

Arginine does not inhibit ADP's effect and, if one adds both arginine and arginine methyl ester to platelet-rich plasma, the inhibition of ADP is less than with AME alone. The explanation for this observation is not clear, but may reside in the differing affinities of arginine and AME for the postulated enzyme.

The proof that platelet aggregation induced by ADP is an enzymatic reaction must await isolation of the pertinent enzyme and identification of its natural substrate. Meanwhile, the demonstration that this reaction can be blocked by a family of compounds typified by arginine methyl ester provides stimulus for further experiments.

Certain of the compounds tested were furnished through the kindness of Dr. Sol Sherry. This work was supported by grants from the U.S. Public Health Service and the American Heart Association. We thank Dr. G. Mechanic for his advice.

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- ¹ Zucker, M. B., and Borelli, J., *J. App. Physiol.*, **14**, 575 (1959).
- ² Born, G. V. R., and Cross, M. J., *J. Physiol.*, **166**, 29P (1963).
- ³ Born, G. V. R., *J. Physiol.*, **162**, 67P (1962).
- ⁴ Lineweaver, H., and Burk, D., *J. Amer. Chem. Soc.*, **56**, 658 (1934).
- ⁵ Gaarder, A., Jonsen, J., Laland, S., Hellem, A., and Owren, P. A., *Nature*, **192**, 531 (1961).
- ⁶ Shermer, R. W., Mason, R. G., Wagner, R. H., and Brinkhous, K. M., *J. Exp. Med.*, **114**, 905 (1961).
- ⁷ Bounameaux, Y., *Rev. franc. d'etudes clin. et biol.*, **4**, 54 (1959).
- ⁸ Käzer-Glanzmann, R., and Lüscher, E. F., *Thromb. Diath. Hæm.*, **7**, 480 (1962).
- ⁹ Grette, K., *Acta Physiol. Scand.*, **56**, Supp. 195 (1962).
- ¹⁰ Schwert, G. W., and Takenaka, Y., *Biochim. Biophys. Acta*, **16**, 570 (1955).
- ¹¹ Salzman, E. W., *J. Lab. Clin. Med.*, **62**, 724 (1963).
- ¹² Nilsson, I. M., Blombäck, M., Jorpes, E., Blombäck, B., and Johansson, S. A., *Acta Med. Scand.*, **159**, 179 (1957).
- ¹³ Salzman, E. W., and Britten, A., *Fed. Proc.*, **23**, 239 (1964).

Abnormal Hæmoglobin in Sheep

IN man there exists a large number of abnormal genetically determined hæmoglobins. To our knowledge no abnormal hæmoglobins are yet reported found in domestic animals. When investigating the hæmoglobin phenotypes of experimental sheep with the technique of starch-gel electrophoresis an animal having atypical hæmoglobin was found.

This abnormal type is shown in Fig. 1, which is a photograph of a gel stained with amido-black and where samples representing the two normal sheep hæmoglobin phenotypes AB and AA have been run for comparison. We have in this communication used the nomenclature of Evans *et al.*¹. For better demonstration of the two bands in the type AB this sample was less concentrated than the other three shown in Fig. 1. The abnormal hæmoglobin type is shown on the right of Fig. 1. It was first found in a sample from a 7-months-old lamb and appeared as a band having a rate of migration on starch gel which was markedly slower than that of the B band. This special band was of approximately the same strength as the A band and has preliminarily been named N (for Norway). The lamb with this band had been heavily invaded by intestinal parasites during the last three months, primarily *Haemonchus contortus*. Accordingly the animal was anæmic, having a hæmoglobin value of only 7.1 g/100 ml. The lamb had lost 5.2 kg in weight in the last three months and had a considerably lower live weight than the average of the lambs in the control group. The parents of the lamb were of hæmoglobin phenotypes AA and AB, respectively. Testing of the lamb was repeated after a few days on a new blood sample and with the same result. However, when investigating another blood sample four weeks later, which is also shown in Fig. 1, the N band was very faint. Unfortunately the death of the lamb prevented any further investigations.

A year later another group of experimental lambs was hæmoglobin typed. One 7-months-old animal was found which also had the N band. This is shown in Fig. 2, which is a photograph of a gel stained with benzidine and where samples representing the three normal sheep hæmoglobin phenotypes were run for comparison. This animal was also anæmic, due to parasite invasion (mainly *Haemonchus contortus*). The lamb has been tested every week for 2 months. Its hæmoglobin value averaged 4.9 g/100 ml. This lamb has not gained any weight during the last four months, whereas the control group showed an average increase of 6.9 kg. The strength of the N band varied from approximately 2 per cent to 15 per cent of total hæmoglobin.

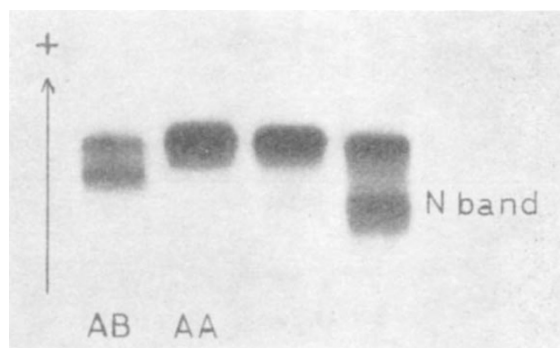


Fig. 1. Photograph of a stained gel showing two examples of the abnormal hæmoglobin type in sheep

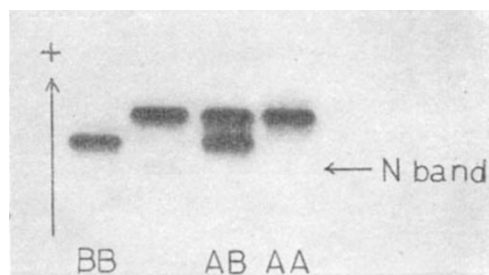


Fig. 2. Comparison between a third example of the abnormal type and the three normal sheep hæmoglobin phenotypes

Recently we have also found the abnormal hæmoglobin type in adult sheep used in special feeding experiments. They did not, however, have any abnormally high number of intestinal parasites.

A definite explanation for the occurrence of this abnormal hæmoglobin type cannot be given at this stage. It is perhaps not genetically determined in the same sense as the many abnormal human hæmoglobins. A condition showing deviations in relative amounts of the two hæmoglobin components has been found in strongly anæmic sheep of phenotype AB by Huisman *et al.*² and Efremov³. Of special interest, in our opinion, would be the possibility of producing the abnormal hæmoglobin type N in sheep experimentally.

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¹ Evans, J. V., King, J. W. B., Cohen, B. L., Harris, H., and Warren, F. L., *Nature*, **178**, 849 (1956).

² Huisman, T. H. J., van Vliet, G., and Sebens, T., *Nature*, **182**, 171 (1958).

³ Efremov, G. D., thesis, Univ. Beograd (1963).