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Diana Borut Stein

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THE DEVELOPMENTAL MORPHOLOGY OF NICOTIANA TABACUM L. CV. WHITE BURLEY AS INFLUENCED BY VIRUS INFECTION AND GIBBERELLIC ACID

by

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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>ii</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>MATERIALS AND METHODS</td>
<td>2</td>
</tr>
<tr>
<td>OBSERVATIONS AND DISCUSSION</td>
<td>5</td>
</tr>
<tr>
<td>- Plant height</td>
<td>5</td>
</tr>
<tr>
<td>- Rates of leaf production</td>
<td>8</td>
</tr>
<tr>
<td>- The pattern of internode elongation</td>
<td>12</td>
</tr>
<tr>
<td>- Changes in leaf size and shape</td>
<td>17</td>
</tr>
<tr>
<td>SUMMARY</td>
<td>36</td>
</tr>
<tr>
<td>LITERATURE CITED</td>
<td>37</td>
</tr>
</tbody>
</table>

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FIGURES

FIGURE I: Growth in height of *N. tabacum* L. cv. White Burley—Run I ........................................... 6

FIGURE II: Growth in height of *N. tabacum* L. cv. White Burley—Run II ........................................... 7

FIGURE III: Rate of leaf production of *N. tabacum* L. cv. White Burley—Run I........................................... 9

FIGURE IV: Rate of leaf production of *N. tabacum* L. cv. White Burley—Run II ........................................... 10

FIGURE V: Growth of internodes of *N. tabacum* L. cv. White Burley—Controls Run I ........................................... 13

FIGURE VI: Growth of internodes of *N. tabacum* L. cv. White Burley—Virus infected—Run I ........................................... 14

FIGURE VII: Growth of internodes of *N. tabacum* L. cv. White Burley—Controls—GA treatment from day 7—Run I ........... 15

FIGURE VIII: Growth of internodes of *N. tabacum* L. cv. White Burley—Virus infected—GA treatment from day 7—Run I ........... 16

FIGURE IX: Correlation of lamina dimensions of four successive leaves (4th-7th youngest) of *N. tabacum* L. cv. White Burley—Controls, day 7—Run I ........................................... 18

FIGURE X: Correlation of lamina dimensions of four successive leaves (4th-7th youngest) of *N. tabacum* L. cv. White Burley—Controls, day 7—Run I ........................................... 20

FIGURE XI: Correlation of lamina dimensions of four successive leaves (4th-7th youngest) of *N. tabacum* L. cv. White Burley—Virus infected, day 7—Run I ........................................... 21

FIGURE XII: Regression of lamina dimensions of four successive leaves (4th-7th youngest) of *N. tabacum* L. cv. White Burley, day 7—Run I ........................................... 22

FIGURE XIII: Correlation of lamina dimensions of five successive leaves (4th-8th youngest) of *N. tabacum* L. cv. White Burley—Controls, day 14—Run I ........................................... 23
FIGURE XIV: Correlation of lamina dimensions of five successive leaves (4th-8th youngest) of *N. tabacum* L. cv. White Burley—Controls + GA, day 14—Run I. .......................... 24

FIGURE XV: Correlation of lamina dimensions of five successive leaves (4th-8th youngest) of *N. tabacum* L. cv. White Burley—Virus infected, day 14—Run I. ..................... 25

FIGURE XVI: Correlation of lamina dimensions of five successive leaves (4th-8th youngest) of *N. tabacum* L. cv. White Burley—Virus infected + GA, day 14—Run I. ..................... 26

FIGURE XVII: Regression of lamina dimensions of five successive leaves (4th-8th youngest) of *N. tabacum* L. cv. White Burley, day 14, untreated—Run I ................. 26

FIGURE XVIII: Regression of lamina dimensions of five successive leaves (4th-8th youngest) of *N. tabacum* L. cv. White Burley, day 14, GA treated—Run I ..................... 29

FIGURE XIX: Average length and width of leaf 8 of *N. tabacum* L. cv. White Burley—Run II ......................... 30

FIGURE XX: Average length and width of leaf 16 of *N. tabacum* L. cv. White Burley—Run II ......................... 32

FIGURE XXI: Average length and width of leaf 23 of *N. tabacum* L. cv. White Burley—Run II ......................... 34
INTRODUCTION

It is well known that one of the common effects of viral infection on plants is a substantial reduction in stature (Bawden, 1956). This stunting has been reversed by treatment with gibberellic acid (GA) in several instances. Maramorosch (1957) demonstrated this using corn stunt, aster yellows, and wound tumor viruses. Chessin (1957) reversed the stunting of Severe Etch Virus (SEV) diseased plants with GA but found that the final height of the treated plants did not equal that of the controls. In these cases other viral symptoms were not reversed. The above studies used over-all plant height as the criterion for the effectiveness of treatment.

Bonnand's study (1956) of Nicotiana tabacum L. cv. White Burley showed that this plant is normally rosetted through leaf stage 15, with stem elongation occurring at leaf stage 18-20, and flowering at leaf stage 30-33. Infection with SEV maintains the rosette condition and reduces the leaf area, without any effect on rate of leaf production (Bawden and Kassanis, 1941).

The object of this study was to determine the morphological changes induced in White Burley Tobacco by SEV and the effect of treatment with GA. Plant height, rate of leaf production, internode elongation, and changes in leaf size and shape were used as assay systems for the changes studied.
MATERIALS AND METHODS

Seeds of White Burley Tobacco were germinated in sterilized quartz sand, watered with $\frac{1}{2}$-strength Hoagland's solution, and provided with continuous incandescent illumination during the course of germination. The young seedlings were subsequently transferred to two and four inch pots in a greenhouse and were treated every two weeks with 50 cc full-strength Hoagland's solution.

Two experimental runs were conducted. In each case about 65 plants were divided into four groups. The plants ranged in leaf stage from 11-13. Leaf number eight was marked with indelible ink to serve as a morphological reference. Since transplanting did sometimes obscure some of the early leaves and since cotyledons might be mistaken for leaves, it is probably more accurate to call this leaf eight plus or minus two. Thirty-two plants were then inoculated on leaf 8 with a Rothamsted strain of SEV. Disease symptoms showed up in about a week. Thirty-two controls were similarly inoculated with water. After two weeks one group of controls and one group of virus-infected plants were sprayed with an aqueous solution of 100 ppm GA. In the first run the entire plant was sprayed to run-off and in the second experiment only the growing point and surrounding leaves were sprayed. When the growing point became a flower bud, a leaf approximately 10 cm in length was sprayed in lieu of the apical region. After the plants stopped growing in height spraying was discontinued. The first run was conducted from February, 1960 to May, 1960 and the second from July, 1960 to January, 1961. No difference in severity of
infection was noted between the two runs. However, in neither case were symptoms as severe as those described by other workers (Bawden and Kassanis, 1941; Chessin, 1957).

All measurements were made with a ruler to the nearest millimeter. Measurements of plant height were made from the soil level to the tip of the youngest visible leaf, except when flowering had occurred. The measurements were then made to the tip of the highest flower bud. After the petals dropped the tip of the highest seed capsule was used as criterion. Leaf stage was determined by counting from the marked leaf number eight to the smallest leaf which could be seen by gently prying apart the terminal bud with a pencil. This leaf was approximately .25 mm long. When leaf eight died an older leaf was similarly marked. Internode measurements were made from the base of one petiole to the base of the next petiole. Internodes less than five mm long were approximated since a ruler could not be placed next to the stem due to the closeness of the nodes.

In Run I the length and width of the fourth, fifth, sixth, seventh, and sometimes eighth youngest visible leaves (counting down from the stem tip) were periodically measured. In this case a changing population was used to assay for early effects due to treatment. In Run II, however, continued effect on the same organ was studied. Therefore, measurements of the length and width of the same leaves were made. Three leaves were selected for every plant: one leaf which was present prior to treatment, one which was a young primordium or leaf presumptive as treatment was
begun, and a third which was definitely initiated some time after
the start of treatment with GA. For each of the four groups of
plants (controls, diseased, sprayed, and unsprayed) both length
and width of the leaf were averaged and plotted against time.
Approximately seven leaves intervened between any two leaves assay-
ed. Measurements were taken weekly or fortnightly.
Growth in plant height during Runs I and II are shown in figs. 1 and 2. Each point represents a minimum of 12 plants. In Run I a difference in height between the virus-infected plants and the controls could be seen within the first two weeks, while in the second run this did not occur until six weeks after inoculation. The plants sprayed with GA showed a height response within a week in both runs. Infection with virus limited the ability of the plant to respond to GA. Thus in Run I a difference in height between sprayed infected and sprayed disease-free plants could be seen at two weeks after spraying, and in Run II as early as one week after treatment. In the case of Run II the difference in stature between the healthy and diseased plants was seen some five weeks earlier in the sprayed groups than in the unsprayed groups. Spraying of the entire plant produced such rapid shoot growth that the root systems which are normally reduced by treatment with GA (Stowe and Yamaki, 1957) apparently could not support the plants and they showed severe wilting after the first month. These plants reached a height in three weeks not attained by the plants of the second run until after four and one-half weeks of treatment. The curves obtained in Run II show the typical "S" shape. Both GA-sprayed groups ceased over-all stem elongation at approximately the same time as the unsprayed controls with growth of the virus-infected plants leveling off over a month later. The final difference in height between the sprayed virus-infected plants and the sprayed controls was almost 300 mm (17%). This agrees with Chessin (1957) who stated that spraying with GA did not completely overcome the stunting induced by the virus. Since spraying of the entire plant did not overcome this
Fig. 1. Growth in height of *N. tabacum* L. cv. White Burley - Run 1.
difference, it appears that either an intermediate amount of spray is needed or that the ultimate difference in size is due to something other than insufficient gibberellin. It is of interest that although the GA-treated plants reached maturity earlier than the controls as indicated both by plant height and flowering they survived for a longer period of time. Five months after the start of the experiment only 25% of the sprayed controls had died, whereas the mortality of the untreated controls was over 60%. To my knowledge this has not been reported previously. None of the virus-infected plants had died at this time. Longer survival of the infected plants would be expected in the unsprayed group since maturation was also delayed, but the reason for longevity in the sprayed group is obscure.

Rates of leaf production for Runs I and II are shown in figs. 3 and 4 respectively. Each point represents a minimum of 12 plants. In both runs the virus-infected plants and the controls had produced approximately the same number of leaves at time of inoculation. Two weeks later increased rate of leaf formation in the virus-infected plants had produced at least one more leaf per plant than in the disease-free tobacco. This difference in leaf number was maintained in both runs until there occurred an increase in rate of leaf production of the healthy plants just prior to flowering. The data presented here are contrary to Bawden and Kassanis' (1941) statement that there is no change in rate of leaf production due to infection with SEV. No quantitative data were supplied by these workers.

Figs. 3 and 4 show an increase in number of leaves produced by the plants treated with GA within one week after treatment. This
Fig. 3. Rate of leaf production of *N. tabacum* L. cv. White Burley - Run I.
Fig. 4. Rate of leaf production of *N. tabacum* L. cv. White Burley - Run II.
increase in number of leaves of GA-treated tobacco has also been reported by Yabuta et al. (1941). Between three and four weeks after spraying there was a sharp increase in number of leaves of the sprayed plants. This is typical of the stage just prior to flowering (Bonand, 1959). The pattern of the curves after this time is somewhat obscure. This is because at this time the flower buds are being initiated and it is difficult to tell whether an organ 0.25 mm long is a leaf or a very young floral bud. The final point in the curve is, however, very accurate since the count for it was made after the bud had opened and elongated and it was, therefore, very easy to count the number of leaves. In the sprayed virus-infected plants one other point should be noted. The early increase in number of leaves, stimulated by infection with SEV, had been amplified by the time of final leaf count to a difference of 2.6 more leaves. This may be partially due to the fact that there was a delay of ten days as compared to the sprayed controls before the sprayed virus-infected plants reached flowering.

It is of interest that there was no difference between the total number of leaves produced by the sprayed and unsprayed controls. It would seem that treatment with gibberellin simply increased the rate of leaf production and maturation without increasing the ultimate number of leaves. This has been described previously in tomato by Rappaport (1957) and Soost (1959). In the case of the virus-infected plants the unsprayed group produced eight more leaves than the unsprayed controls. This is probably because the virus-infected plants which were not sprayed were delayed in flowering. When one
speaks of the reduced surface area of virus-infected plants (Bawden and Kassanis, 1941) one should take into consideration the fact that a greater number of leaves is produced by the diseased plants which may compensate for the smaller individual leaf area. Under severe infection which unfortunately I was not able to obtain, the diseased plant theoretically could produce many more leaves than a healthy plant. Actual measurements of total leaf area would have to be made to ascertain this point.

The pattern of internode elongation is shown in figs. 5-8. Each treatment involved a minimum of 12 plants. Internodes two through six had probably stopped growth by the time these measurements were begun. Internodes seven through ten were capable of limited growth. All these internodes were much shorter than internodes 11 through 34 which show the effect of elongation due to flowering. From the histogram one can estimate that most internodes ceased grossly measurable growth in a period of five weeks. The longest internodes were 19 through 24. This correlates well with the fact that elongation begins in leaf stage 18-20 (Bonnand, 1956). Since these internodes would be very young or not even present at time of the release of the substances which cause elongation, they show the major expansion. The older ones were able to respond only to a limited degree.

Infection with virus shifted this growth pattern upward on the plant. The longest internodes were then 24-29 indicating that elongation and flowering were delayed in the diseased plants. The longest
Fig. 5. Growth of internodes of N. tabacum L., cv. White Burley - Controls.
Fig. 6. Growth of internodes of *N. tabacum* L. cv. White Burley — Virus infected.
Fig. 7. Growth of internodes of N. tabacum L. cv. White Burley—Controls — GA treatment from day 7.
Fig. 8. Growth of internodes of N. tabacum L. cv. White Burley—Virus infected—

treatment from day 7.
internode, number 26, on the virus-infected plants was 25.6 mm as opposed to 37.9 mm for numbers 21 and 22 on the healthy plant. This is a good indication of the reduction in size (33% in this comparison) of the internodes on the diseased plant. Internode 9 is probably the first internode which showed the effect of virus-induced stunting.

Spraying with GA produced a definite response with an internode as old as number 8. The increments in length attained by subsequent internodes in the short period before the plants died were extremely large. In terms of percent increase the longest internode on the sprayed virus-infected plant showed an increase of 179% over the longest internode on the unsprayed diseased plants. The sprayed controls showed an increase of 171% over the longest internodes on the unsprayed controls. However, the longest internode on the diseased plants was still 19 mm shorter than the longest on the sprayed controls. It is of interest that the longest internodes on the sprayed controls were 11 and 12, while those on the sprayed virus-infected plants were 13 and 14. This effect is probably due to the fact that the virus-infected plants produced more internodes than the controls and therefore those internodes responded maximally to GA which were at the same "ripe" stage of development.

Changes in leaf size and shape. As stated under materials and methods two different approaches were used in the study of leaf growth. The relation of length to width of young leaves in untreated plants is shown in the regression of fig. 9. The data were obtained by measurements on all plants at the beginning of the experiment. As could be expected there is a good correlation between the length and width of
Fig. 9. Correlation of lamina dimensions of four successive leaves of N. tabacum L. cv. White Burley.
the leaves. Furthermore, it appears that in this case the fourth to seventh youngest leaves were consecutive stages of an over-all pattern suggesting that these leaves represent the ontogenetic pattern of a single leaf. One week later the controls and virus-infected plants were plotted separately (figs. 10 and 11, respectively). There seems to be a slight suggestion of curvilinearity to the regressions. In order to see whether this change in pattern was real, the data were separated according to leaf number and for each leaf number the ten smallest and ten largest leaves were averaged. The revised graphs are shown in fig. 12. Several points become evident when the data are plotted in this manner. All leaves on the virus-infected plants with the exception of the seventh youngest visible leaf, which presumably was fairly mature at time of infection with virus, were smaller than the corresponding leaves on the controls. This is very early evidence of the virus-induced reduction in growth. The slope values also show clearly that the correlation graph tends to obscure the curvilinearity. The slope of the regression of leaves "four" and "seven" is lower than that of the intermediate leaves. This means that at this stage leaves "five" and "six" had a larger increase in width in relation to length than leaves "four" and "seven". The plants were then divided into four groups and two groups were treated with GA. Data obtained the following week are plotted in figs. 13-16. Since rate of leaf production increased during this period and rate of leaf growth apparently did not, an additional leaf was measured. This permitted the comparison of leaves of similar dimensions, at least in the controls. The correlations are still high and curvilinearity is also evident in these
Fig. 10. Correlation of lamina dimensions of four successive leaves of *N. tabacum* L. cv. White Burley.
Fig. 11. Correlation of lamina dimensions of four successive leaves of *N. tabacum* L. cv. 'White Burley.'
Fig. 12. Regression of lamina dimensions of four successive leaves (4th-7th youngest) of *N. tabacum* L. cv. White Burley.
Fig. 13. Correlation of lamina dimensions of five successive leaves of *N. tabacum* L. cv. White Burley.
Fig. 14. Correlation of lamina dimensions of five successive leaves of *N. tabacum* L. cv. White Burley.
Fig. 15. Correlation of lamina dimensions of five successive leaves of *N. tabacum* L. cv. White Burley.
Fig. 16. Correlation of lamina dimensions of five successive leaves of *N. tabacum* L. cv. White Burley.
graphs. It can be seen that GA reduced the width of the young leaves as early as one week after the first treatment. The second manner of plotting (figs. 17 and 18) shows essentially the same pattern as described above. In the virus-infected material and in both sprayed groups the inclusion of leaf "eight" does not show a decrease in slope. This again correlates with the increased rate of leaf production which was induced by the various treatments. Some other leaf such as "nine" or "ten" would probably have shown the leveling off in slope. Otherwise the pattern is the same as observed in the controls with the intermediate leaves showing an increase in width in relation to length.

While the above data were obtained by the periodic measurement of the immediate products of the shoot apex, it was felt that possibly some additional information might be gained by consecutive measurement of the same leaf throughout its ontogeny. This was done in Run II. In fig. 19 the periodic increase in length and width of leaf eight for the various treatments has been plotted. As previously observed the virus-infected plant had its leaf length sharply reduced (by 30 mm) and the spray did not affect this. The effect of the spray on the leaves of the healthy plants is to make them longer than those of the controls, a result of increased duration of the maximum growth phase. This had not shown up in the previous method of analysis. It can also be seen that the virus-infected leaves were slower to reach ultimate length as they were still growing at least one week after the others had matured. A comparison of width reveals a slight difference (five mm) between the width of the sprayed and unsprayed controls but a large difference (25 mm) between the width of all the virus-infected plants.
Fig. 17. Regression of lamina dimensions of five successive leaves (4th-8th youngest) of *N. tabacum* L. cv. White Burley.
Fig. 18. Regression of lamina dimensions of five successive leaves (4th-8th youngest leaves) of *N. tabacum* L. cv. White Burley.
Fig. 19. Average length and width of leaf 8 of *N. tabacum* L. cv. White Burley.
... types of controls. The curves for the various treatments are generally similar for both length and width with the exception of the control plants which showed the greatest increase in both length and width between day 0 and day 7. This would indicate that initially the leaves of the healthy unsprayed plants had the highest growth rate, although ultimately the GA-treated controls overcame this early spurt. It would be worthwhile to investigate further whether this implies a differential effect on cell division versus cell elongation.

Leaf number 16 (fig. 20) was a primordium or still part of the apical meristem at time of treatment. At maturity leaf 16 of the controls was some 40 mm shorter than leaf seven. It was also the shortest of the four treatment groups. This could be explained on the basis that after a certain point, proximity of leaf position to the inflorescence decreases leaf size. Thus one could expect that leaf number 16 of the controls would be shorter than the same leaf on the virus-infected plants, since the controls will flower after fewer leaves have been formed. Since the controls will still produce some 13 leaves before the flower bud is formed, the reduction in leaf size at this time may be correlated with the elongation process rather than with flowering. The leaves on the sprayed controls were longer than the unsprayed controls which is a predictable result of gibberellin treatment. Leaf 16 of the unsprayed virus-infected plants was only a few millimeters longer than that of the sprayed-diseased plants. Since the unsprayed group produced five additional leaves prior to flowering one would expect a greater size difference between the two groups. This can be explained if one assumes that the GA does lengthen the virus-infected leaves. This would correlate with the reduction in width of the sprayed leaves, a usual response in
Fig. 20. Average length and width of leaf 16 of *N. tabacum* L. 
cv. White Burley.
rice as shown by Ito and Kimura (1931) and described for White Burley by Gray (1957). It should be noted that the curving of the leaf apex to one side, also described by Gray, was not observed in these experiments, although, the concentration of GA applied was five times greater here. The fact that at this time the virus-infected plants had the longest (and widest) leaves indicates that, although, the over-all action of the virus is to reduce plant size, individual organs may actually reach a larger size because of the delay in reaching maturity. The growth in width of the same leaves is also presented in fig. 20. It can be seen that the virus-infected plants had wider leaves than the controls. Narrower than either were the GA-treated plants. It is known that one of the effects of spraying with GA is reduction in leaf width (Ito and Kimura, 1931) and it would be interesting to see whether the lateral meristems are involved in this effect. In all treatments growth in length and width ceased at about the same time. The leaves of the virus-infected plants appeared to reach onset of maturity at the same time although the virus-diseased leaves grew an additional week.

As can be seen in fig. 21 leaf number 23 of the controls is the shortest. Since the control plants are only some six leaves away from flowering one would expect this. The longest leaves are those of the GA-sprayed plants, with the sprayed virus-infected leaves being considerably longer than the leaves of the sprayed controls. In both groups these leaves were formed during the course of treatment, and spraying with GA had the greatest relative effect on them. The size difference between the two groups is again accounted for by the fact that more
Fig. 21. Average length and width of leaf 23 of *N. tabacum* L. cv. White Burley.
leaves were produced by the virus-infected plant prior to flowering. Fig. 21 shows that the widest leaves were the unsprayed virus-infected ones. All the leaves are relatively narrow because of proximity to the flower bud; the widest leaves were those which were furthest removed from the flower.

It can readily be seen that any group of leaves chosen at random (i.e. 8, 16, and 23 in this case) could show differing relationships and hence be responsible for contradictory reports in the literature. In general one can say that the effect of GA on a young leaf primordium of White Burley Tobacco is to lengthen and narrow the resulting leaf. Infection with virus in a relatively mature leaf seemed to prevent the leaf from responding to GA, but leaves produced during the course of treatment with GA were able to respond even though infected with SEV. Leaves present on healthy plants prior to spraying with GA were able to respond with growth in both length and width and this might explain the observation of Yabuta et al. (1941) that the largest leaves on tobacco were found on GA-sprayed tobacco plants, although over-all size of sprayed leaves is smaller. It should be noted, however, that sprayed leaves are really longer, though narrower, so this statement is not quite correct for White Burley Tobacco. Virus infection may reduce the final size of leaves already present at time of treatment but ultimately may result in the production of larger leaves as compared to the same leaf number of the controls. This is due to extended leaf production on the part of the diseased plants.
SUMMARY

Infection of *Nicotiana tabacum* L. cv. White Burley with Severe Etch Virus (SEV) resulted in a reduction of plant height which was overcome to a limited degree by spraying with gibberellic acid (GA). Spraying with GA, while hastening maturity in terms of earlier elongation and flowering, also prolonged the life of treated plants. Infection with SEV caused an increased rate of leaf production, and since flowering was also delayed, the greater number of leaves is produced by infected plants. Spraying with GA also increased the rate of leaf production but did not increase the final number of leaves produced. All groups except the unsprayed virus-infected plants showed a spurt in leaf production just prior to flowering. Individual internodes were measured and a pattern of internode elongation obtained. In general this reflected the stunting properties of the virus and the growth promoting properties of the GA. Internodes which were mature at time of spraying with GA were not affected. Infection with virus generally delayed elongation and shifted the internode pattern.

Infection with SEV tended to reduce the size of leaves already present prior to inoculation, but some leaves produced after infection were actually larger than the same aged leaf on the controls. Spraying a healthy plant with GA made older leaves longer and wider, while less mature leaves at time of treatment tended to be longer and narrower. Spraying with GA reversed the reduction in size caused by the virus only if the leaf was a very young primordium or was formed during the course of treatment with GA.


