

size of fabric fragment for each slide well is 5 mm. × 2 mm.

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¹ Kind, S. S., *Nature*, **185**, 397 (1960).

Non-specific Hæmagglutinin from Cellulose and Cellulose Ion Exchanger

IN our work on the fractionation of ether-treated influenza viruses by chromatography on *DEAE*-columns (N,N-diethylaminoethylcellulose)¹ it was found that samples of cellulose exchangers released different amounts of non-specific hæmagglutinin which has to be removed by thorough washing (refs. 1 and 2 and McCrea, J. F., personal communication). We have found similar non-specific hæmagglutinins in batches of normal cellulose, CAM-cellulose and ECTEOLA-cellulose (Schleicher and Schüll, Dassel, Germany; Serva-Entwicklungslabor, Heidelberg, Germany).

With reference to the increasing importance of the cellulose ion exchangers, for example, for fractionation of hæmagglutinating agents¹⁻³, it seems to be useful to know, and to exclude, this hæmagglutinin.

The dialytic behaviour of the non-specific hæmagglutinin from *DEAE*-cellulose indicates a high molecular weight. The agglutinin was shown to be heat-resistant ($\frac{1}{2}$ hr., 100° C.), not susceptible to the receptor-destroying-enzyme, lysozyme, and trypsin, susceptible to sodium periodate, acid and alkali hydrolyses.

Furthermore it is able to agglutinate red cells of different species, for example, rabbit, mouse, chicken, and guinea pig; also human red cells without respect to the blood-group antigens A₁, A₂, AB and O.

The highest agglutinin titre proved to be at pH 7.2–7.8. The fixation of the hæmagglutinin to the red cell surface appears to be firm, as proved by hæmolysis on elution. It may be assumed that the agglutinin is a pan-agglutinin and that it belongs to the group of polysaccharides, which is further indicated by its susceptibility to sodium periodate and by a positive Molisch reaction.

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¹ Schmidt, B., Hartmann, D., and Grossgebauer, K., *Arch. ges. Virusforsch.* (in the press).

² McCrea, J. F., and O'Loughlin, J., *Viol.*, **8**, 127 (1959).

³ Prager, M. D., and Speer, R. J., *Proc. Soc. Exp. Biol. Med.*, **100**, 68 (1959).

The Rh Factor C in South African Bantu

FOLLOWING the recent work of Sturgeon *et al.*¹ on a variant of Rh antigen C common in Negroes, Allen and Tippett² on factor G, Wiener and Unger³ on the complexities of D and Rosenfield and Haber⁴ on Rh factor Ce, we considered it timely to undertake a re-appraisal of the Rh factor C in South African Bantu. It was planned to test a series of type Cde Bantu blood donors with a variety of anti-C and anti-

CD sera in order to investigate the nature of C and the possible incidence of Allen's G factor. The material consisted of Bantu donors bled during 1959, and, in order to study antibody formation, a series of D-negative Bantu ante-natal patients was also included. In the preliminary stages we referred a number of specimens to Dr. A. E. Mourant for his opinion and we are grateful to him not only for investigating these specimens but also for putting us in touch with Dr. Philip Sturgeon of Los Angeles, who sent us a copy of his paper on the C variant in Negroes and a hitherto unpublished paper extending his original studies; he also gave us a supply of his key serum anti-CN, which we were able to use for the last five months of the investigation.

The specimens were obtained from healthy male and female Bantu donors and ante-natal cases. We screen all specimens in tubes with two anti-D sera, one complete and one incomplete. Negatively reacting specimens are tested further with from two to four additional anti-D sera, after which a check for D^u variants is carried out by the use of two or three incomplete sera and the indirect antiglobulin technique. The D negatives are screened with saline anti-CD and anti-E sera. Those reacting with anti-CD were tested with a panel of anti-C and anti-CD sera and a small series with anti-C^w. The last 64 Cde donors were also tested with Sturgeon's anti-CN.

A total of 10,173 Bantu blood donors was investigated of whom 488 proved to be D-negative, and of these 98 gave strongly positive reactions with saline anti-CD sera. On further testing all 98 specimens gave strongly positive reactions with panels of saline anti-C + D sera and sera which contained saline anti-C + albumin anti-D; variable results were obtained with saline anti-C, two giving negative results while three others gave a proportion of positives. No positive results were obtained with anti-C^w. The really significant finding was that not one of the 64 Cde donors reacted with anti-CN serum although controls on Europeans gave positive results. This is in agreement with Sturgeon's findings on Negroes. In order to find out whether there was an antigenic difference between the C of Cde persons and the C of CDe persons, 100 consecutive Rh D-positive Bantu donors were tested for the C antigen; 16 type CDe were found and of these 1 gave a positive reaction with anti-CN and also strongly positive reactions with the panel of test sera as in the case of European controls, but those negative with anti-CN behaved as the Bantu Cde type, and it would seem that the Bantu CDe group consists of two types of C, a very small minority conforming to the Caucasian type and the great majority belonging to Sturgeon's Negro variant. One is tempted to speculate that the fifteen anti-CN negatives belonged to the genotype C^{nde}/cDe, while the one anti-CN positive was of the type CDe/cde and, furthermore, that C is a characteristic of Caucasians and not of Bantu.

Of the 365 consecutive ante-natal D negative cases, 50 gave strong reactions with anti-CD sera and 30 of those tested with anti-CN were all negative. Of 21 D-negative sensitized Bantu females with Rh antibodies, 5 belonged to the 'Negro' type Cde; two of these have not yet been delivered, in two cases the babies required exchange transfusion, and the fifth case was a patient in the country whose baby was severely jaundiced.

In summary, our findings show that of 853 consecutive Bantu D-negative persons, 148 belonged to