Since this note was submitted in September 1955, there have been rapid developments in the field of haemoglobinopathies: the condition DD has been discovered, and another doubly heterozygous condition (AH+one thalassaemia gene) has been described. More knowledge on the world distribution of the abnormal haemoglobins has been accumulated. Haemoglobin D has been found at a low but regular incidence in north-west Indians and in Gujeratis from what used to be the Bombay Presidency. Haemoglobin E has been traced in more populations in South-East Asia, and, more recently, haemoglobins such as H and J have been found in some populations at a definite though low frequency. More work on the connexion between sickling and malaria has been carried out in Greece, and in French African possessions. Of particular interest for Africa are perhaps the following investigations. Colbourne & Edington [t] have extended their survey from the southern Gold Coast to the northern Gold Coast, an area where haemoglobin C is more frequent than haemoglobin S.[u] In contrast to their observations in the south, no negative correlation between malaria and sickling could be demonstrated in the north. One possible explanation is that, as we have suggested above, haemoglobin C has to be taken into account in such surveys. In East Africa, Raper [v] has brought some initial positive proof supporting the hypothesis that children who are heterozygous for the sickling gene have a greater chance of surviving than normal children when both are exposed to malarial infection. In Kampala, nearly ten times as many non-sicklers than sicklers were admitted to the children's ward for treatment of malaria. Of the 13 sicklers all suffered from uncomplicated malaria; of the 123 non-sicklers, only 70; of the others, 47 had cerebral malaria, and 6 blackwater fever.

[u] Edington, G. M. & Lehmann, H. (1956) *Man*, **56**, 34
[v] Raper, A. B. (1956) *Brit. med. J.* **1**, 965

The Influence of Malarial Infection of the Placenta on the Incidence of Prematurity *

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In his investigation of the effect of malaria on African infants and children in Southern Nigeria, Bruce-Chwatt [a] noted the influence of malarial infection of the placenta on the weight at birth of 310 African infants. He found that of 73 infants delivered from infected placentae, 15 (20.3%) were premature,[b] as compared with 26 under-weight births from 237 non-

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a Bruce-Chwatt, L. J. (1952) *Ann. trop. Med. Parasit.*, **46**, 173

b According to the WHO definition of prematurity, the weight of a premature infant at birth is 5½ lb. (2500 g) or less (see *Wld Hlth Org. techn. Rep. Ser.*, 1950, **27**, 4).

infected placentae (11%). However, since the difference between the two percentages was below the conventional level of significance, it was concluded that additional research was necessary before the influence of malaria on prematurity could be established. Further observations were made over the period 1950-53, in the course of work undertaken by the Nigeria Malaria Service in Ilaro (Abeokuta Province of Western Nigeria). The findings are analysed in the present note.

The women whose accouchements furnished the material for this investigation lived in Ilaro or its surroundings and were delivered by midwives of the local rural health centre. Records are available of 512 deliveries, during which a smear was taken on a blood-slide from the maternal side of the placenta, the weight of the offspring being registered simultaneously. The smears were stained with Giemsa stain and were examined for the presence of malaria parasites.

In 77 cases (15%), the placental smear showed evidence of malaria parasites—mainly *Plasmodium falciparum*. This percentage was consistent with the parasite-rate for adults in Ilaro during the period of the investigation—namely, 14.5% in 1951, 17.7% in 1952, and 12.7% in 1953.

Of 68, apparently full-term, deliveries from mothers with infected placentae, 20 (29.4%) weighed 2500 g (5½ lb.) or less at birth, and were therefore classed as premature. This percentage contrasts with the incidence of prematurity in 395 single, full-term births from uninfected placentae, since in the latter group only 65 (16.5%) were premature. This difference is unlikely to have been fortuitous ($\chi^2 = 5.7$ and $P < 0.02$).

The data presented below show the frequency of various birth weights in children born from malarious and non-malarious placentae. The average weight of those in the former group is 6 lb. (2722 g $\pm$ 312 g), as compared with a mean weight in the latter of 6 lb. 6 oz. (2892 g $\pm$ 454 g).

Weight at birth (pounds)	*Number from malarious placentae*	*Number from non-malarious placentae*
4	—	3
4½	—	5
5	2	21
5½	18	36
6	19	72
6½	14	77
7	8	80
7½	4	54
8	2	35
8½	1	8
9	—	3
9½	—	1
TOTAL . . .	68	395

It is known that first pregnancies are highly likely to yield premature infants, and this is also true, although to a lesser extent, of pregnancies

coming at the end of large families.[c] Consequently, an analysis of the available material was made on the basis of maternal parity. (See the following data.)

Parity of delivery	*Malarious*		*Non-malarious*	
	premature	*mature*	*premature*	*mature*
Not known	1	1	0	10
1	11	24	30	108
2	3	12	20	71
3	2	7	4	72
4	0	4	5	35
5	2	—	5	18
6	1	—	0	9
7	—	—	0	6
8	—	—	1	1

Notwithstanding the exclusion of first-born children, however, prematurity was seen to be more common in the malarious than in the non-malarious group, as was also the case when consideration was limited to deliveries from second to sixth parities inclusive, where the rate of prematurity is normally at its lowest. Of 31 deliveries from mothers of these parities who had infected placentae, 8 (25.8%) were premature, in comparison with 34 (14.4%) from 239 non-infected mothers of the same parities. Although, in view of the number of cases considered, this difference might have been due to chance, the fact that the respective percentages agree well enough with those established for the group as a whole is sufficient evidence to demonstrate the importance of malarial infection.

In addition to the 463 single, full-term deliveries analysed above, of the 512 pregnancies for which records were kept, 11 births, 4 of them multiple, took place before term and 38 multiple pregnancies were delivered at term. Two of these 42 multiple pregnancies resulted in triplets and 40 in twins.

Discussion. The evidence presented in this paper lends weight to the suggestion that malarial infection of the placenta is a cause of premature birth in Africans of south-western Nigeria. In Ilaro, malaria may account for the premature birth of 2% of all infants.

However, it should be remembered that birth weight varies according to race; for example, Negro babies in the USA weigh considerably less than white infants.[d] It might therefore be objected that, in the African, a birth weight of 5½ lb. or less is not indicative of prematurity. Such an objection loses some of its force when it is remembered that the mortality rate for Negro babies in the USA weighing 5½ lb. or less at birth is much the same as that for premature white infants,[e] although the basis of comparison is unsatisfactory in view of the different economic grouping of whites and Negroes in the USA. It seems likely that the disadvantages known to handi-

[c] Ferguson, A. E., Brown, A. C. & Ferguson, T. (1952) *Glasg. med. J.*, 33, 143

[d] Pomerance, W. & Steiner, M. (1950) *Amer. J. Obstet. Gynec.*, 60, 333

[e] Crump, E. P., Wilson-Webb, C. & Pointer, M. B. (1952) *Amer. J. Dis. Child.*, 83, 463

cap the premature baby in developed communities will operate quite as forcibly on the African born under weight in his natural environment. Such disadvantages include not only a high neonatal mortality risk, but also a predisposition to fatal illnesses which lasts until the age of 4½ years.[f]

At all events, the advisability of using chemoprophylaxis as a means of protecting pregnant women in this area of holoendemic malaria, and thereby of reducing the number of premature births, is well worthy of consideration. But before such a policy is prescribed, its possible effects on the women's immunity must be considered. It is now assumed that the mother's immunity to malaria is transmitted to the newly-born infant,[g, h] and any measure likely to interfere with this mechanism should be adopted with considerable caution. What effect malaria prophylaxis, limited to the period of pregnancy, would have on maternal immunity is at present unknown, but it might well be only slight. Generally speaking, however, immunity to malaria is not destroyed by prophylaxis. As evidence of this, it might be mentioned that a group of Lagos schoolchildren, protected for two years with pyrimethamine, retained their active immunity.[i] The same observation was made in the case of a number of Accra graduates who had spent the years of a university course away from the risk of re-exposure to the disease.[j]

On balance, an attempt to reduce prematurity in an area of high malaria endemicity by antenatal chemoprophylaxis seems worth making; but further research is obviously needed, and it would be desirable if investigations in other parts of Africa would fully assess the magnitude of the influence exerted by malaria on prematurity.

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f Douglas, J. W. B. & Mogford, C. (1953) *Brit. med. J.*, 1, 748
g Bruce-Chwatt, L. J. (1952) *Ann. trop. Med. Parasit.*, 46, 173
h Davidson, G. & Draper, C. C. (1953) *Trans. roy. Soc. trop. Med. Hyg.*, 47, 522
i See article by Archibald & Bruce-Chwatt on page 775 of this number of the *Bulletin.*
j Colbourne, M. J. (1955) *Trans. roy. Soc. trop. Med. Hyg.*, 49, 483

Microsporidia in Laboratory Colonies of Anopheles

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Protozoa and other parasites are reported from time to time as affecting mosquitos both in the immature and mature stage, and, as suggested by Jaswant Singh et al.,[a] the presence of such organisms may give rise to confusion in the identification of oocysts and sporozoites of the malaria parasite. As a rule, the infestation is light and affects only a small minority of the insects, though in the case of *Coelomomyces africanus* the infection can be quite widespread, as Dr A. F. Haddow [b] and I noticed in *Anopheles gambiae* in East Africa.

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a Jaswant Singh et al. (1951) *Indian J. Malar.*, 5, 527
b Haddow, A. F. (1942) *Bull. ent. Res.*, 33, 91