tion provide striking evidence to support the importance of the Rh factor in the etiology of erythroblastosis fetalis.

In a general way it may be assumed from these findings that isoimmunization of the Rh negative mother by the Rh factor of the fetus may explain the incidence of erythroblastosis fetalis in 91 per cent. of the families studied. That other blood factors are capable of inducing isoimmunization is shown in one of the 10 Rh positive mothers whose blood contained an atypical agglutinin of an entirely different specificity.[7] Still another blood factor identified by an agglutinin recently described by Levine and Polayes[8] is capable of inducing isoimmunization. This woman, who suffered a transfusion accident during her puerperium of her twelfth pergnancy, had 4 miscarriages but no infants with erythroblastosis fetalis.

The studies on the specificity of various anti-Rh sera produced by isoimmunization in these mothers showed that these sera differ somewhat in their specificity. While the great majority of bloods give identical reactions when tested with various anti-Rh sera, several bloods inactive with a particular serum were found to react with other sera. A similar finding was observed by Landsteiner and Wiener.[9]

Consequently, the bloods of the remaining 9 Rh positive mothers not containing atypical agglutinins will have to be retested with the new agglutinins as well as with a variety of anti-Rh sera.

The correlation of anti-Rh agglutinins and the postpartum interval when the first tests were done, is shown in Table 2.

TABLE II

Incidence of anti-Rh agglutinins in 101 Rh-negative mothers

Interval after last delivery of an affected infant	Agglutinins present	Agglutinins not found
2 months	24	23
2 months to 1 year	3	7
1 year or longer	2	36
No data	0	6
Total	29	72

Atypical agglutinins in the Rh negative mothers were found in 50 per cent. of the cases in tests made within the first two months after delivery of an infant with erythroblastosis fetalis. It may be assumed that the incidence of anti-Rh agglutinins will be higher if such mothers are tested at intervals in the course of subsequent pregnancies which may result in other affected infants.

[7] This case, a patient of Dr. C. Javert, will be published separately.

[8] P. Levine and S. H. Polayes, *Annals Int. Med.*, 14: 1903, 1941.

[9] K. Landsteiner and A. S. Wiener, personal communication.

It is of interest that in 2 cases atypical agglutinins could still be demonstrated 2 and 2½ years, respectively, after the last delivery of an infant with erythroblastosis fetalis.

According to the concept of isoimmunization, the mother's immune agglutinins pass through the placenta and exert lytic action on the susceptible fetal blood. However, this could not occur if the Rh factor had a wide distribution in tissue cells and body fluids, which would specifically bind the anti-Rh agglutinins. Tests made with numerous specimens of saliva and a small number of specimens of seminal fluid and sperm cells of Rh positive individuals indicated that the Rh factor was not present in this material. Thus there is justification, at least for the present, to assume that the Rh factor may be limited to red blood cells.[10] However, a comprehensive study of various organs and body fluids is desirable.

In a future publication evidence will be presented that the familial nature of erythroblastosis fetalis depends upon the heredity of the blood factors involved. The striking incidence in certain mothers and the sporadic occurrence in others depends upon the homozygosity or heterozygosity of the father's blood. That the Rh factor is inherited as a mendelian dominant property was recently demonstrated by Landsteiner and Wiener.[9]

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MOSAIC, CHLOROSIS AND NECROSIS IN VIRUS-INFECTED PERENNIAL PEPPER CAUSED DIRECTLY BY PRODUCTS OF A DERANGED METABOLISM[1]

IN *Capsicum frutescens* L., a tropical perennial pepper, the level of concentration for Nicotiana Virus 1 is relatively low under all conditions of growth studied. When the leaves are wiped with virus local necrotic lesions develop and the leaves absciss.[2] When the inoculated plants are cultured near 32° C. small quantities of virus pass from the inoculated leaf, and systemic infection occurs,[3] but does not when cultured near 23° C. At the high temperatures the branches, stems, taproots and roots become necrotic. These tissues develop a dark brown to black color and death results. Old woody plants are more resistant than young succulent plants.

[10] *Cf.* A. S. Wiener and S. Forer, *Proc. Soc. Exp. Biol. and Med.*, 47: 215, 1941.

[1] Studies conducted under Bankhead-Jones Project S.R.F. 2–17, U. S. Department of Agriculture, Bureaus of Plant Industry and Agricultural Chemistry and Engineering cooperating.

[2] F. O. Holmes, *Phytopathology*, 26: 896, 1936.

[3] H. H. McKinney, *Jour. of Heredity*, 28: 51, 1937.

Before abscission, a secondary chlorosis usually involves the leaf tissues not included in the local infection sites, especially at the higher temperatures with summer sunlight. When one small marginal zone on the leaf or the petiole is inoculated, the secondary chlorosis involves the entire leaf.

Over a period of five years, 125 lots of leaf tissue well outside of the inoculated zones were isolated before and during the progress of secondary chlorosis, and in no case was a sign of virus obtained in *Nicotiana tabacum* L., *N. glutinosa* L., *N. langsdorffii* Schrank, *N. sylvestris* Spegaz. and Comes, or in *Phaseolus vulgaris* L., variety Scotia. Extracts from these pepper tissues were dialyzed to remove any possible inhibiting agent, but no signs of virus were obtained after dialysis.

The inoculated zones, with few exceptions, contained virus. However, 11 lesions per leaf on beans was the highest count obtained; usually the counts were less.

Typical, fully developed light- and dark-green mosaic mottling frequently appeared on new leaves remote from the zones of inoculation when woody pepper plants were cultured near 32° C. This mottling sometimes persisted for several days before the leaves became necrotic. Separate assays made on 10 mosaic leaves revealed no signs of virus before the appearance of necrosis, but when the first faint signs of necrosis appeared in the mottled leaves, virus could be detected in the tissues by means of inoculations into tobacco. Attempts to demonstrate that this mosaic is caused by a virus specific to pepper plants failed.

In the branches, stem, taproot and roots, the virus was most concentrated in or very near to the necrotic cortex or cambium. No virus was detectable in the necrotic xylem of the stem.

When the lateral roots or the tap roots were inoculated and the plants were cultured near 32° C. necrosis resulted but did not advance more than one-half inch up the stem, and no signs of virus were revealed above this point. However, when the lower stem or the upper part of the tap root was girdled and the xylem was solidly necrotic, these plants wilted. Tests indicated that the water-conducting tissue was obstructed as a result of the necrosis. Necrosis occurred considerably in advance of the virus in the xylem, but not in the cortex.

It is concluded that the secondary chlorosis, the mosaic mottling and the xylem-necrosis are induced directly by translocated or diffused products of a deranged metabolism, which in turn is induced by relatively small amounts of virus in remote zones.

Typical fully developed mosaic mottling in tobacco has not been observed to occur in advance of the virus. However, this does not preclude the possibility that mosaic in tobacco is induced directly by the products of a deranged metabolism.

One of the authors (Hills) found that pepper-leaf tissue in the advanced stages of secondary chlorosis contained an increased amount of peroxidase, and reduced amounts of oxidase and catalase, in comparison with alternate normal leaves from the same branches. These alterations in enzyme balance, and the ultimate chlorosis, show that the virus is capable of inciting profound changes in tissues remote from zones of detectable virus, and finally, these changes are so drastic in the necrosed pepper tissues that the virus is completely destroyed soon after these tissues have desiccated.

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SCIENTIFIC APPARATUS AND LABORATORY METHODS

A METHOD FOR CONTINUOUSLY DETERMINING THE RATE OF OXYGEN CONSUMPTION FOR LABORATORY ANIMALS

METHODS now available for determining the rate of oxygen uptake of animals consist essentially of methods of measuring the volume of oxygen taken up over known periods of time. The value for the rate of oxygen uptake calculated from such data gives an average value of the rate over the interval of time employed and tends to obscure any rapid changes that may have occurred. The present apparatus was designed for use in experiments where changes in metabolism occur rapidly and where a prolonged continuous record is desired giving instantaneous values for the rate of oxygen uptake.

The apparatus consists of an air-tight chamber just large enough to hold the animal, lined with wire gauze soda lime containers. These should be arranged so as to assure a rapid and constant rate of absorption of expired CO_2. This chamber is connected with an orifice type flowmeter which consists of a capillary connecting the oxygen supply with the chamber, across which is connected a U-tube water manometer. The rate of flow of oxygen through the capillary is proportional to the pressure across it, so that it is an easy matter to calibrate the reading of one arm of the manometer to read directly in cc/min. This calibration is accomplished by setting the pressure to different values by means of the reducing valve on an oxygen tank and determining the corresponding flow by timing the displacement of water from a volumetric