mately equal amounts in our limited experience to date.

The presence of β-globulin *D* has been established with certainty in serum from two female Negroes from New York (out of 22 males and 27 females tested), and in serum from four male and one female Australian aborigines (out of 17 males and 6 females tested). β-Globulin *D* has not been demonstrated in the serum from any of several hundred Canadians (largely of European ancestry) which have been studied, chiefly by one-dimensional starch-gel electrophoresis. These results suggest the existence of considerable racial differences in the frequency of occurrence in the serum of β-globulin *D*, and indicate that its presence may well prove to be genetically controlled.

I am grateful to Drs. Ralph E. Engle, jun., and James H. Pert of the New York Hospital, New York and to Dr. William R. Horsfall of the Commonwealth Health Laboratory, Cairns, Australia, for their kindness in sending me serum for study.

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Serological Studies on *Fusarium oxysporum* Schl. emend Sn. et H.

Morphological recognition of species of fungi which cause plant diseases present few problems, and it is usually not necessary to use serology in diagnosis. However, within a morphological unit physiological differences exist, and it might be possible to apply sero-diagnostic methods in distinguishing physiological races.

We have studied this problem on various physiological races of *Fusarium oxysporum* Schl. emend Sn. et H., which cause vascular browning and wilting of different host plants. Generally, cross-infection is not possible; for example, isolates of the fungus from *Pisum sativum* do not attack *Lupinus luteus* nor do those from *Lupinus luteus* attack *Pisum sativum*.

Single spore cultures of the isolates were grown on synthetic liquid media (Richard's and Czapek's solutions). Antisera were prepared by intravenous injection into rabbits of suspensions of microconidia, culture liquids and extracts of mycelia. Some rabbits produced specific antibodies which could be demonstrated best in gel diffusion precipitin tests[1,2]. Culture liquids seemed to be the most suitable test antigens in the precipitin tests.

The five tests illustrated in Fig. 1 were carried out simultaneously in one Petri dish; agar was poured into five small glass rings (27 mm. diam.) which had been glued with 'Araldite' to the bottom of the Petri dish. Several holes (4 mm. diam.) were punched into each agar plate. These reagent reservoirs were filled with antisera against *Fusarium oxysporum* f. *lupini* (Linf.) Sn. et H. and *Fusarium oxysporum* f. *pisi* (Linf.) Sn. et H. (further on designated as *L* and *P* respectively) and with the test antigens from these two isolates (*l* and *p*). The plate in the centre of Fig. 1

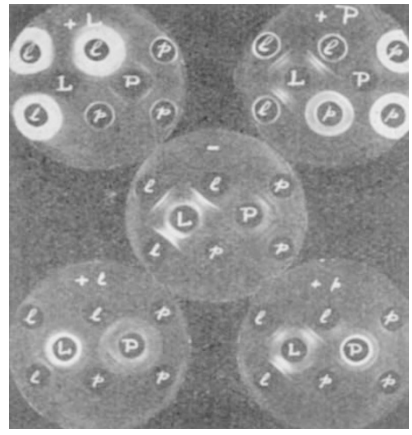

Fig. 1. Comparison of the culture liquids of *Fusarium oxysporum* f. *lupini* (*l*) and *Fusarium oxysporum* f. *pisi* (*p*) in gel diffusion precipitin tests. In the two plates above the agar is mixed with the antisera *L* and *P*, respectively; in the plates below with the antigen *l* and *p*

shows the specific precipitation pattern between *L* and *l* and between *P* and *p*. Frequently, there also appear lines, which represent antigens common to *l* and *p*.

When the agar has been mixed with *L* before pouring (Fig. 1, $+L$), the antigens diffusing from the reservoirs containing *l* are precipitated in a halo around the hole and no specific line between *L* and *l* is formed (specific inhibition of precipitation[3]). The specific reaction between *P* and *p*, however, is not inhibited by the presence of *L* and still results in a precipitation line. The reciprocal pattern is formed if the agar is mixed with *P* (Fig. 1, $+p$). Analogous results can be obtained by introduction of the antigens into the agar (Fig. 1, $+l$ and $+p$).

Further investigations are in progress, and full details will be published later.

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Effect of Acetylcholine and Quinidine on Atrial Cellular Potentials

Briscoe and Burn[1] have shown that acetylcholine will restore the spontaneous contractions of rabbit atria which have been arrested by quinidine. Armitage[2] has suggested that quinidine impairs repolarization by rendering the cell membrane less permeable to potassium ions, and acetylcholine restarts the tissue by restoring the potassium permeability. However, Weidmann[3], Johnson[4] and Johnson and McKinnon[5] have demonstrated that in cardiac muscle quinidine predominantly affected the depolarization phase of the action potential. The maximum rate of rise of the action potential was grossly decreased with little or no concomitant change in the membrane resting potential, indicating that the drug had a direct effect on the sodium-carrying system.