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.

[1] Authorized for publication on November 22, 1949, as Paper No. 1559 of the Journal Series of the Pennsylvania Agricultural Experiment Station.

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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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TABLE 1

INHIBITION OF THE SHWARTZMAN PHENOMENON BY ACTH

Prepreparatory treatment			Preprovocative treatment			Reactions		
Substance	Dose	Hr prior to I.D. inj.† of bacterial filtrate	Substance*	Dose	Hr prior to I.V. inj.‡ of bacterial filtrate	Strongly positive	Doubtful	Negative
..	..	..	..	..	..	26	0	2
..	..	..	P.S.	0.5 oxytocic u. + 0.5 pressor u.	2	4	0	1
..	..	..	"	0.75 oxytocic u. + 0.75 pressor u.	1	5	0	0
..	..	..	"	"	2	6	0	0
ACTH	2 × 5 mg	18 and 1	..	..	..	4	0	0
"	12.5 mg	2	..	..	..	6	0	0
..	..	..	ACTH	2.5 mg	2	1	0	1
..	..	..	"	5 mg	2	2	3	1
..	..	..	"	2 × 5 mg	18 and 1	1	2	1
..	..	..	"	12.5 mg	2	2	0	8

* P.S. = Pituitrin-S, Parke Davis & Co., 1 ml containing 20 oxytocic units and 20 pressor units. ACTH = Adrenocorticotropic hormone, batch H3706, 12.5 mg containing 0.5 oxytocic units and 0.375 pressor units.

† I.D. inj. = Intradermal injection. ‡ I.V. inj. = Intravenous injection.

ously described (*2*). The rabbits were prepared by a single intradermal injection of 0.25 ml of the undiluted filtrate, and 24 hr later injected intravenously with the same filtrate diluted 1: 17.5, in a dose of 1 ml per kilo of body weight. Although the batch of the filtrate employed was not titrated to the end point, it may be safely assumed in accordance with previous experience with numerous similar preparations that the doses employed represented at least 50 minimal phenomenon-eliciting units. Readings of the reaction were made 4 and 24 hr following the intravenous injection. "Strongly positive" reactions were areas of severe confluent hemorrhagic necrosis varying in size only (i.e., 2 × 3 cm to 8 × 5 cm). "Doubtful" reactions were areas showing mild confluent or petechial hemorrhage. The substances under investigation were injected intramuscularly at various intervals of time preceding the preparatory and provocative injections of the bacterial filtrate, as indicated in Table 1.

As may be seen from the results summarized in the table, adrenocorticotropic hormone did not influence the phenomenon when injected prior to the preparatory injection. However, the administration of a sufficient dose of the hormone (12.5 mg per animal) preceding the provocative injection completely suppressed the phenomenon in 8 out of 10 animals tested. Smaller doses of adrenocorticotropic hormone similarly injected produced only irregular inhibition. The results may be considered highly significant when contrasted with the high incidence of reactions obtained in the control groups. It is also obvious that the suppression of the phenomenon cannot be attributed to the presence of oxytocic and pressor substances in the adrenocorticotropic hormone, since surgical pituitrin containing larger amounts of these substances than those present in 12.5 mg of adrenocorticotropic hormone failed to produce this effect. The limited amount of the hormone available did not permit investigations on the optimal time-dosage relationship.

It may be concluded from these experiments that the adrenocorticotropic hormone fails to prevent the elicitation of the state of reactivity following the preparatory injection. It is capable, however, of preventing hemorrhagic necrosis in reactive skin sites following the provocative injection. The interest of the studies reported is twofold: 1) they may afford a new experimental approach for the elucidation of the beneficial therapeutic effect of the adrenocorticotropic hormone in various diseases affecting the collagenous and vascular systems; and 2) they may provide a useful method for bioassaying of the hormone.

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A Simple Tissue Homogenizer

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For the preparation of tissue homogenates we have found that a simple plunger made from pure gum tubing and a glass rod (Fig. 1), and operated by hand in a test tube, is as effective as more elaborate motor-driven homogenizers. A homogenate of rat liver or kidney almost completely free of intact cells and containing nuclei and mitochondria can be prepared by this means in about