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Role of Protein Synthesis in the Senescence of Leaves

II. THE INFLUENCE OF AMINO ACIDS ON SENESCENCE[1]

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ABSTRACT

When the first leaf of the oat (*Avena sativa*) seedling is detached and placed in the dark, yellowing and proteolysis take place rapidly. The earlier finding that D-serine promotes this process has led to a further study of the controlling roles of several amino acids. Since the action of serine was found to be more powerful in presence of kinetin than alone, the effects of other amino acids have been restudied in presence of kinetin. Cysteine emerges as a moderately strong promotor of senescence, with glycine and alanine having definite but weaker effects. The serine effect is antagonized by arginine, especially in presence of kinetin, and so is the cysteine effect. This is considered to indicate that these two amino acids act in the same way. The antagonism exerted by arginine is in turn antagonized by canavanine. The protease activities at two pH regions which increase in the oat leaf during senescence react to both *p*-chlorimercuri-phenylsulfonate and to phenylmethyl-sulfonyl fluoride, and thus may contain both SH and OH groups. The amounts of both these enzyme activities formed in the leaf during 3 days in the dark are increased over 50% by pretreatment with serine, and this increase is very largely prevented by arginine. The amounts of soluble proteins left in the leaf vary as expected in the opposite sense. It is deduced that control of the new formation of proteases plays an important part in senescence. A suggestion is made as to the mechanism of control of senescence in leaves.

It has been shown in preceding papers (9, 12, 14) that the senescence of the detached first leaves of *Avena*, involving both the bleaching of chlorophyll and the massive hydrolysis of protein, can be prevented either by cytokinin or by certain inhibitors of protein syntheses, especially cycloheximide. The process is promoted by darkness and can also be vigorously promoted by L-serine (12). In regard to this action of serine, there are several other points of significance: (*a*) the action is specific for the L-form, (*b*) similar, though weaker, action is exhibited by cysteine, threonine, alanine, and even glycine, but it is shown below that homoserine has no such effect, (*c*) arginine protects against this action of serine, although arginine alone shows little or no real effect on senescence.

The present paper describes a further exploration of these phenomena, especially of the three-way interaction between the amino acids such as serine and cysteine which promote senescence, arginine which antagonizes them, and kinetin which inhibits the senescence process.

In view of the hypothesis (9, 12) that the action of serine may be due to its incorporation into the active center of a protease, particular attention has been paid also to the two proteases of oat leaves whose presence was demonstrated earlier.

MATERIALS AND METHODS

All procedures have been described earlier (9, 12).[3]

RESULTS AND DISCUSSION

Activity of Homoserine. If serine were to act by incorporation as active center into a proteinase, its action should be specific. Although one or two other natural amino acids (including cysteine and threonine) have weak effects, it was shown that no other amino acid was as active as L-serine and in particular that D-serine was inactive. Another, nonprotein, amino acid with otherwise very similar properties is homoserine, which was therefore tested. Table I shows that it is completely inactive in promoting senescence. Taken together with the inactivity of D-serine and β-alanine, this seems to show that only the amino acids of proteins are effective.

Activity of Amino Acids in Presence of Kinetin. A marked characteristic of the action of L-serine is that, when applied alone, its promotion of senescence, though definite, is rather weak, but when applied in presence of kinetin the effect is relatively much greater. Figure 1a shows that the serine effect, as measured by the difference between the chlorophyll contents with and without serine, increases steadily with the kinetin concentration. Indeed, the action of 10 μg/g kinetin can be completely offset by 50 mM L-serine (Fig. 1). Put another way, the amount of chlorophyll remaining becomes independent of the kinetin level.

This finding led to the thought that the apparently weak action of some of the other amino acids mentioned above might prove to be intensified if they were tested in presence of a cytokinin. Those amino acids which had been found to show weak activity, and a representative sample of others, were

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[3] To avoid complicating the picture, the protein values have not been presented in Figures 1 to 5, but in every case they vary in close parallel with the chlorophyll content. The amino-nitrogen values correspondingly vary in the opposite sense (*cf.* [9]). Absolute initial content of the 5 3-cm leaf segments were: protein, 2.61 mg (bovine serum albumin equivalent); free amino nitrogen 1.5 mM; chlorophyll $A_{665\ nm} = 0.90$.

Table I. *Effect of* DL-*Homoserine on Leaf Constituents during Senescence*

Homoserine in 10 μl Droplet	Constituents		
	Chlorophyll	α-Amino nitrogen	Protein
M	*% of initial value after 3 days in darkness*		
0	62	482	47
10^{-3}	59	454	49
10^{-2}	61	418	48
10^{-1}	55	453	49

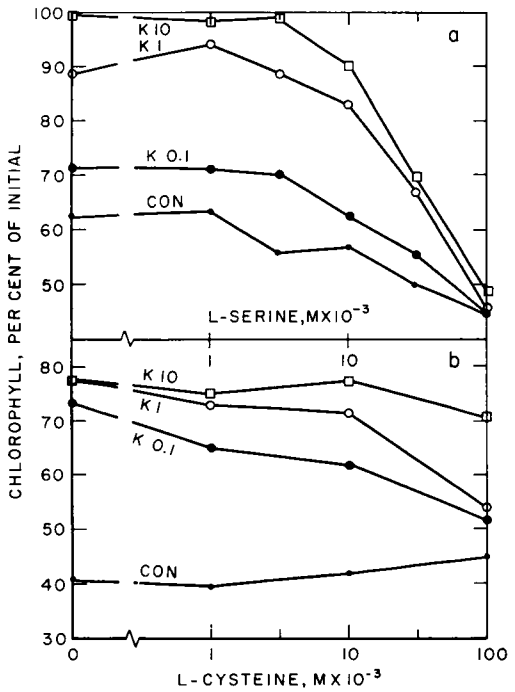

FIG. 1. Promotion of senescence in presence and absence of three concentrations of kinetin, as measured by chlorophyll disappearance. a: L-Serine; b: L-cysteine. Ordinate, chlorophyll content after 3 days in darkness, as percentage of the initial value. In this and Figures 3, 5, and 6, squares represent kinetin 10 or 5 μg/ml; circles, kinetin 1 or 0.5 μg/ml; filled circles, kinetin 0.1 or 0.05 μg/ml; dots, control.

therefore tested in presence of 1 and 10 μg/ml kinetin. The results are shown in Table II and Figure 2; L-cysteine is evidently fairly effective, while glycine and L-alanine have real, though less powerful, effects. Threonine has a still weaker but detectable effect. Dicarboxylic amino acids are truly without effect. Since the droplet of L-cysteine resting on the leaf for 3 days would almost certainly be largely oxidized to cystine, the estimate of the effectiveness of cysteine shown must be considered conservative. However, it is evidently somewhat less effective than serine, for, as Figure 1b shows, cysteine is ineffective without kinetin, and even at 0.1 M it cannot offset the action of 10 μg/ml kinetin, while serine can. (Figure 1, a and b, were done at different times, and the absolute values, though internally consistent, vary somewhat from experiment to experiment.)

Figure 2 gives a more detailed comparison of cysteine with glycine and alanine; at the low concentrations there is little difference, but at 50 mM cysteine has clearly greater effect than the others. The significance of the action of cysteine will be further considered below.

It should be noted that the ability of serine and cysteine to promote senescence in darkness seems quite different from the effect of NAD which (at 1 mM) is reported to promote yellowing only in light (16). It had no effect in darkness. Unlike the action of serine, also, the NAD effect is wholly prevented by cytokinins.

Role of Arginine. The peculiar action of arginine in antagonizing the action of serine called for further study. The experiments to be presented here serve to describe the effect more fully and offer some comparisons, although they do not explain it.

Since the arginine-serine balance was previously studied in absence of added kinetin, the three-way interaction between

Table II. *Chlorophyll Content after 3 Days in Darkness*

L-Amino acids, 30 mM were applied as 10 μl droplet on detached 3-cm apical segments of 7-day-old first leaves of *Avena sativa* (c.v. Victory) over wet filter paper in darkness at 25 ± 1 C. Five segments per test were used at each of three kinetin levels.

L-Amino Acid	Kinetin Concentration		
	Zero	1 μg/ml	10 μg/ml
	chlorophyll as % of initial value		
None	42	79	89
Histidine	45	81	89
Homoserine	45	77	85
Glutamic	43	77	84
Aspartic	42	73	84
Phenylalanine	41	73	84
Threonine	41	73	80
Alanine	40	62	73
Glycine	36	63	68
Cysteine	42	56	70

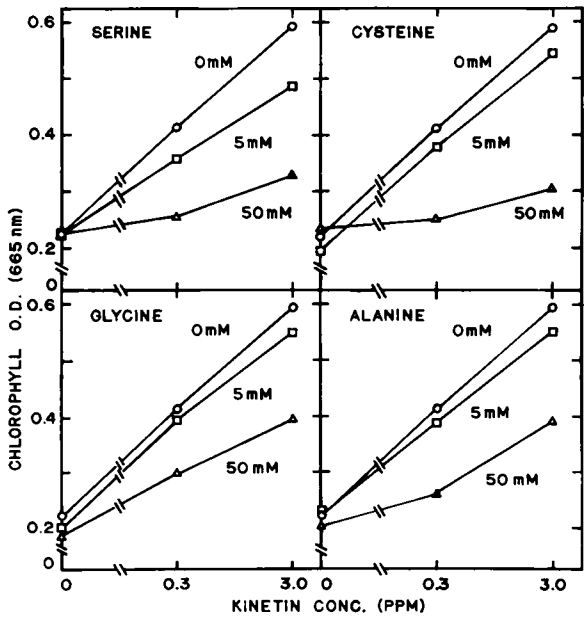

FIG. 2. Comparative effectiveness of the four most active amino acids at 5 and 50 mM in promoting senescence in presence and absence of kinetin 0.3 and 3 μg/ml. All experiments under identical conditions. Ordinate, chlorophyll content after 3 days in darkness.

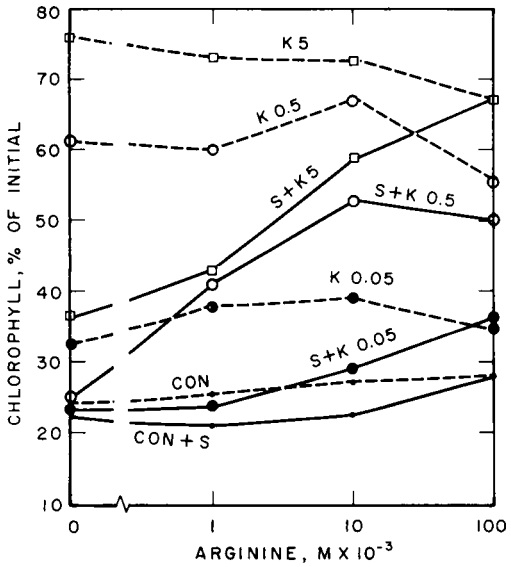

FIG. 3. Antagonism between L-serine (S) and arginine, in presence and absence of kinetin (K). Solid lines, experiments in presence of L-serine 30 mM; dashed lines, without serine. CON, control. Ordinates as in Figures 1 and 4.

arginine, kinetin, and serine was first explored. Figure 3 allows a number of conclusions. In the first place, when the level of arginine is zero, the action of serine is (as was shown in Fig. 1) very strong in the presence of kinetin, but only just detectable in the controls. Serine at 30 mM lowers the chlorophyll (at 5 μg/ml kinetin) from 76% of the initial to 36%—a powerful effect. Secondly, as the arginine concentration increases the action of serine is decreased, until 0.1 M (100×10^{-3} M in Fig. 3) arginine makes the action of serine completely (or almost completely) disappear. In presence of serine the arginine response curves slope up to the right, while without serine they show very little real slope, or even one in the opposite sense. Thus, without serine 0.1 M arginine in presence of kinetin 5 or 0.5 μg/g does promote senescence very slightly (a fact which was not noticed earlier), and this is confirmed in Figure 4.

The fact that L-cysteine, alanine, and glycine of all the amino acids come nearest to L-serine in its senescence-promoting activity is highly suggestive because glycine and alanine are readily convertible to serine (see "Discussion"). According to the literature cysteine is not readily converted to serine and thus may be acting independently. This deduction made it worthwhile to determine whether arginine exerts antagonism on cysteine too. An experiment to answer this question is shown in Figure 4. The interactions between cysteine and arginine are here studied in presence and absence of kinetin. The three lower lines show that cysteine, like serine, exerts little detectable effect alone. In presence of kinetin, and with arginine at 0 or 10 mM, cysteine clearly promotes senescence. Arginine at 0.1 M slightly enhances senescence in presence of kinetin, but nevertheless it completely *prevents* the stronger enhancement of senescence by 0.1 M cysteine. Similarly, 10 mM arginine almost completely antagonizes 10 mM cysteine.

From these results it may be concluded that the actions of cysteine and serine are similar, both promoting senescence much more strongly in presence of kinetin, and both being clearly antagonized by arginine. Since it was postulated that serine may act on senescence by promoting the synthesis of a protease, it is logical to extend this postulate to cysteine. Indeed, either a serine or a cysteine residue forms the active center of most proteases that have been studied.

The action of arginine in antagonizing serine and cysteine is not wholly specific. A survey carried out by Dr. Tetley has shown that citrulline and ornithine have similar (if not quite so strong) effects, while glutamic acid and glutamine act qualitatively in the same direction but very much more weakly. Other amino acids are inactive or almost so.

Many actions of arginine are antagonized by its analog canavanine, in which the —NH linking carbons 5 and 6 is replaced by an ether linkage. The effects of canavanine in the oat-leaf system are very suggestive. Figure 5a shows that canavanine alone has a dual effect; it acts (though more weakly) very much like cycloheximide or 6-methylpurine in that by itself it delays senescence (CON in Fig. 5a); at 0.1 M the chlorophyll content after 3 days has decreased only to 66%, while in the controls its value was 45.5%. However, in presence of kinetin, low concentrations of canavanine somewhat enhance senescence while the highest concentration delays it, giving rise to concave response curves like those for cycloheximide (9).

Being an analog to a natural amino acid, canavanine (like, *e.g.*, *p*-fluorophenylalanine) would be expected to inhibit protein synthesis and hence to inhibit those biological responses which are dependent on such synthesis; for instance, both these compounds inhibit the auxin-induced elongation of coleoptile segments (3, 11). It follows that the delay of senescence by canavanine might be due to its preventing the formation of the necessary proteases. If this is so, then arginine should specifically counteract the effect and, although it has little effect alone, arginine *should promote senescence in presence of canavanine.* Figure 5b presents a parallel experiment to Figure 5a but done in presence of 0.1 M arginine. Comparison of the controls of Figure 5, a and b, shows that arginine reduces the response to canavanine, while in presence of kinetin 0.5 or 5 μg/ml the combination of arginine and canavanine clearly *promotes senescence.* As in Figure 4 above, comparison of the zero level canavanine data in Figure 5, a and b, shows again that arginine in presence of 0.1 μg/ml kinetin has a very modest promoting effect on senescence.

The parallelism between the actions of canavanine and protein synthesis inhibitors such as 6-methylpurine can be brought out by showing the way in which the effect of the latter also varies with kinetin concentration (Fig. 6). The similarity to Figure 5 is clear; in both cases, as also with cycloheximide (Fig. 5 of ref. 14), low concentrations which alone delay senescence

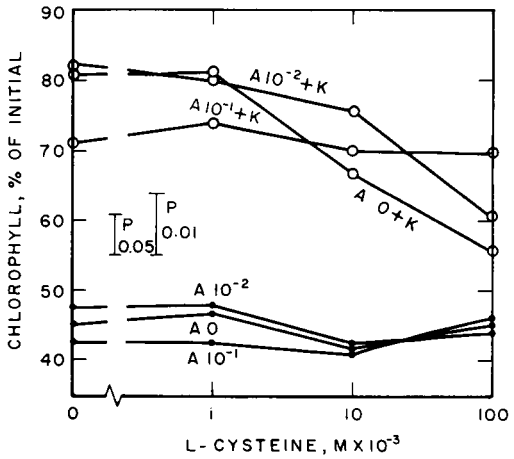

FIG. 4. Antagonism between L-cysteine and arginine (A) in presence and absence of kinetin (K). The three upper lines, experiments in presence of kinetin, 5 μg/ml; the three lower lines, no kinetin. Arginine (A) concentrations are molar. Ordinates as in Figures 1 and 3.

actually promote it in presence of kinetin. Higher concentrations, with or without kinetin, only delay senescence in all cases. The most attractive explanation of these reversals of effect is that the action of kinetin in counteracting the synthesis of proteases itself requires synthesis of a small amount of some other protein, perhaps a proteinaceous enzyme inhibitor. This special synthesis would be antagonized by low concentrations of cycloheximide, 6-methylpurine or canavanine, but at higher concentrations these agents prevent the synthesis of the proteases and thus more directly prevent senescence. Such a proposal, though speculative, would pinpoint the action of kinetin as exerted via a specific inhibitor of proteolytic enzymes.

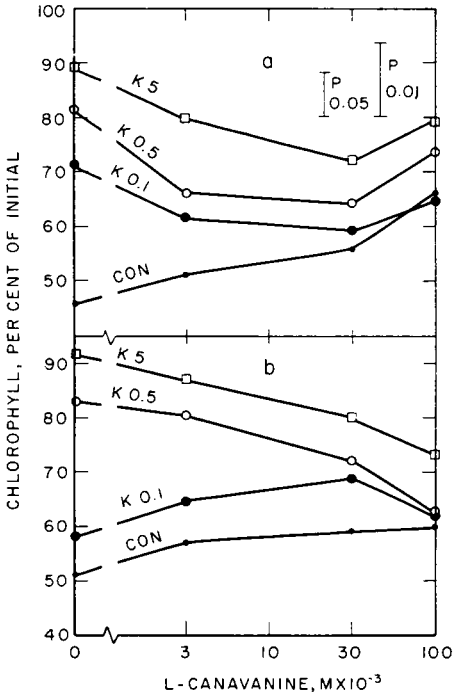

FIG. 5. Action of L-canavanine on senescence in presence and absence of kinetin. Above, L-canavanine alone; below, L-canavanine in presence of 0.1 M arginine throughout.

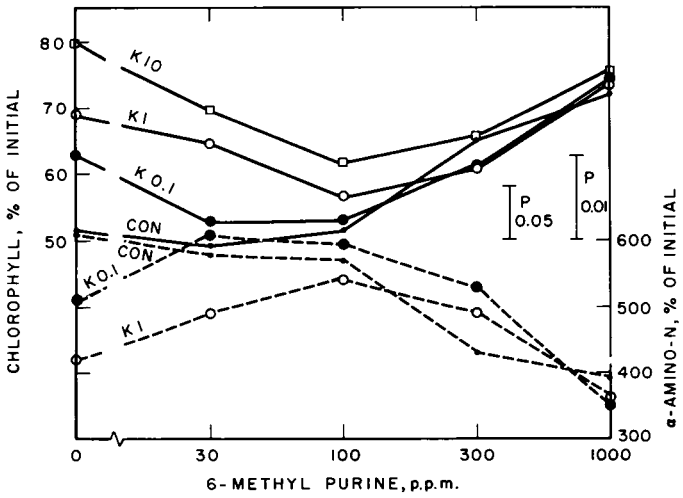

FIG. 6. Action of 6-methylpurine on senescence as a function of kinetin concentration. Solid lines, chlorophyll; dashed lines, amino nitrogen. Note the concave chlorophyll curves and the mirror images presented by the amino nitrogen curves.

Table III. *Inhibition of Oat Leaf-soluble Protease by —SH and —OH Reagents*

Solution	Activity (nmoles amino N liberated per hr)			
	Measured at pH 3	% Inhibition	Measured at pH 7.5	% Inhibition
Na PCMPS and DTE				
No addition	1737		880	
DTE 0.1 mM	1646	NS	811	NS
PCMPS 0.1 mM	503	71	97	89
PCMPS + DTE (both 0.1 mM)	846	51	463	48
PMS in 2.5% ethanol				
No addition	1155		377	
Ethanol 2.5%	1215		457	
PMS 0.08 mM	1205	NS	256	44
PMS 0.8 mM	873	28	194	58
PMS 8 mM	184	85	56	88

Importance of Cysteine Residues in the Oat Leaf Proteases. Among the data supporting the hypothesis that serine acts by incorporation into a protease was the observation that PMS[4] and DFP, which are reagents for peptide-bound serine, inhibit senescence quite effectively (14). Rather high concentrations were needed, and the action on proteolysis was somewhat less marked than that on chlorophyll. Since it now appears that cysteine is also active in senescence, its presence in the proteases was suspected, and the availability of the unpurified protease preparation described in the preceding paper made a direct test of this possible. This preparation contains two peaks of protease activity, one at pH 3 and the other at pH 7.5. The enzyme, prepared as before, was allowed to act upon hemoglobin in presence of PCMPS, and the ability of DTE to counteract its effect was studied. For comparison, similar experiments were carried out with PMS. The results (Table III) show that both the pH 3 and the pH 7.5 enzymes are sensitive to PCMPS, and the inhibition is partly counteracted by DTE. Actually the —SH reagent is the more powerful, since it gives 50% inhibition at well below 0.1 mM, while interpolation in the table shows that for the —OH reagent to produce 50% inhibition would require a concentration of 2.5 mM at pH 3, or 0.3 mM at pH 7.5. It is nevertheless satisfactory that the —OH reagent is here shown directly to act on the proteolytic function, for the previous evidence was only indirect (namely loss of chlorophyll and of protein in the leaf).

Action of Amino Acids on the Leaf Protease. In this section an attempt will be described to secure direct evidence as to the mode of action of the amino acids in controlling senescence. For if serine does indeed act by promoting the synthesis of a protease, we should expect to find increased protease activity in leaves treated with serine. Since arginine antagonizes the serine effect, it should decrease the protease activity in leaves treated with serine; but should not by itself have any obvious effect on the enzyme.

Batches of leaves were therefore treated in the usual way in the dark, with serine, arginine, and a mixture of both (all at 0.1 M); kinetin was added in the hope of making the effects clearer. After the dark period the protease was prepared as before and assayed. The total protein in the extract was also determined. The results for two such experiments, one of 3

[4] Abbreviations: PMS: phenylmethyl sulfonyl fluoride; DFP: diisopropylfluorophosphate; PCMPS: *p*-chloromercuriphenyl sulfonate; DTE: dithioerythritol.

Table IV. *Effects of Serine and Arginine on Leaf Protease Activity*[1]

	Protein in extract	Protease Activity	
		Units	Specific activity
	μg/2 ml	*nmoles amino acid/hr*	*nmoles/hr·μg protein*
Experiment 1			
Initial Value	1663		0.83
Values after 3 days			
Control	829	857	1.04
Arginine	914	870	0.95
Serine	680	1097	1.61
Arginine + serine	709	900	1.27
Experiment 2			
Initial Value	1190	763	0.64
Values after 4 days			
Control	699	943	1.36
Arginine	747	908	1.22
Serine	573	1281	2.23
Arginine + serine	667	1088	1.64

[1] Leaves were held in dark for 3 or 4 days respectively, then treated with a 10-μl droplet of the amino acid, together with kinetin 5 μg/ml in all cases. Enzyme then was extracted and incubated 4 hr with hemoglobin, and free amino acids were determined. Data are corrected for amino acid content of the enzyme.

days and one of 4, are summarized in Table IV. In both cases, (*a*) serine markedly increases the protease activity: when expressed per unit of protein the increase is by some 60%; (*b*) arginine, which is without effect alone, prevents most, though not quite all, of the serine-induced increase. Correspondingly, in both cases, (*c*) the total protein falls to about one-half the initial value during the dark period; (*d*) arginine has no significant effect on this but serine clearly promotes the fall.

DISCUSSION

The most outstanding characteristic of the leaf senescence syndrome is the remarkable series of partly specific antagonisms by agents which are ineffective alone. Kinetin on the attached and lighted leaf has little or no effect within the 3-day period; its effect shows only in presence of detachment and darkness, which it antagonizes. At the second level, serine alone has only a small effect on the detached and darkened leaf; its effect shows mainly in presence of kinetin, which it antagonizes. At the third level, arginine alone has only a slight effect, but its effect shows clearly in presence of serine, which it antagonizes. There is even a fourth level, for canavanine's effect shows mainly in presence of arginine, which it antagonizes. In all those cases where we have the data, the effects on yellowing and on proteolysis go hand in hand, and furthermore the effects on proteolysis all appear to be referable to protease synthesis. This holds for the actions of detachment and darkening, of kinetin, of cycloheximide, of serine, and of arginine.

The spectacle of a group of reactions that seem to be started or stopped either by the environment or by specific substances obviously recalls the phenomena of induction and repression in bacteria. The proteases, and probably other hydrolytic enzymes, would on this basis be considered inducible, and the operon which initiates their synthesis can be repressed, reversibly, by the kinins and derepressed, reversibly, by certain amino acids. If this were the case the serine, or cysteine, might not themselves be incorporated into the enzymes but might exert some controlling function from outside. It will be necessary next to look for more direct evidence bearing on such incorporation.

The basis for the antagonism between serine (or cysteine) and arginine (or citrulline) remains obscure. In a perhaps comparable antagonism between lysine plus threonine and methionine, Dunham and Bryan (5) were able to invoke multivalent inhibition of an enzyme in the pathway common to all three, a phenomenon as yet known only in bacteria. The arginine and serine biosynthetic pathways do not appear to have enough in common for such a relationship, and an action at the level of amino acid utilization rather than synthesis seems more likely. The activity of glycine in promoting senescence may well be due to its ready convertibility to serine by formylation (2), a conversion shown to occur in wheat leaves, both in light and in dark (15), while that of alanine may be due to its serving as amino-group donor to hydroxypyruvic acid in another biosynthetic route to serine.

A point of detail not yet cleared up is whether the serine in young pea roots is sufficiently abundant to be physiologically significant in controlling senescence (12). It has been widely assumed that the cytokinins of roots are important in this connection, following the demonstrations of cytokinin in roots and in bleeding sap (8, 10, 17). Indeed, cytokinin activity has been directly shown in the xylem sap of field peas (4) and in the roots of pea seedlings (13). Corresponding data on the serine, cysteine, and arginine of pea and oat seedling roots are not yet to hand, but in the meantime the amino acids of the bush bean, *Phaseolus vulgaris*, have been reported (7), and the data are highly supportive of our views. For in the roots of these seedlings, whether grown in the dark or in the light, the most abundant free amino acid next to asparagine is serine. This is true at all ages up to 14 days and is largely true also in the shoots. It is suggestive, too, that serine transaminase could not be detected in these plants, so that once formed the serine would not rapidly disappear.

The sensitivity of the proteases to reagents for both —SH and —OH groups is surprising. Two explanations are possible: (*a*) that the —SH reagent is not wholly specific and may combine with —OH groups too, or (*b*) that in fact, *both* groups are in the active center or at least both must remain uncombined for proteolytic activity. In favor of the former is the obvious chemical parallel. In favor of the latter, however, is the remarkable fact that serine and cysteine do in fact occur adjacent to one another in more than one proteolytic enzyme. In papain, two of the active cysteine residues have serine adjacent (1). In pepsin, a recent analysis shows the presence of no less than 5 serine-cysteine units and one of cysteine-threonine-serine (6). Such a frequency can hardly be coincidental. Whatever the explanation, the fact that besides serine the next best amino acid strongly active in promoting senescence should be cysteine, when so far as is known all proteases (except those activated by metal ions) require one of these for activity, certainly strongly supports our general concept of the processes governing senescence.

Acknowledgments—We wish to thank Dr. Richard Tetley for kindly carrying out the survey of activities of amino acids from which Figure 2 was obtained. Thanks are also due to Mark Spritzer for good technical help.

LITERATURE CITED

1. Arnon, R. 1970. *In* S. P. Colowick, and N. O. Kaplan, Methods in Enzymology, Vol. XIX. Academic Press, New York. pp. 226-244.
2. Blakley, R. L. 1955. The interconversion of serine and glycine; participation of pyridoxal phosphate. Biochem. J. 61: 315-323.
3. Bonner, J. 1949. Limiting factors and growth inhibitors in the growth of the *Avena* coleoptile. Amer. J. Bot. 36: 323-332.

4. Carr, D. J. and W. J. Burrows. 1966. Evidence of the presence in xylem sap of substances with kinetin-like activity. Life Sci. 5: 2061–2067.
5. Dunham, V. L. and J. K. Bryan. 1969. Synergistic effects of metabolically related amino acids on the growth of a multicellular plant. Plant Physiol. 44: 1601–1608.
6. Foltmann, B. 1970. The N-terminal and C-terminal amino acid sequence of calf rennin. Phil. Trans. Roy. Soc., B 257: 147–151.
7. Forest, J. C. and F. Wightman. 1971. Metabolism of amino acids in plants. I. Changes in the soluble amino acid fractions of bushbean seedlings (*Phaseolus vulgaris* L.) and the development of transaminase activity. Can. J. Biochem. 49: 709–720.
8. Kende, H. 1956. Kinetin-like factors in the root exudate of sunflower. Proc. Nat. Acad. Sci. U.S.A. 53: 1302–1307.
9. Martin, C. C. and K. V. Thimann. 1972. The role of protein synthesis in the senescence of leaves. I. The formation of protease. Plant Physiol. 49: 64–71.
10. Nitsch, J. P. and C. Nitsch. 1965. Présence de phytokinines et autres substances de croissance dans le sève d'*Acer saccharum* et de *Vitis vinifera*. Bull. Soc. Bot. Fr. 112: 11–18.
11. Noodén, L. and K. V. Thimann. 1963. Evidence for a requirement for protein synthesis for auxin-induced cell enlargement. Proc. Nat. Acad. Sci. U.S.A. 50: 194–200.
12. Shibaoka, H. and K. V. Thimann. 1970. Antagonisms between kinetin and amino acids: experiments on the mode of action of cytokinins. Plant Physiol. 46: 212–220.
13. Short, K. C. and J. G. Torrey. 1972. Natural occurrence of cytokinins in roots of pea seedlings. Plant Physiol. 49: 155–160.
14. Thimann, K. V., H. Shibaoka, and C. C. Martin. 1972. On the nature of senescence in oat leaves. Proc. Int. Growth Subst. Conf., Canberra, Australia (Dec. 1970). In press.
15. Wang, D. and R. H. Burris. 1965. Carbon metabolism of C^{14}-labeled amino acids in wheat leaves. III. Further studies on the role of serine in glycine metabolism. Plant Physiol. 40: 415–418.
16. Waygood, E. R., D. Mishra, and H. R. Godavari. 1968. Responses of nicotinamide nucleotides to kinins in leaves of *Elodea* and wheat. *In:* F. Wightman and G. Setterfield, eds., Biochemistry and Physiology of Plant Growth Substances. Runge Press, Ottawa. pp. 1495–1514.
17. Weiss, C. and Y. Vaadia. 1965. Kinetin-like activity in root apices of sunflower plants. Life Sci. 4: 1323–1326.