Fig. 1 shows the striking inhibitory effect on the cytochrome oxidase activity of the cytoplasmic supernatant of unfertilized eggs, whereas in that prepared from eggs collected 10 min. after fertilization the inhibitory effect is much lower. In the cytoplasmic supernatant of eggs prepared 1 hr. after fertilization the inhibitory activity is negligible. After dialysis, the inhibitor has been quantitatively recovered in the dialysate (after concentration by lyophilization) thus indicating a low molecular compound. The inhibitor is quite stable in the acid-range (and, in fact, can be extracted fully active by 5 per cent trichloracetic or perchloric acid), whereas it is destroyed by alkalinization. The activity is almost completely abolished also by heating at 95° C. for 30 min. A preliminary purification of the inhibitor has been achieved by passing the crude concentrated dialysate at neutral *p*H through a 'Dowex 50' column in the ammonium-cycle. The inhibitor is thus quantitatively recovered in the effluent still contaminated by a few amino-acids but free of substances absorbing in the ultra-violet range between 250 and 280 mμ.

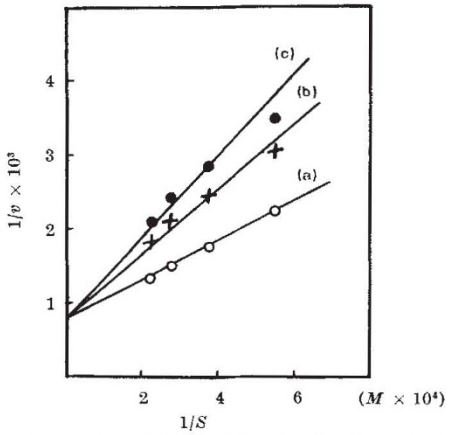

Fig. 2. A Lineweaver and Burk plot of $1/v$ (v, μl. oxygen/hr./mgm. protein) against $1/S$ (S, concentration of cytochrome *c*)). *a*, Control; *b*, with 0·25 ml. of inhibitor; *c*, with 0·5 ml. of inhibitor prepared as described in the text. Other conditions as in Fig. 1

Using this partially purified fraction we have obtained evidence for the competitive type of the inhibition, as is indicated in Fig. 2. Furthermore, spectrophotometric assays using isolated mitochondrial membranes[6] as a source of cytochrome oxidase suggest that the inhibitory action may affect the cytochrome oxidase itself. Work is in progress aiming at the identification and isolation of the inhibitor and examination of the process of its inactivation following fertilization.

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[1] Maggio, R., and Ghiretti-Magaldi, A., *Exp. Cell Res.*, **15**, 95 (1958).
[2] Maggio, R., *Exp. Cell Res.*, **16**, 272 (1959).
[3] Rapoport, S., Hofmann, E. C. G., and Ghiretti-Magaldi, A., *Experientia*, **14**, 169 (1958).
[4] Lord Rothschild, "Fertilization" (Methuen, 1956).
[5] Monroy, A., *J. Cell. Comp. Physiol.*, **50**, 73 (1957).
[6] Watson, M. L., and Siekevitz, P., *J. Biophys. Biochem. Cytol.*, **2**, 639 (1956). Siekevitz, P., and Watson, M. L., *ibid.*, **2**, 653 (1956).

Extended Dormancy of Deciduous Woody Plants treated in Autumn with Gibberellic Acid

Weaver[1] has shown that autumnal application of gibberellin prolongs dormancy of *Vitis vinifera* shoots in the following spring. We[2] recently reported that application of gibberellic acid in late summer and autumn delayed leaf-fall of several woody species; further observations on these plants this spring have shown that in many of them dormancy was prolonged. Thus this unexpected response to gibberellins may not be uncommon.

In our experiments gibberellic acid was sprayed on as a 50 μgm./ml. aqueous soution. Fifteen weekly applications were made between mid-August and late November, 1958. Little or no after-effect was noticed when growth recommenced this spring in the following species: *Acer rubrum*, *Castanea sativa*, *Parthenocissus tricuspidata*, a *Rhododendron* × *molle* cultivar, *Taxodium distichum* and *Ulmus procera*. This list includes nearly all those species which showed no response in autumn to the gibberellic acid treatment but, in addition, two species where leaf-fall was delayed (*P. tricuspidata* and the *Rhododendron* hybrid) and the one species in which leaf-fall was accelerated (*T. distichum*).

Bud-break was delayed by 1–3 weeks in the following species: *Acer pseudoplatanus*, *Betula verrucosa*, *Fagus sylvatica*, *Fraxinus excelsior* and *Sorbus aucuparia*. Many buds and branch-tips of *B. verrucosa* were killed; we believe that this was due to frost damage of young growth induced by gibberellic acid in the previous autumn. In *Liriodendron tulipifera*, bud-break was more or less simultaneous on treated and untreated branches, but leaf-expansion was much slower on the treated branches.

The most striking effects were seen on *Prunus avium*. Here gibberellic acid had little effect on purely vegetative buds, but flower buds were profoundly affected. Most flower buds have remained completely dormant—indeed many are dead. Others have produced distorted or much-reduced flower clusters several weeks after untreated branches had flowered. On branches on the edge of the sprayed area, which probably received reduced doses of gibberellic acid as spray drift, flowering was delayed by about three weeks, though the flowers formed were in all other ways normal.

Of the species showing prolonged dormancy, one (*F. sylvatica*) showed no response to gibberellic acid in the previous autumn. Thus prolongation of dormancy is not necessarily a consequence of, or associated with, delayed onset of dormancy in the autumn. These results support Weaver's view[1] that properly chosen treatments with gibberellin in the autumn might usefully be employed to delay flowering in the following spring, in areas where frost damage is a hazard of fruit cultivation.

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[1] Weaver, R., *Nature*, **183**, 1198 (1959).
[2] Brian, P. W., Petty, J. H. P., and Richmond, P. T., *Nature*, **183**, 58 (1959).