CHAPTER IV

DISCUSSION

Finding of a mean value ± S.D. of serum FABP as 30.76 ± 11.82 pg/ml in 175 blood donors in the present study was in the same order of magnitude as results reported by many authors as shown in table 15. Waxman and Schreiber (1973a) reported a mean serum FABP level in 25 normal subjects as 45 pg/ml. Eichner et al., (1975) also found a similar values of FABP in 38 normal subjects whem 50% were men. They reported the higher value of serum FABP in normal female subjects than those of normal male subjects which was probably due to the hormonal effect. However, these values showed no statistically significantly difference. Colman and Herbert (1976) found a slightly higher value of serum FABP in 20 healthy male and female subjects than that of the present study. They also reported that 3 of 20 normal subjects had serum FABP level higher than 125 pg/ml. Retief et al., (1976) found that the mean FABP in 10 normal sera was 10 pg/ml (ranged from 0 to 40 pg/ml). Gorst et al., (1976) reported that the level of serum FABP in 85% of 94 normal subjects ranged between 20 to 150 pg/ml, with a mean value of 76 pg/ml. The FABP level in 5 normal female subjects was greater than 300 pg/ml.

Results in the present study showed that there was no significant difference between the mean value of serum FABP in male subjects

 $(30.59 \pm 10.13 \text{ pg/ml})$ and in female subjects $(30.91 \pm 13.28 \text{ pg/ml})$. This finding confirmed the result reported by many authors as cited above.

Table 15. The FABP concentrations in normal subjects and Thai blood donors

No.	No. of cases studies	FABP level (pg/ml)		References
		range	mean + S.D.	
1	25	2	45.0	Waxman and Schreiber
				(1973a)
2	38	3.5 - 72.5	21.5	Eichner et al., (1975)
3	10	0 - 40.0	10.0	Retief et al., (1976)
4	20	-	61 <u>+</u> 11	Colman and Herbert,
				(1976)
5	80	20.0 - 150.0	76.0	Gorst et al., (1976)
6	175	12.0 - 72.63	30.76 <u>+</u> 11.82	Present study

A mean value ± S.D. of serum FABP in 45 patients with malaria was found to be 174.40 ± 88.20 pg/ml. This figure was significantly higher than that of the normal subjects. It has been shown that the folic acid deficiency could occur in patients with malaria which was possibly due to an increased utilization of folic acid from hemolysis and fever (Chanarin et al., 1959; Lindenbaum and Klipstein, 1963; Strickland and Kostinas, 1970). This was confirmed by the finding of low serum folate level in the patients with malaria (Strickland and Kostinas, 1970; Areekul et al., 1977). Findings of an increased serum FABP level in patients with malaria may be due to the compensation for the body to bind more folic acid to match the increased requirement in these patients.

The mean serum FABP in patients with pyrexia of unknown origin (PUO) was significantly higher than that of the normal sera. The fever in these patients may play a role as in malaria (Chanarin et al., 1959; Lindenbaum and Klipstein, 1963). The other mechanism may be due to the elevation of white blood cells which caused the increased serum FABP as shown in patients with chronic myelocytic leukemia (Rothenberg and da Costa, 1971; da Costa and Rothenberg, 1974a; Gorst et al., 1976).

The mean value of serum FABP in patients with anemia was significantly higher than that of the normal subjects. A poor dietary intake, an increased utilization of folic acid for increased synthesis of red blood cells may result in low serum folate levels in these patients (Margo et al, 1977; Chanarin et al., 1959; Lindenbaum and Klipstein, 1963). The increased serum FABP may therefore be a compensating mechanism

of the body to bind more folic acid in order to keep more folic acid in the circulation.

In the present study, the mean serum FABP in patients with cirrhosis of liver was significantly higher than that of normal sera. Waxman and Schreiber (1973a) reported that the mean value of FABP in these patients was lower than that of the normal subjects, while Colman and Herbert (1976) reported the higher value in these patients. Patients with cirrhosis usually had low serum folate level which was probably due to a dietary deficiency, and an increased excretion of formiminoglutamic acid in urine (Deller et al., 1965; Retief and Huskisson, 1969; Herbert and Zalusky, 1961; Knowles et al., 1963; Areekul et al., 1977).

Serum FABP in patients with hepatitis, jaundice, hepatomegaly, amoebic liver abscess, opisthorchiasis and carcinoma of liver were also significantly higher than that of the normal subjects. A poor dietary intake and hemolysis, liver cell damage could cause the lower serum folate in these patients (Herbert et al., 1963; Klipstein and Lindenbaum 1965; Retief and Euskisson, 1969; Magnus, 1975; Wu et al., 1975; Areekul et al., 1977). The increased excretion of folic acid in urine in these patients may also be another cause of low serum folate (Retief and Huskisson, 1969). An increased serum FABP in these patients may be the compensating mechanism as in the other diseases.