministic correlation between the biogenesis and role of hormonal factors and that of inhibitors does not appear warranted at present.

This is to reinforce my earlier contention that growth and the endogenous, promotive-regulation of growth constitute a fundamental biologic attribute, but the inhibition of growth and growth cessation are viewed as coincidental and exceptional cases, albeit adaptive.

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