families segregating for at least three distinct molecular species of serum albumin. Additional evidence indicating considerable gene flow in the south-west is the high frequency of the transferrin variant B_{0-1} present among the Navajo[10] and an uncharacterized B group variant high in frequency among the Pima (unpublished observations).

We thank Thomas Hoff and Carol Olson for technical assistance and the Division of Indian Health and the Tribal Council of the Gila River Indian Community for their help.

HERBERT F. POLESKY
Minneapolis War Memorial Blood Bank,
Minneapolis, Minnesota.

DWIGHT A. ROKALA
Department of Anthropology,
University of Minnesota,
Minneapolis, Minnesota.

THOMAS A. BURCH
Clinical Field Studies Unit,
National Institute of Arthritis and Metabolic Diseases,
Phoenix, Arizona.

Received July 22, 1968.

[1] Polesky, H. F., and Rokala, D. A., *Nature*, **216**, 184 (1967).
[2] Melartin, L., and Blumberg, B. S., *Science*, **153**, 1664 (1966).
[3] Melartin, L., *Acta Path. Microbiol. Scand.*, **191** (1967).
[4] Bell, H. E., Nicholson, S. F., and Thompson, Z. R., *Clin. Chim. Acta*, **15**, 247 (1967).
[5] Weitkamp, L. R., Shreffler, D. C., Robbins, J. L., Drachmann, O., Adner, P. L., Wieme, R. J., Simon, N. M., Cooke, K. B., Sandor, G., Wuhrmann, F., Braend, M., and Tarnoky, A. L., *Acta Genet. Stat. Med.*, **17**, 399 (1967).
[6] Melartin, L., Blumberg, B. S., and Lisker, R., *Nature*, **215**, 1288 (1967).
[7] Comess, L. J., Bennett, P. H., and Burch, T. A., *New Engl. J. Med.*, **277**, 894 (1967).
[8] Jenness, D., *Indians of Canada*, third ed., bulletin No. 65, Anthropol. Series 15 (Nat. Mus. Canada, 1955).
[9] Oliver, S. C., *Ecology and Cultural Continuity as Contributing Factors in the Social Organization of the Plains Indians*, Univ. Calif. Pub. Amer. Arch. Ethnol., 48 (Univ. Calif. Press, 1962).
[10] Parker, W. C., and Bearn, A. G., *Science*, **134**, 106 (1961).

Genetic Polymorphism of the Complement Component C′3 in a Bantu Population

A GENETICALLY determined heterogeneity (doubling) of the human complement component C′3, which can be detected by agar gel electrophoresis, has been described in two unrelated families of Flemish stock in Belgium[1,2]. The frequency of this anomaly seems to be extremely low and no clear relationship with a diseased state has been detected. A similar anomaly which does not seem to be genetically determined has also been described[3–5]. The frequency of this anomaly is somewhat greater than the other and its occurrence has been correlated with the presence of neoplastic disease[6].

We describe here the results of similar investigations of a chiefly Bantu population living in Rwanda. Agar gel electrophoresis was applied to the separation of human serum within 12 h of collection using a procedure which has been described before (1 per cent Difco Noble agar in barbital buffer, *p*H 8·4, $\mu = 0{\cdot}05$). We analysed the sera of 688 individuals (aged 0·5–60 yr) attending the University Hospital of Butare. Whenever a duplication was found the individual was reinvestigated—blood was re-drawn and as extensive a family study as possible was made.

We found twenty-four cases of electrophoretic doubling of C′3. In three others doubling was highly probable, although we could not carry out cross-checking. Separation was easily achieved without any special addition to the barbital buffer, even with an electrophoretic run lasting only 30 min (at 20 V/cm with a gel temperature of 15° C). The duplication appeared either as two closely

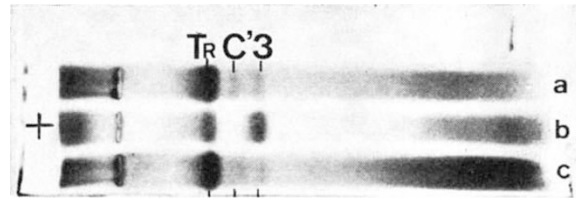

Fig. 1. Electrophoretic separation of C′3 in agar gel. Two cases of doubling (*a* and *c*) are compared with a normal sample (*b*). Tr, Transferrin.

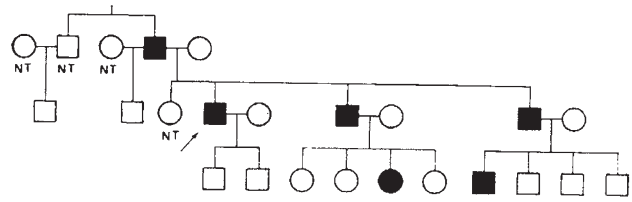

Fig. 2. Typical pedigree of a family with C′3 doubling. ○, Female; □, male; arrow, propositus; NT, not tested; ■ ●, C′3 duplicated.

spaced, very narrow and weak zones or as a rather weak and blurred band with an indication of separation. Prolonged electrophoresis (60 min, other conditions unchanged) produced two zones, always completely separated (Fig. 1).

The supplementary protein always migrated on the anodic side of the conventional C′3 zone. Its identification as C′3 seems justified by a comparison with previous results obtained from a Caucasian population[6], and because of the lability of the supplementary protein which disappeared synchronously with the conventional C′3. We were not able to apply an immunoelectrophoretic identification technique using the Laurell type of electrophoresis, which seems to be indispensable in such a case[2].

For eleven of the twenty-seven individuals with a doubling of C′3 a pedigree could be established. In each case it was possible to see genetic transmission of the autosomal codominant mode. Fig. 2 shows a typical pedigree.

The high incidence of duplication is remarkable, at least in this Rwandese Bantu population. The calculated frequency of 3·9 per cent promotes this anomaly to a genetically determined polymorphism. Looking for a possible association with pathology, we made a detailed investigation of the affected families, in which the frequency of the anomaly was very high (thirty-four variants in seventy-five cases examined). We found no correlation with disease. The biological significance of this phenomenon is thus still obscure.

This work was supported by a grant from the National Foundation for Medical Research in Belgium, and was carried out in collaboration with the National University of Rwanda (Laboratory for Human and Veterinary Medicine, Butare).

R. J. WIEME
J. SEGERS

Laboratory for Protein Chemistry,
Department of Internal Medicine,
Academic Hospital,
University of Ghent, Belgium.

Received July 12, 1968.

[1] Wieme, R. J., and Demeulenaere, L., *Nature*, **214**, 1042 (1967).
[2] Wieme, R. J., Demeulenaere, L., and Segers, J., *Prot. Biol. Fluids* (Proc. Fifteenth Colloq., Bruges), **15**, 499 (Elsevier, Amsterdam, 1968).
[3] Wieme, R. J., *Studies on Agar Electrophoresis*, thesis, 321 (Arscia, Brussels, 1959).
[4] Wieme, R. J., *Agar Gel Electrophoresis*, 238 (Elsevier, Amsterdam, 1965).
[5] Alper, C. A., and Propp, R. P., *Clin. Res.*, **15**, 291 (1967).
[6] Wieme, R. J., *Acta Clinica Belg.* (in the press).