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Physiology of Oil Seeds

I. REGULATION OF DORMANCY IN VIRGINIA-TYPE PEANUT SEEDS[1,2]

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ABSTRACT

The germination and ethylene production by dormant Virginia-type peanut seeds were observed in relation to phytohormone treatments that could conceivably release the dormancy of these seeds. A comparison was made between the effects of these treatments on the less dormant apical seeds and the more dormant basal seeds. Indole-3-acetic acid did not stimulate ethylene production by, or germination of, the dormant seeds to any extent. Gibberellic acid at 5×10^{-4} M stimulated ethylene production by apical seeds to 17 millimicroliters per hour and germination to only 40% above the control. The more dormant basal seeds were affected even less by gibberellic acid than the seeds. Ethylene gas at 8 microliters per liter stimulated germination to 85% above the control for both apical and basal seeds. At this ethylene concentration the physiology of the more dormant basal seeds was altered, so that they behaved in a manner similar to the inherently less dormant apical seeds. 2-Chloroethylphosphonic acid at 10^{-3} and 5×10^{-4} M provided results similar to ethylene gas. Both apical and basal seeds germinated 100% at 48 hours. Among the phytohormones tested in this study, ethylene gas produced the greatest germination at low concentrations, and it appears must directly related to initiating the reactions required for converting the quiescent cells to an active state of growth.

Some varieties of Virginia-type peanut seeds exhibit a "primary" inherent dormancy at harvest. Neither the mechanism nor convenient methods of breaking the dormancy are known. The first method for breaking the "primary" postharvest dormancy of Virginia-type peanut seeds was heating at 40 to 45 C for about 15 days (5). Leaching and treatments with natural aromatic compounds were only partially successful in breaking this dormancy (30).

Shibuya (29) reported that indole-3-acetic in lanolin (0.1 g of IAA/g of lanolin) applied to the exposed radical end of dormant peanut seeds stimulated germination. However, it was necessary to wound the tissue to allow penetration of the hormone since wounding, or IAA in lanolin alone, did not stimulate germination. Also, 90% germination was achieved only after drying the pods under natural conditions for 1 week and determining germination at the end of 2 weeks. With the exception of ethylene, other phytohormones have not, to our knowledge, been used in attempts to break the dormancy of peanut seeds.

The seed coats provide either a diffusion or permeability barrier, since removal of the coats can increase germination of dormant peanut seeds, but this removal appears to be secondary to subsequent conditions or seed treatments (30).

Five per cent CO_2 in air is capable of stimulating germination of dormant peanut seeds, and in either darkness or continuous fluorescent light, 100 μl/liter C_2H_4 induced nearly 100% germination (30). The results with CO_2 were discussed in a previous report by Ketring and Morgan (18) in relation to the discovery that as little as 3.5 μl/liter of ethylene would effectively break the dormancy of NC-13 Virginia-type peanut seeds. In contrast, lettuce seed germination is stimulated by ethylene although it is apparently without effect on dormant lettuce seeds (3).

Thus far the only two effective means of breaking peanut seed dormancy are desiccation by heat and ethylene treatment. Ethylene treatment requires less time, and it is effective at low concentrations and at usual seed-germinating temperatures.

The objectives of this study were to determine: (*a*) the effectiveness of ethylene, over a known concentration range, in breaking dormancy of NC-13 Virginia-type peanut seeds, and (*b*) whether other phytohormones that induce ethylene synthesis in plant tissues might be more directly related to the release of dormancy in Virginia-type peanut seeds than ethylene. In view of recent findings concerning the relation of auxins and gibberellins to ethylene-producing abilities of plant tissues (1, 4, 8–10, 12, 14, 15, 22) and the known effects of these substances on dormancy (19, 31), the type compounds for each of these phytohormones, as well as ethylene and the ethylene-generating substance 2-chloroethylphosphonic acid, were compared for their ability to enhance the germination of dormant Virginia-type peanut seeds.

MATERIALS AND METHODS

The procedures for determining ethylene, the method of germination, and seed source were reported previously (18). All samples contained 50 NC-13 peanut seeds weighing about 0.9 g per seed. Controls were imbibed for 16 hr in distilled water or in the case of 2-chloroethylphosphonic acid in 10^{-3} M glycol. The formulation (Amchem 68-62) used below includes some of the ester and anhydride of the acid with glycol as the carrier solvent. The release of ethylene from the formulation (Amchem 66–329) described by Cooke and Randall (11) and Warner and Leopold (32) differs from (Amchem 68–62) only in that ethanol was the carrier solvent. Ethanol inhibits peanut seed germination, whereas glycol had no apparent adverse affects. Treated seeds were imbibed 16 hr in the growth regulator or CEPA[3] solution, and then moisture

[1] Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the Texas Agricultural Experiment Station, Texas A. & M. University.

[2] Mention of a trademark name or a proprietary product does not constitute endorsement by the United States Department of Agriculture or Texas A. & M. University and does not imply its approval to the exclusion of other products that also may be suitable.

[3] Abbreviations: CEPA: 2-chloroethylphosphonic acid; GA: gibberellic acid.

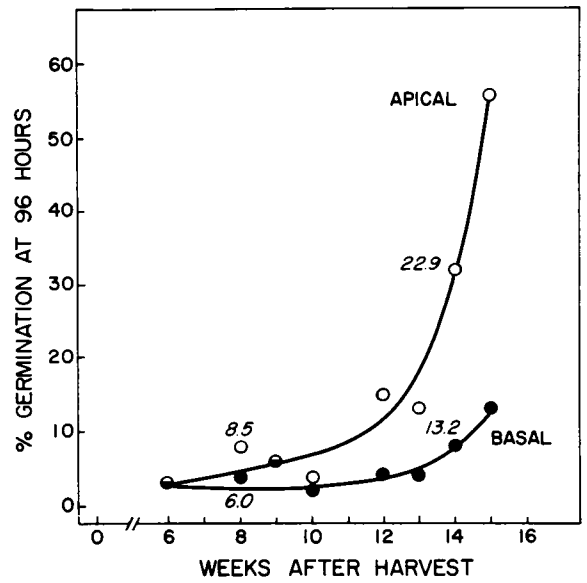

FIG. 1. The time course of germination of NC-13 peanut seeds as the inherent dormancy declines during storage in sealed containers at 3 C. The numbers adjacent to the points at 8 and 14 weeks after harvest indicate the ethylene produced (mμl/50 seeds × hr) by the apical and basal seeds at the times indicated.

was maintained by adding solution as needed throughout the 96-hr germination test. All growth regulator and CEPA solutions were adjusted to pH 6.0. When seeds were treated with ethylene gas, a total of 200 seeds were enclosed in a 55-liter container for a maximum of 48 hr. The concentration of added ethylene in these containers was verified by gas chromatography. Germination was at 28 C except during ethylene treatments when the large containers were at room temperature (23 C). Seeds were maintained primarily in the dark, but no effort was made to exclude light during routine manipulations. Seeds were segregated on the basis of their position in the pod. The seed most distal from the point of attachment of the pod is referred to as the apical seed.

Except in Table I and Figure 1 all data are presented as the increase of germination above the control at 24, 48, 72, and 96 hr. Since we observed that the degree of dormancy of the seeds changes with duration of storage even in sealed containers at 3 C (Fig. 1), the maximum germination of control samples that occurred at 96 hr is reported for each set of data. We attempted to obtain data on the release of dormancy when the condition was most strongly expressed or the seeds were most quiescent. No experiments were performed after the 15th week of storage, by which time the control germination had increased as shown in Figure 1.

The indole-3-acetic acid was obtained from Calbiochem, the gibberellic acid (80 + %) from Eastman Organic Chemicals, and 2-chloroethylphosphonic acid from AmChem Products, Inc.

RESULTS AND DISCUSSION

Natural Increase of Germination (Decline of Dormancy). Figure 1 shows the natural increase of germination of NC-13 peanut seeds during storage. At intervals of 1 or 2 weeks the seeds were removed from sealed containers held at 3 C and tested for germination. In a system that allowed free exchange of gases, apical seeds germinated 15% or less and basal seeds less than 6% up to 13 weeks after harvest. At 15 weeks after harvest the apical seeds germinated nearly 60% while the basal seeds increased to only 13%. It should be emphasized that the percentage germination reported in Figure 1 is for 96 hr, *i.e.*, the maximum germination attained during a germination test. These results and those that follow indicate a major difference in the degree of dormancy exhibited by apical and basal peanut seeds. If the inherently more dormant basal seeds could be induced to germinate in a manner approaching that of the inherently less dormant apical seeds, it would indicate that the treatment applied was close to the natural mechanism for release of dormancy in these seeds. The data that follow bear on this point. Since the basal seeds showed the highest degree of dormancy, the best test of any treatment was the effect of this treatment on the germination of basal seeds.

Effect of Ethylene on Dormancy. Imbibed seeds were treated with 0.1 and 0.2 μl/liter ethylene without noticeable effect on their germination. Table I shows that 1 μl/liter ethylene had no effect on germination of the seeds in the first experiment while 2 μl/liter increased germination of the inherently less dormant apical seeds only about 10%. In the second experiment 1 μl/liter ethylene increased germination about the same as 2 μl/liter in the previous test. Perhaps the seeds became more sensitive to C_2H_4 as they aged in storage. It was apparent that these levels of exogenous ethylene were inadequate to increase substantially the germination of these dormant peanut seeds when the exposure to ethylene was only for 6 hr.

Figure 2 shows the progress of germination as apical and basal seeds were exposed to 5 μl/liter C_2H_4 for 6, 12, 24, and 48 hr. Germination at the end of the 96-hr period increased as the length of exposure time to ethylene increased up to 24 to 48 hr when a maximum was approached. The amount of germination was generally greater for the inherently less dormant apical seeds although the difference was small for seeds treated with ethylene for 24 hr at 48 hr of germination. Apical and basal seeds showed a maximum of 68 and 43% germination above the control after 48 and 24 hr of ethylene treatment, respectively. There appears to have been some inhibition of the germination of the 48-hr ethylene-treated basal seed samples, or in the random selection of samples a more dormant group was obtained. However, the question still remained as to the minimum amount of ethylene required to induce both apical and basal seeds to germinate to the fullest extent. A test for viability (see legend of Fig. 2) showed 100 and 90% germination was possible for apical and basal seeds, respectively. It appeared that the inherently more dormant basal seeds would require a higher concentration or a longer exposure to ethylene, or both, in order to attain the same extent of germination as the inherently less dormant apical seeds.

Table I. *Effect of Ethylene on Germination of Dormant Virginia-type Peanut Seeds*

Experiment	Germination					
	Open control		Enclosed[1] control		C_2H_4 treatment[2]	
	Apical	Basal	Apical	Basal	Apical	Basal
	%	%	%	%	%	%
					1 μl/liter	
1	3 ± 3	3 ± 3	15 ± 9	5 ± 3	14 ± 2	3 ± 3
					2 μl/liter	
	...	...	...	...	24 ± 9	3 ± 3
					1 μl/liter	
2	4 ± 2	2 ± 2	9 ± 3	2 ± 2	25 ± 13	9 ± 9

[1] Seeds were enclosed in containers of about 55-liters volume with or without C_2H_4, and 10% KOH traps were provided in all tests. Apical and basal refers to the position of the seed in the pod.

[2] Ethylene treatments were for two successive 6-hr periods during the first and second 24 hr of germination. The data are for 96 hr of germination. Each datum is the mean of quadruplicate samples of 50 seeds each, except for the controls of experiment 2, which were duplicates.

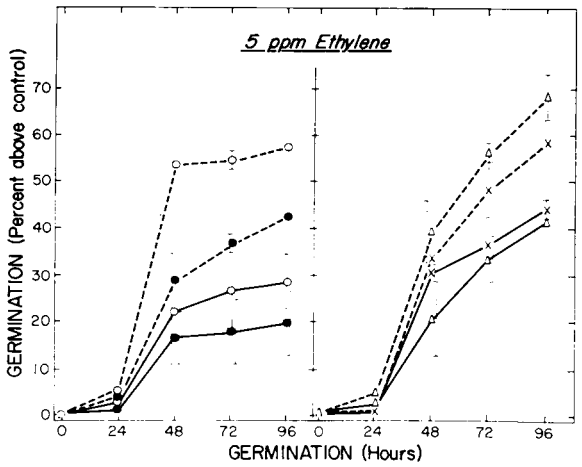

FIG. 2. The increase in germination (difference between mean values for duplicate control and treatment samples) of NC-13 peanut seeds treated with 5 μl/liter C_2H_4 for 6 (●), 12 (○), 24 (×), and 48 (△) hr. Solid lines indicate basal and dashed lines apical seeds. Standard deviations from the mean value are indicated by vertical lines, and SD values of 1% or less are not shown. Control samples were enclosed for 48 hr and germination was 15 ± 3% and 4 ± 0% at 96 hr for apical and basal seeds, respectively. At 96 hr the control seeds that showed no visible signs of germination were treated with 100 μl/liter C_2H_4 for 60 hr. Total germination was 100 and 90% for apical and basal seeds, respectively.

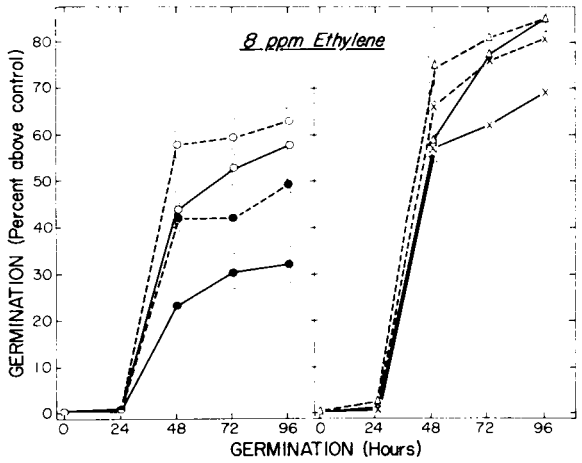

FIG. 3. The increase in germination (difference between mean values for duplicate control and treatment samples) of NC-13 peanut seeds treated with 8 μl/liter C_2H_4 for 6 (●), 12 (○), 24 (×), and 48 (△) hr. Solid lines indicate basal and dashed lines apical seeds. Standard deviations from the mean value are shown by vertical lines, and SD values of 1% or less are not shown. Control samples were enclosed for 48 hr and germination was 13 ± 5% and 4 ± 2% at 96 hr for apical and basal seeds, respectively.

Figure 3 shows that exposure to 8 μl/liter ethylene for increasing time intervals enhanced the amount of germination above that observed with 5 μl/liter ethylene (Fig. 2). As the time of exposure to 8 μl/liter ethylene increased, the difference in the germination processes between apical and basal seeds decreased until the basal seeds germinated to the same extent as the apical seeds. Thus, the physiology of the more dormant basal seeds was altered to an extent so that they behaved like the inherently less dormant apical seeds. This gradual change in the germination behavior of basal seeds is masked if one treats with supraoptimum (100 μl/liter) concentrations of ethylene. In the latter situation dormant apical and basal seeds of equal viability germinate to the same extent (Reference 30 and viability tests reported above). The fact that this gradual change in basal seed behavior can be shown with ethylene treatment indicates that ethylene may be involved in processes that are occurring. This conclusion is supported by our previous observations of ethylene production by germinating peanut seeds and the correlation between release of dormancy and induction of increased ethylene production (18).

Figure 4 demonstrates the behavioral change of the basal seeds in relation to the apical seeds, midway (48 hr) and at the end (96 hr) of the germination period. A rapid rise in germination occurred up to 24 hr of treatment, with 5 μl/liter ethylene at both 48 and 96 hr of germination. However 5 μl/liter C_2H_4 was unable to stimulate germination to the maximum potential of the seeds, particularly for basal seeds. C_2H_4 at 8 μl/liter enhanced the germination of all seeds and decreased the germination ratio of apical to basal seeds to essentially 1.0 after 48 hr of treatment. Thus the concentration of C_2H_4 was a more critical factor than the duration of exposure in inducing the inherently more dormant basal seeds to germinate in a manner similar to the inherently less dormant apical seeds. The maximum increment of germination occurred after 6 hr of ethylene treatment in all cases, the least of which was 16 ± 6% for basal seeds at 48 hr of germination after treatment with 5 μl/liter C_2H_4. The results were comparable to those previously reported for mixed seeds (18). However, the above results indicate a greater activity of ethylene in the release of dormancy than was previously apparent because of the much lower germination of control samples reported here (see legends of figures).

The "after-ripening" processes of dormant peanut seeds occur slowly at 3 C (Fig. 1) and are accelerated at 45 C (5). The fact that the ethylene-producing capacity is at a maximum when the after-ripening processes are complete would suggest that the gas is only a by-product of metabolism. However, the ability of exogenous ethylene to intercede at some step in the after-ripening process and to accelerate the changes taking place suggests that this is not the case (Figs. 2, 3, 4). Since the ethylene-producing ability of the seeds gradually rises as natural dormancy declines, and since the ability to produce ethylene is less for the more dormant basal seeds (Fig. 1), not only is there an indication that ethylene participates in the gradual decline of natural dormancy but there is a quantitative relationship between the amount of ethylene pro-

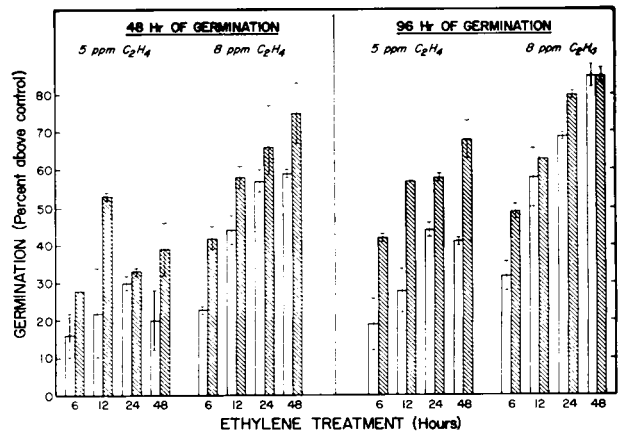

FIG. 4. The increase of germination of NC-13 peanut seeds at 48 and 96 hr of germination after exposure to 5 and 8 μl/liter ethylene for 6, 12, 24, and 48 hr. Open bars: Basal seeds; hatched bars: apical seeds. Standard deviations from the mean values are shown by vertical lines at the top of each bar; lack of a vertical line indicates there was no deviation from the mean value. Control samples for the 5 μl/liter ethylene treatment germinated 2 ± 2 and 4 ± 0% for basal seeds; 10 ± 0 and 15 ± 3% for apical seeds at 48 and 96 hr of germination, respectively. Control germination values for the 8 μl/liter treatment were 2 ± 2 and 4 ± 2% for basal seeds; 11 ± 7 and 13 ± 5 for apical seeds at 48 and 96 hr of germination, respectively.

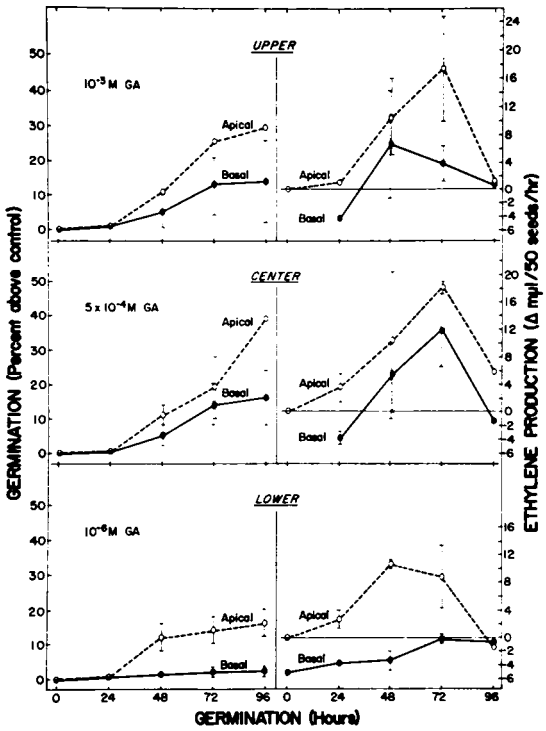

FIG. 5. The effect of 10^{-3} (upper), 5×10^{-4} (center), and 10^{-6} M (lower) gibberellic acid applied for 96 hr on the germination and ethylene production of apical and basal NC-13 peanut seeds. The minus Δ mμl C_2H_4/50 seeds × hr indicates that these seeds were producing the indicated amount of ethylene less than control samples. Germination of control samples at 96 hr of germination was 19 ± 13% and 7 ± 1% for apical and basal seeds, respectively. The vertical lines show the standard deviations from the mean value for two experiments, and the SD values not shown were ±1%.

duced and the degree of dormancy. This correlates with the data of Figure 4 showing that a higher concentration of exogenous ethylene was required to induce basal seeds to germinate to the same extent as apical seeds.

The reduction in differential response between apical and basal seeds by the increased ethylene concentration suggests either that a higher content of some "dormancy factor" may be present in basal seeds, or that the conversion from the quiescent to the active state of growth is less efficient in basal seeds. Since low levels of ethylene are produced by dormant peanut seeds (18), the mechanism for producing ethylene is present even in the dry or freshly imbibed seeds. Therefore, the initial event to release dormancy is probably a biochemical or biophysical change to allow ethylene production to increase to a rate that raises the ethylene level in the tissue to a critical concentration that initiates the reversal of dormancy. Perhaps, as suggested by Huelin and Barker (17) and Burg (7) for the increased production of ethylene by ripening fruit, the process in dormant peanut seeds is also an autocatalytic one that is initiated when the tissue becomes "sensitive" to the low levels of ethylene present in the mature dormant seed. However, a sensitive tissue implies that a change has taken place. Whether such a change is biochemical (2, 14, 16, 27) or biophysical (6, 13, 17, 20, 25–27) remains to be elucidated.

Effect of Indole-3-acetic Acid on Dormancy. In our tests dormancy of peanut seeds was influenced only slightly, if at all, by 10^{-3}, 5×10^{-4}, and 10^{-6} M IAA applied for 96 hr. The largest increase of germination above the control was 6% after 96 hr of germination for apical seeds at 10^{-6} M IAA. The highest ethylene production was 5.2 mμl/50 seeds × hr after 48 hr of germination for the same seeds. Control seeds and those showing no visible signs of germination after auxin treatment were tested with 100 μl/liter ethylene for 60 hr. Apical and basal seeds germinated 97 and 69%, respectively. The germination of basal seeds was not typical of the potential for these seeds as indicated above, but the percentage for apical seeds was as expected.

The dormant peanut seed apparently does not have an ethylene production system that is very readily activated by IAA. This is in contrast to the case of vegetative plant tissue (1, 4, 8–10, 12, 14, 22). Uptake or penetration of IAA into the seeds may be a limiting factor. However, the seeds were imbibed in the IAA solutions rather than a local application of IAA in lanolin as done by Shibuya (29). In our study there was a response of the seeds to both gibberellic acid and 2-chloroethylphosphonic acid under the same conditions. It seems unlikely that IAA was not taken up by the seeds to some extent. These data agree with the findings of Shibuya (29) when his data are considered for a 4-day period. The IAA stimulation of peanut seed germination as reported by Shibuya required 5 to 7 days to show a minimal response. Perhaps over an extended period IAA can induce an enzyme system responsible for breaking peanut seed dormancy mediated either by ethylene or by an entirely different mechanism (1, 4, 23, 24). However, owing to the delayed germination observed by Shibuya and the lack of response to IAA shown by the seeds in this study, an enzyme system induced by IAA would only be remotely related to the reactions stimulated by ethylene gas during the germination processes of peanut seeds.

Effect of Gibberellic Acid on Dormancy. The three concentrations of gibberellic acid tested (Fig. 5) stimulated ethylene production and germination of dormant peanut seeds to some extent. A maximum germination stimulation of about 40% was attained after 96 hr of germination at 5×10^{-4} M GA. In each instance the inherently less dormant apical seeds were induced to germinate to a greater extent than the inherently more dormant basal seeds. There was also correspondingly more ethylene production by the apical seeds. At 10^{-6} M GA the germination of basal seeds was

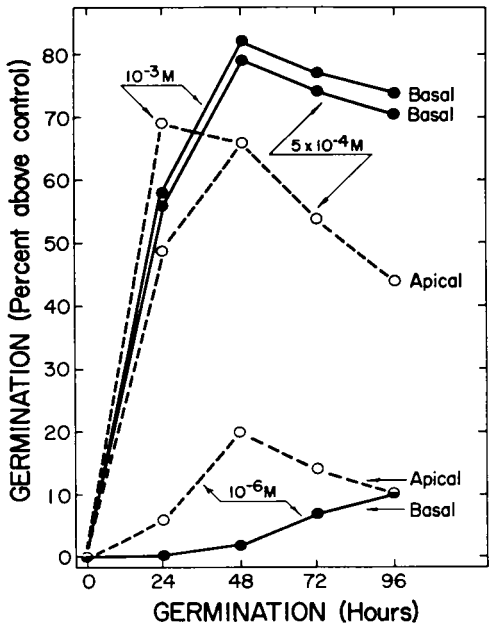

FIG. 6. The effect of 2-chloroethylphosphonic acid applied for 96 hr on the germination of dormant NC-13 peanut seeds. Each point represents the mean of duplicate samples for basal seeds and one sample of apical seeds as a comparison. Germination of control samples at 96 hr of germination was 56% and 26 ± 16% for apical and basal seeds, respectively. Maximum standard deviation from the mean values within and between treatments for basal seeds was ±3% at the two higher concentrations of CEPA.

barely above that of the control, and, correspondingly, the ethylene production was equal to the control only at 72 hr of germination. Both the germination and ethylene production of apical seeds were less at 10^{-6} M GA than at either 10^{-3} or 5×10^{-4} M GA. In both of the latter cases for apical seeds, the rise in ethylene production appeared to precede the increase in germination. Therefore, it seems that when GA was effective in breaking dormancy of peanut seeds, the hormone stimulated the ethylene production of these seeds. The action of GA appears to influence ethylene production by peanut seeds. However, the activity of GA is less directly related to immediate reactions responsible for breaking peanut seed dormancy than the activity of ethylene, since neither the progress of germination nor its magnitude approached that achieved with the lowest effective concentrations of ethylene. In contrast to the antagonistic effect of GA and ethylene in pea seedling growth (12), and lettuce seedling growth and enzyme induction (28), these plant growth regulators act in a similar manner to induce dormant peanut seeds to germinate. Gibberellic acid is known to break dormancy in a variety of seeds (19). Perhaps enhancement of ethylene synthesis by the seeds is one means of GA action in those seeds that produce ethylene as a natural metabolite. The increased ethylene production in the presence of GA agrees with that reported by Abeles and Rubinstein (4) for the first internode of bean stems. Since GA is somewhat effective in breaking dormancy of peanut seeds, while auxin is ineffective during a 96-hr test, it appears that GA is acting independently of auxin in this system.

Effect of 2-Chloroethylphosphonic Acid on Dormancy. This compound is readily converted to ethylene (11, 21, 32) and was very effective in breaking dormancy of NC-13 peanut seeds (Fig. 6). Maximum increase of germination above the control was comparable to that for 8 μl/liter ethylene. The greatest increment of germination occurred during the first 24 hr of germination at 10^{-3} and 5×10^{-4} M CEPA. This was 24 hr earlier than occurred for the ethylene treatments shown above, but this response can be duplicated by 100 μl/liter ethylene (see legend, Fig. 1).

A noteworthy result was the effectiveness of CEPA in breaking dormancy of the basal seeds. One hundred per cent germination was attained by both apical and basal seeds at 10^{-3} and 5×10^{-4} M CEPA at 48 hr of germination. The larger increase of germination of the basal seeds was due to the lower germination of the control basal seeds. The apparent decline in germination after 48 hr was due to the continued germination of the control seeds (Fig. 6). This decline was greater for the less dormant apical seeds. Thus the ability of CEPA to break dormancy of peanut seeds was best expressed by its affect on basal seeds.

Dormancy in peanut seeds can be influenced to some degree by several factors (see the introduction), and ethylene production is only one process among the several metabolic events accompanying germination. Therefore, a hormonal role for ethylene will be fully confirmed only when our criteria for the breaking of dormancy are more carefully defined. Emergence of the radical when ethylene production is near maximum (18) does not establish a causal relationship between ethylene production and the initiation of germination or the breaking of dormancy. However, the facts that endogenous production of ethylene accompanies the natural decline of dormancy (Fig. 1), that exogenously applied ethylene can intervene in the gradual breaking of dormancy (Reference 18 and Figs. 2, 3, 4), and that ethylene can cause the inherently more dormant basal seeds to germinate like the inherently less dormant apical seeds suggest a hormonal role for ethylene.

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