

Symposium: Deterioration Mechanisms in Seeds

Deterioration Resistance Mechanisms in Seeds

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Accepted for publication 30 July 1982.

A starting point for this review of seed deterioration resistance mechanisms might be the question: "Where do we look for deterioration resistance mechanisms?" However, examination of the viability or quality retentions of a wide variety of seeds reveals that large, cultivated, edible seeds such as cereal caryopses and

legume seeds are uniquely vulnerable to deterioration. Seeds of weeds, wild relatives of cultivated species, and even crops cultivated primarily for portions other than their seeds, tend to have superior resistances to seed deterioration compared to those exhibited by the cultivated, edible seeds. Therefore, a more appropriate question might be: "Where did the resistance mechanisms go?" I will return to these questions later.

Although much seed pathogenesis involves conditions and mechanisms commonly associated with saprophytism, seeds seldom are attacked like raw substrate. Evidences of seed resistance to deteriorative processes are many. Anyone who has ever farmed

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or gardened is aware of the persistent reappearance of annual weeds, in spite of efforts to remove all of those that emerge. Many grass seeds, including some of our most troublesome weeds, are enclosed by tightly adherent bracts that may be high in tannins. The role of bract tannins in imparting resistance of seeds to deterioration is largely unexplored. One study (30) has shown that some weed seeds can survive burial for 90 yr in moist soil. Among more economically desirable species, heritable differences in vulnerability to seed deterioration have been shown in barley (54), cotton (7), peas (48), peanuts (27), pine (14), sorghum (22), soybeans (25), and wheat (13). Some mechanisms by which cultivars differ in resistance to seed deterioration have been studied extensively and are fairly well understood. These mechanisms, which most often exclude microorganisms from seeds, will be discussed in detail. Conversely, the mechanisms by which microorganisms are excluded from embryos are poorly understood. I will speculate on possible bases for these resistances.

Within this review I will deal with resistances to seed deterioration processes that can occur from anthesis through planting and germination of seeds, and will refer to seeds in the broad sense, including those with intimately associated ovarian structures, such as the caryopses of grasses. Cases that might appear to be seed resistance to deterioration but are actually due to accessory structures will not be dealt with in detail in this review. These include protection of corn caryopses by tightly enclosing husks that help ward off insect attack, protection of grass seeds from *Claviceps* by rapid shriveling of the stigma following pollination (49), and active protection of many developing dicotyledonous seeds by resistance systems such as phytoalexin synthesis in the surrounding ovaries.

Resistance Associated with the Seed Coat

A survey of the literature on resistance to seed deterioration indicates that the seed coat is the most important component in resistance of seeds to deterioration. I will discuss the role of the seed coat in only two cultivated plants, peas and cotton. The resistances described pertain to numerous other species.

Peas. Stoll (48) first reported that seeds of *Pisum arvense* with dark seed coats were less susceptible to foot rots than seeds of *Pisum sativum* with light colored coats. Subsequently, Sorgel (47) showed that a fungal inhibitor could be extracted only from the resistant seeds. Later studies showed that the inhibitory agents were anthocyanins (8). Hyphae of *Pythium* were unable to penetrate dark seed coats, but readily colonized dark colored seeds with nicks in the coats.

Flentje (12) reported that wrinkle-seeded peas were more susceptible to preemergence rotting than smooth-seeded ones, but that this resistance did not persist once the seed coat was broken. Evidently the seed coats of smooth seeds imparted greater resistance to infection than did those of wrinkled seeds.

Diffusion of nutrients from seeds has been shown to be an important determinant of inter- and intracultivar resistance of pea seeds to attack by soil fungi. Seed coat structure and integrity are major determinants both of nutrient loss and rate of imbibition of seeds. Short and Lacy (44) showed that pea seeds with high rates of solute leakage also had high rates of imbibition and high amounts of seedling decay. Kraft (31) observed that whereas the anthocyanin delphinidin was inhibitory to germination of *Fusarium*, this inhibition was overcome in the presence of glucose. Thus, the seed coat of peas functions as a seed protectant through imposition of a chemical barrier of inhibitory polyphenolic compounds, as a mechanical barrier, and as a barrier to the availability of nutrients to fungi.

Cotton. Infection of ripened, permeable cottonseed occurs almost exclusively through the chalaza (17); it is the only portion of the thick, tannin-impregnated seed coat that is permeable. The seeds of most wild cottons are impermeable to water through the chalaza, whereas cultivated cottons are permeable. The impermeable seed character imparts immunity to weathering (7), as well as to infection by *Aspergillus flavus* and elaboration of aflatoxins (36). Impermeable seed coats also protect seeds against

deterioration in soil; plants of impermeable-seeded lines appear in fields for years following cropping with these lines (11). This does not occur with permeable-seeded lines.

In contrast to ripened cottonseed, which are dark brown and low in permeability, cottonseed within unopened bolls are white and are highly permeable (18). The color and the permeability barriers develop by oxidation of tannins following boll opening. Sadykov (43) reported that up to 12.5% of the dry weight of immature cotton seed coats is composed of catechins and their derived tannins. Inhibition of fungi by these compounds may be partly responsible for a low observed rate of infection of seeds within infected, closed bolls (6).

Other examples of seed deterioration resistances localized in the seed coat follow the same pattern established for these two examples, namely: prophylaxis by physically impenetrable barriers; inhibition of fungal growth, usually by phenolic compounds; and restriction of nutrient availability to microorganisms outside the seeds.

Resistance Systems Localized to the Interior of the Seed Coat

A common observation on many species of seeds is that when seeds do become infected, the fungi first grow within and on the inner layers of the seed coat (eg, 17,33), tissues that frequently are low in their content of polyphenolic compounds. Only after extensive growth within this region do they invade the embryos. These observations suggest the existence of resistance systems within embryos and other tissues inside the seed coat.

The Nucellus. When a cottonseed is infected by a fungus, the hyphae penetrate the chalaza, but typically do not immediately invade the embryo (17). Rather, they grow on the inner portion of the seed coat, an area low in tannins. The embryo is surrounded by a thin tissue called the nucellus, which apparently is derived from crushed endosperm cells. This tissue appears to act as a barrier to fungi, and when the embryo finally is infected, infection occurs primarily within that half closest to the chalaza, which does not contain the embryonic axis. Histochemical staining of the nucellus (18) reveals that there is heavy localization of tannins in that part attached to the chalaza, where the fungus failed to infect the embryo upon entry into the seed, and within that portion covering the embryonic axis. This localized tannin may serve to protect the embryo from attack by microorganisms.

Active resistance systems (phytoalexins). Production of phytoalexins is a well-known defense system in many plants, and their production within seeds would not be surprising. However, examination of reports of phytoalexin production by seed tissues reveals that in all cases (eg, 26) they were produced only at very high moisture levels conducive to seed germination. Pflieger and Harman (39) reported that although pisatin was produced in imbibed pea cotyledons it was not produced at 20% moisture content. Similarly, the cotton phytoalexins gossypol and hemigossypol were produced in cotyledons of germinating seeds and in seeds exposed to prolonged rainfall in the field, but were not produced in seeds incubated at 20% seed moisture (19). Thus, although phytoalexins may be important for protection of germinating or imbibed seeds, they appear to have no role in protection of seeds at moisture levels commonly encountered during weathering and storage deterioration.

Enzyme inhibitors. Many seeds are known to produce inhibitors of hydrolases, especially proteases, but the functions of these inhibitors are poorly defined. Although a major function of protease inhibitors is thought to lie in regulation of seed metabolism, Ryan (42) made the interesting observation that many protease inhibitors in seeds are not inhibitory to seed proteases, but are inhibitory to proteases of other organisms. Kirsi (29) reported that a protease inhibitor from barley embryos was inhibitory to *Aspergillus* proteases. Halim et al (21) observed that trypsin inhibitor from corn inhibited growth of fungi and proposed that it may serve to protect the seeds against fungal invasion. Hwang et al (24) observed rapid release of protease inhibitors and lectins during early stages of soybean germination, and proposed that both groups of compounds may serve as inhibitors of microbial growth.

Lectins. Plant lectins are large proteins that contain multiple sites for binding of carbohydrates. These compounds have received much attention in recent years as possibly being involved in pathogen recognition responses in various plant tissues. Little attention has been given to the fact that they may function directly as resistance-imparting systems in seeds, which usually are the richest sources of these compounds. Hajj and Stevens (16) proposed that seed lectins function as inhibitors of viruses. Mirelman et al (37) observed that wheat germ lectin bound to hyphal tips of *Trichoderma viride* and inhibited its growth. They proposed that the lectin may serve to protect wheat against pathogens during germination. The extreme toxicity of castor bean seeds is well known to most botanists. This toxicity is due to the lectin ricin, which is a potent inhibitor of protein synthesis (32). The role of this and most other seed lectins in protecting seeds from microorganisms has not been studied. Soybeans might provide an ideal system for studying these interactions since a number of lines lacking specific lectins have been identified (eg, 41).

Lipids. Several studies have shown antifungal activity of seed lipids. Pflieger and Harman (40) reported that a complex lipid fraction from pea seeds prevented sporulation, but not hyphal growth of several fungi. Homma (23) reported that lecithin from soybeans caused abnormal germination, hyphal growth, and conidiation of *Sphaerotheca* and *Pyricularia*. Doskotch et al (10) reported that the 10-carbon fatty acid, capric acid, from elm seeds had antifungal activity. Cotton and other members of the Malvaceae contain the cyclopropenoid fatty acids malvalic and sterculic acid, which exhibit some biological toxicity. Interestingly, they are not uniformly distributed throughout embryo tissues, but are highly localized within the embryonic axis (2). It is tempting to speculate that they may have a role in protecting the dormant and germinating embryonic axis from attack by microorganisms.

Phytic acid/zinc. Marsh et al (35) demonstrated that zinc is required for production of aflatoxin by *Aspergillus parasiticus*. Phytic acid is a common constituent of seeds and supposedly functions as a phosphate reserve. It also binds polyvalent cations, including zinc. Soybeans are high in phytic acid, low in zinc, and are poor substrates for aflatoxin formation. Gupta and Venkatasubramanian (15) studied this interrelationship and concluded that phytic acid, through binding of zinc, was responsible for the failure of *Aspergillus parasiticus* to form aflatoxins in soybeans.

Seed Dormancy

There is evidence that dormancy of seeds contributes to deterioration resistance. Although dormancy may not contribute to resistance to microbial degradation, it could aid in preventing degradative changes of seed origin. A major problem in some crops is premature or precocious germination of seeds before harvest. Gordon (13) reported that in wheat, only red wheats exhibited dormancy and absence of sprouting damage. He speculated that this was due to flavanols, which functioned not as direct inhibitors of germination, but rather as contributors to hypoxia of the embryos. King (28) proposed that abscisic acid functions as a germination inhibitor in developing wheat embryos, as it does in many other species.

An interesting related finding was reported by Castor and Fredericksen (4). They observed that sorghum heads infected by *Fusarium* had a higher incidence of sprouting damage than uninfected heads. Thus, fungi may function to overcome dormancy, and seeds resistant to infection by fungi may be better able to maintain dormancy and sustain less sprouting damage.

Villiers (52) reported safe storage of seeds of lettuce and green ash by complete immersion in water. Whether the lack of deterioration was due to dormancy or anoxia is not known, but survival of the seeds under these conditions, supposedly conducive to microbial growth, seems remarkable. Toole and Toole (50) reported prolonged survival of imbibed dormant lettuce seeds; however, similar samples incubated at high humidity deteriorated. This suggests that dormancy may protect these seeds from their own degradative processes, but that complete imbibition protects

them from attack by microorganisms, possibly through anoxia or such mechanisms as phytoalexin synthesis.

Whatever the protective mechanisms in dormant imbibed seeds, they must be very effective; Kivilaan and Bandurski (30) reported that seeds of mullein survived 90 yr of burial in moist sand. Since these seeds apparently were dormant rather than hard-seeded, dormancy must have contributed to their survival.

Other Mechanisms

There is extensive literature concerning the fact that seeds with low rates of solute leakage are less susceptible to seed rots and seedling diseases than those with high rates of leakage. However, as discussed earlier, most of these differences are due to seed coat characteristics rather than to properties of the embryos. Loria and Lacy (34) reported that exudates from pea seeds with "bleached" or light-colored embryos rendered peas more vulnerable to attack by *Fusarium solani* than exudates from those with green embryos. This may be a case of differences in solute loss due to embryos, rather than seed coats, determining relative resistance or susceptibility to deterioration.

Several studies have compared the susceptibility of normal and high-lysine (opaque) endosperm corn caryopses to infection by *Fusarium moniliforme*. Two of the reports (46,53) stated that opaque endosperm caryopses were more susceptible, whereas the third (38) stated that there were no differences between the two types.

Earlier, I discussed that flavanols and their derived tannins in seed coats can function as fungal inhibitors. Related compounds also occur in embryos. Turner et al (51) reported that the flavanol, dimethoxyisoflavone, extracted from peanuts, inhibits growth of *Aspergillus flavus*. Flavanols and flavanol glycosides have been isolated from cotton embryos (3), but any role they may have in deterioration resistance has not been studied.

Unidentified volatile compounds from lentils (5) and members of the Umbelliferae (45) have been reported to inhibit growth of various fungi, and an unidentified fraction from cottonseed has been reported to inhibit growth of *Clostridium perfringens*.

Nutritional status of plants has been shown to influence susceptibility of seeds to infection. Crittenden and Svec (9) reported that fertilization with potassium reduced the incidence of soybean seed infection by *Diaporthe sojae*, and Ooka and Kommedahl (38) reported that nitrogen fertilization reduced the incidence of corn kernel infection by *Fusarium moniliforme*. Conversely, Agrawal et al (1) reported that nitrogen fertilization increased the susceptibility of rice to infection by *Trichoconis padwickii*. The concentrations of these nutrients within seeds and their possible roles in controlling incidences of infections are not known.

Caution should be used in ascribing resistance-imparting properties to an observed system. An antimicrobial system, for example, should be in the right place, in the right form, and present at the right time to do the job. An example of an antibiotic system in the wrong place is the terpenoid aldehyde gossypol in cottonseed. Until recently, all commercially produced cottonseed contained fairly high concentrations of gossypol, which is also a phytoalexin in cotton. However, gossypol in seeds is highly localized in discrete glands throughout the embryo tissues, and thus poses no barrier to infection by, and ramification of, pathogens. Halloin et al (20) observed no difference between susceptibilities to preharvest deterioration of glanded and nonglanded (gossypol-deficient) cottonseed.

Using Resistance Mechanisms in Plant Improvement

The most effective of the various systems that seeds have for protection from deterioration are those associated with the seed coats. These also may be the easiest ones to work with in breeding programs. Several programs have undertaken to develop resistances to various fungi. Useful resistances are available in corn, soybeans, cotton, peanuts, peas, and barley. Most of these involve improved seed coat properties and are broadly based to the extent

that they impart resistance to weathering, storage loss, and postplanting seed rot.

In many instances it may prove useful to look for factors such as reduced seed coat permeability or increased seed coat tannin or flavanol content, rather than for resistance to specific pathogens. Where applicable, embryo dormancy also may be a useful character.

Where Did the Resistance Mechanisms Go?

This leads back to the opening questions: where do we look for deterioration resistance mechanisms? and where did the resistance mechanisms go? Seemingly, in order to look for mechanisms of resistance, one should have clearly demonstrated resistance. Given such resistance, one can search for mechanisms among those reported for other tissues of the same plants. In searching for mechanisms to incorporate into plants, one can turn to weeds or less "domesticated" members of the same species or closely related species. However, there is an alternative and potentially more productive approach that will provide answers to both of the above questions: determine the properties of seeds that agronomists, seed technologists, and food scientists deem "undesirable." A list of some "undesirable" seed characteristics follows:

1. Impermeable seed coat—gives problems with volunteer plants, erratic germination, and requires extra processing of seeds.
2. Too much seed coat—reduces percentages of proteins and oils.
3. Flavonols and tannins—impart off-colors to foods and bind with proteins, especially free amino groups of lysine, and impart astringency to foods.
4. Protease inhibitors—provide antidigestive properties.
5. Lectins—some, like ricin, are toxic, others are known hemagglutinins.
6. Cyclopropene fatty acids—in addition to some toxicity, they function as cocarcinogens with aflatoxins and inhibit some dehydrogenases.
7. Phytic acid—anti-nutritional, due to binding of divalent cations.
8. Dormancy—necessitates prolonged storage or specialized handling in order to achieve high germination rates.

Thus, when someone wants to breed some undesirable property out of a seed, it is fair to ask teliolegically why it was there in the first place. Obviously, selecting against many of these characteristics has increased the vulnerability of seeds, especially edible seeds, to deterioration. With starvation or malnutrition occurring in various parts of the world, often in climates highly conducive to seed deterioration, it may be well to think of reincorporating some of these "undesirable" properties into seeds.

LITERATURE CITED

1. Agrawal, V. K., Singh, O. V., and Modgal, S. C. 1975. Influence of different doses of nitrogen and spacing on the seed-borne infections of rice. *Indian Phytopathol.* 28:38-40.
2. Berry, S. K. 1980. Cyclopropene fatty acids in some Malaysian edible seeds and nuts: 1. Durian (*Durio zibethinus*, Murr.). *Lipids* 15:452-455.
3. Blouin, F. A., and Cherry, J. P. 1980. Identification of color-causing pigments in biscuits containing cottonseed flour. *J. Food Sci.* 45:953-957.
4. Castor, L. L., and Fredericksen, R. A. 1977. Seed molding of grain sorghum caused by *Fusarium* and *Curvularia* species. (Abstr.) *Proc. Am. Phytopathol. Soc.* 4:151.
5. Catsaka, V., and Vancura, V. 1976. Gaseous metabolites of germinating seeds of some plant cultivars resistant and susceptible to phytopathogenic fungi. *Folia Microbiol.* 21:214.
6. Ciegler, A., Lee, L., Klich, M., Jacks, T., and Hensarling, T. 1981. Expression of *Fusarium semitectum* in cottonseed. (Abstr.) *Phytopathology* 71:867.
7. Christiansen, M. N., and Justus, N. 1963. Prevention of field deterioration of cottonseed by an impermeable seedcoat. *Crop Sci.* 3:439-440.
8. Clauss, E. 1961. The phenolic constituents of the testas of peas and their importance for resistance to the agents of root rot. *Naturwissenschaften* 61:106.
9. Crittenden, H. W., and Svec, L. V. 1974. Effect of potassium on the incidence of *Diaporthe sojae* in soybean. *Agron. J.* 66:696-697.
10. Duskotch, R. W., Keely, S. L., Jr., and Schreiber, L. R. 1975. Isolation and identification of an antifungal agent from seeds of American elm. *Phytopathology* 65:634-635.
11. Endrizzi, J. E. 1974. Survival of seed of *Gossypium thurberi*. *Cotton Grow. Rev.* 51:35-38.
12. Flentje, N. T. 1964. Pre-emergence rotting of peas in South Australia. I. Factors associated with the seed. *Aust. J. Biol. Sci.* 17:643-650.
13. Gordon, I. L. 1979. Selection against sprouting damage in wheat. III. Dormancy, germinative alpha-amylase, grain redness and flavanols. *Austr. J. Agric. Res.* 30:387-402.
14. Grzywacz, A. P., and Rosochacka, J. 1980. The colour of *Pinus silvestris* L. seed and their susceptibility to damping-off. I. The colour and quality of seeds and fatty acids content of the seed coat. *Eur. J. For. Pathol.* 10:138-144.
15. Gupta, S. K., and Venkitasubramanian, T. A. 1975. Production of aflatoxin in soybeans. *Appl. Microbiol.* 29:834-836.
16. Hajj, B. and Stevens, W. A. 1979. The effects of legume seed extracts on plant virus infection. *Experientia* 35:1460-1462.
17. Halloin, J. M. 1975. Postharvest infection of cottonseed by *Rhizopus arrhizus*, *Aspergillus niger*, and *Aspergillus flavus*. *Phytopathology* 65:1229-1232.
18. Halloin, J. M. 1982. Localization and changes in catechin and tannins during development and ripening of cottonseed. *New Phytol.* 90:651-657.
19. Halloin, J. M., and Bell, A. A. 1979. Production of nonglandular terpenoid aldehydes within diseased seeds and cotyledons of *Gossypium hirsutum* L. *Agric. Food Chem.* 27:1407-1409.
20. Halloin, J. M., Turner, J. H., and Hoskinson, P. E. 1978. Comparison of seed and fiber properties and yield of glanded and glandless cottons. *Crop Sci.* 18:519-520.
21. Halim, A. H., Wassom, C. E., Mitchell, H. L., and Edmunds, L. K. 1973. Suppression of fungal growth by isolated trypsin inhibitors of corn grain. *J. Agric. Food Chem.* 21:1118-1119.
22. Harris, H. B., and Burns, R. E. 1973. Relationship between tannin content of sorghum grain and preharvest seed molding. *Agron. J.* 65:957-959.
23. Homma, Y. 1979. The mode of action of soybean lecithin against pathogenic fungi. Abstracts of Papers, 924. Ninth Int. Congr. Plant Prot., 5-11 August 1979, Washington, DC.
24. Hwang, D. L., Yang, W.-K., and Foard, D. E. 1978. Rapid release of protease inhibitors from soybeans. Immunochemical quantitation and parallels with lectins. *Plant Physiol.* 61:30-34.
25. Keeling, B. L. 1974. Soybean seed rot and the relation of seed exudate to host susceptibility. *Phytopathology* 64:1445-1447.
26. Keen, N. T. 1975. The isolation of phytoalexins from germinating seeds of *Cicer arietinum*, *Vigna sinensis*, *Arachis hypogaea*, and other plants. *Phytopathology* 65:91-92.
27. Ketring, D. L., Benedict, C. R., and Yeager, M. 1976. Growing season and location effects on water uptake and drying rates of peanut seeds from genotypes resistant and susceptible to invasion by *Aspergillus flavus*. *Agron. J.* 68:661-665.
28. King, R. W. 1979. Abscisic acid synthesis and metabolism in wheat ears. *Austr. J. Plant Physiol.* 6:99-108.
29. Kirsi, M. 1974. Proteinase inhibitors in germinating barley embryos. *Physiol. Plant* 32:89-93.
30. Kivilaan, A., and Bandurski, R. S. 1973. The ninety-year period for Dr. Beal's seed viability experiment. *Am. J. Bot.* 60:140-145.
31. Kraft, J. M. 1977. The role of delphinidin and sugars in the resistance of pea seedlings of *Fusarium* root rot. *Phytopathology* 67:1057-1061.
32. Liener, I. E. 1976. Phytohemagglutinins (Phytolectins). *Annu. Rev. Plant Physiol.* 27:291-319.
33. Llyos, M. B., Dhingra, O. D., Ellis, M. A., and Sinclair, J. B. 1975. Location of mycelium of *Diaporthe phaseolorum* var. *sojae* and *Cercospora kikuchii* in infected soybean seeds. *Plant Dis. Rep.* 59:17-19.
34. Loria, R., and Lacy, M. L. 1979. Mechanisms of increased susceptibility of bleached pea seeds to seed and seedling rot. *Phytopathology* 69:573-575.
35. Marsh, P. B., Simpson, M. E., and Trucksess, M. W. 1975. Effects of trace metals on the production of aflatoxins by *Aspergillus parasiticus*. *Appl. Microbiol.* 30:52-57.
36. Mayne, R. Y., Harper, G. A., Franz, A. O., Jr., Lee, L. S., and Goldblatt, L. A. 1969. Retardation of the elaboration of aflatoxin in cottonseed by impermeability of the seedcoats. *Crop Sci.* 9:147-150.
37. Mirelman, D., Galun, E., Sharon, N., and Lotan, R. 1975. Inhibition of fungal growth by wheat germ agglutinin. *Nature* 256:414-416.
38. Ooka, J. J., and Kommedahl, T. 1977. Kernels infected with *Fusarium moniliforme* in corn cultivars with opaque-2 endosperm or male-sterile cytoplasm. *Plant Dis. Rep.* 61:162-165.
39. Pflieger, F. L., and Harman, G. E. 1975. Inability of storage fungi to

- invade pea embryos: Evidence against phytoalexin involvement. *Phytopathology* 65:642-643.
40. Pflieger, F. L., and Harman, G. E. 1975. Fungal antispore activity of a complex lipid fraction extracted from pea seeds. *Can. J. Bot.* 53:1625-1629.
 41. Pull, S. P., Pupke, S. G., Hymowitz, T., and Orf, J. H. 1978. Soybean lines lacking the 120,000-dalton seed lectin. *Science* 200:1277-1279.
 42. Ryan, C. A. 1973. Proteolytic enzymes and their inhibitors in plants. *Annu. Rev. Plant Physiol.* 24:173-196.
 43. Sadykov, A. S. 1972. Polyphenolic compounds of *Gossypium* and *Hibiscus* species. *Chem. Natural Prod.* 1972:256-264 (In Russian).
 44. Short, G. E., and Lacy, M. L. 1976. Carbohydrate exudation from pea seeds: Effect of cultivar, seed age, seed color, and temperature. *Phytopathology* 66:182-187.
 45. Singh, D. B., Singh, S. P., and Gupta, R. C. 1979. Antifungal effect of volatiles from seeds of some Umbelliferae. *Trans. Br. Mycol. Soc.* 73:349-350.
 46. Sivasanker, D. V., Asnani, L., Lal, S., and Agrawal, B. D. 1975 (1976). Dosage effects of opaque-2 gene on the susceptibility in maize to seed rots by *Cephalosporium acremonium* and *Fusarium moniliforme*. *Indian Phytopathol.* 28(2):235-237.
 47. Sorgel, G. 1952. Über die ursachen der unterschiedlichen resistenz verschiedener erbsensorten gegenüber den krankheitserregern *Ascochyta pisi* Lib., *Ascochyta pinodella* Jones und *Mycosphaerella pinodes* (Berk. et Blox.) Stone. I. Vergleichende Untersuchungen zum Verhalten der Pilze an einer schwach anfälligen sorte. *Züchter* 22:4-26.
 48. Stoll, K. 1950. Resistenzprüfungen an leguminosen gegenüber dem fusskrankheitserreger *Ascochyta pinodella* Jones. *Z. Pflanzenzucht.* 29:175-192.
 49. Thakur, R. P., and Williams, R. J. 1980. Pollination effects on pearl millet ergot. *Phytopathology* 70:80-84.
 50. Toole, V. K., and Toole, E. H. 1953. Seed dormancy in relation to seed longevity. *Int. Seed Testing Assoc. Proc.* 18:325-328.
 51. Turner, R. B., Lindsey, D. L., Davis, D. D., and Bishop, R. D. 1975. Isolation and identification of 5,7-dimethoxyisoflavone, an inhibitor of *Aspergillus flavus* from peanuts. *Mycopathologia* 57:39-40.
 52. Villiers, T. A. 1974. Seed aging: Chromosome stability and extended viability of seeds stored fully imbibed. *Plant Physiol.* 53:875-878.
 53. Warren, H. L. 1978. Comparison of normal and high-lysine maize inbreds for resistance to kernel rot caused by *Fusarium moniliforme*. *Phytopathology* 68:1331-1335.
 54. Wilcoxson, R. D., Rasmusson, D. C., Banttari, E. E., and Johnson, D. A. 1980. Feasibility of selecting for resistance to kernel discoloration in barley. *Plant Dis.* 64:928-930.