tions of adrenalin around the tumors; and in 9 of the 21 rats in which the subcutaneous tissue had been damaged by injections of adrenalin prior to implantations of tumor grafts. The challenge grafts grew in the 6 rats in which chloretone had inactivated the tumor tissue implanted on the right side. The results showed that resistance to growth of subsequent grafts of native tumor tissue had become established in 40 of the 61 rats treated with adrenalin in the experiments described. The tumor inhibitory action seemed to be caused by damage to the vascularization of the tumor tissue, followed by its absorption.

The characteristic loss of hair previously noted in rats that received minced adrenal glands (*1*) took place in these rats treated with adrenalin.


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## Erythrocyte Aggregation Factor in the Plasma and Serum of Patients with Acute Lupus Erythematosus


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It is the purpose of this preliminary communication to report a phenomenon heretofore not described, namely, aggregation of washed "O" group Rh-positive erythrocytes in a mixture of egg white and fresh plasma and serum obtained from patients with acute lupus erythematosus. The aggregation phenomenon occurs following refrigeration for ½ hr.

The erythrocyte aggregation factor is not destroyed by activating the plasma or serum at 56° C for 30 min. Strong positive plasma or serum kept at room or refrigerator temperature for 10 days gave weak to negative reactions; when kept in the frozen state for one month, positive reactions were obtained which compared favorably with the original result.

The plasma and serum samples containing the erythrocyte aggregation factor also produced the so-called L. E. cell phenomenon (*1–3*).[2] No red blood cell aggregation occurred in a control series of plasma and serum samples obtained from 150 normal subjects.

The test is carried out as follows. Fresh egg white is filtered through 2 layers of gauze, and 10 ml of the filtrate is diluted with 90 ml of physiological saline solution. The egg white is placed in the refrigerator for 24 hr. Group "O" Rh-positive erythrocytes are washed in saline solution, and a 10% suspension is made and stored in the refrigerator for 1 day.

To assure an even mixture of the egg white, the solution is shaken, and 1 ml is placed in a Wassermann tube. To the egg white 0.5 ml of test plasma or serum and 1 drop of the red blood cell suspension are added. The tube is shaken until the erythrocytes are well dispersed. The test is then placed in the refrigerator for ½ hr. Upon removal, the test is centrifuged at 2,000 rpm for 1 min. The tube is gently shaken until the red blood cell button is completely broken up. A positive test shows easily visible clumps of erythrocytes. A normal control is simultaneously shaken along with the test, since the reading is started when the erythrocytes in the control are homogeneously dispersed.

A 5-tube quantitative test may be set up, and the degree of clumping recorded as 1, 2, 3, and 4+, adding amounts of plasma or serum corresponding to the test-tube number—that is, 1 drop in the first tube, 2 drops in the second tube, and so on.

A positive test may be kept in the refrigerator for several days without an appreciable breaking up of the red blood cell clumps. The clumps disappear when the test is placed in a water bath at 56° C for 1 min or at 37° C for 5–10 min. The erythrocyte aggregation phenomenon can be reinduced by placing the test in the refrigerator for ½ hr, then centrifuging and shaking as above.

Whether the cold phenomenon reported here and the so-called L. E. cell phenomenon are produced by the same factor, and whether this factor is common to diseases other than lupus erythematosus, are being investigated.


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## Insect Transmission of Western X-Disease of Peaches[1]


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Western X-disease has been reported as the most serious virus disease of peaches in the northwestern United States and British Columbia (*1*). Similar diseases occurring on peach have been described under the name leaf casting yellows in California (*2*) and X-disease in northeastern United States and adjoining areas of Canada (*3*). The disease on peach in

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[2] The initial work of H. R. Wolfe from 1947 to 1949 received support from the Washington State Department of Agriculture.

[3] The work of E. W. Anthon has been supported in part by the USDA.

California has been related to the buckskin disease of cherry occurring there (*2*). Similarly, Western X-disease occurring on peach has been related to Western-X–little-cherry in Oregon (*4*) and Utah (*5*), and there is indication that there may be relationship between the two in Washington (*6*). Western X-disease, like X-disease in the East, affects chokecherries, producing red-leaf symptoms (*3,5*).

The search for vectors during the 7 years ending with 1947 failed to incriminate any insects, despite nearly 1,000 tests with 200 species. Emphasis was placed on the Cicadellidae, with 124 species being placed on trial, and upon the Aphididae, represented by 16 species.

Extended work in 1948 and 1949 produced the first evidence of insect transmission of the Western X-disease. This was through the geminate leaf hopper, *Colladonus geminatus* (Van D.).[4] Surveys that showed it to be common in the commercial peach-growing areas of the Northwest directed attention toward this leaf hopper. Although its favorite host plants in orchards appear to be legumes and grasses, it is one of the few leaf hoppers that can be maintained indefinitely on stone fruit trees, at least when caged on them. It is found on peach during the spring and summer and occasionally becomes abundant by early autumn.

The first indication that this leaf hopper is a vector was obtained at The Dalles, Ore., in a field test plot operated by the Oregon State Agricultural Experiment Station[5] in cooperation with the United States Department of Agriculture. One J. H. Hale peach tree test-inoculated with *C. geminatus* between August 2 and September 10, 1948, gave a positive chemical test (*7*) for Western X-disease in October 1948 and produced distinct Western X-disease symptoms by June 21, 1949. The symptom-showing branch was growing from precisely the place where the leafhopper cage had been attached. This tree was the only one at this location that showed any reaction out of 28 peach trees exposed to this species of leaf hopper.

Additional transmissions were achieved by December 1949, with small, potted Lovell peach seedling trees in the greenhouse of the Tree Fruit Experiment Station[6] at Wenatchee, Wash. Six cases of Western X-disease were produced in one series of 18 trees test-inoculated with *C. geminatus*, and 6 additional trees developed fair to questionable symptoms and gave positive reactions to chemical tests (*7,8*). In the present report only the trees which developed typical symptoms on leaves are being regarded as cases of transmission (Table 1).

All the 15 check trees in the series are still normal.

[4] The leaf hoppers used in these experiments were identified by P. W. Oman, U. S. Bureau of Entomology and Plant Quarantine, Division of Insect Identification.

[5] The Oregon State Agricultural Experiment Station made possible the Oregon studies, with J. A. Milbrath, plant pathologist, H. J. O'Reilly, leader of The Dalles Experimental Area, and S. C. Jones, entomologist, collaborating. The work of L. S. Jones was done at this location.

[6] The work of H. R. Wolfe and E. W. Anthon was done at this location. The writers wish to express their appreciation to R. C. Lindner, E. L. Reeves, E. C. Blodgett, and H. C. Kirkpatrick for their helpful assistance and aid in diagnosis.

TABLE 1

TRANSMISSION OF WESTERN X-DISEASE TO LOVELL PEACH SEEDLINGS BY *Colladonus geminatus* (VAN D.)

| Symptoms | No. trees |
|---|---|
| Typical | 6 |
| Fair | 3 |
| Questionable | 3 |
| None | 3 |
| Tree died | 3 |

Two of the test trees showing typical symptoms were approach-grafted to normal Lovell peach seedlings of the same age. One of the approach-grafted trees displayed symptoms within 60 days.

Significant facts relating to the 7 cases of Western X-disease recorded as transmitted by *C. geminatus*, one in the field test plot in Oregon and 6 in the greenhouse in Washington, are itemized below:

1. In order to insure the maximum possibility of acquiring the virus, the leaf hoppers were allowed to feed on infected cherry as well as on infected peach before being confined on the healthy peach test trees that reacted. Whether the leaf hoppers acquired the virus from cherry or peach, or both, in this series of experiments is not known.

2. Sixty to 130 days elapsed between the first exposure of leaf hoppers to inoculum and their final feeding on the peach test trees. In the greenhouse tests, the insects fed on inoculum as nymphs and adults, but fed on the test trees only as adults.

3. In the 6 greenhouse tests a period of 56 days elapsed from the time the insects were removed from the inoculum until the time they were caged on the small peach test trees to which they transmitted the disease. During this period they were confined upon normal peach and cherry trees in the field. This indicates retention of the virus in the vector for this period.

4. In 2 cases where infective leaf hoppers were first allowed to feed on a small Lovell peach test tree in the greenhouse for 11 days, and then moved to a second Lovell test tree for 14 days, the second tree developed typical Western X-disease symptoms, whereas the first tree developed only questionable symptoms.

5. One test tree became diseased following feeding by a single adult leaf hopper. As many as 25 leaf hoppers were fed on the other test trees.

6. The first symptoms on the 6-in. Lovell peach seedlings in the greenhouse appeared 46 days after exposure to the vectors.

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