proximal to the abscission zone, abscission was accelerated. The acceleration followed application of droplets containing 10, 105, and 525 mg IAA/l, the degree of acceleration varying only slightly with the concentration. Fig. 2 shows (*a*) the course of abscission in untreated explants, (*b*) the course (acceleration) that followed the application of IAA proximal to the abscission zone, and (*c*) the course (retardation) that followed the application of IAA distal to the abscission zone.

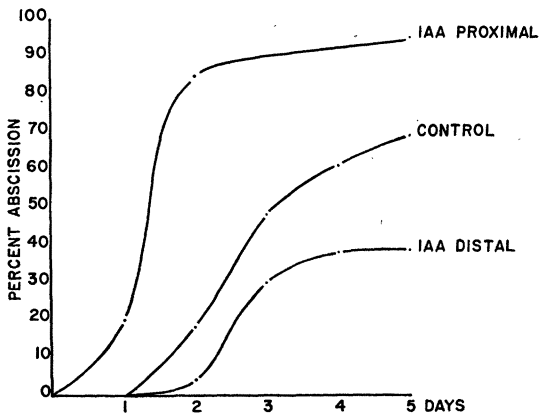

FIG. 2. Effect of the site of application of IAA on abscission in excised abscission zones of beans: Application proximal to the abscission zone, accelerated abscission; application distal to the abscission zone, retarded abscission. The control curve was obtained from data on the abscission of several hundred explants following the application of water distal to the abscission zone. The curves of proximal and distal application of IAA were obtained from 40 and 160 explants, respectively, and 105 mg/l was used in the experiments.

When the application of IAA was simultaneously distal and proximal to the abscission zone of the explant, the effect was identical with the effect of distal application only. When a distal application of 105 mg/l was made, abscission was retarded to the same extent, whether it was accompanied by a proximal application of zero concentration (water), of equal concentration, or of a concentration 5 times as high (525 mg/l). When a distal application of a concentration of 525 mg/l was made, abscission was inhibited, whether it was accompanied by a proximal application of zero concentration, of a weak concentration, or of an equal concentration.

A related experiment was conducted by Swets (*5*) in the greenhouse. He applied IAA-lanolin to the surface of the stalk proximal to the leaflet abscission zone of intact and debladed leaves. In the intact leaves he found abscission retarded; in the debladed leaves it was accelerated. Swets suggested that the retardation of abscission in the intact leaves was due to the absorption of IAA and its movement in the transpiration stream to the leaflet blade. He offered no explanation for the acceleration of abscission in the debladed leaves.

The results here reported comprise: (1) a confirmation of the ability of IAA to retard abscission, (2) a demonstration that the extent of this retardation is positively correlated with the concentration of the applied IAA, (3) a demonstration of the ability of IAA to accelerate abscission, and (4) a demonstration that the effect of IAA is dependent, at least in part, on the site of its application. This indicates physiological polarity of the leaf stalk in the vicinity of the abscission zone.

There is considerable evidence that a high concentration of IAA in a tissue results in the mobilization of physiologically active compounds in that tissue. Much of the evidence is indirect, but certain experiments of Went's (*6*) offer direct support. With this function of IAA in mind, the results here reported suggest that the mobilization of carbohydrates and other substances in the pulvinus, distal but close to the abscission zone, tends to keep the latter healthy and intact, whereas the mobilization of carbohydrates and other substances in the stalk, proximal to and several mm distant from the abscission zone, tends to bring about its degeneration and ultimate separation. These ideas are being investigated in histological and physiological studies. The results, with the details of the experiments here summarized, will be reported in a later communication.

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The Inhibition by Furacin of Adaptive Enzyme Formation in *Mycobacterium butyricum*

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Mycobacterium butyricum, like certain other mycobacteria (*1*), oxidizes benzoic acid by the formation of an adaptive enzyme. When the compound is added to a washed suspension of the cells in the Warburg vessel, the oxidation proceeds slowly for 30–45 min. After this the rate increases until the amount of oxygen required for complete combustion is taken up. If the cells are preincubated with a small amount of benzoate, the latent period—i.e., the period during which the adaptive enzyme is being formed—is greatly reduced. The extent of this reduction is a function of the amount of benzoate used for adaptation and the length of time employed.

Eadie, Bernheim, and Fitzgerald (*2*) have studied

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TABLE 1*

Minutes	Adapted with 10 γ benzoate					Adapted with 20 γ benzoate					Adapted with 30 γ benzoate				
	No drug	1:50	1:100	1:150	1:300	No drug	1:50	1:100	1:150	1:300	No drug	1:50	1:100	1:150	1:300
60	33	6	17	23	29	137	66	93	102	124	183	57	139	139	161
90	26	3	8	15	21	71	31	44	58	62	107	36	73	83	93
120	6	−2	0	1	2	8	−2	1	3	2	54	16	35	41	45

* The percentage increases in the rates of oxidation of 5 μm of sodium benzoate by washed cells of *M. butyricum* at indicated intervals after adaptation to various concentrations of benzoate, with and without Furacin, as compared with non-adapted, drug-free cells (drug values = concentration of Furacin × 10^{-3}).

the kinetics of this process, but almost nothing is known concerning the mechanisms involved. To gain some insight into the various metabolic factors of the organism involved in this process, a series of highly specific enzyme inhibitors would be desirable. Thus, by testing the effect of each inhibitor, one could determine which is effective in delaying or stopping the process altogether.

Furacin[2] (5-nitro-2-furaldehyde semicarbazone) has recently been shown to inhibit many dehydrogenases, particularly those involved in carbohydrate metabolism (*3*). Other bacterial enzymes such as esterases, proteases, catalase, transaminase, and amino acid decarboxylase were only slightly if at all affected. The inhibition does not appear to be a drug-substrate competition, since increasing amounts of substrate do not alter the inhibition produced by a given amount of the drug. The purpose of this work was to study the effect of Furacin on the formation of the benzoate oxidase by *M. butyricum*.

The organism used was obtained from the Department of Bacteriology at Duke Hospital. It was grown in Long's synthetic medium for 3 days at 37.5° C, using 25.0 ml medium in 250-ml Erlenmeyer flasks. To prepare a suspension of the cells, the contents of each culture flask were placed in two 20 × 100-mm test tubes and centrifuged at 2,000 rpm for 15 min. Following this, the supernatant fluid was decanted, distilled water was added, and the cells were stirred vigorously with a small glass stirring rod. The contents of each test tube were then divided equally between two Hopkins tubes. Afterwards, the cells were centrifuged again for 10 min, and decanted. Buffer (0.2 *M* KH_2PO_4 with 0.2 *M* NaOH; pH 6.8) was then added, and the contents of each tube were stirred again. After a final centrifuging for 10 min, the liquid was decanted, and fresh buffer added so that 0.05 ml of packed cells was suspended in 1.0 ml. One ml of this suspension was used in each Warburg vessel, which had a fluid volume of 3.2 ml.

Cells were adapted to benzoate by incubation with 10, 20, or 30 γ, added to the main compartments of the vessels, for 45 min. Various concentrations of Furacin were also added to this compartment during the adaptation period, as shown in Table 1. Controls contained neither Furacin nor benzoate during the incubation period. At the end of this time, benzoate (5.0 μm/vessel) was added from the side arm, and the oxygen uptakes were measured.

Table 1 shows the percentage increases in O_2 uptakes as compared with controls for each set of conditions at given times. When the cells were incubated with 10 γ, the degree of adaptation was less, and the percentage inhibition by Furacin more. The highest concentration of drug gave the greatest percentage of inhibition in all cases. It will be noted that when the cells were incubated with 20 γ benzoate, the percentage increase in O_2 uptake at 60 min was 137%, and 66% with 1:50,000 Furacin. When 30 γ was employed, the percentage increase in the vessel without the drug was 183% and 57% with 1:50,000 Furacin. This is in accord with the findings of Asnis and Gots that the inhibition does not appear to be a drug-substrate competition.

[2] Thanks are due to the Eaton Laboratories for supplying a generous sample of Furacin.

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The β-Glucuronidase Activity of Chemically Induced Rat Hepatoma

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Evidence has been produced by Fishman and his colleagues (*1, 2*) which indicates that in many cases cancer tissue has a higher β-glucuronidase activity than the normal tissue of origin, and it was suggested on the basis of these findings that an elevated β-glucuronidase activity may be characteristic of malignant tumors. These findings have also been taken as support for the hypothesis of Levvy and his colleagues (*3*), who have postulated a relationship between the β-glucuronidase activity of a tissue and the degree of cellular proliferation in that tissue.

One great difficulty in the interpretation of work on tumors is that of obtaining adequate controls of a