use of a conversion factor is insisted upon, then carbon, not nitrogen, should be used as an indicator.

The amount of carbon or nitrogen present in the organic matter of sediments is dependent upon numerous factors. Some of the more obvious are (i) the carbon or nitrogen content of the original supply of organic detritus, (ii) the ratio of the various organic compounds or groups, (iii) the carbon or nitrogen content of the various organic compounds or groups present in the sediments, (iv) the amount and type of decomposition that has affected the organic matter during and following deposition, and (v) the degree of resistance that various organic compounds or groups display. All of these factors will vary according to the environmental conditions existing or preexistent in the area sampled. It is thus quite possible that n number of investigations, undertaken to determine a single constant for converting organic carbon or nitrogen to total sedimentary organics, will produce approximately n number of constants.

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Effect of 2,4-D on Respiration and on Destruction of IAA in Oat and Sunflower Tissues*

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It has been established that the naturally occurring hormone or auxin, indolacetic acid (IAA), and 2,4-dichlorophenoxyacetic acid (2,4-D) stimulate certain general respiratory responses in *in vitro* experiments with plants (*1, 2*). Miller and Burris (*2*) showed in addition that for pea sections, concomitant with the increase of respiration caused by the presence of IAA, there occurred a gradual disappearance of IAA from the medium, which was accompanied by an equivalent decline in respiratory rate. Later, Miller and Henderson (*3*), reported the identical situation for oat coleoptile sections. The disappearance of IAA finds credence in the work of Tang and Bonner (*4*) and Wagenknecht and Burris (*5*).

Experiments with Avena *coleoptile sections.* By use of the direct method of Warburg, results with varying concentrations of 2,4-D and IAA indicate that both substances increase proportionately the oxygen uptake of *Avena* coleoptile sections, but that in the case of IAA there is a sharp decline in the rate after the fourth or fifth hour (Fig. 1); the same response does not occur with 2,4-D. Figure 1 represents a typical determination in which oxygen uptake of *Avena* sections was measured in the presence of IAA and 2,4-D, singly and combined. The broken curve shows the theoretical additive oxygen uptake that should be expected by combination of the two substances. It is easy to observe that this is not the case. Indeed, after 6 hr the oxygen uptake for the two combined (upper solid curve; 100 ppm IAA and 500 ppm 2,4-D) showed a continued rise at a very rapid rate, thus revealing a "synergistic effect." At this time the oxygen uptake by IAA alone had dropped to the endogenous rate.

Figure 2 shows the effects of varying concentrations

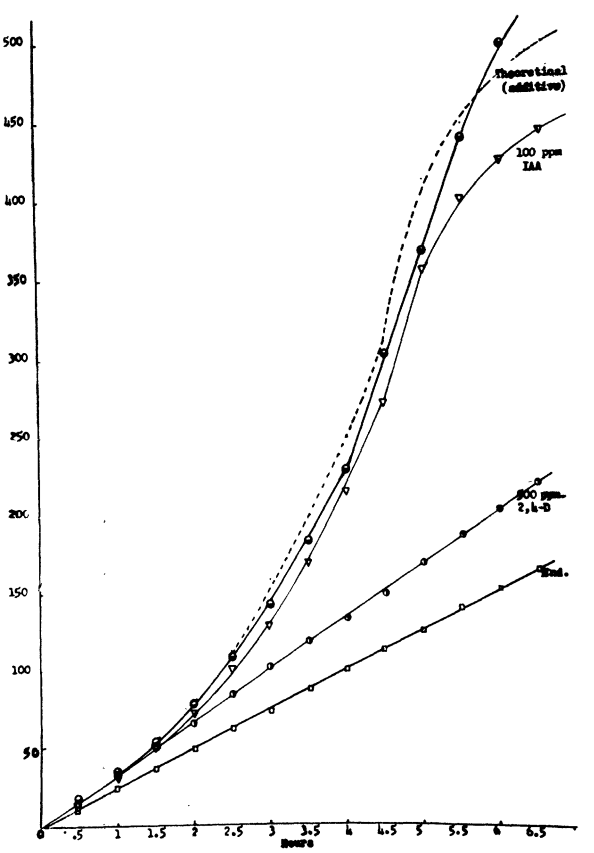

Fig. 1. Respiration of oat sections with IAA and 2,4-D. Abscissa: time in hours. Ordinate: μlit O_2 uptake. The three lower solid curves represent: endogenous; 500 ppm 2,4-D; and 100 ppm IAA, from bottom to top, respectively.

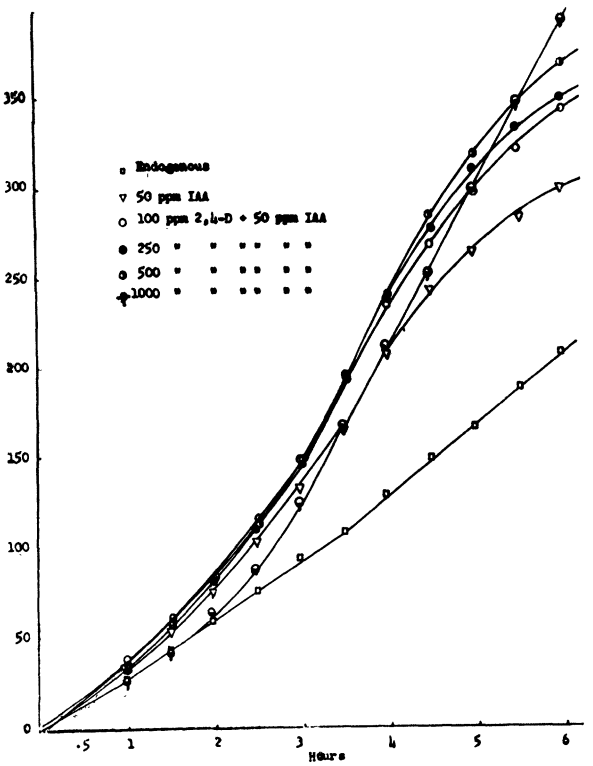

Fig. 2. Same as Fig. 1. At the sixth hour the curves read as follows (from bottom to top): endogenous; 50 ppm IAA; then, 50 ppm IAA + 100, 250, 500, and 1000 ppm 2,4-D, respectively.

of 2,4-D in combination with 50 ppm IAA. A gradual increase in the total oxygen uptake with increase in concentration of 2,4-D may be noted. Analyses of the mediums indicated that this increase in oxygen uptake was accompanied by a gradually declining rate of IAA destruction with increasing concentrations of 2,4-D. Larsen (*6*) reported previously that *Avena* sections destroy IAA.

To test further the latter phenomenon (auxin inactivation), experiments were designed to measure proportionately on a macro scale the disappearance of IAA in the presence and absence of 2,4-D. The observations are summarized in Table 1. It is shown that IAA destruction is prolonged with increasing concentrations of 2,4-D (*7*). These results indicate that 2,4-D influences the rate of disappearance of IAA and that the increased respiratory response resulted from the sparing action of IAA by 2,4-D in higher concentrations (*8, 9*).

Table 1. Time in hours to cause complete destruction of IAA (50 mg/lit) in 120 sections of 3 mm length (total volume of medium 12 ml). Phosphate buffer, *p*H 6.5. Continuous shaking at 110 oscillations/min at 30°C.

Species	Concentration of 2,4-D			
	0	250	500	1000
Oat	4.5	5.0	6.0	> 9.0
Pea	6.0	4.5	4.5	6.0*
Sunflower	4.0	3.5	3.25	4.0*

* Destruction possibly inhibited by disorganization of the tissues in the sections owing to high concentration of 2,4-D.

Experiments with sunflower sections. Several workers (*10, 11*) have reported that 2,4-D enhances the destruction of IAA *in vitro*. This is the opposite response to the one just reported and may lend credence to a species difference based on the influence of 2,4-D on the naturally occurring IAA-oxidase system that destroys added IAA *in vitro*. (*In vivo* results will be reported separately.)

Sunflower hypocotyl sections showed an increase in oxygen uptake, similar to that of oat sections, with increased concentration of IAA. At the lower concentrations of IAA the oxygen uptake reached a plateau; at higher concentration an increase in rate is still evident after 6 hr. This suggests that in the case of the former, possibly the IAA is being destroyed by the sections. Indeed, when analyses were made for this destruction at the 50-ppm level, it was found that the IAA had been destroyed by the fourth hour, causing the decline in respiratory rate (Table 1).

When 2,4-D and IAA were added together to sunflower sections, instead of the respiratory response being greater than additive, it was actually less. For example, at the end of 6 hr of respiration, 50 ppm IAA showed 24-percent increase over control, 500 ppm 2,4-D gave 15 percent, and the two combined gave 29 percent. The additive response would be 39 percent. This would seem to indicate that no sparing action by 2,4-D of IAA exists in sunflower sections (lack of "synergistic effect"). Thus, the possibility exists that an increased destruction of IAA occurs in sunflower sections, in contrast to a sparing effect in the oat sections, as a result of presence of certain concentrations of 2,4-D.

Indeed, when the analyses were made for the disappearance of IAA, this proved to be the case for both sunflower and pea sections (Table 1). At concentrations of 250 to 1000 ppm of 2,4-D, IAA at 50 ppm is destroyed as rapidly—and in some cases more rapidly—than in the control containing no 2,4-D.

Thus, it appears that there is a possible difference in the reaction involving 2,4-D and IAA (via the IAA-oxidase system) in monocots and dicots and that this difference may be the basis for the selective nature of certain growth regulators or "weed killers," notably 2,4-D.

References and Notes

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8. This response, although not obtained from enzyme breis preps., would seem to obviate the findings of Goldacre *et al.* (*9*) that the presence of 2,4-dichlorophenol in samples of 2,4-D is largely, if not solely, responsible for the enhanced inactivation *in vitro*. Further, analysis of the sample of 2,4-D used in the present investigations showed less than $3 \times 10^{-6} M$ DCP, as given by the Folin-Ciocalteau reagent.
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Production of Dissecting Aneurysms in Rats Fed *Lathyrus odoratus*

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A toxic factor in sweet peas (*Lathyrus odoratus*) has been shown to exert pronounced effects on the skeletal development of growing rats (*1–5*). The first to record any alteration in the aorta were Ponseti and Baird (*5*), who described aortic aneurysms. Because of this effect which the *Lathyrus* diet apparently exerts on the integrity of the aorta, it seemed important to study further the factors responsible for the arterial changes.

In the initial phase of our investigation (*6*), extracts of sweet pea meal were assayed for toxicity in rats. The casein concentration in the diets that contained the extracts was either 10 or 20 percent. After a crystalline toxic factor had been isolated by E. D. Schilling and F. M. Strong, it was characterized as B-(γ-L-glutamyl) aminopropiononitrile and subsequently synthesized by these workers (*7*). The assays indicated that the crystalline factor from pea meal and the synthetic compound were just as effective in producing skeletal deformities as was crude pea meal (*8*). However, we observed aneurysms in only three of 60 test rats, a much lower percentage than reported by Ponseti and Baird.

Examination of the diets employed suggested that the casein in the basic ration might have exerted an inhibitory effect on the production of arterial lesions in rats fed preparations that contained the *Lathyrus* factor. It was observed that aneurysms developed only in those animals fed a diet with the casein at a 10-percent level. No aneurysms developed when the casein level was 20 percent. Diets in this study were prepared, therefore, with 10-percent casein.

Seventeen control rats were fed aqueous alcohol-extracted pea meal, and 28 test rats were given crude pea meal. The *test diet* follows: 10 percent Borden's crude casein; 10 percent Pabst Brewer's yeast; 50 percent ground pea meal (*9*); 24 percent cerelose; 4 percent Wesson salt; 2 percent olive oil containing 0.21 mg of vitamin-A acetate, 0.26 IU of vitamin D, 10 mg of a-tocopherol, and 0.15 mg of 2-methyl-1-4 naphthoquinone per kilogram of diet. The control diets were similar in all aspects except that aqueous alcohol-extracted pea meal was substituted for the crude material.

Table 1. Effect of feeding crude pea meal on the development of aortic aneurysms. M, male; F, female.

No. and sex	Days on diet	Cause of death	Starting weight (g)	Autopsy weight (g)
Control: aqueous alcohol-extracted sweet pea meal				
126M	40	Killed	41.0	135.5
127M	36	Killed	41.8	134.5
128F	100	Killed	59.1	162.0
129F	100	Killed	54.1	184.0
145M	91	Killed	41.4	163.4
146M	91	Killed	46.5	209.0
147M	49	Died, cause not established	48.6	
148F	32	Killed	50.4	128.5
149M	61	Killed	42.6	143.3
150F	28	Killed	48.3	125.0
Test: crude sweet pea meal				
122M	35	Ruptured aorta and hemothorax	40.8	105.0
124M	37		39.9	119.0
125M	60	Paralysis and malnutrition	36.0	68.0
130F	62	Ruptured aorta and hemothorax	59.0	135.0
135M	68		60.3	169.4
136M	43		59.4	148.0
140M	27		59.0	125.7
141M	48		47.1	95.0
144F	31		51.7	125.0
154F	48		42.3	75.1

The data in the table show the initial weights, days on the diet, weight gains, and cause of death in 20 of the 45 animals that were studied. Neither bony deformities nor ruptured aortic aneurysms occurred in any of the 17 control rats. In 28 test rats, 14 animals died of aortic rupture and massive hemothorax. Two other animals, which died of malnutrition and upper respiratory infection, also had aortic aneurysms. Three test rats were killed, one of which had an aortic aneurysm. Two others died after developing hernias; two others, following a hemorrhage into the urinary bladder and a fractured leg. Five test rats were placed on commercial pellets and are recuperating from malnutrition.

Practically all of the dissections occurred along the arch of the aorta. All of the test rats developed moderate to marked bony deformities. Partial paralysis of the hind limbs, secondary to deformities of the vertebral column, occurred in less than 20 percent of the animals. Microscopic examination of formalin-fixed tissues revealed degeneration and fragmentation of the elastic connective tissue fibers. In addition, there