Technical Papers

Blossoming of Fruits Delayed by Maleic Hydrazide[1]

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The flowers of many fruit plants often are killed by late spring frosts, resulting in enormous losses to the industry. If flowering could be delayed 10 days or more in many instances the danger of frost would be past. Winklepleck (*5*) and Hitchcock and Zimmerman (*1*) concluded that naphthaleneacetic acid and its derivatives were promising as means for delaying blossoming, but these compounds were found by Mitchell and Cullinan (*3*) and Marth *et al.* (*2*) to injure the plants. Recently Schoene and Hoffmann (*4*) reported maleic hydrazide to be a unique growth regulant, exerting a pronounced, but temporary, inhibiting effect on plant growth.

On April 27, 1949, dilutions of 1000, 1500, 2000, and 3000 ppm of the diethanolamine salt of maleic hydrazide in water were sprayed on certain fruit plants. Unfortunately, peaches, cherries, currants, and gooseberries were already in flower and could not be tested during this season. Flowers of the Golden Delicious apple were in the early pink stage and all applications of maleic hydrazide were followed by early abscission of the fruit. No retardation of vegetative or floral development was apparent. Second year plants of Premier strawberry had many flowers formed but not open when sprayed on April 27. Although these flowers proceeded to open, one month after treatment check plants continued to blossom profusely, whereas treated plants ceased to blossom. About one week later the plants treated with 1000 ppm resumed blossoming. Vegetative development was definitely retarded, but no specific injury was apparent. Unfortunately, these plants were a part of another experiment and had to be plowed under before additional observations could be made. It seems likely, however, that early spring applications on the strawberry would delay bloom.

Two-year-old vigorous Bristol black raspberries afforded the best example of the effects of maleic hydrazide in delaying blossoming. Leaflets were expanded to about 1 cm and side branches were just beginning to appear on April 27, when plants were sprayed. The treated plants blossomed 24 to 38 days later than check plants, and matured their fruit 16 to 23 days later than check plants. Fruit set was good and no difference in size or flavor of the berries was apparent. Vegetative development was temporarily inhibited and although new canes appeared late they grew rapidly and by midsummer no vegetative differences were apparent. No injury to the foliage was associated with dilutions of 1000 and 1500 ppm, but slight burning was evident with 2000 ppm, and considerable burning with 3000 ppm.

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Inhibition of the Shwartzman Phenomenon by Adrenocorticotropic[1] Hormone (ACTH) from the Adenohypophysis

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In the basic experiment, the Shwartzman phenomenon (*2*) is characterized by severe hemorrhagic necrosis at the skin site prepared by an intradermal injection of a bacterial filtrate (preparatory injection) following the intravenous injection of the same or another potent bacterial filtrate (provocative injection). Although nonanaphylactic in nature, the phenomenon is due to a profound alteration in vascular reactivity. Direct and indirect clinical evidence strongly suggests that the phenomenon demonstrates the mechanism underlying the production of a variety of spontaneous diseases and syndromes of known and unknown etiology, in which vascular lesions are a predominant feature. A large number of different substances fail to suppress the phenomenon—i.e. pharmacological substances variously affecting the vascular system, such as antihistaminics, hormones, vitamins, antivitamins, proteins, amino acids, and British antilewisite. There are, however, a few notable exceptions. According to Thomas and Stetson (*3*), a single application of bromobenzene to the prepared skin site completely inhibits the phenomenon (possibly by interfering with the activation of the tissues' protease). Becker (*1*) suppressed the phenomenon by means of nitrogen mustard, benzol, and x-rays, whose individual effects on blood-forming organs and the reticulo-endothelial system are nearly identical.

In view of this consideration it seemed to be of particular interest to carry out the following studies on the effect of the adrenocorticotropic hormone upon the Shwartzman phenomenon.

The phenomenon was elicited with the meningococcus 44B "agar washings" filtrate made in the manner previ-

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