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Effects of Gibberellic Acid and Sucrose on the Growth of Oat (*Avena*) Stem Segments

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ABSTRACT

Gibberellic acid induced growth in *Avena* (oat) stem segments within 35 minutes after hormone application. The total elongation elicited by gibberellic acid was greater than 15 times the control growth. The sensitivity of the segments to low concentrations of gibberellic acid (1 pmole) and the specificity of the segments to the gibberellin class of hormones suggest that oat stem segments would be a valuable tool for gibberellin bioassays. Both gibberellic acid-induced growth and control growth are temperature-dependent and showed a Q_{10} of two or greater. Although the most apparent effect of gibberellic acid was to promote the uptake of water into the internode, the hormone also promoted transport of endogenous substrate and the uptake of exogenous substrate into the growing region. The growth promotion was accomplished without an apparent increase in osmotic pressure.

The internodal growth in excised *Avena* stem segments which contain the intercalary meristems has been shown to be strikingly stimulated by exogenous applications of gibberellins, whereas little elongation is induced by auxins and cytokinins when applied singly (15, 17). The GA-promoted growth in these segments was found to be primarily due to an increase in rate of cell elongation (18). Since gibberellins generally promote the growth of intact plant stems much more markedly than the growth of excised plant stems (5, 25, 29), the exceptionally large response and specificity to gibberellins observed in excised *Avena* stem segments presents a unique opportunity to study the mechanism of gibberellin-stimulated stem growth.

An earlier attempt to correlate the gibberellin-promoted growth with the gibberellin-stimulated invertase activity indicated that the GA-enhanced invertase activity does not account for all of the gibberellin promoted growth in the segments (21). We are, thus, far from understanding the primary mode of action of gibberellins in growth stimulation of *Avena* segments. Therefore, attempts are made here to analyze the basic physiologic processes associated with gibberellin-stimulated growth in order to shed light on the mechanism of gibberellin action in stem elongation.

MATERIALS AND METHODS

Unless otherwise mentioned, plants of *Avena sativa* cv. Victory were grown in a greenhouse for about 6 weeks until the internode below the peduncle (p-1 internode) was 1 to 3 cm long. One-cm segments of these internodes along with their subtending nodes and encircling sheaths were isolated with a razor blade cutting device and used for this study (18, 19, 21). The segment thus includes a growing internode with its basally located intercalary meristem and nongrowing node and sheath.

Segments were placed with nodes down on a piece of filter paper placed in a styrofoam disk in a plastic Petri dish which contained 15 ml of test solution. The cover of the Petri dish was perforated so as to support the segments vertically. Petri dishes were incubated in a closed moist container (with provision for some air exchange) throughout the experimental period. The experimental solutions used in this study were Hoagland's solution (see ref. 3), H[2] containing 0.1 M sucrose (HS), H containing 30 μM GA (HG), and H containing 0.1 M sucrose + 30 μM GA (HSG). Routine measurements of growth were made with a ruler to the nearest 0.5 mm on the segments which had been treated with a given test solution in a Sherer-Gillet growth chamber for various periods. The growth chamber was programmed for 18 hr light (750 ft-c at the tissue level with fluorescent and incandescent lamps) at 21 C and 6 hr dark at 16 C.

In order to determine rapid growth responses to various treatments, an apparatus (termed "microgrowth" apparatus) was constructed with a compound microscope (Fig. 1). One segment at a time was used in this apparatus in the following manner: tight fitting polyethylene tubing was forced over the node of the segment, which was then fitted snugly into a short section of glass tubing so that the polyethylene tubing formed a water-tight seal with the glass. Before placing the segment into the glass tube (D), the sheath of the segment was partially removed, and a glass collar with an attached pointer was placed around the top of the segment. A small triangle of aluminum foil cemented onto the pointer provided a sharp edge to which the horizontal microscope (E) was focused. A test solution, added through the reservoir (A), was circulated to the base of the segment by means of a Chemical Rubber Company vibrostaltic pump (B) whose flow rate was controlled by a rheostat. Temperature control during the experiment was achieved by passing liquid of a given temperature through the outer jacket of the condenser (C) to which a Neslab water bath (F) with a portable bath-cooler (Model PBC-4) was connected.

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[2] Abbreviations: H: Hoagland's solution; HS: H containing 0.1 M sucrose; HG: H containing 30 μM GA; HSG: H containing 0.1 M sucrose + 30 μM GA.

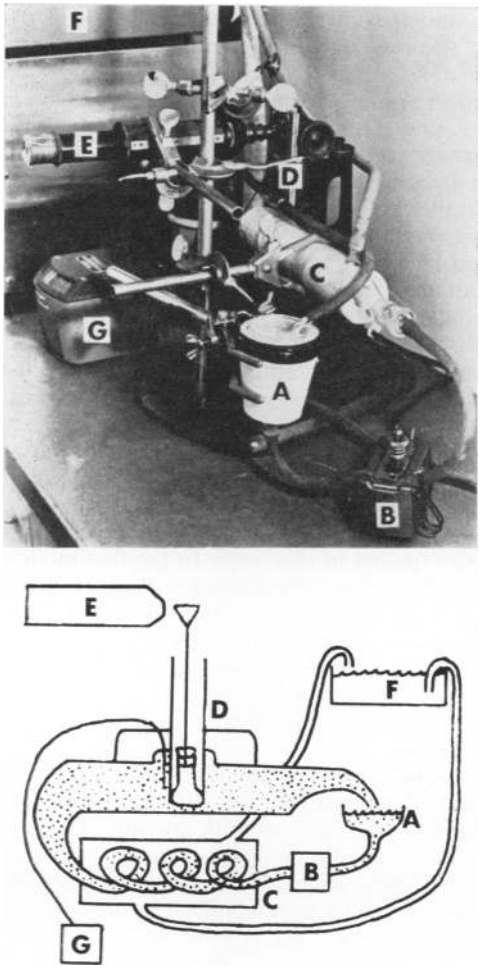

FIG. 1. Microgrowth apparatus. See "Materials and Methods" for explanation.

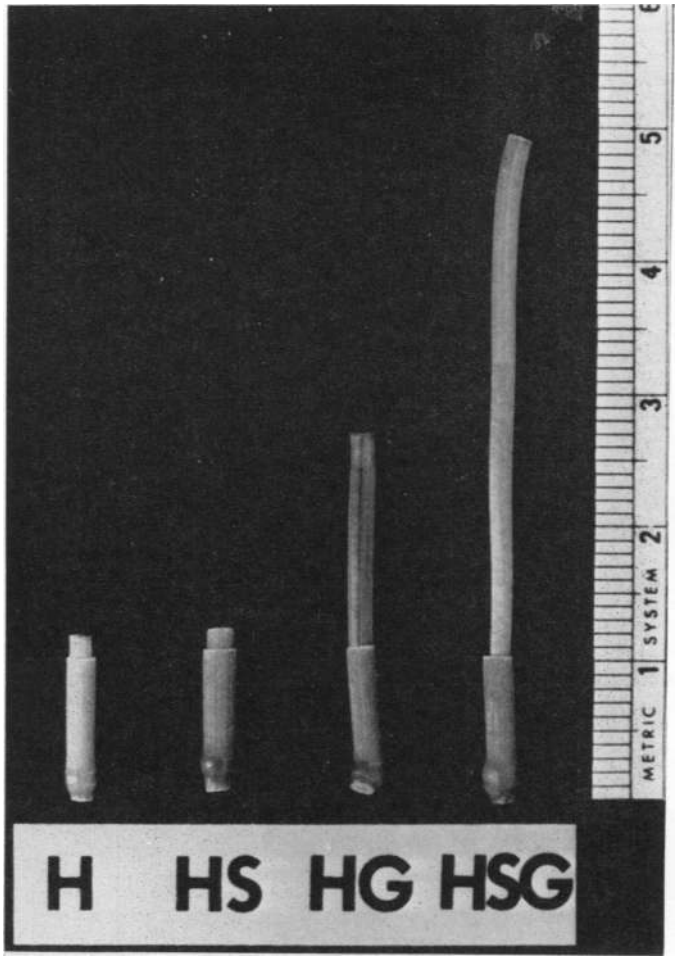

FIG. 2. Effect of GA and sucrose on the growth of 1 cm stem segments from greenhouse-grown plants. The segments are shown after 60 hr of treatment in Hoagland's solution (H), Hoagland's + 0.1 M sucrose (HS), Hoagland's + 30 μM GA (HG), and Hoagland's + sucrose + GA (HSG). A centimeter ruler denotes actual size.

The temperature of the segment was read on a galvanometer (G) connected to a thermocouple placed through the rubber stopper and immersed in the solution surrounding the segment. Unless otherwise stated, experiments were conducted under diffuse room light at 30 C.

Fresh and dry weight changes were determined for the growing (internode) and the nongrowing (node-sheath) portions separately. The segments grown in the routine manner were removed at prescribed times from the Petri dish, the internodes were separated from the sheaths and nodes by means of a razor blade, and fresh weight was measured for each part. Each part was then quickly frozen and lyophilized in a Thermovac freeze dryer, and dry weights were determined to 0.01 mg with a Mettler balance (Type M5).

The determination of solute concentrations in the internode was carried out with a freezing point depression method (28). The segments were removed from Petri dishes after a given period of incubation, and the internodes, freed from the nodes and sheaths, were immediately frozen in a test tube. The frozen internodes were then ground completely in the same test tube, which was then placed in a boiling water bath for 1 min to precipitate most proteins and then placed in an ice bath while the particulates settled out. About 0.2 ml of the clear supernatant was used to determine the solute concentration by means of the freezing point depression with a thermocouple connected to a microvoltmeter.

All inorganic chemicals and sucrose were of analytical reagent grade. Gibberellic acid was purchased from Nutritional Biochemicals Corporation. "Victory" oats were obtained from Allmanna Svenska Utsades A.B., Svalof, Sweden. Statistical analysis of the results were performed according to Edwards (11).

RESULTS

Macroanalysis of Growth. Figure 2 illustrates segments grown in the four standard treatments (see "Materials and Methods") for 60 hr. H and HS treatments without GA produced about 0.2 cm elongation in the segments. When GA was included in the medium, the segments grew 1 to 2 cm without sucrose (HG) and 3 to 4 cm with sucrose (HSG). The growth in HSG was greater than 15 times the growth in HS. A similar response was achieved when glucose or fructose at 0.2 M was substituted for the 0.1 M sucrose in the HSG medium (1). Thus, a marked growth response was induced by GA, especially in the presence of exogenous substrate.[3]

[3] It should be mentioned that in later experiments, it was observed that the growth response of segments is strongly influenced

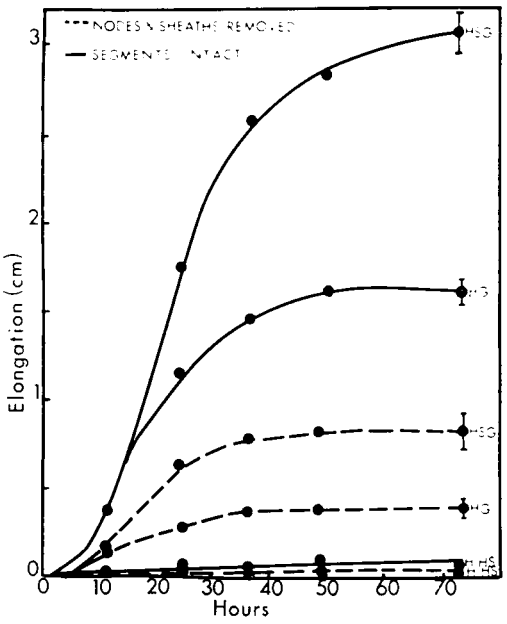

FIG. 3. Time course of internodal elongation in oat segments ± node-sheaths. Values represent means ± S.E. of 25 replicates.

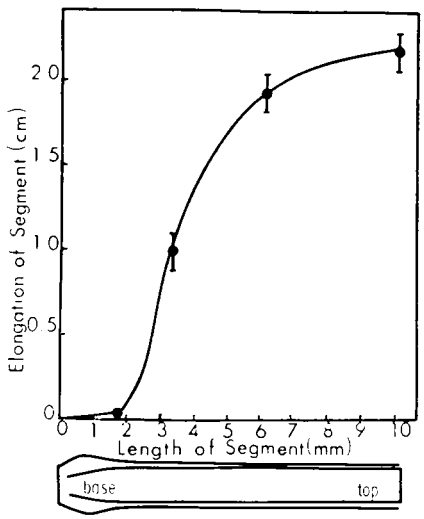

FIG. 4. Distribution of growth potential along the internode. Segments of varying lengths were grown in HSG for 60 hr at which time growth was measured. Basal 1.5 mm portion of segment represents the node. Values represent means ± S.E. of 10 replicates.

Although the oat segments comprised a simplified system compared with that of the whole plant, they still remained highly complex and diversified. The most obvious differentiation of tissues is the growing internode as compared with the nongrowing sheath and node. In order to determine the influence of the nongrowing portions on the internodal growth, the nodes and sheaths were removed prior to the start of the experiment, and the internodes were treated with the four test solutions. The results in Figure 3 clearly show that the growth of HG- and HSG-treated internodes was reduced to about 25% of the growth of intact segments. A growth reduction was even noted in those segments cultured without exogenous GA. Thus, the elongation of the segments was markedly influenced by the node and sheath, while the basic pattern of growth responses to the four treatments remained the same.

The distribution of the growth zone in the internode was determined by excising the segments at various distances from the base of the node and growing them in HSG for 60 hr. Figure 4 indicates that essentially all of the elongating cells are contained within the 1-cm segments. As expected, very little elongation occurred in the node alone, but more than 60% of the growth occurred between the 2nd and 4th mm from the base. Thus, the primary growing region for the segment was restricted to a rather small basally located portion of the internode. Similar results were reported earlier from this laboratory on the basis of cytological observations (18, 20).

The growth response of the segments to varying amounts of GA was determined in two ways. In the first method, GA was supplied to the bases of the segments (through the nodes) by a range of concentrations in the basal solution. Figure 5A shows that the segments responded to GA concentrations above 10 nM and reached a maximum above 1 μM. The half-maximal response was found at about 80 nM. A concentration of 30 μM GA was thus used in later experiments to ensure a maximal growth response.

In the second method, discrete quantities of GA were added apically to each segment in 3 μl droplets. Figure 5B indicates that a response was detected with amount of GA as low as 1 pmole; a half-maximal response occurred at 20 pmole; the maximal response occurred above 300 pmole. Somewhat different dose-response data were obtained in previous studies when GA was added apically via agar blocks (18, 19) and when the segments were floated horizontally in GA solutions (21).

Microanalysis of Growth and the Effect of Temperature. The rapid growth response to GA was studied with the microgrowth apparatus as described in "Materials and Methods." The data (Fig. 6) indicate that it took 35 to 40 min for the induction of growth by GA to occur; subsequently, the growth accelerated from a base growth rate of 200 μ/hr to 630 μ/hr at 115 min after GA addition.

A single segment was used with the microgrowth method to determine growth rates at several temperatures. In no case was a lag time detected in the temperature-induced changes in growth rate in either control or hormone-treated segments. The growth rates of typical HS- and HSG-treated segments were plotted against temperature (Fig. 7). As expected, the most obvious differences in the two curves was the higher growth rate in the presence of GA. Under these conditions the maximal growth rate in HSG treatment was about eight times that of HS treatment. In both instances this maximal growth rate was obtained at about 35 C.

The calculation of temperature coefficients (Q_{10} values) from these curves gave values of about 4.5 for HS growth and about 2.5 for HSG growth from 11 C to 25 C. In order to examine the difference in Q_{10} values further, 14 segments were treated with HS or HSG and subjected to altogether 50 different temperatures. These results showed the same trend: the Q_{10} values were lowered by GA treatment. An analysis of the regression coefficients on the Arrhenius plots indicated that the null hypothesis could be rejected at only the 5% level with the t test (1). If these results are indeed significant, they suggest that the most rate-limiting steps leading to growth are modified by GA.

Weight Analysis. The general mobilization and incorporation of substrate was examined by determining the effect of GA and

by the conditioning of the plants *prior* to experimentation. If the oats were grown from seed in a growth chamber (4000 ft-c, 16 hr day at 22 C, 8 hr night at 18 C) with daily treatment of Hoagland's solution, segments from 15 shoots elongated in 66 hr of treatment to 0.30 ± .05 cm in H, 0.35 ± .05 cm in HS, 2.50 ± .15 cm in HG, and 7.45 ± .25 cm in HSG. These results indicate a maximum growth response in HSG to be about double that of greenhouse-grown plants.

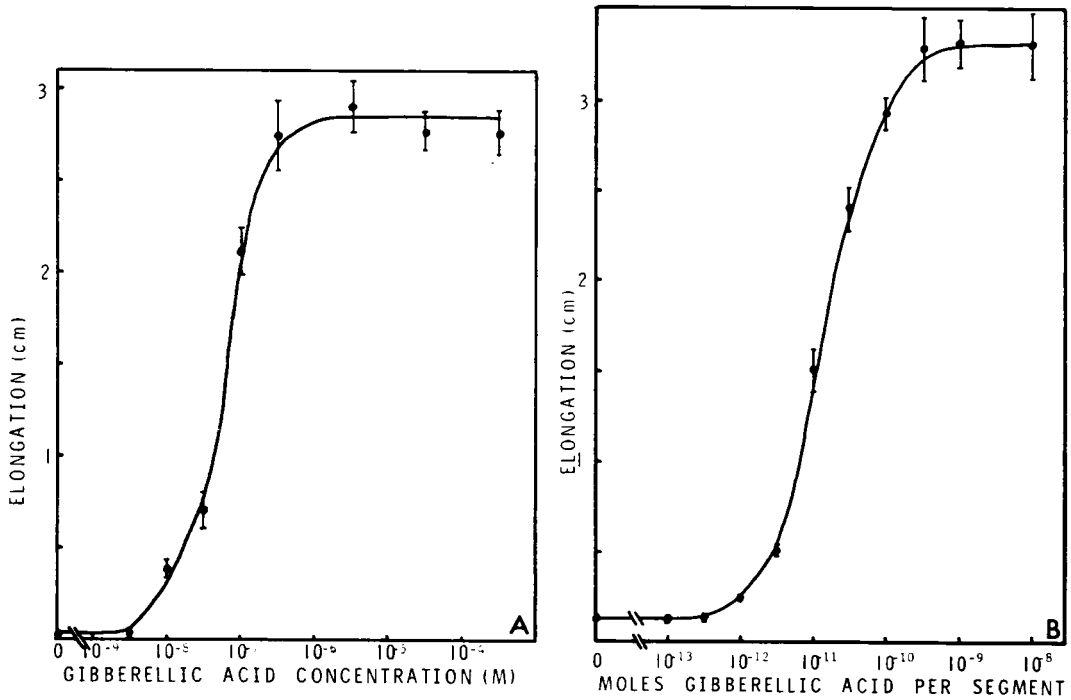

FIG. 5. A: Elongation of segments as a function of GA concentration. Segments were measured after 70 hr of treatment. All treatments included sucrose in Hoagland's solution. Values represent means ± S.E. of 15 to 20 replicates. B: Elongation of segments as a function of discrete amounts of GA added to individual segments. Segments were measured after 70 hr of treatment. All treatments included sucrose in Hoagland's solution. Values represent means ± S.E. of 15 replicates.

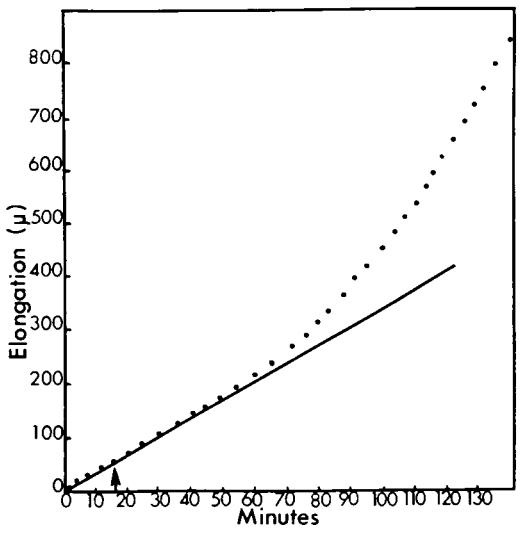

FIG. 6. Detailed kinetics of GA-induced growth. Arrow indicates the time of addition of 30 μM GA into HS solution in which the segment was pretreated.

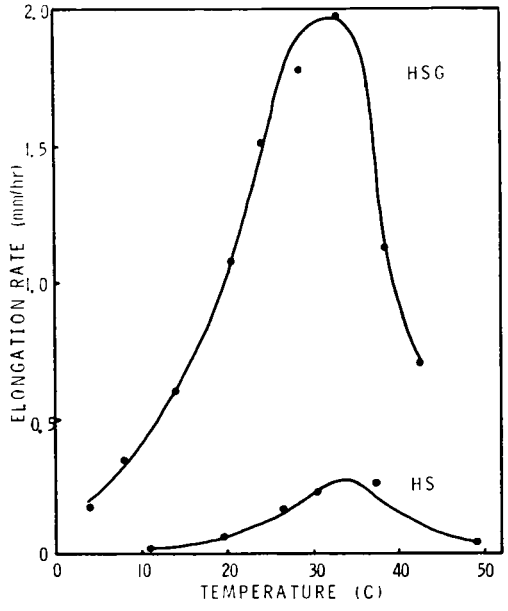

FIG. 7. Elongation rate of segments treated with HS and HSG as a function of temperature.

sucrose on the dry weights of segments grown in the routine procedure (see "Materials and Methods"). Sixty segments were treated with each of the four standard solutions, and 20 segments were removed after 10, 30, and 60 hr of growth, separated into internode and node-sheath parts, and each part freeze-dried. The weights are expressed in Figure 8 as per cent change with reference to the initial (t_0) fresh weight. In these experiments the absolute values of dry weight changes can be estimated from the following average dry weight of each segment at t_0: 0.8 mg/internode and 3.7 mg/node-sheath.

The data for total segments (Fig. 8A) reveal that dry weight changes differed significantly from the fresh weight changes apparent in Figure 3; while the dominant fresh weight changes took place in HSG and HG, the dominant dry weight changes took place in HSG and HS. It is apparent that the capacity of the segments to incorporate large quantities of exogenous substrate exists irrespective of the presence of GA. A dominant effect of GA treatment thus appears to be an increase in the uptake of water by the segments.

While the dry matter content in the total segment increased similarly for both HS and HSG treatments, a clear difference is seen in the two treatments when the dry weight levels in

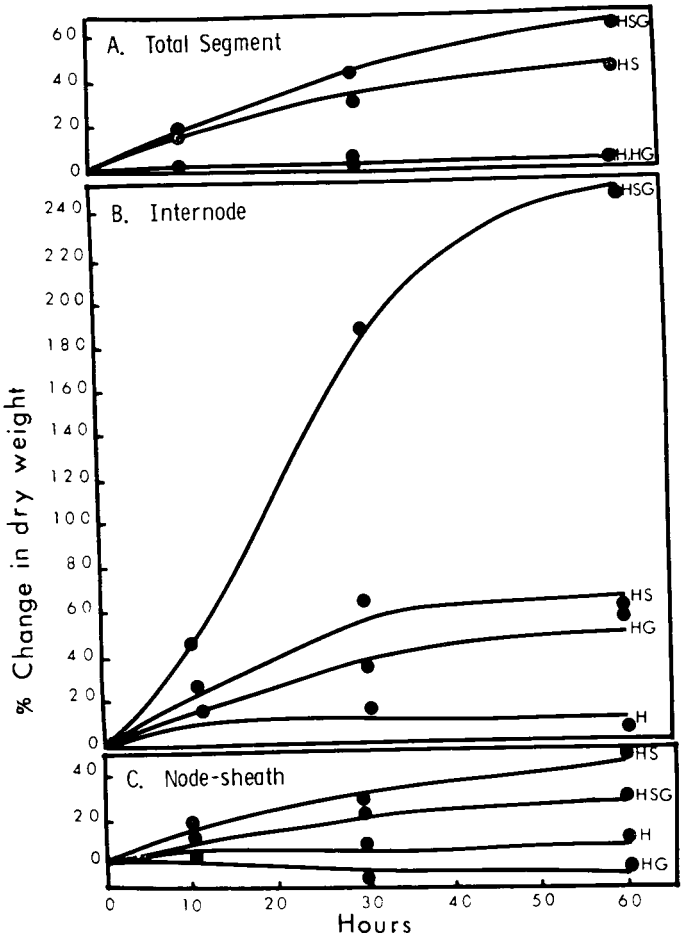

FIG. 8. Time course of dry weight changes in the oat segment upon treatment with H, HS, HG, and HSG. Each value represents 20 pooled segments.

internodes is compared with those in node-sheaths. Increases in internodal dry matter were about four times greater in HSG than in HS (Fig. 8B), and node-sheath dry matter increased about two times more in HS than in HSG (Fig. 8C). Since the dry weight of the node-sheath portion of the T_0 segment is 4.5 times greater than that of the internodal portion, the internodes of HS segments contain 30% and the node-sheath 70% of the total dry weight increase over the T_0 value. A similar calculation for the HSG segments reveals 80% for the internode and 20% for the node-sheath. Since the increase in dry weight should reflect the increased uptake of sucrose, these calculations show that GA causes a shift of the primary site of sucrose uptake from the node-sheath to the internode.

Although the total dry weight did not change appreciably during the experimental period when the segments were treated with H and HG (Fig. 8A), changes in the distribution of endogenous dry matter in these treatments can be seen in Figure 8B, C. With HG treatment the internodal dry weight increased, whereas the node-sheath dry weight decreased; *i.e.*, about 10% of the dry matter in the total segment at T_0 was lost from the node-sheath and reappeared in the internode. The H treatment caused a much smaller change in dry matter distribution. It can be concluded, therefore, that GA promotes growth of these segments not only by stimulating the uptake of exogenous substrates into the internode but also by mobilizing the substrates in the node-sheath to the internode.

From the previous data, it is clear that the uptake into the internode of both water and substrate is involved in its growth. The relative contribution of these is expressed in Figure 9 as percentage of change in the ratio of dry weight/fresh weight or "density" of the tissue grown in the four standard treatments. The internodes had a T_0 density of 0.082 which increased 30% in HS and decreased 50% in HG in 60 hr of growth. The internodes treated in H and HSG decreased only about 15%. These large differences are particularly interesting in the GA-treated tissue. Without an exogenous supply of substrate (HG treatment), the percentage of increase in water exceeded the percentage of increase in dry matter by nearly four times. With HSG treatment, however, the percentage of increase in internodal water content was only slightly greater than the percentage of increase in dry matter. Thus, while the tissue had the capacity to decrease its density greatly, the density remained quite constant with GA treatment if substrate was available.

The relation of water and substrate uptake was further analyzed by determining the osmotic activity of the internodal cells of segments grown in the four standard treatments (Fig. 10). The tissue solute concentration of untreated internodes was about 0.26 M. The two treatments in the absence of GA caused an increase in osmolarity; H-treated tissues increased to slightly above 0.3 M, and HS-treated tissues increased to nearly 0.5 M. HSG treatment may induce an initial increase in osmolarity before the major growth response occurs. However, in both tissues treated with GA, the osmolarity ultimately decreased to about 0.2 M. Thus, the tissues responding to GA had less solute than the controls, which indicates that the mechanism of GA-induced growth apparently does not directly involve an increase in osmotic pressure.

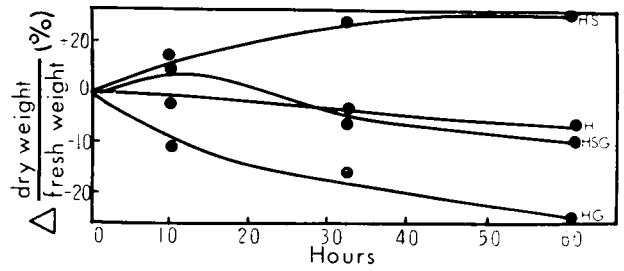

FIG. 9. Time course changes in the density ratio (dry weight/fresh weight) in the internodes of oat segments treated with H, HS, HG, and HSG. This figure was compiled from data presented in Figure 8B plus the fresh weight data of the same segments.

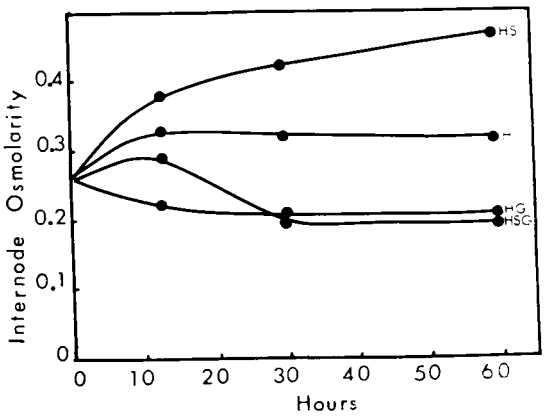

FIG. 10. Time course changes in internodal osmolarity of segments treated with H, HS, HG, and HSG. The data represent the combined results of four separate experiments, each of which showed the same trend.

DISCUSSION

In *Avena* stem segments, growth was stimulated by GA and this growth was further enhanced by exogenous substrate (Figs. 2 and 3). Exogenous substrate did not, however, markedly promote elongation when supplied without GA (Figs. 2 and 3), even though it was taken up to a considerable degree (Fig. 8) and was thereafter both respired and incorporated into cell walls (1). Broughton and McComb (8) reported that exogenous glucose stimulated growth in pea internodes to an extent similar to the promotion by exogenous GA. They proposed that GA acts by causing enzymatic changes favoring the production of substrates for general metabolism and cell wall synthesis (7, 8). It is clear from our data that the hormone does not induce growth in oat stem segments by increasing the level of endogenous carbohydrates for growth metabolism.

When exogenous substrate is present, the growth of *Avena* stem segments treated with GA is greater than 15 times that of control growth (Figs. 2, 3, and 5). The major gibberellin bioassays, such as the dwarf pea test (4), the dwarf corn test (27), the lettuce hypocotyl test (12), and the cucumber hypocotyl test (6) all show growth responses less than five times that of the control, which is true of practically all intact plants studied. An exceptionally large response is found, however, in the case of GA-induced bolting (24).

If one compares the growth of oat stem segments with those of other isolated plant parts, the differences are even more striking. Brian and Hemming (5) reported that GA caused a 3-fold increase in growth when applied to intact dwarf pea plants, but there was no increase when GA was supplied to isolated stem sections of the same plant. Other workers (14, 16, 30, 31) have noted only small responses of isolated plant parts to gibberellins. Purves and Hillman (29) were able to improve the response of pea stem segments to the hormone by using more apical internodes, but even here, the growth response was less than two times the control level. The growth response of *Avena* stem segments is almost certainly the largest ever reported for GA effect on the growth of isolated plant tissues.

Besides the large response to GA, *Avena* stem segments have other desirable characteristics for gibberellin studies. They have a high sensitivity to GA, responding to solution concentrations as low as 10 nM (Fig. 5A) and to discrete amounts as low as 1 pmole (Fig. 5B). In addition, the segments are highly insensitive to auxin and kinetin in the elongation response (15, 19), whereas gibberellins A_1, A_3, A_4, A_5, and A_9 induce a marked growth response in them (19). The magnitude of the growth response, the sensitivity of the tissue to small amounts of the hormone, and the specificity to the gibberellin class of hormones indicate that the *Avena* segment is ideal material for use as a bioassay of gibberellin-like substances in plant extracts. (See ref. 2 for a review of current bioassay systems.)

The response to GA in about 35 min was obtained frequently with the microgrowth procedure. This approaches the 24-min response recently reported by Warner and Leopold (33). Included in *Avena* response time is the transport of the hormone through the dense non-growing node, which may represent a significant time component. Under special conditions to minimize the transport time, preliminary indications suggest that the induction time in *Avena* can be reduced appreciably.

The temperature studies cited here provide some basic insights on the nature of the *Avena* growth response. Figure 7 indicates that both HS- and HSG-treated tissues respond greatly to temperature changes in both directions. These data, coupled with the fact that the Q_{10} of growth is about 2 or higher, indicate that the growth of both tissues is limited metabolically and that the growth-inducing process must be viewed within a metabolic framework.

In this study GA was shown to have a marked influence on both fresh and dry weights of *Avena* stem segments. The dominant effect of GA was a linear extension of the internodal tissue which was the result of a large increase in the uptake of water. This uptake could greatly exceed the relative assimilation rate of dry matter into the internode. However, it is also clear that an increase in dry matter of the growing region was very important in the elongation response. This was apparent in HG-treated segments, in which a large mobilization of materials occurred from the node-sheath into the internode (Fig. 8) and in which a pronounced limitation of growth occurred because of the eventual depletion of endogenous substrates (Fig. 3). Therefore, both the uptake of water and the requirement for substrate were clearly involved in GA-induced growth, but the relative contribution of these to the induction of the elongation response by the hormone remains unanswered.

One physiological mechanism of growth induction which could relate to the requirement for substrate has apparently been eliminated by this study: namely, an increase in osmotic pressure of the internodal cells. This has been suggested as a possible mechanism of hormone-induced growth by a number of workers (9, 10, 32). This idea has been supported circumstantially by observations that stem elongation is accompanied by massive hydrolysis of storage products in some tissues (23, 26). Most of the actual measurements of tissue solute, however, show that the expanding tissue declines in osmolar concentration (13, 22, 34). This was also true here in elongating internodes of *Avena* stem segments. Not only did the osmotic concentration decrease in the GA treatment, but it also increased in the control tissue. The presumed relationship was thus reversed: an increase in the solute concentration in internodal cells did not induce growth, and a decrease in solute concentration did not prevent the GA-induced growth.

The importance of the node-sheath in the internodal growth was clearly established in this study, but its specific role remains to be examined further. Although the node-sheath has essentially no elongation potential itself (Figs. 2 and 4), it increases the potential of the internode to elongate by fourfold (Fig. 3). This enhancement of internodal growth probably involves the mobilization and transport of basic substrates (see Fig. 8). However, since exogenous sucrose alone cannot replace the effect of the node-sheath (Fig. 3), it is likely that this mobilization and transport phenomenon elicited by GA involves more than sugar alone.

LITERATURE CITED

1. Adams, P. A. 1969. Studies on gibberellic acid-induced growth in *Avena* stem segments. Ph.D. thesis. University of Michigan, Ann Arbor.
2. Bailiss, K. W. and T. A. Hill. 1971. Biological assays for gibberellins. Bot. Rev. 37: 437-479.
3. Bonner, J. and A. W. Galston. 1952. Principles of Plant Physiology. W. H. Freeman and Co., San Francisco.
4. Brian, P. W. and H. G. Hemming. 1955. The effect of gibberellic acid on shoot growth of pea seedlings. Physiol. Plant. 8: 669-681.
5. Brian, P. W. and H. G. Hemming. 1958. Complementary action of gibberellic acid and auxins in pea internode extension. Ann. Bot. (N. S.) 22: 1-17.
6. Brian, P. W. and H. G. Hemming. 1961. Promotion of cucumber hypocotyl growth by two new gibberellins. Nature 189: 74.
7. Broughton, W. J., E. O. Hellmuth, and D. Yeung. 1970. Role of glucose in development of the gibberellin response in peas. Biochim. Biophys. Acta 222: 491-500.
8. Broughton, W. J. and A. J. McComb. 1971. Changes in the pattern of enzyme development in gibberellin-treated pea internodes. Ann. Bot. 35: 213-228.
9. Cleland, R., M. Thompson, D. L. Rayle, and W. K. Purves. 1968. Difference in effects of gibberellins and auxins on wall extensibility of cucumber hypocotyls. Nature 219: 510-511.
10. Czaja, A. T. 1935. Wurzelwachstum, Wuchstoff und die Theorie der Wuchsstoffwirkung. Ber. Deut. Bot. Ges. 53: 221-245.

11. EDWARDS, A. L. 1954. Statistical Methods for the Behavioral Sciences. Rinehart, New York.
12. FRANKLAND, B. AND P. F. WAREING. 1960. Effect of gibberellic acid on hypocotyl growth of lettuce seedlings. Nature 185: 255–256.
13. HACKETT, D. P. 1952. The osmotic change during auxin-induced water uptake by potato tissue. Plant Physiol. 27: 279–284.
14. HAYASHI, T. AND Y. MURAKAMI. 1953. The biochemistry of the bakanae fungus. 29. The physiological action of gibberellin. V. The effect of gibberellin on the straight growth of etiolated pea epicotyl sections. J. Agr. Chem. Soc. Japan 27: 675–680.
15. JONES, R. A. AND P. B. KAUFMAN. 1971. Regulation of growth in *Avena* stem segments by gibberellic acid and kinetin. Physiol. Plant. 24: 491–497.
16. KATO, J. 1956. Effect of gibberellin on elongation, water uptake, and respiration of pea-stem sections. Science 123: 1132.
17. KAUFMAN, P. B. 1965. The effects of growth substances on intercalary growth and cellular differentiation in developing internodes of *Avena sativa*. I. The effects of indole-3-acetic acid. Physiol. Plant. 18: 424–443.
18. KAUFMAN, P. B. 1965. The effects of growth substances on intercalary growth and cellular differentiation in developing internodes of *Avena sativa*. II. The effects of gibberellic acid. Physiol. Plant. 18: 703–724.
19. KAUFMAN, P. B. 1967. Role of gibberellins in the control of intercalary growth and cellular differentiation in developing *Avena* internodes. Ann. New York Acad. Sci. 144: 191–203.
20. KAUFMAN, P. B., S. J. CASSELL, AND P. A. ADAMS. 1965. On the nature of intercalary growth and cellular differentiation in internodes of *Avena sativa*. Bot. Gaz. 126: 1–13.
21. KAUFMAN, P. B., N. GHOSHEH, AND H. IKUMA. 1968. Promotion of growth and invertase activity by gibberellic acid in developing *Avena* internodes. Plant Physiol. 43: 29–34.
22. KETELLAPPER, H. J. 1953. The mechanism of the action of indole-3-acetic acid on the water absorption by *Avena* coleoptile sections. Acta Bot. Néerl. 2: 387–444.
23. KOMISSAROW, D. A. 1961. The effect of gibberellic acid on arboraceous plants. Dokl. Akad. Nauk. USSR 136: 1241–1244.
24. LANG, A. 1956. Stem elongation in a rosette plant, induced by gibberellic acid. Naturwissenschaften 43: 257–258.
25. LOCKHART, J. A. 1965. Cell extension. *In:* J. Bonner and J. E. Varner, eds., Plant Biochemistry. Academic Press, New York. pp. 826–849.
26. NANDA, K. K. AND A. N. PUROHIT. 1965. Effect of gibberellin on mobilization of reserve food and its co-relation with extension growth. Planta 66: 121–125.
27. PHINNEY, B. O. 1961. Dwarfing genes in *Zea mays* and their relation to the gibberellins. *In:* Plant Growth Regulation. Iowa State University Press, Ames. pp. 489–501.
28. PROSSER, C. L. ed. 1950. Comparative Animal Physiology. W. B. Saunders and Co., Philadelphia.
29. PURVES, W. K. AND W. S. HILLMAN. 1958. Response of pea stem sections to indoleacetic acid, gibberellic acid, and sucrose as affected by length and distance from apex. Physiol. Plant. 11: 29–35.
30. RADLEY, M. 1958. The distribution of substances similar to gibberellic acid in higher plants. Ann. Bot. 22: 297–307.
31. VAN OVERBEEK, J. AND L. DOWDING. 1961. Inhibition of gibberellin action by auxin. *In:* Plant Growth Regulation. Iowa State University Press, Ames. pp. 657–663.
32. VAN OVERBEEK, J. 1966. Plant hormones and regulators. Science 152: 721–731.
33. WARNER, H. L. AND A. C. LEOPOLD. 1971. Timing of growth regulator responses in peas. Biochem. Biophys. Res. Comms. 44: 989–994.
34. YODA, S. 1961. Effect of auxin and gibberellin on osmotic value of pea stem sections. Plant Cell Physiol. 2: 435–441.