responsible for the reflexions must exceed 3.7×10^4 electrons/cm.³ in order to be observed at this frequency. Somewhat similar echoes from aurora borealis have been reported by Lovell, Clegg and Ellyett¹ using transmissions on 46 Mc./s. Also, ultra-high-frequency long-distance transmission during auroral storms has aroused considerable interest in amateur radio circles².

Fig. 1 shows the 17.31-Mc./s. record of August 8, 1950, 0130-0400 E.S.T. at Concord, Massachusetts. The height-markers here are each 20 km. and the total sweep represents 660-km. slant range. The minimum range associated with the auroral pattern is about 220 km. (The horizontal black line at 500 km. is an extraneous marker from some neighbouring equipment.) Local U.S. Weather Bureau observers reported strong visual aurora between 0055 and 0350 (beginning of dawn). The reflexions in Fig. 1 begin very faintly at 0050, become strong at 0107, and become intermittent at 0235; a faint trace is noticeable at 0344. A number of reflexions due to meteors are evident below the broad auroral pattern, notably the one at 0157 at 100 km., and the one at 0237 at 210 km.

While auroral reflexions on the lower frequencies are commonplace at this latitude, they ordinarily do not yield clear and distinct patterns owing to other complicated background reflexions normally present. Fig. 2 shows one of the clearer recordings received at 3.5 Mc./s. on September 28, 1946. In this figure the height-markers are 15 km. each and the total sweep is 210 km.

Since August 8 we have continued to record 17.31-Mc./s. transmissions with a long sweep, and we have occasionally noticed more reflexions of the type mentioned here, although none so spectacular as those of Fig. 1.

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¹ Lovell, Clegg and Ellyett, *Nature*, **160**, 372 (1947).

² See "The World Above 50 Mc.", *QST*, **24**, No. 10, 40 (October 1950).

Action of Trypsin on Rh-Positive Human Red Cells

It is well known since the work by Pickles¹, and Morton and Pickles², that cholera filtrates and trypsin alter the Rh-positive cells with the result that they become agglutinable in saline by anti-Rh pure blocking antibodies. On the other hand, Race and Sanger³ have recently described a chromosome alteration in an individual lacking all antigens at the Cc and Ee loci, with an extraordinarily strong D antigen, as judged by its avidity for anti-D. This particular blood was found to be agglutinated by pure blocking anti-D diluted in saline.

We have tested the hypothesis that trypsinization would increase the available quantity of agglutininogen (in the widest sense of the word) at the surface of the cell, and that agglutination of trypsinized cells by pure blocking antibodies would be merely a quantitative phenomenon taking place in the zone

of optimal proportions between agglutinogens and antibodies.

By absorption experiments we found eventually that trypsinized cells absorbed more anti-D than fresh cells, this absorbing power increasing (up to a certain point) when greater amounts of enzyme had been used in the preliminary trypsinization. Absorption was carried out with 1:4 vol. of cells (packed) for 1 vol. of serum.

The method of trypsinization used was that described by Dausset and Vidal⁴. A solution of trypsin ('Difco' 1:250) in 8.5 per thousand saline is allowed to stand for an hour, then adjusted to pH 7.3 with Sørensen's phosphate buffers (20 per cent M/15 disodium hydrogen phosphate and 0.5 per cent M/15 potassium dihydrogen phosphate). It is mixed with one-third of three-times washed and packed cells, and incubated for one hour in the water-bath at 40° C. During incubation the tubes are frequently shaken. The cells are then washed three times in normal saline.

No haemolysis was observed, and both the negative controls for agglutination and the morphology of the cells as seen under a high-power microscope were satisfactory.

It is remarkable that absorption of anti-Rh sera by trypsinized cells brings down the titres of agglutination in both saline and in albumin, the titre of agglutination in saline with trypsinized cells and also the sensitization as judged by the indirect anti-globulin test. It is therefore most likely to be specific and seems to justify our hypothesis upon the mode of action of trypsin. (Since this communication was submitted, we have received a paper by Wheeler, Luhby and Scholl⁵ stating that D trypsinized cells absorb more anti-D than fresh cells.)

Thus it appeared possible to vary the statistical 'load' of agglutininogen at the surface of the individual cell and to obtain all the degrees of agglutinogenic strength between highly trypsinized and normal cells. I have pointed out recently⁶ that such an opportunity affords a means of studying the agglutininogen-antibody reaction with both constant antigen and constant antibody techniques. It will be remembered that, using the indirect antiglobulin test, we demonstrated 'prozones' in the antigen excess and the antibody excess ratios, and that the agglutination pattern suggested a linear relationship between the optimal proportions of antigen and antibody. Our conclusion was that 'prozones' could be more satisfactorily explained by an excess of antibody than by the action of a blocking antibody.

The same results have been achieved with anti-D blocking sera and D trypsinized cells. They suggest that in the case of Rh-antibodies also, the 'prozones' and the blocking effect are more satisfactorily explained by a quantitative theory.

A fuller report of these investigations will be published elsewhere.

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¹ Pickles, *Nature*, **158**, 880 (1946).

² Morton and Pickles, *Nature*, **159**, 779 (1947).

³ Race and Sanger, "Blood Groups in Man", 277 (Blackwell, Oxford).

⁴ Dausset and Vidal, *C.R. Soc. Biol.*, **144**, 679 (1950).

⁵ Wheeler, Luhby and Scholl, *J. Immunol.*, **65**, 39 (1950).

⁶ Hubinont, communication at the International Congress of Haematology, Cambridge, August 1950; *Brit. Med. J.* (in the press).