family Muscidae have a diploid complement of 2 n = 10. 4 of the 7 species in the tribe Phaoniini that have been studied cytologically, have 2 n = 10 (Boyes and van Brink[3]) while the remaining have 2 n = 12. The 2 n = 10 complement of *P. heterochaeta,* however, does not resemble those of the other 2 n = 10 species. In *P. heterochaeta* 2 n = 10 complement has very short pair I chromosomes, and an apparently large pair V chromosome, whereas the other 3 species have medium sized, metacentric pair I and their pair V chromosomes are not large.

The presence of both 2 n = 12 and 10 complements in the tribe is rather interesting. It seems that as far as chromosome number and form is concerned the tribe has retained a flexibility and is not, as yet, completely fixed at 2 n = 12 level, but has produced karyotypes which may represent more specialized states.

1 Acknowledgments. Thanks are due to Dr Adrian C. Pont of British Museum (Natural History), London for identifying our specimens through the courtesy of Dr Rokuro Kano, Dean, Faculty of Medicine, Tokyo Medical and Dental University, Tokyo, Japan. We also thank Dr U.S. Srivastava, Professor and Head, Dept. of Zoology, Univ. of Allahabad for providing the necessary laboratory facilities.

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## Studies on the late-pre-β-lipoprotein of human serum[1]

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*Summary.* Very low density lipoprotein (VLDL) isolated from 3 healthy normolipidaemic subjects had a raised VLDL cholesterol to triglyceride ratio. The VLDL fractions gave 2 pre-β-bands on agarose gel electrophoresis. Family study of the subjects appears to indicate sex linkage of this trait and a possible polygenic type of inheritance.

In a previous study[2,3] we demonstrated that VLDL composition (in terms of cholesterol to triglyceride ratio) is high in type III and type IIb hyperlipoproteinaemic subjects. During a population study[4], however, 3 healthy normolipidaemic subjects were observed to possess a raised VLDL cholesterol to triglyceride ratio ($\geq 0.38$). These 3 subjects were therefore studied in detail as regards lipid and lipoprotein concentration as well as family disposition to the trait.

*Materials and methods.* Subjects: The 3 probands M.R. (a 28-year-old mechanical engineer), F.P. (a 44-year-old plumber) and E.A. (a 59-year-old house-wife) were healthy and had normal lipid concentrations. None was taking any drug known to affect lipid metabolism.

Analysis. VLDL precipitation and measurement was done as reported previously[5]. Preparative ultracentrifugation was done according to the method described by Carlson[6]. Agarose gel electrophoresis was made according to Noble[7]. Blood was obtained after 12–14 h fast. Accessible first degree relatives (parents, siblings and children) of the probands were investigated for lipid and lipoprotein concentrations.

VLDL cholesterol: Triglyceride ratio in a normal population is $0.32 \pm 0.06$ (mean ± SD). *Results.* Lipoprotein and lipid concentration. The 3 probands had normal lipid concentrations in serum, VLDL, LDL and HDL. Their VLDL cholesterol to triglyceride ratios were however high.

Electrophoresis: Figure 1 shows the electrophoretic pat-

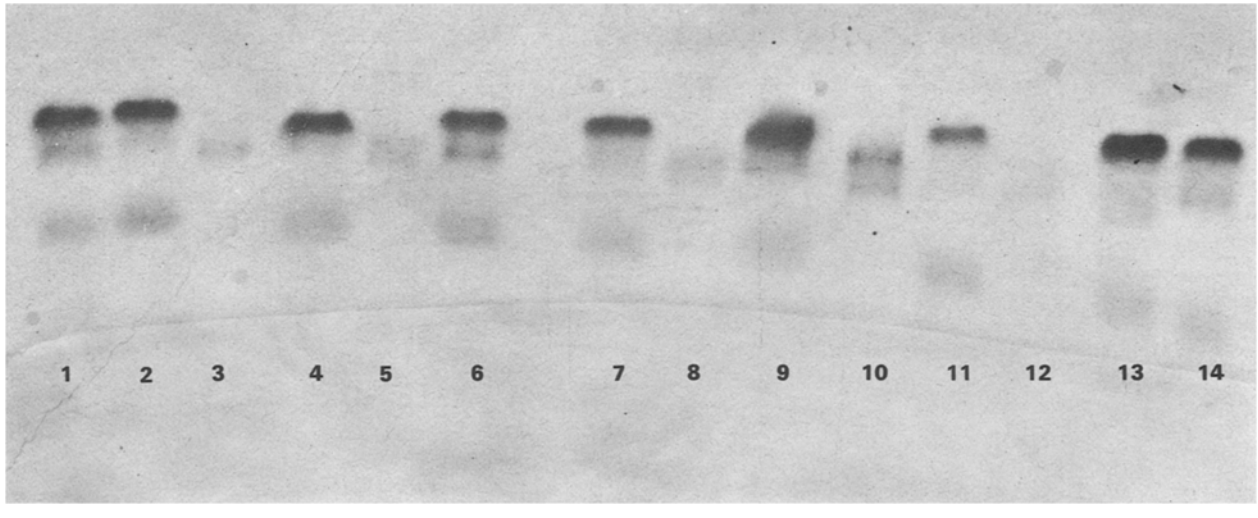


Fig. 1. Agarose gel electrophoresis showing the abnormal VLDL. Numbers 1, 2, 6, 11, 13 and 14 normal sera and numbers 3 and 12 are normal VLDL. Numbers 4, 7, 9, and 5, 8, 10 are sera and VLDL respectively of affected subjects, E.A., M.R., and F.P. respectively.

Table 1. Lipid and lipoprotein concentrations of probands

| Subject | Serum | | Very low density lipoprotein (VLDL) | | | Low density lipoprotein (LDL) | | High density lipoprotein (HDL) | |
|---|---|---|---|---|---|---|---|---|---|
| | Cholesterol | Triglyceride | Cholesterol | Triglyceride | Cholesterol/ triglyceride | Cholesterol | Triglyceride | Cholesterol | Triglyceride |
| M.R. | 232 | 112 | 30 | 68 | 0.44 | 150 | 34 | 57 | 7 |
| F.P. | 215 | 126 | 37 | 80 | 0.46 | 121 | 30 | 53 | 19 |
| E.A. | 230 | 138 | 35 | 78 | 0.45 | 145 | 41 | 59 | 21 |

Concentrations in mg/100 ml.

Table 2. Lipid and lipoprotein concentrations of relatives

| Subject | Serum | | Very low density lipoprotein (VLDL) | | Low density lipoprotein (LDL) | | High density lipoprotein (HDL) | |
|---|---|---|---|---|---|---|---|---|
| | Cholesterol | Triglyceride | Cholesterol | Triglyceride | Cholesterol | Triglyceride | Cholesterol | Triglyceride |
| P.J. (sister of M.R.) | 252 | 75 | 12 | 36 | 152 | 27 | 73 | 11 |
| F.R. (father of M.R.) | 275 | 159 | 20 | 71 | 186 | 41 | 80 | 16 |
| L.R. (mother of M.R.) | 279 | 115 | 16 | 50 | 190 | 38 | 80 | 14 |
| D.R. (brother of M.R.) | 270 | 160 | 35 | 82 | 196 | 58 | 41 | 21 |
| M.P. (wife of F.P.) | 209 | 84 | 16 | 50 | 113 | 23 | 73 | 10 |
| L.P. (son of F.P.) | 120 | 66 | 10 | 33 | 52 | 25 | 46 | 10 |
| C.P. (son of F.P.) | 166 | 106 | 16 | 58 | 77 | 40 | 69 | 15 |
| S.P. (daughter of F.P.) | 163 | 53 | 8 | 30 | 72 | 18 | 78 | 8 |
| T.P. (son of F.P.) | 197 | 58 | 8 | 30 | 108 | 21 | 80 | 9 |
| G.P. (brother of F.P.) | 209 | 102 | 31 | 62 | 135 | 23 | 48 | 17 |

Concentrations in mg/100 ml.

terns of serum and VLDL fraction of these subjects with raised VLDL cholesterol to triglyceride ratio. The direction of run is from top to bottom.

Instead of 1 electrophoretic band, 2 bands were obtained with the VLDL fraction separated at density less than 1.006 in the preparative ultracentrifuge. One of the bands had a characteristic pre-$\beta$-mobility while the other had a mobility between $\beta$ and pre-$\beta$-lipoproteins. Numbers 1, 2, 6, 11, 13 and 14 are normal sera and numbers 3 and 12 normal VLDL from healthy controls. Numbers 4, 7, 9 and numbers 5, 8 and 10 are the sera and VLDL respectively of the 3 subjects, E.A., M.R., and F.P. respectively, with raised VLDL cholesterol to triglyceride ratio.

Family studies: The families of 2 (M.R. and F.P.) of the 3 probands were available for study. The lipid concentrations of the relatives are given in table 2. The pedigrees of the 2 families are represented in figure 2.

Family of M.R.: His father (F.R.) is well at 76 years. He has a raised serum cholesterol but normal serum triglyceride concentrations. The VLDL cholesterol to triglyceride ratio is normal. His mother (L.R.) at 59 years has cornea arcus but no xanthomas, angina or claudication. She has raised serum cholesterol level. The VLDL cholesterol to triglyceride ratio is normal. His sister (P.J.) at 35 years is well and has normal serum cholesterol, normal serum triglyceride concentrations and normal VLDL cholesterol to triglyceride ratio. His brother (D.R.) at 32 years is well with raised serum cholesterol level and raised low density lipoprotein cholesterol level. VLDL cholesterol to triglyceride ratio is also raised.

Family of F.P.: The father of F.P. died of coronary thrombosis at the age of 70. His mother is well at 67 years but was not available for study. His brother (G.P.) at 57 years has normal serum cholesterol and triglyceride levels. His VLDL cholesterol to triglyceride ratio is however high. His children (L.P., S.P., C.P., and T.P.) have normal lipid and lipoprotein concentrations for their age. Their VLDL cholesterol to triglyceride ratios are normal. His wife (M.P.) at 39 years is well with normal lipid and lipoprotein concentrations. Her VLDL cholesterol to triglyceride ratio is normal.

*Discussion.* Carlson and Ericsson[8] and Carlson and Carlson[9] reported the occurrence of a $\beta$-VLDL in a normal healthy population. They referred to this abnormal VLDL as late-pre-$\beta$-lipoprotein because of its slow mobility in zone electrophoresis. Also by zone electrophoresis on cellulose acetate, a slow moving pre-$\beta$-lipoprotein called pre-$\beta_1$-lipoprotein[10], dense pre-$\beta$-lipoprotein[11] and additional pre-$\beta$-lipoprotein[12] has been reported in a healthy population. This trait is beleived[13] to be under genetic control. However, a slow pre-$\beta$-lipoprotein band appears to represent more than one entity.

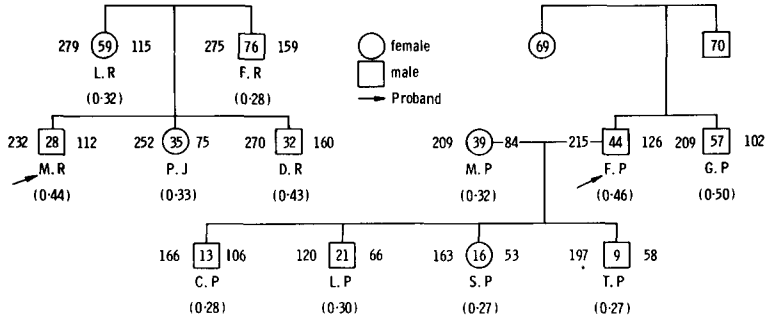


Fig. 2. Pedigree of 2 families in which probands have high VLDL cholesterol; triglyceride ratio and slow moving VLDL component on agarose electrophoresis. Figures inside, left side and right side of block represent age in years, serum cholesterol and triglyceride in mg/100 ml respectively. Figures in brackets represent VLDL cholesterol; triglyceride ratio.

1 source of such a band is the presence in high titre of the Lp (a) - like variant[14] which is a genetically determined trait[15]. This variant is poor in triglyceride and has a density of 1.05-1.09. This is, however, distinct from the slow pre-$\beta$ material of density < 1.0006 found in the 2 families reported here. Data from the present study indicate that this trait is sex-linked. There is also indication that families of probands with this VLDL of 'abnormal' composition are susceptible to hyperlipidaemia, especially of hyper-cholesterolaemic nature. Only one of the 1st degree relatives of each proband was affected by this abnormal VLDL composition. This gives the proportion of affected first degree relatives in the F.P. and M.R. families as 20% and 25% respectively. This low proportion of affected 1st degree relatives suggests according to Falconer's postulate[15], a polygenic type of inheritance. Further family studies have, at any rate, to be carried out to elucidate this mode of inheritance.

°1 Acknowledgment. This investigation was supported by a grant from University of Nigeria, Nsukka. I am grateful to Prof. Barry Lewis, St. Thomas Hospital Medical School, London, for his advice.

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## Bisalbuminemia in a bottlenosed dolphin *(Tursiops truncatus)*

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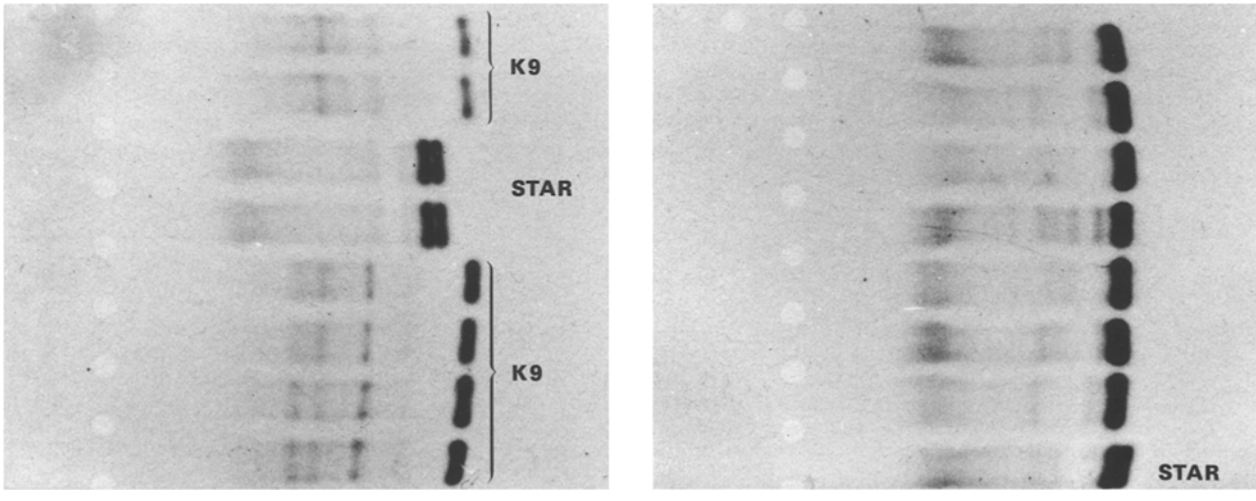
*Summary.* Bisalbuminemia was found in a female bottlenosed dolphin *(Turiops truncatus)* on routine examination. There is no association with disease.

On routine electrophoretic examination of blood sera from a number of dolphins *(T. truncatus)*, 1 animal was found to have 2 albumins (Bisalbuminemia) (figure). The animal (Star) with this anomaly is a female believed to be between 10 and 12 years of age. She was captured in January, 1977 in the Gulf of Mexico. There is no association, to our knowledge, of this finding with disease in this animal. Bisalbuminemia is an inherited blood protein disorder associated with 2 serum albumins differing in their physical responses to electrophoresis. Many studies have been done to show similarities and dissimilarities between the slow-moving albumin B and the fast-moving albumin A. In 1 study[2] the addition of $I^{131}$ thyroxine to bisalbumin sera resulted in thyroxine-binding by albumin B (slow-moving) but not by albumin A. The failure of albumin A to bind added $I^{131}$ thyroxine led to the speculation that, in the family reported, neither albumin A nor B are identical to normal human albumin.

In another study, the albumins were separated in the Tiselius apparatus at pH 8.6. The albumins could not be




Cellulose acetate electrophoretic separation of serum proteins of the affected animal as well as from many other dolphins for comparison. The bifid albumins in 'Star' can be seen easily. The dog sera separations are for comparative purposes only.