the rate of inactivation of insulin by the liver but appears to be a result of an increased secretion of insulin. This conclusion is in agreement with Milner's report (*8*) that $10^{-5}M$ ouabain stimulated insulin secretion in pancreatic tissue in vitro. The exact mechanism of this action of ouabain must still be clarified. Whether it is related to an effect of ouabain on sodium and potassium active transport, as postulated by Milner, or to an effect of the sugar molecule which is part of the glycoside, remains to be established.

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Autoimmune Chorioretinitis in Rhesus Monkeys

Abstract. *Monkeys injected with monkey retinal tissue incorporated in Freund's complete adjuvant developed ocular lesions characterized by choroiditic patches in the fundus periphery and sheathing of retinal vessels. Bovine retina, monkey choroid plexus, and guinea pig kidney were ineffective in this respect.*

An unusual complication of experimental autoimmune encephalomyelitis (AE) has been described in rhesus monkeys; the lesion, a hemorrhagic retinopathy (HR) occurs during the early stages of the AE (see *1–3*). A matter of primary consideration was whether the retinal damage represented an intrinsic feature of the AE syndrome, or an indirect and secondary complication of AE, or a separate disease entity. The possibility that central nervous system and retina could share common antigens provided the first point for experimental attack; another possibility was that the vascular damage in the retina was a response to sensitization with antigens of vascular origin rather than to central nervous system or retinal antigen.

Ten juvenile rhesus monkeys (*Macaca mulatta*) of both sexes were given a single immunizing dose of 100 mg (wet weight) of freshly dissected and emulsified monkey retina incorporated in Freund's complete adjuvant containing 0.5 mg of dried, killed *Mycobacterium tuberculosis*. The total volume of 0.5 ml of emulsion was injected into four sites subcutaneously over the scapular and nuchal regions. The animals were housed and fed as described (*1–3*) and examined at frequent intervals for signs of central nervous system disease and for eye lesions. Control groups of rhesus monkeys of equivalent age received the following tissues emulsified in Freund's complete adjuvant and injected in similar fashion: monkey choroid plexus (16 to 40 mg) or guinea pig kidney or spinal cord (0.25 ml of 50 percent suspensions). Other control monkeys received the complete adjuvant without antigens. In experiments to determine the species specificity of retinal antigen, bovine retinal tissue (100 mg) was substituted for that of monkey (*4*). After retinal lesions developed, histological examination was performed on the eyes and optic nerves.

Monkeys immunized with monkey retinal tissue developed ocular lesions in all instances, whereas bovine retina, monkey choroid plexus, and guinea pig kidney were ineffective. The first clinical signs of ocular disease consisted of choroiditic patches of varying size in the fundus periphery and sheathing of retinal vessels. There was no clear correlation between the extent of the lesions in the two tissues. In three eyes, a hemorrhagic component complicated the picture, and occasionally lid or periorbital edema and conjunctival hyperemia preceded the onset of fundus changes. Histologic examination established the presence of perivasculitis in the retina and mild or severe uveitis, especially located in the pre-equatorial portion of the choroid (Fig. 1). The optic nerve was free of inflammatory changes with the exception of the large vessels on the disk which in several instances showed a moderate accumulation of lymphoid cells in the adventitial tissue (*5*).

The lesions produced in the monkey eye by immunization with homologous retinal tissue differed sharply from those produced with guinea pig spinal cord. Immunization with this heterologous antigen resulted often in destructive hemorrhagic vasculo-occlusive retinopathy without perivasculitis, minor involvement of the uvea, and the typical widespread inflammation in the optic nerve.

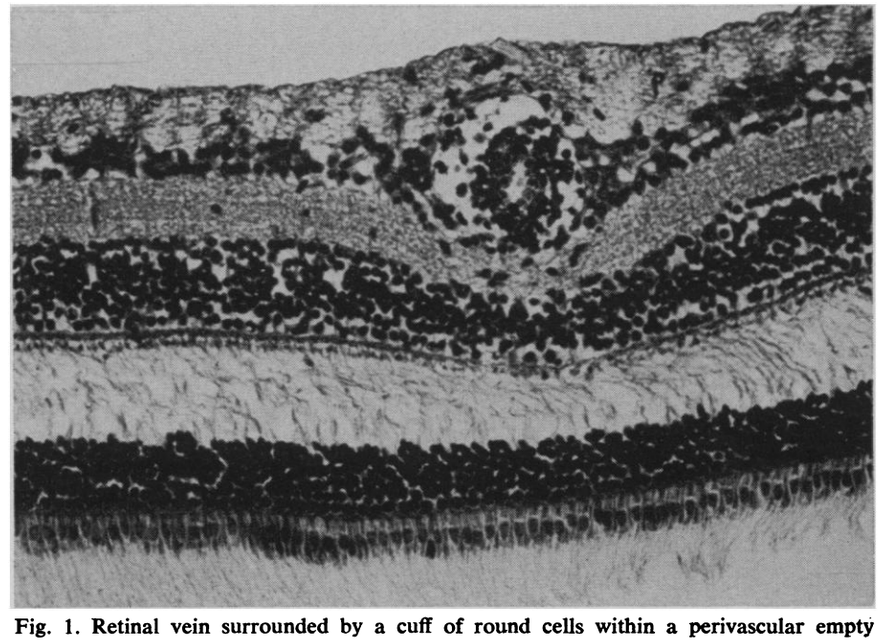

Fig. 1. Retinal vein surrounded by a cuff of round cells within a perivascular empty space.

In preparing the retinal antigen, a clear-cut separation of the retina from adjacent tissues, particularly the uvea at the ora serrata, is difficult if not impossible. The contamination of the retinal antigen could not be quantitated; variations in the admixture of nonretinal tissue may have influenced the ocular response.

A possibility other than the inadvertent contamination of retinal antigen with uveal antigen, or that of cross antigenicity, is the natural anatomic association of disparate tissue antigens as exemplified by the presence of myoid cells in thymic tissue (*6*). Bullington and Waksman (*7*) and Wacker and Lipton (*8*) reported the induction of uveitis by immunization with optic nerve and with retinal antigens, respectively. Although Bullington and Waksman (*7*) and Wacker and Lipton (*9*) failed to produce uveitis in rabbits by immunization with rabbit uvea, this has been done by Aronson (*10*). Thus, the suggestion by Wacker and Lipton (*9*) that the antigen responsible for the uveitis is a primarily retinal component remains to be confirmed.

After sensitization with retinal antigen, the eye was the only organ to show disease change; no involvement of the central nervous system or any other organ system was noted clinically in any of these animals.

The mechanism of the retinopathy (*1*), which is elicited by injection of central nervous system tissue, remains obscure. The possibility had to be considered that monkey retina might contain myelinated components as is the case in the rabbit retina (*7*). The absence of AE in monkeys injected with monkey retina and Freund's complete adjuvant is an argument against this possibility, although quantitative considerations prevent excluding it completely. To examine the possible role of vascular antigenicity in HR in producing eye lesions, heterologous kidney, which contains large amounts of vascular tissue and basement membrane, and homologous choroid plexus were used as controls. The choroid plexus was particularly suitable since it is a highly vascular tissue located within the brain itself. Although data are lacking for clarification of the pathogenesis of the HR, our results with the chorioretinitis suggest that the lesion induced in rhesus monkeys by injection of homologous retinal tissue in Freund's complete adjuvant is an autoimmune disease of organ- and species-specific nature.

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Pesticide Mobility: Determination by Soil Thin-Layer Chromatography

Abstract. *Pesticide movement was evaluated by the comparison of* R_F *values on thin layers of soils. Results from the new technique correlated well with existing information on pesticide movement, facilitating the grouping of pesticides into classes on the basis of mobility. Thin-layer chromatography may have broad applicability in soils research.*

Movement of a pesticide from its site of application is one cause of environmental contamination. Thus, mobility data are useful in evaluating the persistence of applied chemicals, as well as in defining conditions associated with pesticide use. Such information, however, is limited by the expense and difficulty of conducting leaching studies (*1*). Lack of a standard method makes comparisons among pesticides and soils difficult.

We report a new approach to the investigation of pesticide mobility—the use of untreated soil as an adsorbent in thin-layer chromatography (TLC). The new method is termed "soil thin-layer chromatography." Soil TLC is rapid, reproducible, and inexpensive; requirements for equipment, laboratory space, test chemicals, and soil are modest. A quantitative index of relative pesticide mobility, based on R_F values, correlated well with published observations on movement.

Three soils from Ap horizons (a layer 0–6 inches, cultivated soil) which differ substantially in texture and content of organic matter were studied: Lakeland sandy loam (12.0 percent clay, 0.9 percent organic matter), Chillum silt loam (26.3 percent clay, 3.1 percent organic matter), and Hagerstown silty clay loam (39.5 percent clay, 2.5 percent organic matter). Medium sand (> 250 μ thick) was removed by dry-sieving from Chillum and Hagerstown soils, and coarse sand (> 500 μ) by dry-sieving from Lakeland sandy loam prior to chromatography.

Conventional TLC apparatus was used to prepare most soil plates. Immediately before spreading, a slurry of soil and water was prepared. Chillum and Hagerstown layers (500 μ thick) were prepared with a variable-thickness spreader, and the Lakeland layer (750 μ) was prepared with a glass rod moved over masking tape along the plate edges (*2*). Six or seven ^{14}C-labeled pesticides (3 to 10 μg each) were aplied to a plate (20 by 20 cm) and developed 10 cm with water by ascending chromatography. The plates were visualized by autoradiography with "no-screen" medical x-ray film.

The relative mobilities of six herbicides are readily differentiated in Fig. 1. Little tailing was noted for the highly mobile dicamba (*3*), indicating that the adsorption isotherm is nearly linear. Compounds of slightly lower mobility (for example, amiben, fenac, and 2,4-D) exhibit increased tailing, resembling the movement of tritiated water in a clay soil (*4*). With monuron and other less mobile compounds, movement is seen as a continuous streaking or elution from the origin.

In the comparison of pesticide compounds, we measured R_F values as the front of a streak or spot. This value changed only slightly in the range of sample size from 0.5 to 200 μg. Streak-