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HAEMOSTATIC AND HAEMORHEOLOGICAL ABNORMALITIES IN TYPE II HYPERLIPOPROTEINAEMIA. B.M. McArdle, G.D.O. Lowe, M.M. Drummond, K.A. Whigham, P. Stromberg, A.R. Lorimer, C.D. Forbes and C.R.M. Prentice. University Departments of Medicine, Biochemistry and Cardiology, Royal Infirmary, Glasgow, Scotland.

Thirty subjects with type II hyperlipoproteinaemia had significantly higher levels of the three major determinants of blood viscosity (haematocrit, fibrinogen and alpha-2-macroglobulin), the two major inhibitors of plasmin (fast antiplasmin activity and alpha-2-macroglobulin), and plasminogen, compared to 30 controls matched for age, sex and smoking habit. Fibrinogen, fast antiplasmin activity and plasminogen correlated significantly with total and LDL (low density lipoprotein) cholesterol. In vitro, purified LDL did not affect these assays. Levels of factor VIII coagulant activity and related antigen, anti-thrombin III, platelet count, platelet aggregation to ADP and adrenaline, and red cell deformability did not differ significantly in the two groups. Haematological variables were unrelated to HDL cholesterol, VLDL cholesterol, or triglyceride. We suggest that these abnormalities might promote arterial disease in type II hyperlipoproteinaemia by three mechanisms - increased blood viscosity, inhibition of fibrinolysis, and arterial wall infiltration by macromolecules (fibrinogen and alpha-2-macroglobulin as well at LDL)

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CONGENITAL DEFICIENCY OF CYCLO-OXYGENASE IN A WOMAN WITH GENERALIZED ARTERIOSCLEROSIS. Z.Boda, E. Tamás, L. Altorjay, Gy. Pflieger, K.Rak. Second Department of Medicine, University Medical School, Debrecen, Hungary

A 52-year old woman with congenital cyclooxygenase deficiency who had moderate bleeding tendency and severe generalized arteriosclerosis is reported.

Diagnosis based on the following data: secondary platelet aggregation induced by adrenaline, ADP, ristomycin, collagen, thrombin was absent; platelet completely failed to aggregate by arachidonic acid while aggregation induced by calcium ionophore A23187 was normal; no malondialdehyde formation could be detected in the platelet rich plasma /PRP/ in four different times /for excluding any drug effects/. The abnormal adrenaline and ADP induced aggregation were not corrected when patient's PRP was mixed in equal proportions with that of a normal subject ingesting aspirin.

Although our patient had been free from severe thrombotic episodes, expressed signs of generalized arteriosclerosis could be detected.

Until now only five cases of congenital deficiency of cyclo-oxygenase have been described /Malmsten et al., Weiss and Lages, Lagarde et al., Pareti et al./. However, in these patients no signs of arterial vascular diseases were mentioned.

The special importance of this new case comes from the fact that life-long deficiency of cyclo-oxygenase enzyme could not protect from progressive vascular disease which might prove again that chronic intake of large doses of aspirin cannot prevent arterial disorder.

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COMPLEMENT C3 POLYMORPHISM IN GREENLAND ESKIMOS. K.A. Jørgensen, J. Dyerberg, E. Stoffersen and G. Nymand. Coagulation Laboratory, Aalborg Hospital, 9100 Aalborg, Denmark.

The $C3^F$ gene has been associated with atherosclerotic vascular disease. Greenland Eskimos have a very low frequency of acute myocardial infarction (AMI). The C3 polymorphism phenotypes were therefore determined by high-voltage agarose gel electrophoresis in 125 Greenland Eskimos originating from different parts of Greenland and compared with a control group of 1,554 Caucasian Danes. The $C3^F$ gene frequency in the Eskimos (0.056) was significantly lower ($p < 0.001$) than in the control Danes (0.201). These results indicate that genetical factors may also play a role for the low AMI frequency seen in Greenland Eskimos.

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INHIBITION OF CHOLESTEROL ATHEROSCLEROSIS IN RABBITS BY AMINO IMINO PROPENE DIACETATE B.I. Weigensberg, S. Katz, J. Lough and R.H. More. Department of Pathology, McGill University, Montreal, Canada.

Previous studies have shown that in vitro blockage of the ϵ amino group of lysine in LDL by acetoacetylation enhances LDL uptake by macrophages and liver cells and inhibits uptake of LDL by fibroblasts in tissue culture. Amino imino propene diacetate (AIPD) is a compound which is absorbed into the blood after feeding and which blocks the ϵ amino group of lysine in LDL for short periods of time after which the AIPD dissociates from LDL and is excreted in the urine. AIPD does not inhibit absorption of either ^{14}C labelled or unlabelled cholesterol from the gastrointestinal tract. In a preliminary study it was found that 19 rabbits fed 300 mg crude AIPD along with 500 mg cholesterol per day in their food for 14 weeks showed 13.0 ± 3.3 (mean $\pm$ S.E.) % of their total aortic intimal surface area covered with grossly visible atherosclerotic plaques. In comparison 13 control rabbits fed 500 mg cholesterol per day in their food for 14 weeks showed 33.9 ± 6.0% of their total aortic intimal surface area covered with atherosclerotic plaques. ($P < 0.01$) These in vivo observations suggest that the inhibition of atherosclerosis by AIPD might be related to a reduction in the number of free lysine amino groups in LDL. In previous studies, we reported that dietary induced lysine deficiency could inhibit atherosclerosis. These and other studies suggest that blocking or reducing the number of free lysine ϵ amino groups in LDL appears to enhance LDL uptake and clearance by liver and macrophage type cells and reduces LDL uptake by arterial smooth muscle cells and fibroblasts in vivo. The combination of these effects appears to offer some protection against the development of cholesterol atherosclerosis.