COMMUNITY BASED MALARIA CONTROL THROUGH CHEMOTHERAPY AND CHEMOPROPHYLAXIS IN SARADIDI, KENYA

Thesis submitted in accordance with the requirements

of the University of Liverpool for the degree of

Doctor of Philosophy

by

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CONTENTS

PREFACE

This thesis was based on research supervised by Dr. D.H. Smith of the Liverpool School of Tropical Medicine. The research was funded by the UNDP/World Bank/WHO Special Programme for research and Training in Tropical Diseases (TDR) Laboratory investigations were carried out at the Division of Vector Borne Diseases Laboratory in Kisumu, Nyanza Province, Kenya. The laboratory services were backed up by the Kenya Medical Research Institute, the Clinical Research Centre, Nairobi, Kenya with further assistance from the Parasitic Division of the Center for Disease Control, Atlanta, Georgia, United States of America.

Data analysis was carried out at the Centre for Disease Control for the first part of the study and at the Liverpool School of Tropical Medicine, Department of Tropical Paediatrics, Liverpool.

The main aim of this thesis was to document the process of involving a community in health care activities with particular reference to malaria control through chemotherapy and chemoprophylaxis for pregnant women

The community mobilisation started in 1979 when a baseline community health survey was carried out by the Department of Community Health, University of Nairobi. This was followed by further discussions with the community which led to a complete census and household listing.

Before the chemotherapy and chemoprophylaxis project was started, the sensitivity of the local P.falciparum to chloroquine phosphate was tested in vivo and in vitro. This was done periodically throughout the research period. Various surveys were carried out to determine the use of chloroquine in the area, factors affecting it; the impact of the antimalarials on mortality, morbidity. parasite prevalence and antimalarial antibodies in the community. In 1985 a study was initiated to find out the effectiveness of a monthly chemoprophylactic scheme for pregnant women.

A regular surveillance was kept on the sensitivity of the local

P.falciparum to chloroquine and other commonly available antimalarials. The demographic data were obtained from six monthly updates of the census information which had been compiled in 1980-81. The six monthly updates were complemented by periodic community health surveys which were carried out in 1979, 1982 and 1984.

Some of the findings during the period 1980-1984 were analysed and published as a series of papers in the special issue of the Annals of Tropical Medicine and Parasitology, volume 81, supplement 1, 1987.

Statistical Analysis

Analysis of variance

Chi Square Test (X^2)

Fisher s exact test

Student's t-test

Logistic regression analysis

Computer Programmes Used

Minitab version 5 1

Minitab Inc., 3081 Enterprise Drive, State College, PA 16801, USA

SPSS X

SPSS Inc., Suite 3300, 444 North Michigan Avenue, Chicago, IL 60611, USA

Computer Programmes for the demographic analysis

Washington DC, U.S goverment printing office 1976

Abbreviations

ANC - Ante natal care

CHW - Community health worker

CBR - Crude birth rate

CDR - Crude death rate

CMR - Child mortality rate

IMR - Infant mortality rate

ELISA - Enzyme linked immunosorbent assay

IFAT - Indirect fluorescent antibody test

IGA - Income generating activities

PHC - Primary health care

SD - Standard Deviation

SRHDP - Saradidi Rural Health and Development Programme

VHDC - Village Health and Development Committee

VHH - Village health helper

AGM - Annual general meeting

VHC - Village Health Committee

CAWs - Community agricultural workers

UNICEF - United Nations Children's Fund

WHO - World Health Organisation

ABSTRACT

A community based primary health care and development programme was established in Saradidi, Kenya. The process of implementation which involved the community in identifying their problems and mobilising their resources to address the problems started in November 1979. Leaders and Community Health Workers (CHWs) were identified by the community and trained by resource people invited by the community. Their activities included health education, treatment of malaria and recording vital events.

The CHWs started providing chloroquine to community members who had fever and requested treatment. Soon the CHWs became the main source of chloroquine in Saradidi as a survey showed that 80% of adults and 65% of children needing chloroquine obtained it from the CHWs. Women of child bearing age were the main users. Chloroquine induced pruritus was so frequent in Saradidi (17%), that it interefered with chemoprophylaxis.

Malaria transmission went on all the year but varied from 10% per month in January-March to 65% per month in April-August and tapered off to 10% in September-December.

P.falciparum is the dominant malaria species in Saradidi. In the 7 years of the study there had been a gradual decrease in P.falciparum sensitivity to chloroquine and by 1987, 32% of school children and 80% of children under two years infected would not respond to treatment at 25 mg/kg.

The recommended weekly chemoprophylaxis for pregnant women was difficult to administer and maintain even in a community based PHC system. Only 29.1% used it in Saradidi. Chloroquine phosphate given monthly at a dose of 10 mg/kg at antenatal clinics proved adequate protection against malaria. Women on monthly chloroquine did not suffer febrile illnesses more frequently, or have higher parasite prevalence counts than those receiving weekly chloroquine. All treatment groups had higher parasite prevalence in primigravidae (54.4%) than multigravidae (33.3%) parasite prevalence decreased with gestational age and with increasing parity.

The mean birth weight was lowest for primigravidae (2.91 kg) and particularly those who were not on regular chemoprophylaxis. 15.1% of newborns who had infected placentae had low birth weights as compared to 5.9% for those with parasite negative placentae.

48% of low birth weight babies died in the first year of life. Although their reported causes of death were common to all children, their risk of dying was increased threefold as compared to their normal birth weight counterparts.

Placental parasitaemia was found more frequently among younger women with low parity. Primigravidae had the highest rate; 32.7%, as compared to 15.3% among multigravidae. The women who received monthly chemo-prophylaxis had the lowest rate of placental parasites; 13.3% as compared to 36.1% for weekly and 45.7% for those who were only treated when ill. This difference was most marked among the primigravidae.

65% of mothers with peripheral parasitaemia had placental parasitaemia. This rate was 80% for women who were from outside Saradidi project area. Cord parasitaemia was 10.6% for Saradidi women but much higher (37%) for non-Saradidi women.

55% of the infants had parasites by 6 months and 71% of them by one year. Their nutritional status was good at 6 months, based on their weight for age, weight for height and height for age z scores. By 12 months, these anthropometric measurements were already showing nutritional deficit in comparison to international standards. The downward trend continued up to the end of the third year of life when catch-up growth started slowly.

ACKNOWLEDGEMENTS

I wish to thank Dr. D.H. Smith for his kind encouragement, guidance and support during my field work. data analysis, writing and final presentation of the thesis. I am also indebted to Ms Sara Macfarlane, Mr. Barry Moody and Mrs. Wanda Russell for their patient assistance during data analysis and presentation

Many people participated in the work at Saradidi and could not all be named in the available space, but I wish to thank Dr. Harrison Spencer and Dr. David Brandling-Bennett for their collaboration in the work presented in this thesis. I thank also the field staff: Mr. Gilbert Ongayo Onyango. Mr. Sylvanus Oteku and Mr. Alfred Luoba for their dedication in supervising the women, following up cases. collecting data and specimens and carrying out the investigations and examinations that were required.

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Without the pregnant women and the Community Health Workers in Saradidi this study would have been impossible. I wish to thank them very sincerely for their co-operation and time.

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CHAPTER 1

PRIMARY HEALTH CARE, ITS CONCEPT AND PRACTICE

1.1 GENERAL INTRODUCTION

Saradidi Rural Health Development Programme (SRHDP) as a primary health care, research and development programme has been going on for nearly nine years now. The work that has been done at Saradidi has covered many areas of health care, and general development. It is beyond the scope of this volume to describe in detail all the aspects of the Saradidi programme. I have deliberately limited myself to the studies on malaria control within primary health care with particular emphasis on community based chemotherapy and chemoprophylaxis for pregnant women. The rest of the studies have been described elsewhere (Kaseje et al 1987; and Kaseje and Sempebwa, 1988).

The volume is divided into two sections: the first section (Chapters 1 to 6) deals with the general concept and practice of Primary Health Care (PHC) as applied to the work at Saradidi, but also as it forms a basis for community based malaria control activities. Also discussed is the impact of the community based chemotherapy and limited chemoprophylaxis programme during the period 1979-1987 and the factors that appear to have influenced that impact. The second section (Chapters 7 to 11) deals with the overall evaluation of the impact of the project based on a few selected indicators of health improvement and presents the results of a major trial of three schemes to protect pregnant women from the consequences of malaria during pregnancy using chloroquine as a chemoprophylactic and chemotherapeutic agent.

1.2 THE CONCEPT OF PRIMARY HEALTH CARE

Primary health care was defined as 'essential health care made universally accessible to individuals and families in the community where they live and or work by methods which are scientifically sound yet acceptable to them and through their full participation and at a cost that the community and

government can afford at every stage of its development in the spirit of self-reliance and self-determination' (WHO/UNICEF 1978). This was at the meeting organised by the World Health Organisation and UNICEF at Alma Ata in the USSR and attended by representatives of more than 150 ministries of health from all over the world. The definition is subject to many and varied interpretations and misinterpretations, which is not necessarily a bad thing since no one definition of PHC would suit it for all possible situations and conditions in the world of health care and development today. In this section is described the features that should characterise the implementation of primary health care, the interpretation of its definition according to the condition in which it is implemented. These features are set out below.

1.2.1 Community participation

A distinction was made in 1981 between the phrases 'community participation' and community involvement by the World Health Organisation (WHO 1981). The term community involvement was given preference because 'participation' may involve a mere passive response to imposed services like family planning and other similar services. Whereas involvement implies active participation which must have mechanisms, methods and processes that enable people to become fully involved by taking responsibility for decisions made by themselves and taking actions that would meet their own needs. Health professionals would blend their ideas and skills into this process in such a way that the skills, knowledge and experiences contributed by the people are expressed and utilised to the maximum. It is implied in this, that involvement is not simply donating money, materials and/or time. The people benefitting from a PHC programme should have control over decisions directly affecting them and thus determine the direction these activities must take. This is only possible when the means to bring about the desired results are in the hands of the people. It is difficult to see how the community can be fully involved in

decisions concerning external resources which are usually provided by a support system, e.g. the ministry of health or donor agencies; the people should be able to choose what resources they need, when they need them and for what purpose. They should decide on the objectives to be achieved and plan how they would be achieved. Maximum use of internally generated resources from within the community would allow them an even greater scope for maximum community involvement. When involvement simply means taking control of and or having access to external resources then involvement is often limited to a few individuals, such as the local elite who already wield a lot of power in the community and are the least needy.

Community participation in this sense then means taking responsibility for decision making, for mobilising resources, managing and evaluating activities which meet the health needs of the community as identified and prioritised by themselves.

A PHC programme should seek to empower the whole community to be involved to the extent possible in their situation. This should lead to equitable sharing of power, resources and services. This approach may not always achieve the goal of efficiency and effectiveness of a programme in achieving tangible outcomes of service coverage; it may not even have quantifiable results quickly enough but its results will be deeply rooted in the community and will be sustained indefinitely as they will become part of the culture of the people involved.

The majority of health projects tend to over emphasise the objective of effectiveness i.e. motivating people to use services (Paul 1987). The mechanism for doing this often involves community health workers (CHWs) and health committees. These groups often take over involvement from the rest of the community. Efficiency and effectiveness as defined by the external health providers are over emphasised and makes it difficult to avoid approaches that limit community involvement.

The level to which involvement is possible is further determined by the political system, a factor often taken for granted (Midgley 1986). Various political systems prescribe ways by which their people can influence decisions over important aspects of their own lives. The recent development towards the district focus for rural development in Kenya is one such example. While these methods may be able to mobilise people to action they have also been criticised as methods of social control (David Werner, 1983). Real participation in these systems often leads to conflict with the interest of the state which therefore tends to define the extent to which participation is allowed. One other factor that may affect participation is poverty. Very poor people may find it difficult to participate because they have neither the time nor the resources to free themselves from the demands of day to day living.

Fortmann (1983) makes the statement that many villages are littered with carcasses of moribund committees and organisations, a situation that is true of Saradidi.

In SRHDP, intense voluntary group activity was a key to the success of the health project: women and church groups initiated and undertook many of the health activities and were an important element in sustaining the programme. The main lesson from the project was that it is unnecessary to reorganise the community, and set up new structures: it is best to strengthen and work within the existing leadership and community organisation and structures (Kaseje and Spencer, 1987). The committees which are often set up in response to an external demand, usually do not know their responsibility, neither do they know their role (Navallo, 1987) and their authority is usually non-existent. They usually do not have resources under their contol but where they have money, mismanagement abounds. (Chan et al, 1987). Reasons why there is mismanagement of resources may vary from one programme to another, but must include the following:

a) The committee does not feel accountable to the community or system

that elected them because the resources that they are managing are from a source outside the community. The community have no power over the committee which often would identify more with the external source of resources than with the community.

b) The external support system has inadequate understanding of how things work in the community and usually have no legal or social authority over the committee.

c) The community lose confidence in the committee and they thus are left isolated and to their own selfish designs. The wise among them take advantage of the situations and maximise their own individual gain.

d) There may be a genuine lack of skills and or knowledge on resource management which may be deliberately perpetuated only to plead ignorance when mismanagement is exposed.

It is therefore important that a local community based committee be in charge mainly of resources which are generated or mobilised from within the community. This enhances community awareness of their rights and would ensure committee accountability to the community. The committee members themselves need to have a say in what they are looking after. With all the above suggestions, must be training and regular supportive follow up to enable the committee to manage the resources of the community responsibly.

1.2.2 Equity

Equal access to services and resources and the power to choose is the second concept that goes with PHC. It is to be noted that not the whole community can have equal power to make important decisions at the same time. They can all participate in delegating this power to popularly elected members who are subject to them through mechanisms set up by themselves, with necessary requested inputs from professionals. Those who make decisions would thus be subject and accountable not to some authority structure in the

city but to the community itself. The poor and marginalised members of any community who are often the majority and who find it difficult, to express themselves and to get involved in new activities must be given special consideration by setting up a process that would enable them to participate.

Thus health outcomes in the population that are described even in this volume, are not due solely to the vertical interventions like distribution of chloroquine and health information but they also depend on how the whole of the implementation process is administered. It is also very difficult to relate certain inputs to certain outcomes in an effective way, since inputs are never pure, and so interpretations of results of studies in PHC must be handled with care (Isley, 1984). Political and economic settings of the community will also influence outcomes (McAnamy, 1978).

1.2.3 Appropriate technology

The third concept in PHC which is central but also related to the above is the concept of using the most appropriate means in meeting the needs of the people. This already implies the involvement of the beneficiaries in deciding on the method most appropriate to them. Thus malaria had to be identified as a problem by the people of SRHDP and they have been involved through their elected leaders and community health workers (CHWs) in trying the best approach, to protect pregnant mothers, the most vulnerable group among them from the consequences of malaria.

The technology selected may not be the most effective but, if selected by the community with requested inputs from professionals then it will be successful in the long run since it will be a method with which the people are familiar and can sustain.

1.2.4 Intersectoral co-ordination and collaboration

The equal participation of the community in making decisions that affect

their lives means that the selected package of services by a given community may involve skills and resources that are not found in one sector like the Ministry of Health. The skills of working with the community themselves may not be available in the health sector. It is therefore important that persons from various sectors team up in the process of PHC implementation.

The above concepts imply that in PHC the focus is no longer on the service providers but on the beneficiaries, their needs, their choices and their priorities. To bring about a situation in which beneficiaries are the senior partners in the process of meeting their health needs is not easy and requires skills in communication.

Communication plays a role in providing people with information that they may need in making their choices, providing available alternatives, what change can occur, and providing the methods, information, means and benefits involved in new ideas and ways. O'Sullivan adds that communication may also be needed to teach new skills demanded by choices that the community have made, for example, the training of CHWs in certain skills (O'Sullivan 1974).

Appropriate communication can bring about greater informed participation in health activities if it focuses on those that are most needy and when it does not impose but aims to inform in a process of free dialogue where professionals and community members are both learners (Roger, 1976 and Roling 1976). The end result of this is that the poeple are empowered to have greater control over their environment and can effectively mobilise resources available to them to meet their needs (Rogers and Shoemaker 1971).

Health interventions which are selected by the beneficiaries, and are introduced and implemented in this way would eventually lead to a slow and permanent change towards adopting ways and means that become assimilated into the habitual practice of the people. Havelock concludes that user initiated change is the strongest and most permanent, (Havelock, 1971).

Thus people's participation in planning and implementing a malaria control programme in areas like Saradidi where its eradication is impossible

may be the only strategy which would have a reasonable chance of success. The research activities at Saradidi were aimed at working with the community in finding appropriate technologies and alternatives that could form part of the body of knowledge and skills communicated and developed in the process of community dialogue.

The community in Saradidi included malaria among their priority problems that they wanted to address. They were also looking for ways and methods that would enable them to deal with the major causes of morbidity and mortality among them. They selected community based chemotherapy and chemoprophylaxis limited to pregnant women, but they were also interested in a research activity that would enable them to discover drawbacks, for example the poor parasite response to treatment, and the best way to approach chemoprophylaxis for pregnant women. They also wanted to assess their impact on mortality and morbidity at every stage of programme development.

The community priority in Saradidi was in agreement with that of the World Health Organisation, who decided that malaria was to be a high priority in the implementation of PHC in the areas of high endemicity (WHO, 1979).

1.3 BACKGROUND AND CHARACTERISTICS OF THE PROGRAMME AND STUDY AREA

1.3.1 Geography

The project area is situated in Siaya District, Nyanza Province in Western Kenya. The project extended to two administrative units called Asembo East and Asembo West locations in Bondo division. This area lies along the shores of Lake Victoria (Figure 1.1).

The annual precipitation in the area is characterised by two rainy periods: the long rains in March to June and short rains in October and November. The average annual precipitation is 600-1000mm with temperatures of 20°C to 30°C all the year. However, rainfall is generally unreliable and for

most years, the expected amount is not achieved.

The climate is ideal for transmission of malaria all the year round and parasite rates remain the same i.e. 50% for adults and 80% for children. The area is thus holoendemic for malaria.

1.3.2 Administrative structure

Kenya is divided into eight large administrative units called Provinces, each of which is divided into a number of districts.

A district is divided into divisions which are made up of locations and these are divided into sub-locations. The smallest administrative unit is therefore the sub-location. The sub-locations are made up of smaller units, which may be termed 'villages' (or Gweng, in Luo).

The villages are made up of several homesteads, usually belonging to people of the same clan. Each clan has its own clan-head. Leadership at the village level is often based on influence but at times it follows the traditional set-up. The leader of the village or the village elder (Jaduong' gweng') and the village formed the basic organisational unit for health activities in Saradidi.

1.3.3 Population

In Siaya District where Saradidi is located, more than 90% of the inhabitants obtain their livelihood from subsistance agriculture. Less than 10% of the Saradidi population has a regular income from salaried or self employment.

The pattern of agricultural production has persisted from the colonial set up which designated the whole of Western Kenya a reservation area, allowing only subsistance farming for household consumption. Hence there is still very limited cash cropping. The 20 years of independence has not changed this pattern. The population of the area has continued to grow rapidly and the

exert pressure on the land which has accelerated soil exhaustion. The soil is now unable to produce enough food for the peoples' consumption to last them from one harvest to another. Apart from soil exhaustion, traditional farming methods have persisted and soil erosion is a great hazard. Nyanza Province is inhabited mainly by the Luos, who are a Nilotic ethnic group and who comprise the second largest group in Kenya in terms of population size. The average literacy ratio in Nyanza Provinces is 50.3% but this is lower among females as described by the Central Bureau of Statistics (1979).

The population in the study area was about 50,000. For project activities the area was divided into three, each area comprising approximately 15,000 people. The population structure was characteristic of a developing country being pyramidal in shape with 50% of the total population being under 15 years of age; 20% of these are children under five years and 4% under 1 year. Another 20% of the total population are women of child bearing age (15-49).

The infant mortality rate at the start of the activities of the project was very high, 180 per 1000 live births. Overall,more than half the deaths in the community occurred under five years. The crude death rate was 15 per thousand while crude birth rate was 50.4 per thousand mid year population.

In 1981 the Central Bureau of Statistics, estimated the infant mortality rate (IMR) for Siaya District, in which Saradidi is located, as 147 per 1000 live births, basing their estimate on the information gathered during the 1979 national census (CBS, 1981).

1.3.4 Health and Health Services

The health status in Siaya is generally poor. Malaria is the commonest diagnosis in health facilities and is associated with high mortality in children under the age of five years. The other common causes of mortality and morbidity are measles, diarrhoea and vomiting and malnutrition.

The health services were inadequate, inaccessible and

unavailable and often not acceptable to the community. The situation was compounded by poor transport services. The three locations in the study area were served by one small Government dispensary and a Mission Health Centre with maternity services. The referral hospitals are about 40 kilometers at Siaya District Headquarters and 60 kilometers in Kisumu town.

1.3.5. Origin of the programme

The health and development activities by the community arose out of a Development Education Programme of the Anglican Church of the Province of Kenya, the Diocese of Maseno South.

This programme aimed at making simple drugs available to the people and also providing promotive health education. The church programme covered the whole of Nyanza Province which comprises an administrative region. The Saradidi Programme was an intensification of the Diocesan programme in a small and well defined area. The Saradidi Anglican congregation took initiative to mobilise the whole of their community to be involved in health care and development activities. They did this by talking to other church denominations, various leaders, individuals and families. They undertook this so effectively that within three months, the community in the area was aware of what was developing and their role in it. The first in a series of community meetings was held 3 months after the Saradidi Church Committee's mobilisation efforts. From then on, activities were initiated which included problem identification and prioritising, identification and donation of resources, selection of CHWs and identifying training needs.

1.3.6 Organistion of the programme

It was felt that the participation of all those living in the project areas required community organisation. This would work best if the functional community unit was small enough.

For this reason, the programme committee, with some facilitation from the local administration and outsiders, divided the area into smaller units which were called 'villages'. These divisions took into consideration geographical, administrative and clan set-up. Where there was conflict over boundaries, they were adjusted to conform to the peoples' wishes. There were a total of 56 villages in the programme area. Each of these villages elected leaders for programme activities.

As the community organised themselves, a leadership pattern emerged which included church leaders, village elders and other influential community members. Leadership was organised at three levels:

a) at the village level was the Village Health Committee (VHC), each VHC was responsible for planning, organising and implementing health and development activities in their village as well as supporting the Community Health Workers (CHWs);

b) at the next level were four joint programme leadership committees which were task orientated and responded to the needs of all the villages according to their tasks;

c) at the top was an overall Executive Board which had representation from the task committees (Figure 1.2).

Having identified health promotive behaviour as one of their needs the community realised that they did not have adequate knowledge needed for their behaviour change. This led to the selection of some individuals based on 'villages' who would receive training relevant to identified community needs and would in turn, train the community (CHWs). There were 126 CHWs by the end of 1985 and 135 CHWs by 1987.

One aspect that the study set out to demonstrate was the process, the outcome and the impact of community involvement in malaria control and factors influencing the above.

In order to demonstrate this, the study area was divided into three, namely A, B and C during the first three years of the study (1980-1984) as summarised in Table 1.1.

1.3.7 Establishment of the health centre

It should be clear from the above description that the Saradidi Project was so community based that it literally had no roots in any institutionalised support structure. It was therefore important for the project to develop its own support structure. This support structure required a physical base for central administration, technical support referral back-up to the community based health activities and storage of information, materials and supplies; the centre was also a meeting place for involving all villages and to providing clinical primary health care for example, immunisation, growth monitoring, ante-natal care and clinical family planning. The Saradidi community recognised their needs for the centre and mobilised their resources to establish it. Three farmers donated portions of their land to provide the five acres on which the centre now stands. Funds were raised by villagers to put up buildings. They have been able to house clinics, maternity services, offices, stores and a laboratory. They also have staff housing for five families and a hostel for visiting personnel and students.

Staffing of the project has been provided for by donor money. Some members of staff are now supported by funds generated from the project, for example from fee-for curative services and agricultural activities. It is hoped that all staff will eventually be supported from within the project when donor funding ceases.

1.4 RESEARCH IN PRIMARY HEALTH CARE, THE SARADIDI EXPERIENCE

1.4.1 Statement of the problem, and its general characteristics.

In hyper and holo-endemic areas of malaria it is a very important cause of mortality and morbidity. Malaria mortality and morbidity is concentrated among children under five years, pregnant and lactating mothers.

Malaria was identified by the Saradidi community as one of the major problems to start with. It was also one of the activities for which external assistance was sought. As malaria intervention was to be carried out through the use of anti-malarial drugs at the community level, a system of distribution had to be worked out. This included selecting and training the CHWs who were charged with the responsibility of supplying the drugs.

Early chemotherapy of clinical malaria with chloroquine phosphate at the dosage of at least 10mg per kg. body weight was effective in the study area, in preventing the death of that person from a clinical episode (Spencer et al, 1987). This was therefore the highest operational priority (World Health Organisation, 1984).

In addition to chemotherapy, chemoprophylaxis is known to be effective in reducing morbidity particularly among pregnant women, with the greatest impact among primigravidae (McGregor, 1984).

Mass chemoprophylaxis cannot maintain high coverage indefinitely at an acceptable cost (Onori 1983). Regular chemoprophylaxis also delays development of antimalaria immunity and the magnitude of delay depends on the regularity of administration (Bruce-Chwatt 1985). The use of anti-malarials in large quantities in an area may favour the selection of resistant parasites; the selection is enhanced by high coverage and low dosage. These are some of the reaons why mass chemoprophylaxis was not included in this study because it would not be feasible to apply it outside a research setting. Chloroquine was considered as being still an effective antimalarial drug for treatment and

chemosuppression of malaria infections in the study community (Spencer et al, 1987).

Apart from chemotherapy and chemoprophlaxis, the community had to initiate and participate in community based malaria control and other health promotive activities (Kaseje and Spencer 1987).

1.4.2 Rationale for the study

Malaria remains the most prevalent disease in the tropics with more than 2.3 billion people living in endemic areas and more than a million children dying of malaria each year in the same areas (Payne et al 1976). In Kenya, malaria is known to contribute significantly to mortality in infancy and childhood in high endemicity areas which include the study area. This is supported by the following:

a) World Health Organisation (WHO) Fenitrothion Study 1973-76 demonstrated a reduction of infant mortality rate by more than 50% in the study region through using malaria control as the only intervention (Payne et al 1974);

b) the highest under 2 year mortality rates in Kenya are found in areas of highest malaria endemicity as shown by the two years mortality calculated from 1979 population census (Kibet, 1981).

It was considered impossible to reduce the incidence of the malaria, Saradidi being a holoendemic area. However, it was necessary to find an appropriate means of reducing at least the malaria mortality in Kenya and other similar countries.

The study intended to explore ways of developing an effective malaria control strategy within the Primary Health Care (PHC) framework. PHC, in the Alma Ata sense, aims at bringing health care as close as possible to individuals, where they live and work through their participation (WHO/UNICEF 1978).

The study also aimed to document the process of this strategy and its impact on the community, since PHC is a strategy that is likely to be established in most countries of tropical Africa where malaria is among the greatest of health problems.

We therefore carried out a careful assessment of malaria control in PHC to find out whether its operationally feasible; possible constraints and the determinants of effectiveness. The study also set out to document the impact that can be achieved in terms of selected indicators for example, utilisation drugs and reduction in mortality and morbidity.

The major questions that the study sought to answer were:

1. Can the community distribute chloroquine for treatment of those with symptoms of malaria and for prophylaxis of pregnant women in such a way that there would be measurable reduction in mortality and morbidity, and what is the most effective regimen for pregnant women?
2. Can the chloroquine made available in this way accelerate the change in the sensitivity of the parasite, to chemotherapeutic doses of chloroquine phopshate?

Chloroquine for chemoprophlaxis and chemotherapy was made available at the community level through the CHWs in areas A & B to ensure that it was within easy reach for those who needed it i.e. chemotherapy for the sick and chemoprophylaxis for pregnant women.

Knowledge, attitude and practices about malaria would affect the participation of the community in the utilisation of chloroquine, and other malaria control activities hence the final outcome.

It was assumed that there was a relationship between resource availability and the continuation of the programme. The willingness of the people to get involved and their perceived advantages of the programmes would also affect continuation of the programme.

This could be achieved only if the people continued to be involved at

every step in developing, implementing and maintaining effective interventions with appropriate inputs from the support system.

The ability of the community to monitor and evaluate their activities is related to how effectively they can be involved and also to the programme achievements. Some of the indicators for the communities self-assessment include utilisation of available services and reduction in levels of morbidity and mortality.

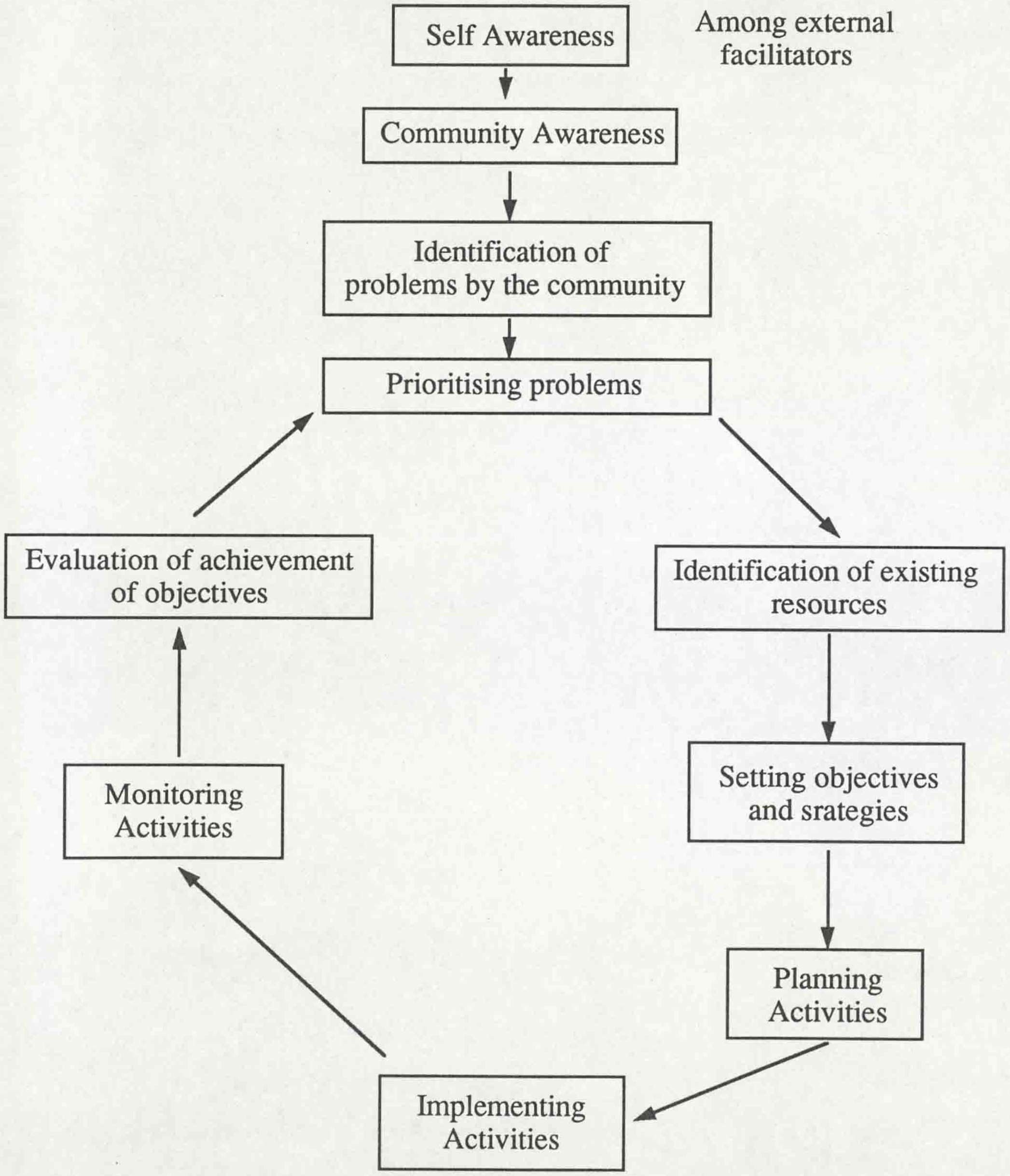
1.4.3 Project objectives

1. To document the community process in setting up and participating in antimalarial chemoprophylaxis and chemotherapy using chloroquine in such a way that morbidity and mortality due to malaria in the community is reduced.
2. To assess the changes in the knowledge, attitudes and practices about malaria in the community and how they affect community participation in the project in general and utilisation of the community based antimalarial activities in particular.
3. To determine demographic and parasitological impact.
4. To demonstrate the most effective chemoprophylactic regimen for pregnant women.

1.4.4 Research Design

Introduction

The Saradidi Programme was a community based project. Although there were some external inputs, the community took the overal responsibility of the project planning, implementation, monitoring and evaluation. This process can be summarised as below:


Self Awareness
Among external facilitators
Community Awareness
Identification of problems by the community
Prioritising problems
Evaluation of achievement of objectives
Identification of existing resources
Monitoring Activities
Setting objectives and srategies
Planning Activities
Implementing Activities

The community or its representatives were involved at each step. The role of outsiders was to facilitate the process, and together with the community, to foster an atmosphere that maximised effective implementation. This process was based on a conceptual model as presented below:

a) <u>The community process</u>

Outside involvement was kept to a minimum and was introduced only within a process of dialogue where everyone involved listened to everyone else and learned from everyone else. The professionals and the community were going through a process of increased awareness simultaneously. Ideas from outside were not imposed on the community but were introduced as part of the dialogue so that final decisions were from the community. Thus, the design of the programme was from the community who also acknowledged this fact since they participated in all the activities and decisions of the programme including problem identification, prioritisation, selection of solutions, planning, mobilising resources, implementing activities, monitoring and evaluating of the project.

b) <u>The Community Dialogue</u>

The process of information, education and communication has been continuous up to now and will continue as it is vital in a community based programme.

c) <u>The Community Organisation</u>

This was necessary to enable the community to work together and was facilitated by the community leaders with some assistance from professional extension personnel both Government and Non-governmental organisations. This ensured that the resulting organisation structures maximised co-ordination and collaboration among all partners.

d) The Community Leadership

The community elected leaders of their own choice from among themselves and the professionals continued to work with the leadership as established by the community. Other local resources mobilised and used included financial contributions and time spent in attending meetings and carrying out disease control work. Maximum use of local resources was important as it enabled the community not to act as helpless, passive recipients of external aid but as active contributors to their own health and development. For example, they had a free choice, without any criteria from outside, in selection of leaders and CHWs and they provided food and money for their training and support.

e) The Community Supervision and Facilitation

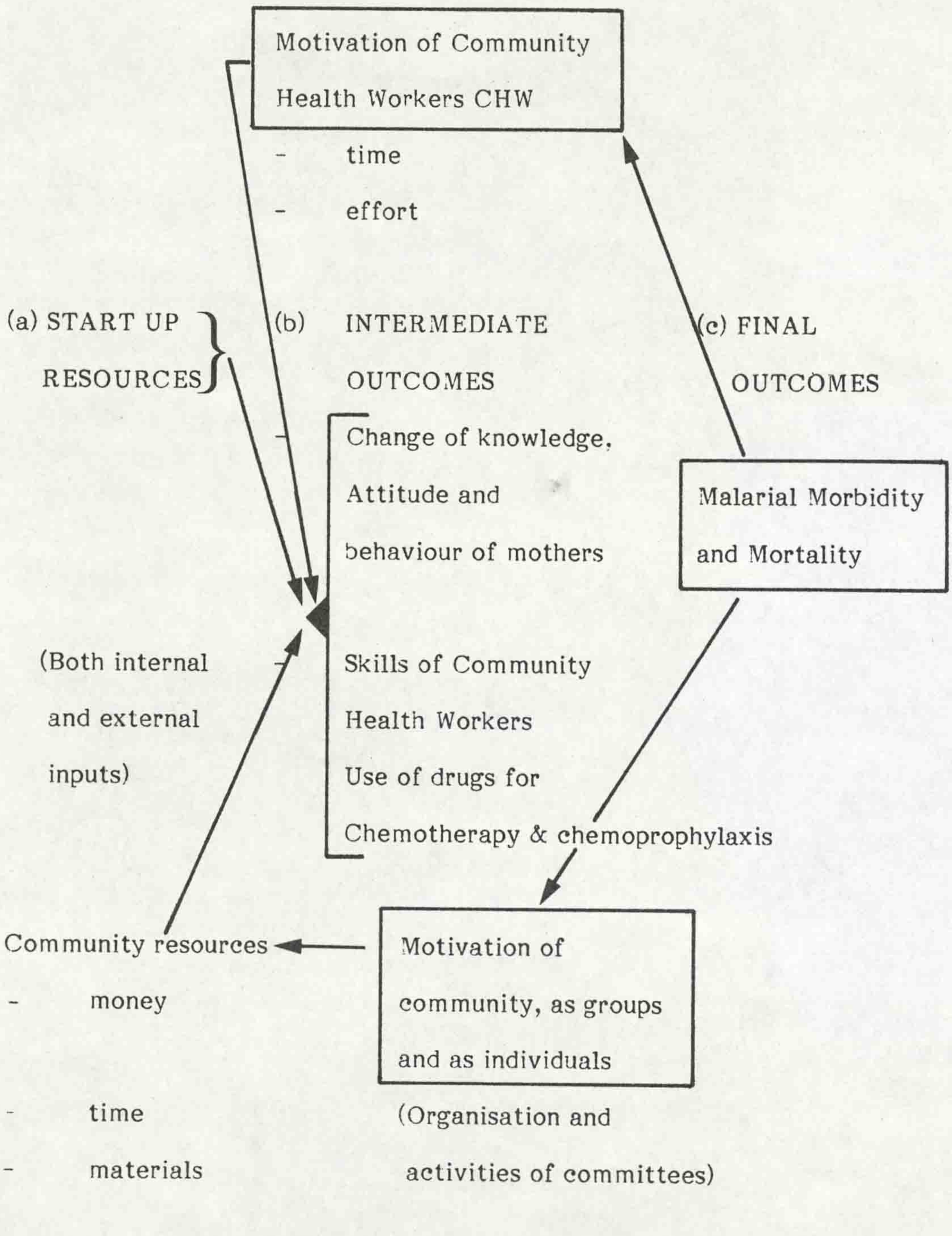
Outside supervision was maintained at a low level and within the context of dialogue so that there was always feedback and discussion with the community. The management activities in the project were left to the community leaders but professional guidance was available to them when needed.

f) The Community Involvement in Research

Being involved in monitoring and evaluation was a motivational and educational tool. The community fully participated in collecting information and keeping records for monitoring and evaluation which was used for programme management. The records kept were utilised in the documentation of the process of community participation. Any data or information that were not of immediate use to the community were collected independently of them but with their approval, (for example, testing of the sensitivity of malaria parasites to chloroquine). All the findings were fed back to the community and used as educational materials and also for planning future activities.

The model is summarised below:

The Conceptual Model



It was postulated that the initial inputs in mobilising the community, training CHWs and providing chloroquine for chemotherapy and chemoprophylaxis would lead to a decline in malaria mortality and morbidity. The decline in mortality and morbidity would in turn motivate the community and this would lead to increased community organisation for action. It would also

motivate CHWs to spend more of their time and effort in providing information, education and drugs to the community.

The motivated community would in turn make available resources such as (time, money and materials which would make more chloroquine available and would support the efforts of CHWs. With support forthcoming, the CHWs would be encouraged in their work thus enhancing the community's confidence in demanding services from the CHW, Government facilities and elsewhere. The resources so generated in the community would operate the community system except for professional guidance and referral where necessary. The impact of the programme would provide motivation for greater change in the community's knowledge, attitude, behaviour and involvement in malaria control as well as other development activities.

1.4.5 Methods of Data Collection and Analysis

The Saradidi Rural Health and Development Project (SRHDP) was not planned by the people as a purely research programme. The research component was built into the programme to complement community efforts and to provide lessons which would enhance planning and education at the national level and other parts of the world where similar programmes are implemented.

a) Baseline

Baseline data gathering was carried out in August, 1979. Information was collected on:

- Environmental conditions
- Health service availability and utilisation
- Immunisation coverage
- Nutrition status and food availability;
- Knowledge, attitude and practice about malaria and other health problems

- Knowledge and use of family planning services
- Demographic and economic situations.

The data was analysed manually and it formed the basis from which progress and achievements were to be measured. The data also helped in identifying areas where there were gaps in knowledge or great need for intervention which were later considered during planning. We formed the following from the baseline survey.

i) Environmental Conditions

Most of the households (65%) had temporary dwellings while 60% had windows in some rooms. The most common sources of water were unprotected.Less than 1% of the respondents had access to safe drinking water. Over 75% of the households used more than 10 litres per day at home. This was considered an underestimate of water usage as many people usually washed clothes at water sources. The water brought to the house was mainly for cooking, drinking and washing utensils. More than 50% of the population walked for more than one hour to their water source. This affected the volume of water used in the households. Twenty percent of the households visited had no pit latrines. The remainder had latrines and were using them.

ii) Utilisation of Services

Over 70% of deliveries occurred at home and without trained assistance. However, most of the women (90%) attended antenatal clinics at least once.

The knowledge of family planning was very low (less than 20%) and less than 1% of the eligible population were currently using a modern family planning method.

Thirty percent of the women interviewed did not know the cause of Kwashiorkor in children. Seventy percent did not know what causes malaria neither did they know how it could be prevented.

iii) Immunisation Coverage

Of 1596 children under the age of 5 years examined, only 27% had immunisation cards. Only 11% of the children were fully immunised.

iv) Nutrition Status

Nine percent of the children examined were severely malnourished while 45% were moderately malnourished by the Harvard Standard of weight for age. The direction or trend of curve was not looked at.

v) Demographic and Economic Status

Household size was 4.2, with a population structure characteristic of developing countries. Fifty percent of the households were headed by women. The main source of income for household heads was subsistance farming. Only 11% of the population had regular income either from self or wage occurring employment.

During the past 12 months, 472 births were reported to have occurred in the project area giving a crude birth rate of 58 per 1000 population in the sample.

Deaths in the past 12 months were 102 giving a crude death rate of 17.6 per 1000 population sampled. The infant mortality rate was 181 per thousand live births in the sample in the past 12 months. The main causes of death were diarrhoea and omitting, measles and malaria.

b) Data Collection at the Community level

Efforts were made to ensure that data collection by the community was not cumbersome. The community identified people who needed to keep data and information and these included:

- various committee members who kept dairies
- CHWs
- the project staff
- outsiders

The information required from each was that which was relevant to their activities, for example,

- the CHWs collected information on
- children born and their birth weight
- deaths indicating age and cause of death
- pregnant women
- environmental status
- malaria treatment by age and month
- family planning users and drop-outs by method.

Raw data was collected from them, analysed by the project staff who gave them feedback monthly. Identified areas of weakness were often discussed with individuals who needed particular help. Areas for further training were identified during these feed-back sessions.

c) Community Self Assessment

To understand fully what was going on, the community, with some facilitation from outside, was encouraged to evaluate its own activities through 'self assessment' sessions which were organised by competition and where the winning villages were rewarded.

d) Specific Studies

A number of scientific studies and surveys related to malaria were carried out and the results are summarised in various subsequent chapters. The main research results presented and discussed are those of malaria chemoprophylaxis to pregnant women as provided by Community Health Workers in Saradidi and the most effective regimen and mechanism of supply.

e) Observations and Discussions

These were carried out by various categories of people. Areas for observation and discussion included social, anthropological, economical, environmental and behavioural concerns.

The observations were usually complemented by discussions using check-lists or questionnaires.

f) Census and Updates

A complete counting ('census') and household listing of the total project area was carried out between 1980-82. Each household in the area was allocated a number and each individual obtained an identity number which distinguished the area, village and household any individual in the project area came from. The demographic structure of Saradidi was determined from this exercise. Updates were carried out through semi annual visits to each household to record changes and other happenings i.e. births, miscarriages, deaths by cause and migrations were obtained. This information is presented in Chapter 6.

The information was also shared with the communities and used to guide allocation of resources in the communities. In addition to direct calculation of these rates for the appropriate dates, the 'census' gathered adequate information to enable the use of indirect methods of estimation for retrospective childhood mortality as developed by Brass and Trussel 1972.

g) Sample Surveys

Specific sample surveys and studies were carried out from time to time and the sample size for each was based on the indicators of assessment. Structured questionnaires, check-lists and observations, clinical examination (for example, weighing of children) and collection of various specimens (for example blood samples) were used in these surveys.

The completed household listing was used as a sampling frame for sample surveys.

h) Periodic Parasite Prevalence Surveys

These were conducted three times a year, mainly in Primary Schools to provide information on seasonal trends of parasite prevalence rate by parasite types, sex and age of the pupils.

Knowledge, attitude and practice (KAP) surveys were carried out twice after the baseline survey.

Utilisation surveys were carried out three times to determine sources of chloroquine for treatment of malaria and the effectiveness of CHWs as distributors. The utilisation of antimalarial chemoprophylaxis provided by CHWs to pregnant women was also surveyed.

Specific studies were carried out to determine the epidemiology of chloroquine pruritus, symptoms associated with malaria in the community and response of the malaria parasite to chloroquine and other antimalarials used in Saradidi area. The results of these surveys are presented in Chapter 2.

1.5 COMMUNITY LEADERSHIP AND PARTICIPATION IN THE SARADIDI RURAL HEALTH DEVELOPMENT PROGRAMME

Community participation in Saradidi was both a 'means' and an 'end'. It was considered the best means for solving health problems. In addition, community participation, involvement and committment was viewed as critical for developing a self-reliant community and thus became one goal of the programme.

The process of community participation in Saradidi, (for example, who was involved, how, in what activities, why and when) was documented. The results show that the organisation of the area into villages

had to be sensitive to existing community organisation structure such as geography, religion, kinship and administrative boundaries. Age and social status were important factors in accepted leadership role in Saradidi; most of those selected for leadership were over 45 years of age and were of higher economic status than the average for Saradidi community.

Table 1.2 shows the positions of the people considered to be most influential in the villages in the project activities. Most of them held a post within the programme

Some groups within the community such as women and youth were not included in leadership positions. These people, however, were often the most aware of certain village problems. Women's groups, most of which existed before the programme was set up, played an important role for community development; they supported the volunteer community health workers and carried out many village health and development activities. The conditions which were found to favour community participation included the following:

i) the idea of the development programme was conceptualised within the community and not by outsiders;

ii) the programme was led by a community health doctor who knew that community participation was vital and encouraged it;

iii) decisions were taken at traditional community meetings open to all residents, many of whom attended these meetings;

iv) the people were ethnically, religiously and politically homogeneous and many were from similar economic situations;

v) local resources were mobilised and the community was motivated to donate land, time, labour, materials goods and money;

vi) outside resources were available for training, and other essential interventions to solve priority problems and brought tangible benefits, while the contribution of the community was never overshadowed by the external resources;

vii) support was available at the programme centre, from the Executive Board and from the Programme Development Committee. The centre at which a clinic was later added, provided a strong backup and referral system for the community based activities;

viii) community needs were readily identified and verified. There was a readiness for change as the community had been primed by the Diocesan programme;

x) religious affiliation was a strong bond in the community and it also provided the necessary spiritual commitment to voluntary work for the good of others.

Many village based health committees selected at the start of the programme did not function effectively. CHWs volunteered a significant proportion of their time despite poor support by village health committees.

The central project structure and the training they received compensated for the lack of guidance by village health committees.

Village income-generating activities which were meant to support village health activities were not very successful; group involvement in income raising ventures proved to be inefficient many ended up as income draining activities. It was observed that village group income projects must be well selected relative to the skills and resources available and the ability of the product to be marketed.

Those based at the programme's centre were more successful perhaps because of greater investment in skills, money and marketing.

1.6 COMMUNITY HEALTH WORKERS

1.6.1 Introduction

The role of community health workers in enabling communities through information and education to identify their own health needs and to take actions to solve their problems is central to primary health care (World Health Organisation and United Nations Children's Fund, 1978; World Health Organisation, 1984; Ofosu-Amaah, 1983).

The community health worker also links the community, of which she is part, to the health services that provides her with technical and referral support. This innovative concept of involving the community has given a new dimension to the provision of health care, and was the basis for the implementation of the Saradidi Health Development Programme (Hagmann and Kaseje 1987). This section describes the characteristics and functions of community health workers in the Saradidi programme.

Based on the experience in Saradidi, this analysis identified characteristics that determine an effective community health worker and the activities that are most appropriate for them to carry out in communities similar to Saradidi. Each village in Saradidi elected a village health committee and one or more CHWs.

By 1983 there were 126 CHWs who served a total population of more than 43,000 in 56 villages distributed over a total area of about 22km^2 (Spencer et al, 1987). The CHWs were the core of health activities and contributed the most amount of time in implementing the development programme, even though they were volunteers and were to work in such a way that they still catered for their own families.

1.6.2 Data Collection

Data were obtained in the following ways:

1. Records of personal information (age, sex, education, etc) collected from villages at the time CHWs were selected and enroled for training.
2. Retrieval of information from the records of CHW activities which were submitted to the project once every month.
3. Results of a survey carried out on a random sample of 36 CHWs for assessment of attitudes, important activities undertaken, training, support by village, reasons for continuing and problems encountered. The survey was carried out by a female Luo-speaking social scientist well known to the CHWs. With the exception of those items included for identification, the questions were open-ended.

1.6.3 Results

a) Selection and Support

CHWs were selected by their village, generally by the village health committees (VHCs) formed in each village (Kaseje & Spencer 1987). There were no criteria given to the village for the selection of CHWs but they formed their own criteria, for example, that CHWs were to be perceived as mature, compassionate, willing to help the people in the village voluntarily that they would be permanent residents in the village. There were no formal educational requirements or reading and writing skills required of CHWs. The villages were also keen that if they were young or middle aged women. One way of proving their attitude was their ability to look after their mothers-in-law well. These conditions were determined by discussion and vote at a community meeting open to all residents of Saradidi. What emerged, therefore, was a group of people with varying ages and educational attainments. All were to be supported materially and administratively by their village.

b) Sex and marital status

Most of the CHWs were female; only 3 out of 126 were males. Virtually all of them were married, the exception being 1 who was a divorcee.

c) Occupation

Out of the 123 female CHWs 120 were housewives and/or subsistance farmers; 2 were tailors, and 1 was a nursery school teacher. The 3 males were subsistance farmers. The CHWs knew all the people in their respective villages.

d) Age

The oldest CHW was 64 years while the youngest was 21 years. Only 12 (9.5%) were aged 24 years or less. Seventy eight (or 61.9%) were between 25 to 34 years old; 17 (or 13.5%) were 35 to 39; and 19 (or 15.1%) were aged 40 years or more (four of these were 50 years of age or older).

e) Education

Although it was not required that a CHW be literate, most of them were better educated than other residents of the community. The majority (101 or 80.2%) had 8 or more years of education. Only 9 (or 7.1%) CHWs had no formal education, but even these had attended adult literacy classes so that they were able to read and write to a certain extent, 16 (or 12.7%) CHWs had between 1 and 4 years of education.

f) Activities of the CHWs

The activities expected of the CHWs were decided by the community members meeting which was open to all Saradidi residents (Table 1.3) (Kaseje & Spencer, 1986). To appreciate the responsibilities of the CHWs one must examine the tasks of the average woman in Saradidi. Most women in Saradidi carry out the following each day.

1. Care for children
2. Clean the homestead
3. Cultivate food for the family; this involves long hours of work daily during planting, weeding and harvesting seasons.
4. Cultivate cash crops for sale; many are involved in petty trade to provide basic resources for their families.

5. Carry water to the house (usually from 3 to 10 km away).
6. Collect firewood (often from long distances).
7. Prepare at least 2 hot meals for the family per day.
8. Look after their in-laws who often live nearby.
9. Participate in village activities.
10. Look after their husbands.

Most of each woman's day was already committed to these activities. Handicrafts and related activities were usually accomplished as the women walked, collecting water and firewood.

The female CHWs had all the responsibilities of women and mothers and, in addition, were asked to perform the functions of their volunteer responsibility in the health programme.

The CHWs were supposed to be available to people living in the village at any time of the day and they even received calls at night. They provided chloroquine phosphate to any person wishing treatment for malaria (Spencer et al, 1987). Thus a female CHW could be asked to leave her work in the field to come and give malaria treatment or for another task such as to assist in delivering a child. Several hours each week had to be reserved for home visiting.

During these visits she could be asked to help people in environmental health improvements, such as digging pit latrines or garbage pits, constructing dish-racks and clearing bush and stagnant water from around homesteads. In addition CHWs were requested to keep accurate records of the activities to be submitted to the programme centre. The CHWs also participated in training, assisted in mobile health clinics and worked at the programme clinic. Most spent 5 to 10 days each month on the health programmes's activities.

By 1983, many CHWs had been performing these tasks voluntarily for

four years. The original plan was that each village would support its CHW from the income they obtained from income-generating activities. In fact, through 1987 no village in Saradidi had financially supported any CHW on a regular basis.

g) Training

The process of training the CHWs and methods used are described elsewhere (Kaseje et al, 1987). All CHWs received training. Of the 36 CHWs selectively interviewed only 1 had been a CHW for only 1 year; 12 had served from 2 years, 11 for 3 years and the remaining 12 for 4 years. Training by the programme had been continuous; 6 CHWs had received training over a period of less than 1 year, 22 for 1 to 2 years, 4 for 3 years, and 4 for 4 years. Many, 22 or 61.1% had also been trained as village birth attendants.

h) Size of the population CHWs serve

Each household in the project area was numbered. Each person had a unique number identifying the area, the village, the household and the particular person. The average number of people per household in Saradidi was 4.0 and the average village size was 764 persons (Spencer et al, 1987).

Each CHW was supposed to be responsibile for a maximun of 100 households. Thus many villages had 2 or more CHWs. Of the 36 CHWs interviewed, 2 said that they served 250 people or less, 15 said they served between 250 and 500 people and 19 said they served more than 500 people. In addition to the people from her/his village, the CHW could provide services to others. A few malaria patients and family planning clients came from beyond the boundaries of the programme area or the CHWs area of operation because they knew that such health services were available within Saradidi villages.

i) Activities as a CHW

The CHWs were supposed to submit monthly reports on all their

activities to the staff at the programme centre. However, only 49 or 126 CHWs submitted reports. The need for reports and the importance of each type of information required from this was emphasised to them in their training.

During household visits, the CHW discussed a wide range of issues with household members. With the family she identified health problems and planned solutions. These household visits usually occupied 1 to 2 hours each. Therefore the CHW could not visit more than 2 homes in one afternoon. Each CHW visited about 15 households each month and thus needed an average of 7 months to visit all her 100 households. However, additional discussions on health issues occurred when people came for malaria treatment and health advice.

The CHWs also attended more than one meeting each month. The CHW often walked to these meetings and could be away from home all day. Each CHW also made over 10 family planning contacts per month: 1.7 of these on average became acceptors.

CHWs in 36 of the 56 villages provided chloroquine phosphate for treatment of malaria (Spencer et al, 1987). This activity reduced congestion at the health service delivery points and decreased distances travelled and the time of illness before beginning treatment.

(j) Important activities

The 36 CHWs interviewed considered home visiting, as a disease prevention activity, treatment of malaria, promotion of maternal-child health clinic attendance, environmental health education, nutrition education and recording of vital events as their most important activities. They said the home visits were an important element in establishing contact with people and in identifying health problems. The CHWs reported that mothers talked more freely in their homes especially about family planning. Other important activities identified were malaria control and treatment, village health committee responsibilities and assisting with home childbirths.

However, the CHWs said they considered all their functions to be important. On occasion, the CHWs were requested to participate in the research activities. They viewed their involvement in such studies, particularly sample surveys which were not part of their normal routine, as least important.

k) Use of training

The following topics covered in their training were considered by the CHWs to be the most useful and are listed in order of stated importance:

1. Malaria control and treatment
2. Family planning
3. Nutrition education
4. Disease prevention
5. Home visiting
6. Antenatal and postnatal care
7. Immunisation
8. Conducting home deliveries
9. Home hygiene
10. First aid
11. Treatment of simple diseases
12. Identification of diseases common diseases.

The CHWs training consisted of discussions, role plays, field work, talks/lectures, demonstrations and the use of audio-visual aids (Kaseje et al, 1987). All of the CHWs said they needed more training.

e) Problems as a CHW

In the sample survey the CHWs were asked to list the problems they encountered in their work (Table 1.4).

CHWs were frequently called at night and during bad weather conditions. The CHWs were not provided with equipment such as flashlights, rain coats or gumboots for movement in such conditions. The programme area was large (about 225km^2). For many people in Saradidi the programme's clinic was more

than 5km to 10 km away; no transport was available in the village; the CHWs found the lack of transport and distance a hindrance when they had to refer people to family planning clinics, to maternal and child health clinics or when someone was seriously ill.

People came to the CHWs with many different health complaints. The CHWs said they felt inadequate in many instances since for most problems they could offer only advice; chloroquine phosphate for treatment of malaria was the only drug they had available. A serious drought beginning in 1982 led to inadequate good water supplies in Saradidi. Teaching about nutrition and hygiene were difficult when food and water were scarce.

The CHWs were mothers and wives who volunteered their much-needed time to help the community. Some of them (7 of 36) said that they would like to be paid, especially for delivering babies. The staff at the centre were relied upon by most of the CHWs to solve their problems; 29 of 36 said they went to the centre first when they had problems. Although the (VHCs) were originally intended to fulfill this role, only 5 CHWs said they went to these committees; 2 would go to the assistant chief. Thus the CHWs looked first to the centre and not to their own village leaders.

m) Co-operation with other people in the village

The 36 CHWs who participated in the sample were asked how different categories of people influenced their work. Their responses were as stated below for each category:

1) The Programme Development Committee was supposed to develop work plans for the programme, advise villages on development and solve village problems. Many CHWs said they worked together with the programme committee in these activities.

2) The Village Health Committee were intended to call village meetings, organise and supervise village development projects and advise and

supervise the CHW. However, 7 of the 36 CHWs said that they did not work with the village health committee because it was inactive and a total of 31 CHWs said that their village committee was less suppportive than expected.

3) The Elders in the villages were responsible for interactions with government, advising on development. calling meetings and disseminating information, promiting health and keeping law and order. Seventeen of 36 CHWs said that they worked frequently with the village elders.

4) Programme Centre health staff provided training, advice, referral services, mobile clinics, help with problems and supervised and evaluated the CHWs work.

Most CHWs mentioned support from the centre health staff, women and children in the village. From these three groups. the CHWs received training, co-operation, appreciation and much needed moral boost and encouragement; they were the three most important groups for the CHWs.

n) Reasons for continuing as a CHW The CHWs were asked why they have continued as CHWs; their responses are shown in Table 1.5. All of them found the training to be so beneficial to themselves and their families as well as to the community that they were willing to continue working despite lack of financial support. The second major reason given was that the CHWs had agreed to serve the villages when they were chosen and would not like to go back on their word. Some of them added that they like the work as well as the training they recieve. Another reason for continuing was that they felt they had had an impact on the life and health of their village. Occasional allowances was also a reason for continuing for those who received them.

The CHWs believed community training and health education had changed many traditional but erroneous beliefs. Some of these beliefs reported

changed as a result of CHW training were as follows:

Children should not eat eggs

Children with measles should not eat meat, eggs and other proteins.

Kwashiorkor/marasmus are incurable.

There is no need to attend antenatal and immunisation clinics.

A pregant mother cannot breastfeed and

Family planning produces abnormal children.

These are the same people who would be asked to provide antimalarial chemoprophylaxis weekly to pregnant women (in the currently recommended regimen). They were over-worked, unsupported materially and unable to keep records and submit reports regularly. It is not surprising that they did not perform this function effectively when it was assigned to them. (Kaseje et al, 1987).

o) Responsibilities

The major tasks community health workers are expected to perform are very similar in different countries and have been shaped to a large extent by the declaration at Alma Ata. (World Health Organisation and United Nations Children's Fund. 1978). Tasks almost universally required include first-aid; treatment of accidents and simple illnesses; dispensing of drugs; anti and post natal advice and motivation; child-care advice and motivation; nutrition motivation and demonstration; promotion of immunisations, and assistance during clinics; promotion of family planning; motivation for environmental sanitation, personal hygiene and general health habits; communicable disease screening, referral and prevention; referral of seriously ill patients, treatment failures and difficult cases; maintenance of records and compilation of reports; home visits; and participation in community meetings. This is too long a list for a worker who is essentially a volunteer.

The CHWs in Saradidi were no exception to the general rule that community health workers are asked to do too much. The priority of health

problems, other responsibilities, the distance to travel, the terrain and technology involved should be considered in deciding on duties. The experience in Saradidi emphasises that community health workers should only perform activities of direct relevance to the top priority problems in their village. They should not be the only active participants in a community based programme. Others in the community must also take actions aimed at solving community recognised health problems. A way must be found to reduce the burden on the volunteer CHWs. This is why an antenatal care based on regular monthly prophylaxis for the most at risk members of the community would be a better approach than that based on CHWs. CHWs could then improve it by following up defaulters.

p) Population coverage

The population served by CHWs in Saradidi (about 500 people) was similar to that elsewhere. The fewer and less time-consuming tasks are, the larger the population that can be covered. Population limits need to be well-defined. Given the findings in this survey a volunteer CHW should ideally look after a maximum of 20 households. This implies such a large number of CHWs that the supervisory load it would lay on the health staff would perhaps prove unbearable. The answer must lie in reducing the tasks of CHWs and increasing the participating actors within the community.

In conclusion, the homogeneity of the Saradidi community, the community preparation and organisation that occurred prior to selection of CHWs, the support of the programme director and staff at the centre to them, and the continous training provided contributed to the success of the programme.

The ingredients for a successful volunteer programme such as this one are present in many areas. At the minimum, a strong programme centre, community support from women's or other groups, back-up for problems, referral capability, on-going training, adequate supervision, on the job evaluation and a sense of purpose must be provided.

1.7 TRAINING OF COMMUNITY HEALTH WORKERS

1.7.1 Introduction

Community participation in primary health care often involves volunteer community health workers (World Health Organisation. 1987). Although it is agreed that the roles and performance of community health workers are directly related to the training they receive, there is no concensus as to what constitutes appropriate training (Ofosu-Amaah, 1983; World Health Organisation 1984). Interest in the training of community health workers is recent, the training has been in many cases experimental and there has been little careful evaluation (Li et al, 1984). The training of the CHWs at Saradidi was carefully documented and analysed so that the lessons learned can be useful in other similar situations.

The cornerstone of community health development was the work of volunteer community health workers (CHWs) chosen and supported by each village (Kaseje et al, 1987). From the beginning of the programme, continuous training was considered to be a priority for the health development programme by the community; this was emphasised in the community meetings open to all residents (Kaseje & Spencer. 1987).

1.7.2. Who were trained

The CHWs were the main trainees. However, community leaders and programme centre staff received orientation to the programme to enable them to understand the training objectives content and methods, so that they could support the CHWs more effectively. The orientation also emphasised their roles in the programme. The characteristics of the CHWs chosen have been described (Kaseje et al, 1987). Four groups of the CHWs had completed training through July, 1984 and a fifth one was undergoing training. Each class numbered about 25. The class was divided into 4 groups (6 to 8 people in each including trainers), which met at different sites in the area.

1.7.3 Venue

The training was located as close to the villages of the trainees as possible so that it was carried out within the environment of the problems under discussion. The CHWs, most of whom were mothers, were expected to continue with their normal household activities. The division of each class into 4 small groups made it easier to locate training near the villages of the participants. Thus, the CHWs could return home after each daily session to fulfil their other roles and responsibilities in the community.

1.7.4 Duration

The CHWs were given 2 weeks of introductory training followed by two one-day sessions each month and a one-week workshop each year until they had received at least 3 months total training spread out over 2 years.

Supplementary training sessions of 1 or 2 days each month continued intermittently after the three-month basic course. These were carried out at the programme centre. Timing was flexible and always determined by the trainees, training was suspended during any period of intense household socioeconomic activities such as the planting, weeding and harvesting periods.

1.7.5 Training course

Training content, methods and activities were developed with an education specialist and a community health doctor both of whom belonged to the Saradidi community; they also participated in training sessions and were particularly involved in the training of the trainers. Two community health nurses, who were full-time staff members did much of the training. Other trainers included local residents, programme staff such as the clinical officer and community leaders.

From 1983, CHWs who had previously received training began to participate as trainers. The training of trainers was done through seminars and workshops held at the programmes centre. Facilitators from the Ministry of

Health, the University of Nairobi, the United Nations Children's Fund (UNICEF). the African Medical Research and Education Foundation (AMREF) were involved in the seminars to train trainers.

1.7.6 Training methods and materials

These were made deliberately flexible to fit the age, education and social status of the trainees. The process was problem-orientated leading to the development of skills and was based on sharing of experiences in response to specific problems posed. The training programme had broad objectives and a curriculum but did not identify specific training activities. The aims of training were:

1. to help CHWs identify and respond to the important health problems presented to them in their villages, e.g. 'What can I do about children who die of fever in my village';
2. to teach specific skills needed for specific situations e.g. 'how do I prepare oral rehydration fluid for children suffering from diarrhoea and vomiting in my village and what do I teach their mothers'; the aim was to teach the correct way to perform this skill;
3. to respond to and build upon community needs, perspectives and programmes, e.g. learning sessions led to specific actions and activities in the village. The trainer usually began with problems that the CHWs and the community could do something about with relative success and tangible results e.g. treatment of fever. The trainees agreed to go and carry out an agreed action at the end of each session which also included a practical demonstration of the intended action;
4. to develop the empirical and analytical skills appropriate to the situation of CHWs in their individual villages by teaching, through analysis of common village health problems, appropriate information needed in the situation, the reason the problem occurs and what can be done about the problem;

5. to make learning a group activity in which individual members were supported by the efforts, skills, insights, wisdom and experience of the other group members recognising the fact that adults are rich in relevant knowledge, skills and experience to their local situation.

All sessions were based on discussion conducted in Luo, the local language. The trainer participated as a member of the group. Experience of trainees were elicited, shared and extended in an attempt to identify, analyse and suggest solutions to their health problems. The sessions included discussions on how to identify patients who should be referred (e.g. those who failed initial treatment and or were seriously ill).

The main approach was to minimise barriers of communication by making the training process a dynamic one, emphasising participation (both verbal and non-verbal) (Keesing, 1981). This process in which the target audience actively participates helps in the assimilation of new ideas because these are presented in a form to which the trainees can relate. The participation method also minimises the effects of culture since new information is introduced in the context of the trainee's environment and in a form consistent with their perception, their concepts of self and their world-view.

1.7.7 Role of trainers

The main roles of the trainer were to organise sessions for learning; to direct discussions based on perceived health problems; to provide information as appropriate and as part of the learning process and to demonstrate skills to be learned. The trainer also synthesised and summarised what had been learnt and helped the trainees to recognise what learning had taken place. The details of the methods are as described by Kaseje et al, 1987.

1.7.8 Content

The content of the training course covered three main areas: new knowledge, demonstration and practice of skills, and discussion of what to do with the knowledge and skills acquired. The topics to be covered and length of time allocated for each were decided on through:

a. problems identified by the community during community meetings and confirmed and defined by community surveys and

b. the CHWs were assigned tasks and functions in the process of solving these problems (e.g. malaria control activities).

1.7.9 Evaluation

The results of training on the knowledge of CHWs about selected topics on their attitude, behaviour and skills and their impact at the village level were evaluated. Tests were given before and immediately after initial training and then again after 2 years. To assess change of behaviour and impact at the household and at the village level the community health nurses visited CHWs villages and houses to determine changes that had occurred following training activities.

The trainees evaluated the training and trainers in terms of the relevance of the course content, how well the trainer helped them to learn and which aspects of the process interfered with their learning. CHWs also suggested ways to improve learning; useful suggestions by trainees' knowledge, practices and behaviour, in their homes and villages which were not significantly influenced by their age, sex or formal education background Tables 1.6 and 1.7.

1.7.10 Conclusion

The participatory approach to the training of community health workers was found to be effective in Saradidi. The emphasis on local public health

problems and solutions and on teaching methods appropriate to the CHWs was important. This training experience in Saradidi utilised techniques similar to successful programmes elsewhere (Storms, 1979, Lit, et al, 1983-84) The size of the training group was restricted to allow close trainer-trainee contact. Trainers had undergone training themselves and understood the trainers and the local problems. The trainer encouraged active trainee participation in 'learning-by-doing' and continually stimulated the trainees to improve on the way things were done in the past. The programme provided the CHWs with continuing instruction, systematic supportive technical supervision and on-the-job evaluation. These training activities have been shown to be crucial to the success of community-based programmes (Ofosu-Amaah, 1983).

Table 1.1 Malaria intervention in Saradidi by area, villages and population, 1982-1984.

Area	No. Villages	Initial Population	Malaria Invervention in each village
A	23	16,560 (1981)	Chemotherapy to all fever cases and chemophrophylaxis to pregnant women. **IEC regarding community based anti-malarial activities
B	13	11,052 (1981)	Chemotherapy to fever cases and IEC
C	20	15,182 (1982)	IEC

** IEC = Information Education and Communication.

Table 1.2 Position of named influential people in 46 selected villages of the village health committees and in the community development project, Saradidi.

Position	Village Health Committees No.	Community Development Project*
None	75	168
Member	106	49
Chairman/Vice Chairman	19	7
Secretary/Vice Secretary	13	0
Treasurer/Vice Treasurer	14	0
Not stated	2	5
	229	229

* Includes overall project committee as well as working committees.

Table 1.3 *Activities of the CHWs in Saradidi.

1. Home visiting
2. Environmental health education
3. Promoting utilisation of maternal and child health clinics (immunisations and ante natal)
4. Treatment of malaria
5. Antimalarial chemoprophylaxis for pregnant women (in selected villages only)
6. Health education in village meetings and in schools.
7. Family planning education and services
8. Serving as midwives
9. Weighing newborns in the village
10. Recording vital events (births and deaths)
11. Nutrition education
12. Participation in meetings
13. Encouraging development in village and participation in income generating activities
14. Visiting and advising the sick
15. Participation in project clinic and mobile clinics
16. Attending training sessions
17. Preparation and submission of monthly reports
18. First aid and other first-line treatment of the sick in the village

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* As decided in meetings open to all residents of Saradidi.

Table 1.4 Problems that Community Health Workers perceive as hindrance to the performance of their responsibilites, Saradidi.

		% mentioned
1.	Lack of transport, e.g. ambulance, bicycles	94.4
2.	Difficulties in getting around at night or during rain (no flash light, gumboots or raincoats)	83.3
3.	No first aid kit	83.3
4.	People take long to understand and accept new ideas	55.5
5.	Does not have drugs for other diseases - only malaria	50.0
6.	Project clinic too far from her village	41.7
7.	Nutrition - lack of food so malnutrition	41.7
8.	Too many disease in the village	41.7
9.	Village not united, people do not attend meetings	27.8
10.	Environment hygiene - some people still not using latrines, dish racks, etc.	25.0
11.	Husbands interference in family planning activities	25.0
12.	Weak or non-functioning Village Health Community	22.2
13.	Lack of development in the village	22.2
14.	Water problems - village has no safe water	22.2
15.	Lack of teaching materials	19.4
16.	No payment for the work they do	19.4
	Total number	36

Table 1.5 Reasons for continuing as a Community Health Worker, Saradidi.

Reason	% mentioning
Training	100
Desire to help in the village	36.1
Family planning allowances	22.2
Likes the work	19.4
Effect/impact on the village	16.7
Personal development	13.9
Total number	36

Table 1.6 Mean and range scores before and immediately after the introductory training session. Community Health Workers (CHWs) by age, sex and education, Saradidi 1980-83.

Age	Number	Pre-test Mean	(range)	Post-test Mean	(range)	Paired t test	p
less than 20	5	80.2	(56-95)	86.2	(80-92)	1.22	0.29
21-30	75	66.3	(0-98)	79,6	(40-98)	6.21	0.0001
31-40	32	62.8	(0-98)	77.6	(8-98)	4.14	0.0002
40 or more	7	68.0	(36-88)	82.4	(60-96)	3.82	0.009
Uknown	2	77.5	(65-90)	81.5	(65-98)		
	103						
Sex							
Male	3	83.3	(80-90)	90.7	(85-95)	3.14	0.009
Female	100	65.8	(0-98)	79.2	(8-98)	7.84	0.0001
	103						
Education years							
None	4	51.0	(0-88)	63.5	(20-96)	4.35	0.022
1-	13	53.4	(0-84)	67.5	(8-98)	2.83	0.016
5-8	68	64.6	(0-98)	76.5	(40-98)	5.84	0.0001
9 or more	18	79.1	(56-98)	89.0	(76-98)	4.15	0.0007
	103						

Table 1.7 Mean difference between Pre-Score and Post-Score two years after initial training by age and education

Education (years)	Mean difference Number	In scores	P value
None	4	12.5	0.022
1-4	13	13.1	0.102
5-8	74	11.5	0.0001
9 or more	26	6.3	0.0001
	118		0.0001

Age (years)	Mean difference Number	In scores	P value
20-29	57	10.3	0.0001
30-39	45	11.6	0.0057
40 or more	14	11.1	0.0033
Total	118	11.0	0.0001

Figure 1.1 The map of Kenya showing the location of Saradidi community

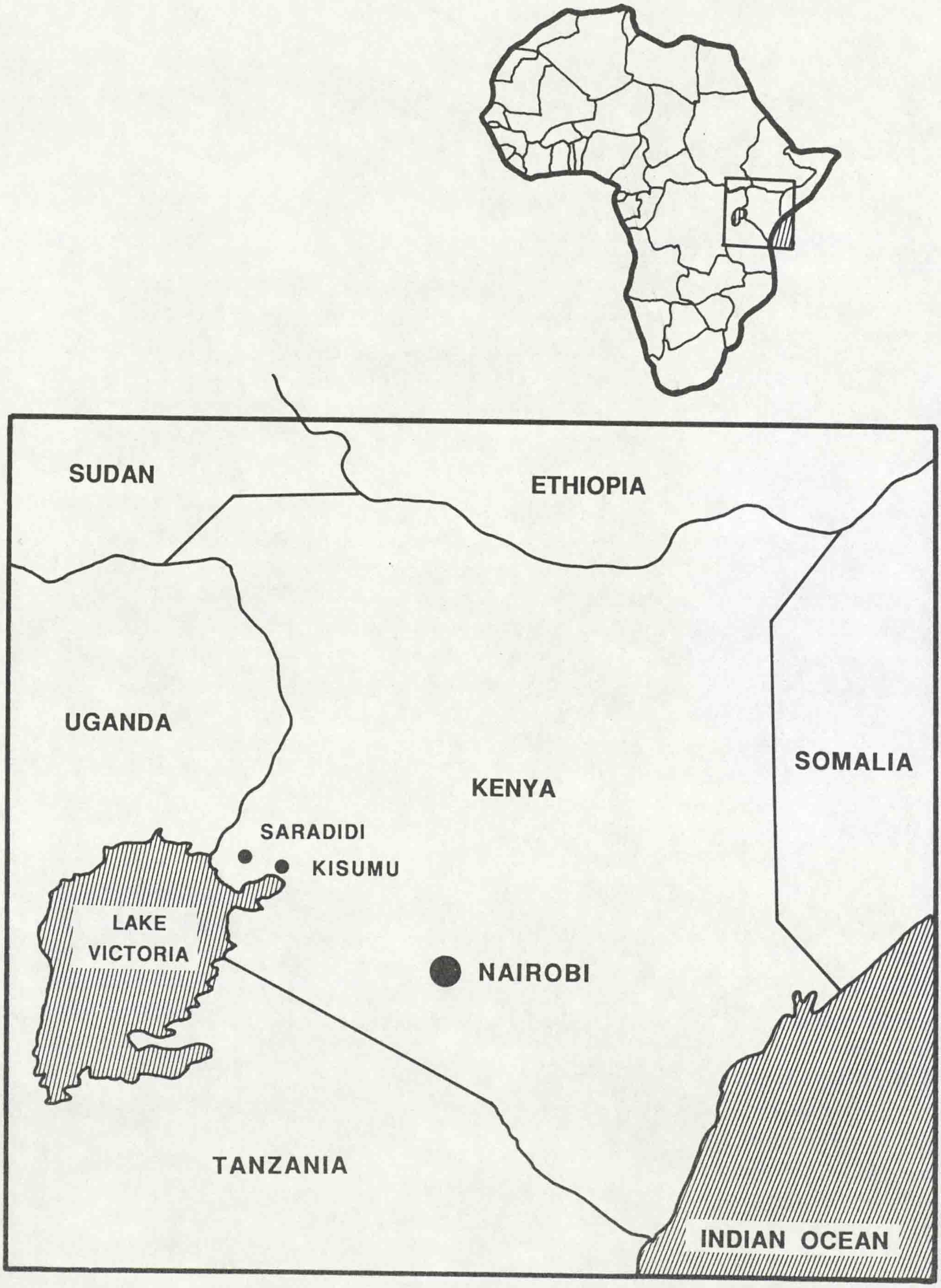


Figure 1.2

SARADIDI RURAL HEALTH PROGRAMME ORGANISATION CHART

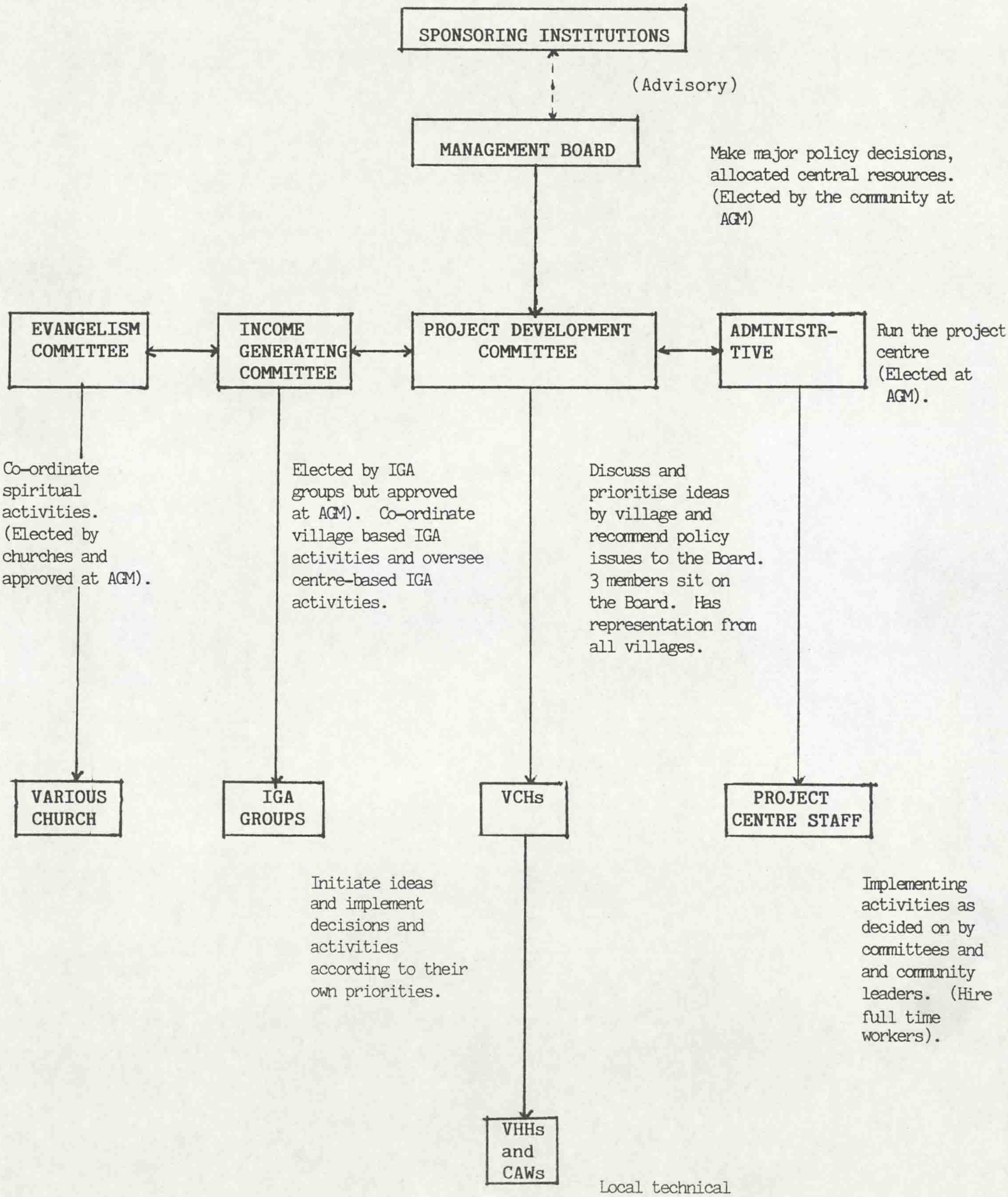
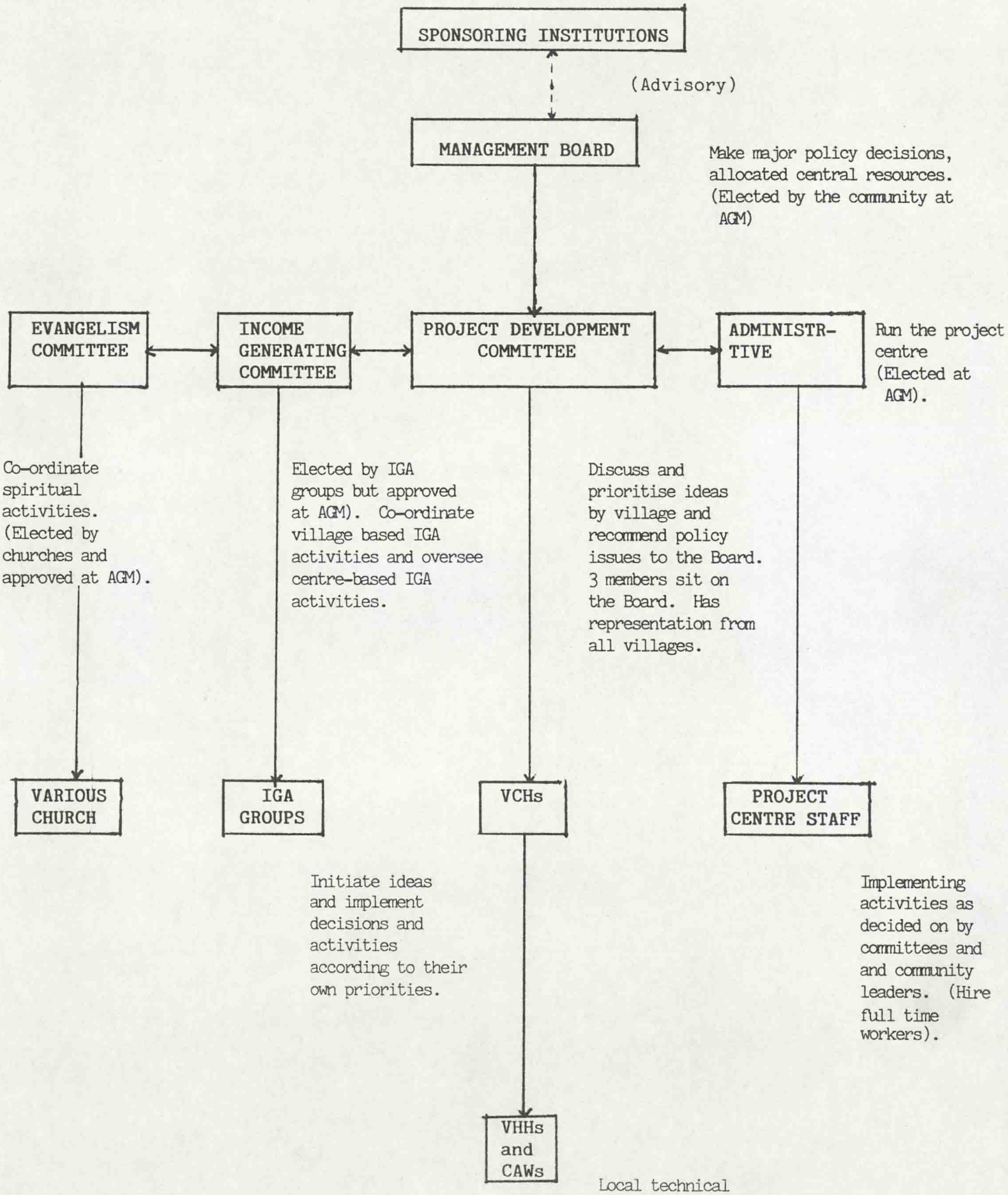


Figure 1.2

SARADIDI RURAL HEALTH PROGRAMME ORGANISATION CHART



CHAPTER 2

USAGE OF COMMUNITY-BASED CHLOROQUINE TREATMENT FOR MALARIA AND CHANGES IN SOURCES OF ANTIMALARIALS 1982-1987 AND FACTORS AFFECTING USAGE

2.1 INTRODUCTION

In May 1982, a community-based malaria control project was initiated in 36 villages in Saradidi. Treatment of malaria was provided in each village at no charge by volunteer community health workers (CHWs). Previous surveys done in the area had identified the major sources of medicine for malaria, to be shops, Ministry of Health facilities, and a mission hospital. Since the pattern of utilisation of treatment in the village would determine the effect on health, it was important to know if people used chloroquine whenever they thought they had malaria and whether they obtained it from the CHWs or they continued to use alternative sources.

It was also important to know not only the reaons for not obtaining the drugs from the CHWs, who provided them free of charge, but other factors that might have influenced use or non-use of chloroquine.

The Community Health Workers (CHWs) in areas A and B were provided with chloroquine phosphate. In both, tablets and syrup were provided to give to all symptomatic persons who came to them requesting treatment. In Area A, the CHWs also provided chloroquine phosphate to pregnant women for chemophrophylaxis. This system was backed up with information and education regarding the causes, prevention and treatment of malaria.

In this chapter the results of several studies are presented.

2.1.1 Sources of chloroquine

Repeated surveys to determine change in sources of chloroquine used for the treatment of malaria in Saradidi community during the study period 1982-

1987. Four surveys were done in areas A and B and three surveys in area C. The details of methodology have been described elsewhere (Mburn et al, 1987) and the survey questionnaires can also be found in a report prepared for the World Health Organisation who funded the project (Kaseje and Sempebwa, 1987). The surveys were carried out in April 1982. The surveys were carried out in April 1982, June 1983, December 1984 and December 1986.

2.1.2 Reasons for not using CHWs as a source

This study verified the shift in the main sources of chloroquine for malaria treatment and brought out the main reasons for not using CHWs as a source.

2.1.3 Consumption of chloroquine from shops in areas A, B and C

This study was designed and was carried out in 1985 to estimate the consumption of chloroquine from the shops and whether there were changes in the chloroquine sales that would correspond to the findings of the above two studies.

2.1.4 Consumption of chloroquine provided by CHWs

The CHWs kept records that enabled the study of chloroquine consumption by age, sex and season. The details of methodology and results have been presented elsewhere (Spencer et al 1987).

2.1.5 Itching induced by chloroquine phosphate

The epidemiology of this problem was studied as a factor that would affect any malaria control strategy based on chloroquine phosphate either for chemotherapy alone or in combination with selective chemoprophylaxis.

2.1.6 Symptoms associated with malaria by Saradidi community

One of the major problems with the use of CHWs to provide chloroquine

for the treatment of malaria is the fact that such a system relies on the people to diagnose themselves and their children and then go to the CHW for treatment. The CHW did not have to decide whether the illness is malaria or not, her job was to supply the chloroquine at the recommended dose. This study attempted to find out the extent to which the people of Saradidi could diagnose malaria based on the symptoms they associated with it and whether they could distinguish it from other diseases which may have similar symptoms.

The details of methodology and results of the study have been presented elsewhere (Spencer et al, 1987).

2.2 SOURCES OF CHLOROQUINE FOR THE TREATMENT OF MALARIA IN SARADIDI, 1982-1987.

2.2.1 Survey Method

A questionnaire was developed to determine the proportion of persons with malaria during the previous 2 weeks who obtained treatment from the CHWs, the usage of alternative sources of chloroquine and the reasons people failed to obtain treatment in their village from the community-based programme, and what people knew and did about malaria. The interviews were conducted by 2 female fourth-year Luo-speaking medical students. The interviewers were trained and the questionnaire was pretested in the community.

Results of field-testing were analysed and discussed with the interviewers and community members. The first survey began after the questionnaire was modified and was completed in 4 weeks. The same instrument was used in subsequent surveys. Villages and households in the chosen villages were randomly selected from the census records using a table of random numbers; 4 additional households close to the one selected were also visited. All persons aged 10 years and over were interviewed. Mothers were

asked the same questions on behalf of their children under 10. Households were revisited on at least 3 different occasions to find persons not available at the first visit.

2.2.2 Results

The people who were interviewed in each of the surveys may not have been the same but were similar in terms of age and education (Tables 2.1 and 2.2).

In the first survey (April 1982) most people were obtaining antimalarials from shops (Table 2.3). After May 1982 most people went to CHWs in their villages in 1986 people in areas A and B used more of the health facilities for their chloroquine while those in area C used mainly the CHWs. This was due to the fact that CHWs in areas A and B were recovering the cost of the drugs from the users beginning January 1985 while those in area C started providing chloroquine free of charge also beginning in January, 1985. Area C had been a control area during the period 1982 to 1984 and had been promised a free supply after the first phase of the study the main part of which ended in December, 1984.

2.3 REASONS FOR NOT USING CHWs AS A SOURCE OF CHLOROQUINE

Ten of the 36 villages with treatment provided by CHWs were selected and 100 households visited. A total of 222 persons 10 years of age and older were interviewed; 113 (50.9%) gave a history of malaria in the previous 2 weeks and 82 (36.9%) had taken medicine for malaria. Most, 133 (59.9%) of those interviewed were women, but no statistically significant difference by age or sex in the proportion of people with a history of malaria or who had received treatment was found.

The majority (82 or 72.6%) of the 113 individuals who thought they had

malaria took medicine. The differences in the proportions receiving treatment by age were not statistically significant. More than half sought and received treatment from the CHWs (Table 2.3). The other 2 major sources were shops and health facilities. A few obtained treatment from a family member or already had chloroquine at home.

The most frequent reasons for not getting medicine from the CHWs were that the CHW was not at home or had no drugs (Table 2.5). A total of 65 (79.3%) patients obtained or first tried to obtain chloroquine from the CHW (41 obtained from CHW; for 14 the CHW was not there; for 9 CHW had no drugs). In 4 (10%) instances, the patient was felt to be too sick for the CHW to treat; all these patients obtained antimalarials from a health facility usually the Saradidi clinic. Three individuals thought the CHW was 'no good', did not have adequate training and 2 said that it was more convenient to obtain chloroquine from a shop that was near to them when they became ill.

Similar results were found regarding treatment of children (Table 2.6). Of 103 children less than 10 years of age whose mothers were interviewed; 67 (65.0%) had had malaria in the previous 2 weeks; 59 (88.1%) of these 67 received antimalarial treatment.

Children 0 to 4 years had the highest proportion of any age group treated (72.4% of 54). The CHW was the largest source of treatment for children (30 of 56; 50.8%) followed by health facilities (20.3%) and shops (18.6%). Three (5.1%) children were given a drug already present at home. and for 3 children the source of antimalarial treatment was unknown.

As with adults. the 4 children who were considered too sick for the CHW to treat were all taken to health facilities, usually the Saradidi Clinic. Thus, a total of 41 (69.5%) of 59 children considered to have malaria went first to the CHW for antimalarial treatment.

The community-based programme rapidly became the main source of antimalarial treatment in Saradidi (Table 2.3). Drug consumption was high in both adults and children, perhaps because the survey was done in June which is the season of peak malaria transmission. The main reason for failure to obtain treatment from the CHWs were mostly logistical or organisational, such as that the CHW was not home or that she/he had no drugs. Since 96.8% of CHWs are women who have the same household responsibilities as other women in Saradidi, (Kaseje et al., 1986), it is not suprising that they were sometimes not at home. Lack of drugs is difficult to understand since the Saradidi centre maintained reserves to supply the CHWs. However, some CHWs live as far away as 20km from the clinic, and had to find time to go for their supply of drugs. An important reason for failure to use the CHW was the perception that the patient was too sick to be taken to the CHW. All of these individuals were taken to health facilities. This fact may be important in determining how effective this programme can be in reducing mortality rates due to malaria if the sickest patients continued to be treated as before. The dispensaries, hospitals and health centres these patients were taken to may on occasion not even have had antimalarials available. For most people in the Saradidi community the nearest health facility was more than 5km away and transport was not readily available even for very sick patients. The first survey done in April 1982 indicated small local shops to be the main source of antimalarial treatment; these shops stock various brands of chloroquine phosphate. The survey also showed that a number of patients received the drug from family members or already had antimalarials in their homes.

Thus, if the CHW was able to be available and have drugs all the time, then most members of the community, with the exception of those who were considered to be too sick at the time they sought treatment, or who already had chloroquine in their own homes, would use the community source of chloroquine. The CHWs seemed to be well accepted by the village since only 3

of the 222 persons interviewed in these 10 villages gave mistrust of these workers as a reason for not going to the CHW. Drug consumption during this period was high. A possible reason is that the survey was done in June 1983, a time of peak malaria transmission, as demonstrated in Chapter 3. Although the use of questionnaires can be fraught with difficulties in obtaining reliable information, the results presented here correlated well with those from records kept by CHWs of consumption of chloroquine phosphate discussed later in this chapter and with surveys of sources of malaria treatment done in Saradidi in 1982, 1984 and 1987. These results suggest that volunteer community health workers such as CHWs in Saradidi can be effective in supplying antimalarial treatment.

The third survey first confirmed the findings of the second survey. Although the local shops were important as sources of antimalarials, as some people had good reasons for going to them (Table 2.4) the community seemed to realise that they would not be able to provide adequate guidance regarding the choice of dosages and drugs . The drugs supplied by the CHWs were always available and accessible to the community and the CHWs were also well informed regarding the dosages. The drugs were also affordable. Hence one can conclude that the CHWs system made chloroquine available, accessible, affordable and acceptable to the community and the people were more willing not only to use chloroquine but also to complete the course of treatment.

In 1985 and 1986 the CHWs continued to provide chloroquine but they had to recover the costs from the users in order to:

a) ensure continuous supply of chloroquine at the village level;

b) ensure availability of chloroquine to those who genuinely needed it.

The fourth survey carried out in 1987 on sources of chloroquine used in the community still showed that CHWs were more important as source of antimalarial drugs (24%) than local shops (7%) and that most people now obtained their chloroquine from the Mission or Government clinics (29%) or Saradidi clinic (27%). They also now felt that the sources with the best

medicine for treating malaria were Saradidi clinic (33%) Mission and/or Government clinics (31%) and CHWs (23%). Only 3 people (6%) mentioned local shops as a source of good medicine.

The main factors mentioned by the community as determining the quality of the source was effectiveness in treating fever (40%) distance (33%) and cost (5%).

2.4 CONSUMPTION OF CHLOROQUINE FROM SHOPS IN AREAS A, B AND C

In 1985, it was felt necessary to examine the consumption of chloroquine phosphate from the shops in all the three study areas. Using questionnaires, information was gathered from the shops to try and estimate total chloroquine consumption in the three areas of study, apart from the clinics and community health workers.

Three methods were used:

a) retrospectively - we enquired how much chloroquine had been sold during a three month period before the onset of the study;

b) prospectively - a sample of shop-keepers were requested to keep records of all chloroquine sales for a period of three months;

c) the shop-keepers were asked how long it took them to sell 1000 tablets of chloroquine to try and double check on the findings from a and b above.

The results, standardised to 30 shops per area, are presented on Table 2.7. This table shows that there is higher consumption in area B. This could, however, be explained by the fact that in area B was the large Ndori shopping centre and market. It is a major centre drawing people from a large area beyond the project area and hence the higher rate of consumption. It was also clear that there was no marked difference between areas A, B (who have been receiving chloroquine phosphate from the CHWs since 1982) and area C where

provision of chloroquine by the CHWs began late in 1985. This study confirmed the fact that there is high consumption of chloroquine in Saradidi.

2.5 CONSUMPTION OF CHLOROQUINE PROVIDED BY CHWs

The malaria chemotherapy programme started in May, 1982. Chloroquine phosphate for treatment of malaria was provided in each village by community health workers (CHWs) who were chosen and supported by people living in the village (Kaseje et al., 1987). The dosage of chloroquine phosphate was 10mg base per kg given in a single dose. The number of pills or teaspoons of syrup (for small children) given were based on age and designed to ensure at least 10mg base per kg was taken (e.g. adults were given 5 tablets each of 150mg base). The dosage chosen was based on results of *in vivo* and *in vitro* drug sensitivity studies carried out in Saradidi prior to initiating malaria control activities (Spencer et al., 1983). Any person wishing treatment for malaria went to the home of the CHW. The antimalaria drug was taken in the presence of the CHW; none could be carried home. Exceptions to this rule were made for acutely ill individuals; treatment for these persons could be given to a family member to deliver to the patient.

For each treatment given the CHW recorded the date, name, age, and sex of the patient, where the patient lived in the village, the household and personal numbers, the village of residence if not the village where treatment was obtained, the number of pills or teaspoons of syrup given, whether the patient had requested previous treatment and, if so, the date. The printed record books were each numbered. Each page also had information indentifying the CHW, the village and the area. Each page had a unique number (e.g. the book began with the number 101, another with 201 etc.).

We selected a one-year period 1 September, 1982 to 31 August, 1983 to analyse the user records in order to determine the pattern of chloroquine consumption i.e. the rate of use by age and sex. The mid-year population was

obtained from census information as previously described (Spencer et al., 1987). 40,649 treatments were given to village residents. The treatment rate per person in the mid-year population was 1.24. However, at least 41.8% of the mid year population of 32,650 did not receive a single treatment. Multiple treatments were given to 50.5% of persons treated at least once and 13.4% of 13,879 persons treated at least once received 5 or more treatments during the year. Consumption patterns were not random; they were higher in females, and in the area with greater community organisation and community participation (Table 2.8).

The lowest rate of antimalarial treatments were in persons 30 years.

Females had significantly higher treatment rates than males in both areas (Table 2.8); may be because the mothers requested treatment when they took their children for treatment over and above treatments they obtained when they themselves were ill. It is also conceivable that the fact that CHWs who provide treatment were mostly women 15 to 59 years, might have determined utilisation by women as observed.

The age group with the highest proportion of the mid-year population treated at least once were children less than 5 years as was expected (Table 2.9).

The proportion of treatments varied by month. The lowest monthly per cent of treatments occurred in April-June, 1983. In 1983, the rainy season in Saradidi did not begin until May.

The results suggest that CHWs were effective in providing antimalarial treatment in spite of over use in certain age groups.

2.6 ITCHING INDUCED BY CHLOROQUINE PHOSPHATE

Another important factor that would affect the use of chloroquine for

treatment or prevention of clinical malaria is the itching often induced by chloroquine ingestion. This problem has been reported in Africans and non-Africans (Berliner et al., 1984, Ekpechi & Okoro, 1964, Olatunde, 1977, Olatunde & Obih, 1981, Spencer et al., 1983, Isifo, 1984). When chloroquine phosphate was made available in May 1982 in Saradidi, Kenya in each village as part of a community-based malaria control programme reports of chloroquine-induced pruritus began to appear. Because of the concern that this side effect would influence drug usage, a study was undertaken to determine the prevalence and some of the epidemiologic features of chloroquine-induced pruritus in Saradidi.

2.6.1 Materials and methods

Two investigations were carried out, one in school children (January 1983) and the other in adults (March 1984). The methods were the same in both studies.

Subjects were informed that they would receive treatment for presumptive malaria and should return for examination the next day. Consent was obtained from the adult patients or from parents or teachers of the children. A thick blood film for malaria parasites was taken, and a urine sample was examined for 4-aminoquinolines by the Dill-Glazko test (Lelijveld & Kortmann, 1970). If this test was negative, patients were weighed and then given either 10mg base per kg of regular chloroquine phosphate (white tablet of 150mg base), 10mg base per kg of enteric-coated chloroquine phosphate (red tablet of 300mg base), 10mg base per kg of amodiaquine (yellow tablet of 200mg base), or 300mg of enteric-coated ferrous sulphate (green tablet). Since enteric-coated chloroquine is not available in Kenya, the participants in the study did not know what these pills contained. Enteric-coated ferrous sulphate is not available in rural Kenya, although it can be purchased in urban areas.

The same day, the blood films were stained with giemsa and examined for malaria parasites. On the following day, a repeat Dill-Glazko test was done

on urine sample from each individual. Then each person was asked 3 questions in the following order: 1) Have you had any problems since treatment yesterday? 2) If yes, what? 3) Have you had itching? (The last question was asked only if itching was not mentioned in the answer to the second question). The interviewers were members of a field team who had been working in the area 3 or 4 years and who were well known to the study population. Interviews were conducted in Luo, the local language, and all subjects were interviewed separately. Interviewers had no knowledge of parasite results or which drug the person had received. Itching was considered to be present if the person mentioned it in response to the second question or if they replied affirmatively to the third question. Persons who had received iron and who had a blood film showing malaria parasites were treated with chloroquine. Persons who had a positive Dill-Glazko test before treatment or those who received iron but had a positive Dill-Glazko on follow-up were excluded from analysis, as were all persons without a urine sample at 24 hours.

For the study in children, every pupil in a randomly selected school was examined. In the adult study, persons were asked to come to places where a community meeting or mobile health clinic was being conducted. The 3 sites selected for the adults were each at least 5km from the other.

2.6.2 Results

There were 426 children in the school study and 193 persons in the adult study. Eleven persons under 18 years of age enrolled in the adult study for the purpose of analysis these 11 were considered to be children, giving a final figure of 437 children (less than 18 years) and 182 adults (18 years or more).

More adults (20.3%) reported itching than did children (12.8%) ($p<.05$); and there was no significant difference between males and females noted, (Table 2.10). A history of itching after 24 hours after treatment was not significantly more common in persons with malaria parasitaemia. Pruritus was

more frequent in those receiving regular chloroquine (21.5% of 186) and enteric-coated chloroquine (17.8% of 118) than after amodiaquine (11.6% of 173) or iron (8.5% of 142) ($p<.005$). Amodiaquine which is a 4-aminoquinoline like chloroquine did not appear to cause significant pruritus in this population. These results demonstrate that chloroquine-associated pruritus is experienced frequently in Saradidi.

This side effect of malaria treatment could influence usage of chloroquine phosphate provided by community health workers.

2.7 SYMPTOMS ASSOCIATED WITH MALARIA BY SARADIDI COMMUNITY

The priority of the malaria control activities at Saradidi, a holoendemic area in tropical Africa, was according to WHO recommendations the reduction of mortality through making chemotherapy as readily available to every member of the community as possible. This was done in Saradidi through the community health workers selected by the community but trained by the health services personnel. This system depended completely on the community members recognising their or their childrens need of chemotherapy and then going to the community health worker for it. One factor determining the use of the chemotherapy programme was the symptoms that the community felt to be associated with malaria.

We attempted to investigate what these symptoms would be.

2.7.1 Method of the study

a) Symptoms and Diseases

A list of common diseases and symptoms was elicited as follows: Villages and the Saradidi Programme Centre were visited and people at random were asked to name diseases and symptoms (in Luo, the local language) they considered to be common in Saradidi. Lists were merged and a final list generated which consisted of those diseases and symptoms recognised as

'common' by a majority of people interviewed. This final list was then pre-tested in the field and further revised as required by the results of the pre-test. One problem encountered was that occasionally a symptom was synonymous with the name of a particular disease in Luo.

b) Study Population

Thirty-six women were interviewed. Women were chosen because they were most often in the household and because they are responsible for health care for themselves and for children under 5 years in this community.

Nine villages were selected using a table of random numbers. Households were then chosen randomly in the same way and 1 woman in each household was interviewed. Four women from each village were interviewed; 1 from each of the age groups 15-29 years. 30-44 years, 45-59 years and 60 or more years. Households were visited a total of 3 times before an alternative to the selected women was interviewed.

The woman was asked whether she knew the disease. If she said yes, then she was asked the symptoms one by one and if a particular symptom was caused by that disease or not; responses were recorded as yes, no and unknown.

c) Interviews

The study was carried out by a male Luo-speaking technologist, and interviews were conducted in Luo.

d) Analysis

Approximate English translations of the Luo terms for the common symptoms and diseases were obtained. In an effort to group 'like' disease based on the constellation of symptoms perceived associated, an average linkage hierarchical cluster analysis was carried out using the Statistical Package for the Social Science (SPSS) Programme D'Andrade et al., 1972, Nie et al., 1975,

Young, 1978). This technique sorted 'like' diseases together based symptom responses according to the number of clusters. For example, if there were 6 clusters, all diseases were put into one of the 6; diseases in 1 cluster were more closely associated by response than those in another.

2.7.2 Results

a) Study population

Twenty-five (69.4%) of the women interviewed were those randomly selected: 7 (19.6%) were first alternates, 3 (8.3%) were second alternates and 1 (2.8%) was third alternate.

b) Diseases known

Measles, malaria and influenza were known by all 36 women (Table 2.11). Tetanus was recognised by only 8 women. Woman 60 years and older knew a higher proportion of the diseases. Of a possible 108 positive responses (9 women x 12 diseases). Women 60 years of age and more had 92 (85.2%). Those 45 to 59 years had 77 (71.3%), those 30 to 44 years had 68 (63.9%) and those 15 to 29 had 76 (70.4%). These differences were statistically significant (Chi square = 13.95, df = 3, $p<.0005$).

c) Associations of diseases and symptoms

The association of symptoms with diseases were very similar among the different age groups of women and so the results were combined. More than 90% of women associated headache, fever, vomiting, joint pain, loss of appetite, tiredness, and death with malaria. Madness was associated with malaria by 77.8%. Many symptoms associated with malaria were also associated with measles and influenza. Major distinguishing symptoms appeared to be rash and ulcers in the mouth for measles and 'runny' nose and sneezing for influenza. More details have been previously described (Spencer et al, 1987).

e) Cluster analysis

Malaria and measles were distinct at any grouping level with 3 or more clusters and malaria, measles and influenza were not associated when 4 or more clusters were formed. Tuberculosis and whooping cough were the most closely associated. (Spencer et al, 1987).

Based on symptoms, the women interviewed clearly distinguished malaria from measles and influenza. The constellation of symptoms associated with malaria were those expected. The symptoms associated with diseases other than malaria were also reasonable based on known clinical manifestation.

Diseases could be separated based on perceived symptoms. The results suggest that if people in Saradidi do not obtain treatment from community health workers, it is not because they do not recognise the clinical symptoms of malaria. They seem to have enough knowledge of the symptoms to effectively use the chemotherapy programme. Cluster analysis has been used successfully in examining the properties defining disease states (D'Andrade et al, 1972; Young, 1978, Welles, 1983 & 1984 and Spencer et al, 1987). A great variety of properties may serve as distinctive features in defining disease states. These include type of agent, body, location, symptom types and order of appearance, duration of illness, degree of disability and others. Different cultures deal with, think about, and integrate disease into their wider system of beliefs, values and behaviours in different ways. Thus, while these results present associated symptoms they give little insight into disease concepts and factors in Saradidi that would predict their health seeking behaviours and their choice of the source to use for various ailments. Attitudes and beliefs regarding available health services (including the traditional health system in general and the community based system in particular) will influence drug use and behaviour.

2.8 DISCUSSION

2.8.1 Sources of chloroquine

To determine the changes in source of antimalarial treatment and perceptions about malaria after the initiation of a community based malaria control programme in Saradidi, Kenya; three identical surveys were carried out, one in April, 1982 (before the project began in May 1982), the second in December, 1984 during the community based chemotherapy with free drugs and the third one in December 1986. The three areas were involved: area A and B had antimalarial treatment provided by community health workers (CHWs) and area C had CHWs who did not provide treatment. Two groups of randomly selected women aged 15 to 59 years were interviewed: 45 in survey 1 and 92 in survey 2. A decided change in the source of malaria treatment was observed. In the first survey, 52.9% of the respondents from areas A and B combined purchased antimalarial medicine from shops; other sources were government health facilities, mission clinics, and the Saradidi community clinic. By the second survey, 85.2% of the respondents in areas A and B obtained treatment from the CHWs; no significant change occurred in area C. In both surveys the leading reason given for people purchasing drugs from shops was that the distance to health facilities was great, that no transport was available and that shops were open when illnesses occurred. The shopkeeper frequently advised which drug to take and the dosage as well as selling the drug. For family illness of unknown aetiology most people (82.2% in survey 1 and 78% in survey 2) went to a hospital or clinic. These results demonstrate that the malaria control project in Saradidi influenced both the source of antimalarials and attitudes people have about malaria. In Saradidi, people chose to obtain antimalarial treatment and advice mainly from community health workers. However, consumption of chloroquine from shops was also still quite high perhaps indicating the fact that people were now more aware of the need to treat malaria with drugs and what drugs to use so they could purchase them when needed.

We changed the drug supply scheme in areas A and B from free supply to a system where the users except pregnant mothers, had to pay for chloroquine. The third survey was conducted to examine the impact of this change on sources of antimalarial treatment and their perceptions about malaria in December 1986. The results showed that people obtained drugs from Mission or Government clinic (29%) Saradidi clinic (27%), CHW (24%), and local shops (7%). Local shops have remained an insignificant source of drugs. The CHWs rapidly assumed prominence as source of drugs in area C as soon as they started supplying them. The source of best medicine for malaria was felt to be Saradidi Clinic (33%) Mission or Government clinics (31%) and CHW (23%).

The community determined where to go due to the quality of the source in its effectiveness of treating malaria (40%), distance (33%) and cost (5%). It should be noted that the CHWs only stocked chloroquine and no other drugs. Increasing the variety of drugs stocked by the CHWs would have improved their attractivity even more as compared to the local shops, particularly when they had to recover the cost of drugs from the consumers.

The three surveys done: one before the community-based chemotherapy was launched, two during its implementation and a last one after the active phase ceased demonstrated marked and lasting changes in reported behaviours of people. This change was also reflected by the records of drug use kept by CHWs. While the shops were the principal source of antimalarials in 1982, CHWs became the main source by June, 1983 and remained so as determined again in 1984 and 1986. The use of shops dropped considerably and remained negligible. This change was not observed in the control area in 1984 where more than half the people with malaria still obtained their chloroquine from the shops. People liked the shops because they were near and had the drugs all the time. A system to replace the shops must meet the needs met by the shops i.e. be close to the people and ensure availability of drugs at all times but also offer a variety of drugs. The CHWs succeeded in doing this at Saradidi. Their

effectivenesss can be enhanced by giving them a variety of drugs.

The shops are not necessarily bad sources of chloroquine but the shop-keepers would need training, guidelines and supervision if they were to be effective in giving people advice to their clients. We tried organising training for shop-keepers in Saradidi without much success as there was no incentive for them to be trained. They saw training, as wasting their valuable time.

The malaria control programme at Saradidi has influenced the source of antimalarials used, the knowledge of the cause and treatment of the disease; and their attitude and practice regarding malaria. People obtained treatment and advice from CHWs. People were willing to pay for the drugs which continued to be available with the CHWs at cost. The CHWs source of malaria is still preferred to local shops.

2.8.2 Chloroquine consumption

The results suggest that CHWs were effective in providing antimalarial treatment in Saradidi. More than 42,000 treatments were given during the study year. The treatment rate per person in the mid-year population was 1.24. These rates were higher in area A which had had a longer period of the community based programme (Spencer et al, 1987).

The incidence of malaria in this community was worked out in 1985 and the results are presented in Chapter 3. The children under five years who were continuously exposed to infection without any protection had high rates of symptomatic attacks but attack rates of clinical malaria among adults were much lower (Molineaux and Gramiccia, 1980). Females 15-49 were more often treated than males in the same age groups. More than half of the persons treated had multiple treatment. All these indicate a factor of wastage of drugs. Mothers, for example may have requested treatment from the CHW whenever they brought the child and this would add to the treatments given to adult females.

This excess use was another strong indication for making the drugs available to the community at cost. This would select users who are in real need of treatment. It can be concluded that the volunteer CHWs were effective in providing chemotherapy at their villages although there was excess use by females in the child bearing age.

2.8.3 Pruritus

Chloroquine-associated pruritus was experienced frequently by the population in Saradidi. Its occurrence in 20.1% of 304 persons receiving regular chloroquine was similar to the rate of 8% to 28% reported from Nigeria (Olatuude, 1977, Olatuude and Obila, 1981). In a previous study (Osifo, 1984), the maximum intensity of pruritus occurred $\pm$ 12 hours after ingesting chloroquine. Therefore, the true rate may have been slightly underestimated in this study, since all persons were interviewed 24 hours after treatment. Amodiaquine the 4-aminoquinoline like chloroquine was not demonstrated to cause pruritus. Since 8.5% of the people who received ferrous sulphate itched it means that there is a certain amount of false itching. This could have caused over estimation in this study. The real rate of itching may be closer to 12% in the Saradidi community.

Pruritus occurred less frequently in children than adults but there was no sex difference. The presence or absence of malaria parasites also did not affect chloroquine associated itching.

These results mean that the selection of drugs for use in the control of malaria in Primary Health Care (PHC) either for community based chemotherapy or for chemoprophylaxis for vulnerable groups like pregnant women should take this factor into account. People who itch with chloroquine are unlikely to comply with prescriptions of the drug either for prophylaxis or therapy. The mothers who itch may also withold the drug from their children. It may be necessary to discuss the problem and its remedies with the users to enable them to comply with the required medication.

2.8.4 Recognition of malaria

Based on symptoms, the women interviewed clearly distinguished malaria from measles and influenza. The constellation of symptoms associated with malaria were those expected. The symptoms associated with diseases other than malaria were also reasonably based on known clinical manifestations. Diseases could be separated based on symptoms. The results suggest that people in Saradidi could make a reasonable self-diagnosis of malaria and should be able to take appropriate action to treat it by going for antimalarials at the sources available.

Cluster analysis has been used in examining the properties defining disease states (D'Andrade et al, 1972, Young, 1978, Wells, 1983, 1984). The present study was only interested in symptoms as the community-based chemotherapy was mainly interested in the availability of the required ability for reasonably accurate self-diagnosis and to take an appropriate decision to seek help. It is to be expected that attitudes and beliefs regarding health, illness, available services will influence drug used and behaviour. These factors must be considered when planning, implementing, and evaluating similar community based disease control activities such as Saradidi. Thus providing chemotherapy through CHWs appears to be the best approach to malaria action in PHC at this time, (Jeffrey, 1984). The CHW must be supervised and supported by the supplies and a referral base. Some problems of implementation would include those identified by MacCormack, and Lwihula, 1983 e.g.

- logistic problems
- side effects of the drug e.g. itching
- effectiveness of the drug which has become more important since the emergence of chloroquine resistance.

The involvement of the community in the total process of planning implementations, monitoring and evaluation of the scheme would minimise

these problems. The chemotherapeutic activities should be supported by an effective educational process and community based antivector activities suggested by Stevens (1984).

Table 2.1 Selected characteristics of persons interviewed about antimalarial treatment Saradidi, 1983.

Age	No interviewed	History of malaria* No. (%)	History of taking medicine for malaria* No. (%)	Proportion of those with malaria taking medicine
0 - 4**	54	42 (77.8)	39 (72.2)	92.9 (39/42)
5 - 0**	49	25 (51.0)	20 (40.8)	80.0 (20/25)
10 - 14	59	30 (50.8)	19 (32.2)	63.3 (19/30)
15 - 29	59	25 (42.4)	22 (37.3)	88.0 (22/25)
30 - 44	42	25 (59.5)	18 (42.9)	72.0 (18.25)
>45	62	33 (53.2)	23 (37.1)	69.7 (23/33)
	325	180 (55.4)	141 (43.4)	78.3 (141/180)

* In the previous 2 weeks
** Information obtained from mother

Table 2.2 Age and education of women interviewed by survey, Saradidi.

Age in years	Survey 1 (March 1982) %	Survey 2 (December 1984) %
15 - 29	49.7	25.0
30 - 44	28.9	39.1
45 - 59	24.4	35.9
Total Number	45	92
Education in years		
None	44.4	32.6
1 - 4	13.3	31.5
5 or more	42.2	35.9
	45	92

Table 2.3 Source of antimalarial treatment by area and year of survey, Saradidi.

Source	Area: A and B combined, Survey yr. 1982	1983	1984	Area: C, Survey yr. 1982	1984
Shop	52.9**	28.0	1.6	45.5	61.3
CHW	-	51.2	85.2	-	-
Other***	47.1	20.8	13.1	54.5	38.7
No. of respondents	34	82	61	11	31

** Percentage of group with this source.
*** Includes Saradidi clinic, Mission clinic, and Ministry of Health Facilities.

Table 2.4 Reasons why people buy medicine for malaria from shops by area and survey, Saradidi.

Reason	A and B combined Survey		Area C Survey	
	1*	2*	1	2
Other sources too far/no transport	32.4**	-	45.5	41.9
Necessary in emergencies	17.6	8.2	45.5	48.4
Shop medicine good	14.7	-	-	3.2
Shops are cheap	5.9	-	-	-
Hospital/dispensary has no drugs	11.8	-	9.1	-
Other	11.8	-	-	-
Do not know	5.9	1.6	-	-
Most do not use	-	90.2	-	6.4
Number of respondents	34	34	61	11	31

* Survey No. 1 conducted in March, 1982; Survey No. 2 conducted in December, 1984.

** Percentage of group responding.

Table 2.5 Reasons that 40 persons (> 10 years) failed to obtain chloroquine for treatment of malaria from the community health worker (CHW) Saradidi.

Reason	Percent
CHW not there	35.0
CHW had no drugs	22.5
Patient too sick	10.0
Drugs already available in home	10.0
'Not registered' with CHW	10.0
CHW 'no good'	7.5
More 'convenient' to obtain elsewhere	5.0

Table 2.6 Reasons why 30 children (< 9 years) did not obtain antimalarial treatment from the community health worker (CHW) Saradidi.

Reason	Percent of Children
CHW not at home	23.3
CHW had no drug	13.1
Treated with drug already at home	20.0
Treated in school survey	13.3
Child 'too sick' for CHW	13.3
Too far from CHW when became ill	3.3
Unknown	13.3

Table 2.7 Chloroquine consumption from the shops by area - 1985

Area	Total Amount Chloroquine used	Number of Shops	Average Amount sold per shop
A	Tablets	30	371.3 tablets
	11,138		
	Syrup		
	7,115 mls	30	237.2 mls
B	Tablets		
	15,687	30	522.9 tablets
	4,691 mls	30	156.5 mls
C	Tablets	30	388.6 tablets
	11,658	30	10 mls
	Syrup		
	300 mls	30	10 mls

Table 2.8 Mid-year population, number of malaria treatments given and treatment rate per person by sex, Saradidi - 1st September, 1982 to 31st August, 1983.

	Area A		Area B		Area C	
	Males	Females	Males	Females	Males	Females
Mid year population	9233	10658	6046	6610	15279	17268
Number of treatments given	11457	15557	5771	7488	17228	23045
Treatment rate per person	1.24	1.46	0.95	1.13	1.13	1.33

Table 2.9 Antimalarial treatments per person* by age, Saradidi - 1st September 1982 to 31st August 1983.

Age (years)	1	2	3	4	5 to 9	10 or more	Number of persons Treated at least Once in age group
<1	55.3**	20.2	8.7	4.7	9.5	1.6	633
1 - 14	52.7	20.8	10.1	5.2	8.8	2.4	5898
15 - 29	51.9	20.3	10.6	6.5	9.1	1.6	2941
> 30	42.6	18.6	12.4	7.8	14.2	4.4	4382

* Areas A and B combined

** Persons with absent identification numbers excluded. Analysis based on recorded identification numbers.

** Percent of age group treated this many times.

Table 2.10 Itching by type of drug in school children and adults Saradidi by prompted and unprompted answers.

	Unprompted		Prompted			
Drug 2	No.	% itching	No.	% itching	Both	
Iron	102	7.8	40	10.0	142	8.5[3]
Amodiaquine (AD)	126	10.3	47	14.9	173	11.6[3]
Enteric-coated chloroquine (ECH)	65	13.8	53	22.6	118	17.83
Regular chloroquine (CH)	144	18.1	42	33.3	186	21.53
	437	12.84	182	20.34	619	15.0
	Chi square = 6.56		Chi square = 8.05		Chi square = 13.3	
	df = 3		df = 3		df = 3	
	p<0.1		p<.05		p<.005	

1 History of itching by either direct or indirect questioning

2 See text for dosages

3 AD compared with CH Chi square = 5.67 df = 1 p<.05

AD compared with ECH Chi square = 1.76 df = 1 NS*

ECH compared with CH Chi square = 0.4 df = 1 NS

Iron compared with AD Chi square = 0.5 df = 1 NS

Iron compared with ECH Chi square = 4.2 df = 1 p<.05

Iron compared with CH Chi square = 19.8 df = p<.01

4 Adults compared with children Chi square = 5.68 df = p<.05

* Not signficant p>0.05

Table 2.11 Proportion of 36 women who knew a particular disease Saradidi.

Disease	Proportion of women
Malaria	100
Measles	100
Influenza	100
Ear infection	86.1
Worms	86.1
Gastroenteritis	65.0
Whooping cough	72.2
Tuberculosis	63.9
Chickenpox	58.3
Cholera	55.6
Schistosomiasis	50.0
Tetanus	22.2

CHAPTER 3

MALARIA TRANSMISSION AND CONTROL IN SARADIDI

3.1 INTRODUCTION

Human malaria is caused by the four major Plasmodium species: P.falciparum, P.vivax, P.malariae and P.ovale. In most parts of the world, the prevailing parasite is P.falciparum which causes the most severe form of acute disease that is often fatal depending on the immune status of the host. The parasite is transmitted by various species of the anopheline mosquito from one infected host to another susceptible human host. The sporozoite, the infectious form of the parasite, released from mosquito salivary glands, initiates the exoerythrocytic cycle in the liver. Subsequently the developed merozoites invade erythrocytes, and the asexual cycle continues through the course of the infection. A subset of the erythrocytic form of the parasite develops into male and female gametocytes. The gametocytes when taken up by the female anopheles mosquito develop into sporozoites which result in disease transmission.

Malaria is one of the major infectious diseases in the world, with acute malaria affecting more than 100 million people each year according to World Health Organization (WHO) estimate (WHO Statistical quarterly, 1984).

Elimination of malaria worldwide has proved impossible to achieve and thus the current efforts are devoted to the control of malaria, World Health Assemby (1969). Even in the few instances where eradication was attempted the achievement was only transient. The main objective was therefore changed from eradication to long term control as in the official resolution of the World Health Assemby of 1969. The long term control strategy must also be carefully planned in accordance with the local malaria situation and the extent of development of primary health care services. The principles involved include:

- formation of clear objectives based on the local malaria situation;
- easy access to antimalarials for treatment to every member of the

community living in malarious areas and with enough information and education to enhance proper use of chemotherapy;

- adequate case detection and treatment within PHC;
- community participation in all antimalarial activities;
- adequate referral and technical back up support team;
- assessment of impact the results of which are fed back into planning and operation of activities.

The control measures to be implemented must be those that can be sustained by the local system indefinitely and must differ from place to place according to various environmental, socioeconomic and biological factors that determine the stability of malaria transmission.

In this chapter an attempt is made to define various malaria situations, present how malaria transmission can be determined in a geographical area, and to discuss control strategies appropriate for each situation. The Saradidi Community based malaria control project is presented in summary: how it was carried out and how its impact on parasite prevalence and antimalarial antibodies was assessed.

3.2 MALARIA TRANSMISSION

It has been known for the better part of this century, that malaria transmission is related to climatic conditions as summarised by Gill (1938). This knowledge was there even before the mode of transmission was worked out and before the disease agent was known. The name of the disease itself suggests its relationship to the climatic conditions. This was reviewed by Russell and his colleagues in 1963 (Russell et al., 1963). In 1938 Gill defined four climatic zones of malaria in the world (Gill, 1938). Later, in 1963 Macdonald distinguished 12 epidemiological malaria zones in the world. During the same period malaria maps were produced in Kenya and Tanzania by their survey departments (Survey of Kenya, 1956; Survey of Tanganyika, 1956).

Various determinants of malaria transmission have been used to define these zones of malaria: the length of the transmission season; level of endemicity based on spleen enlargement and parasite prevalence rates and the ecological systems that depend heavily on the climatic conditions as described by Weng (1985). Malariologists have found it necessary to identify, characterise and delimit malaria situations in order to work out appropriate control strategies. This is done by studying the local variability at the meterological level in order to improve the effectiveness and efficiency of the malaria control as described by Koznetsov (1986).

This effort to identify, characterise and delimit the malaria situation in different areas has been termed the stratification of malaria. The stratification idea recognises that the problem of malaria today is complex and is getting even more complex. The determinants of malaria distribution are no longer simply biological or climatic but also include sociological and economic factors and more recently the response of the malaria parasite (particularly Plasmodium falciparum) to treatment.

3.2.1 Epidemiological determinants

Rainfall, temperature, altitude, malaria vectors, prevalence, incidence and related mortality and morbidity and their distribution in the population.

3.2.2 Socioeconomic determinants

Demographic patterns (population structure and movement), socio-cultural characteristics of various communities, economic activities and projects (e.g. irrigation and hydroelectric schemes), housing (where sited and how constructed) and economic activities of individuals (e.g. fishing).

3.2.3 Resource distribution, availability and effectiveness for malaria control

Availabilty of primary health care services and response of the local parasite to available treatment.

It can be seen from these determinants that the malaria strata that may be defined cannot be discreet but must overlap greatly as various factors will exist in varying combinations.

3.2.4 Definition of malaria strata

a) Stratum 1

Rural areas with traditional agricultural system and stable perennial malaria transmission (e.g. in the forest belt of tropical Africa). In this stratum the incidence and prevalence of malaria are both very high. Morbidity and mortality are concentrated in children under the age of 5 years, pregnant women (particularly those pregnant for the first time), and visitors from non-malarious areas. The adults are clinically immune but have malaria parasites circulating in their peripheral blood. These are the areas described as holoendemic using the endemicity classification suggested by WHO at the first conference on malaria in Africa in Kampala, Uganda in 1951 (WHO, 1951). Saradidi community belongs to this stratum.

b) Stratum 2

Rural areas with traditional agricultural system but with stable seasonal malaria transmission. This is found in savanna areas and is characterised by high prevalence and incidence most of the year but with marked seasonal variations. For this stratum, morbidity tends to be concentrated to a greater or lesser extent, depending on the level of overlap with the first stratum above, among children under age 5 years, pregnant women, particularly those pregnant for the first time and immigrants from non-malarious areas. These areas are described as hyperendemic.

c) Stratum 3

Arid or semi-arid rural areas with traditional agricultural system with unstable transmission due to aridity. Morbidity and mortality in these areas extends to adults and may vary greatly from year to year. The areas are characterised by recurrent epidemics.

d) Stratum 4

High altitude rural areas with traditional agricultural system and unstable malaria transmission and with recurrent epidemics.

e) Stratum 5

Populations of nomadic or semi-nomadic pastoralists who may belong to more than one stratum. They will also tend to experience recurrent epidemics affecting all age groups.

f) Stratum 6

Modern irrigation schemes tend to extend transmission period and stabilise malaria situation. The widespread use of insecticides in agriculture may render the local mosquito vector prone to developing resistance to insecticides that may be used for mosquito control. Migrant labour in the area may influence transmission, incidence, prevalence, morbidity and mortality. The extent of this influence depends on the place of origin of the migrants and how they are settled in their new settlement or work areas.

g) Stratum 7

Temporary development projects e.g. bridge and other construction works. The development activities may increase breeding sites and may also bring about migration and migrant labour that may create a unique malaria transmission situation.

h) Stratum 8

Urban and suburban areas.

This classification is a modified form of the one suggested by Lysenko (1983).

Since drug resistance is rapidly developing in sub-Saharan Africa (Onori, 1984), further consideration is now also given to the response of the local parasites (P.falciparum) to chloroquine. Each of the above may be described further as having malaria parasites that:

- are fully sensitive to chloroquine
- have low level resistance to chloroquine

- have high level resistance to chloroquine
- have high level resistance to chloroquine and resistance to Fansidar.

This has been suggested by the World Health Organization (1984).

Whereas the global malaria situation which got worse in the period 1973-76 has tended to improve in the last 15 years, the malaria situation in Africa south of the Sahara has remained either unaffected or deteriorated. In most of rural Africa, malaria transmission is at its highest, maintained by the most efficient vectors, Anopheles gambiae, An.arabiensis and An.funestus. Nevertheless, considerable geographical and seasonal variations occur due to altitude, natural drainage or the extent of the dry season as described by Fontaine (1987), and economic factors described above, and explained by MacCormack (1984).

3.3 A METHOD OF DETERMINING MALARIA TRANSMISSION

A study designed to define transmission of malaria by studying its incidence in Saradidi, Western Kenya, demonstrates one method that can be used. The study correlated incidence of malaria among children under 6 years of age with entomological and meteorological data gathered during the same period of time (January to December 1986).

3.3.1 Method of study

Every four weeks a cohort of about 47 children aged 6 months to 6 years were enrolled in the study. They were examined at enrolment and a thin and thick blood slide was made.

All the children were treated with Fansidar (sulfadoxine/pyrimethamine combination) and were seen weekly or whenever the child was unwell thereafter for 12 weeks. This was to detect any new illness and ensure that no antimalarial drugs were given them except within the study. Each time the child was seen for any illness malaria slides were repeated and treated promptly if positive. Other

illnesses were treated but with drugs known to have no effect on malaria parasites.

Additionally, routine malaria smears were made on the children on weeks 1, 2, 4, 6, 8 and 12 after the initial treatment. All the smears were stained with Giemsa and 200 oil immersion fields were examined independently by two observers. Children who developed recurrent parasitaemia on or before day 28 following treatment were considered to have had a recrudescence, and were treated and dropped from the study. The remaining children were checked biweekly for new parasitaemia and were treated as soon as found positive and dropped from the study. The rest were followed for 12 weeks after their enrolment.

The incidence of new malaria infections was calculated for the biweekly periods, after week 4, and was expressed as the proportion of children who developed parasitaemia during the two week period among those who were negative at the beginning of the period.

Concurrent studies were performed on Anopheles vectors of malaria in the community once a week. Six pairs of men collected mosquitoes from 6 houses, one pair per house. Each member of the pair collected mosquitoes from his partner for half an hour and then rested for half an hour. Thus each person collected mosquitoes for alternate half hour intervals from 6 p.m. until 6 a.m. The mosquitoes collected were examined the following day in the laboratory for identifiation of their species and were dissected for parity determination.

Only three species An.gambiae sensustrictu, An.funestus and An.arabiensis were found. The entomological innoculation rate was calculated by multiplying the average number of Anopheles collected per man per night and the proportion with sporozoites in their salivary glands.

Maximum and minimum temperatures, relative humidity and rainfall were recorded at two sites in the community. See Table 3.1 and Figure 3.1a for rainfall distribution in Saradidi, 1986.

3.3.2 Results of the study

The monthly incidence of P.falciparum infections was 0.1 in January and rose to a plateau of 0.65 in April-August and dropped to 0.1 again in September (see Table 3.2 and Figure 3.1c).

Anopheles biting rates averaged less than one per man per night from all night human-bait collections from January to March. The rate rose to 15 mosquitoes per man per night in May and slowly declined to 3 mosquitoes per night by August and to less than one by September (see Table 3.3 and Figure 3.1a).

Sporozoite rates were about 0.2 in January, fell to 0.01 in March and began to increase again in April to a peak of 0.3 in August and then declined again.

The entomological inoculation rate was 0.2 in January, fell to 0.005 in March and rose to a peak of 1.4 in June and then fell to 0.01 by September.

Little rainfall was recorded in January, September and December. The heaviest seasonal rains occurred in March-July and tapered off in August (Figure 3.1a). The number of mosquitoes collected per month followed the rainfall pattern (Figure 3.1b).

From these results it can be seen that the seasonal patterns of the incidence of P.falciparum malaria and the entomological inoculation rates were similar except for the multiple simultaneous P.falciparum infections occurring at the peak of the transmission season and which could not be detected.

It was concluded that the incidence and hence the transmission of P.falciparum malaria was highest in the period April to August. This increase was preceded by increased rainfall, which started in March and peaked in May, and increased the numbers of Anopheles and higher entomological rates all of which peaked in early April (see Figure 3.1d) just before the increase in incidence in late April and early May.

Thus the timing and duration of high transmission can be precisely

defined from year to year using this method and appropriate antimalarial actions planned and undertaken in communities like Saradidi.

3.4 MALARIA CONTROL

The stratification of malaria situation, as already described, is for the purpose of designing malaria control measures within primary health care (PHC) suitable for each stratum.

The choice of a strategy is made only after evaluating the local epidemiology, socioeconomic conditions, resources available including the state of the development of the health service infrastructure. This way one can determine not only what is necessary but also what is possible.

Four priority levels have been described by the World Health Organization in the control of malaria and are usually referred to as tactical variants (WHO, 1979)

3.4.1 Tactical Variant 1

This is the first priority in all malarious situations and is aimed at controlling mortality. In PHC this refers to making effective chemotherapy available in the community for presumptive treatment of all fever cases. The microscopic confirmation of diagnosis in such circumstances may neither be practical nor desirable in places which are holoendemic and hyperendemic. The success of such a programme depends on:

- availabilty of appropriate antimalarials;
- availability of competence within the community, e.g. trained Community Health Workers (CHWs) to provide appropriate treatment as soon as possible after a malarial attack;
- availability of information and education that would enable informed use of antimalarials by the community, families and individuals;
- adequate technical supervision and referral back up;

- adequate surveillance system to monitor the malaria situation and the response of malaria parasite to treatment.

Saradidi Rural Health Programme was an attempt to demonstrate the feasibility of this tactical variant.

3.4.2 Tactical variant 2

This aims at not only controlling mortality but also morbidity. It has limited impact on controlling prevalence. It is achieved through chemotherapy as in tactical variant 1, but also chemoprophylaxis on the most high risk members of the community. For Strata 1 and 2 chemoprophylaxis is usually limited to the non-immune or relatively non-immune members of the community: visitors from non-malarious areas and pregnant women (mainly first exposed pregnancy). Children under the age of five years may no longer be considered target groups for chemoprophylaxis because:

- such a programme is not sustainable indefinitely in most African countries;
- if given for five years enough chloroquine may be deposited in the tissues to cause toxicity;
- it may hinder the development of immunity against malaria;
- it may accelerate the development of resistance to chloroquine in the local parasite population due to drug pressure.

The chemoprophylaxis to pregnant women remains one of the most important activities in PHC because it prevents anaemia in pregnancy, it improves the health of the mother, it prevents low birth weight and hence reduces perinatal and post perinatal infant mortality. The chemoprophylaxis can also be provided by community health workers (CHWs), traditional birth attendants (TBAs) and antenatal clinics. The relative ineffectiveness of the CHW system was demonstrated in this study, if they were to do it in villages by themselves (Kaseje et al., 1987). It was because of this that a monthly regimen

given at ANC clinics could be the better strategy since the weekly supply of chemoprophylaxis to pregnant mothers seemed to place an unmanageable demand on the time of the part-time voluntary workers. The monthly regimen needs to be carefully worked out and must be supported technically, logistically and by an effective information and education system. This approach was studied at Saradidi and is presented in Chapters 8 to 11.

Thus for both tactical variants 1 and 2 a strong support health service system is necessary to provide referral support, supplies, supervision, training, laboratory backing and detection of resistance.

During epidemics in strata 3, 4, 5, 6, and 7 already described, similar antiparasite activities (chemoprophyalxis and chemotherapy) may be appropriately applied to limit the mortality and morbidity during transmission periods. It may often be necessary to provide mass treatment whether weekly, fortnightly or monthly according to feasibility based on the available resources. In this case, antimalarials should include schizontocidal and gametocytocidal agents (e.g. chloroquine and primaquine). The drug distribution should be continued until the end of one month after the transmission season. The effectiveness of CHWs in this exercise was outside the scope of the present study. Their effectiveness will also depend on information, education and training given to them and to the rest of the community.

3.4.3 Tactical variant 3

This aims at reducing not only mortality and morbidity by the antiparasitic methods described above, but also at prevalence of parasitaemia by interrupting the actual transmission. This variant can be complex, expensive and dependent on such high level of supervision and support that it is not commonly found in developing countries. Antivector activities should include individual protection methods which are feasible at the family or community level and should be encouraged in all the strata. These methods may include bed nets

(better still those impregnated with insecticides), mosquito repellents, screening of houses (which is limited to permanent houses), and siting of dwelling houses. These individual protection activities are also dependent on socioeconomic conditions, information and education and supplies being available.

The most effective antivector method which is key to this tactical variant is insecticide spraying. In our day this is only used in very special circumstances because it is too costly and has too many logistic problems for most developing countries in Africa to handle. It is also heavily dependent on technical expertise. Insecticide application can be by indoor residual spraying (the most common) or outdoor space spraying. Ideally the concept of integrated pest management described by Olsen (1979) should be applied if socioeconomic conditions allow its application.

Whenever this is to be implemented it is of vital importance to define carefully roles and responsibilities of various actors involved (Fontaine, 1985). The actors should include the community, individuals and the health services. This method can be used in strata 2, 4, 6 and 7 during risk of epidemics. In the context of PHC, the community should be fully involved in planning, executing and evaluating the activities. The involvement of the community in antivector operations has been described in Ethiopia, Sudan, Thailand and Vietnam according to reviews by Jeffery (1984) and Najara (1986).

Outdoor spraying operations are only effective in urban centres where they are used to reduce the population of adult infected mosquitoes. They should be properly timed for them to be effective. This usually demands a special malaria unit for its adequate management.

The control of vector breeding is another antivector method which is commonly used. The peridomestic removal of breeding sites and sanitation should be done by the community following information and education to them.

In tropical Africa adequate reduction of breeding sites operation is also only possible in special circumstances as in urban areas, irrigation schemes, and

in the arid areas (strata 3, 6, 7 and 8). Other methods of source reduction include larviciding and environmental modification. These activities should always be planned into the development projects in malarious or potentially malarious areas.

3.4.4 Tactical variant 4

This is the total eradication of malaria which is now considered virtually impossible at least in tropical Africa. This has to do with both antiparasite and antivector activities.

In recent years the approach of integrated vector control (IVC) has been advanced by Laird and his colleagues (1983). This approach recommends the use of all appropriate technology to bring about an effective degree of vector control. This approach must be based on sound ecological understanding of the environment including the availability and adequacy of the health system. It is also very important to have a thorough knowledge of breeding sites and mosquito habits. This is the only way to decide the best strategy as described by Kligler more than 50 years ago (Kligler, 1930).

In the context of the PHC the methods to be applied must be those that are suitable for the community to carry out given their competence and resources. Other workers have also stressed the appropriateness of methods not only with regard to what the community can do but also a very sound understanding of vector control. Understanding of vector control implies:

a) a sound understanding of the target vectors;

b) a quantitative analysis of costs and benefits in terms of impact on man and his environment;

c) possibility of source reduction;

d) integrated, complementary control measures possible;

e) monitoring system to detect impact;

f) ability to feed back the results of monitoring and evaluation to on-going antivector or antimalarial activities.

A good vector control effort should reduce the transmission of malaria to levels acceptable to the community. The monitoring system should include surveillance to be able to predict the risk of impending transmission and to assess regularly the vectorial capacity and factors affecting it like vector population density, longevity, anthropophilic behaviour and the length of the sporogenic cycle. The understanding of these are vital to the selection of which IVC methods are to be employed.

One of the main lessons learnt from the attempt to eradicate malaria from the world is the high degree of local variability not only in intensity of the malaria problem but also in its response to control interventions.

The main aim of epidemiological approach to malaria control is to identify appropriate technologies for malaria control as part of the development of PHC. The concept of appropriate technology implies its being scientifically sound for the solution of the problem and adapted to the society considering its application in terms of acceptability and affordability. It is for this reason that a malaria control package must be well suited to the stratum with adequate understanding of all the factors that determine the problem and those that would influence the process of implementing control measures and the response of the disease parasites and its vectors to the intervention strategies.

It is because of this that the studies presented in this thesis were undertaken at Saradidi, to determine and monitor the malaria situation and to test the effectiveness of a community based chemotherapy and chemo-prophylaxis programme.

3.5 COMMUNITY BASED MALARIA CONTROL IN SARADIDI

The process that led to the community based malaria control as implemented in Saradidi has been described in greater detail in Chapter 1. In this section is summarised the activities that were involved. Through a process of discussion and open dialogue with the community, the people were engaged in

a process of thinking deeply about their health status which they had realised was not as good as it could be. Asking open ended questions, reflective questions and probing questions enabled the community to critically review not only the problems of health but also the resources that they had among themselves as a community and within their reach.

This exercise led the community to decide on certain activities in taking more responsibility for improving their health, among which were antimalarial activities. The antimalarial activities included:

3.5.1 Participating in information education and communication regarding malaria.

Opportunities were created to enable learning to take place either in groups (e.g. church meetings, womens meetings, open 'barazas'), families during home visits, not only by health personnel but also by the CHW and other community members that had been given these responsibilities by the rest of the community and at individual level in clinics and other informal meeting places as markets and water points.

It was recognised in this project that learning is a two way process where all participants contributed as equals. In this context, the messages about the causation, the treatment and the prevention of malaria were assimilated into the local thought processes. The message learnt and applied was therefore a hybrid of the local traditional concepts and in-puts from trained facilitators.

3.5.2 Taking actions as a result of their learning to respond to their problems

The community carried out those activities that were permitted by their economic status and which fitted into their priorities. Some of the prevention and control activities undertaken were:

- removing mosquito breeding sites
- screening of houses

- sleeping under nets
- using repellents both traditional and modern
- taking drugs either when sick or giving them to those that are more likely to develop untoward consequences of malaria infection.

3.5.3 Recognising those who are sick with malaria and giving them drugs or taking them to health facilities for treatment

These control activities at Saradidi did not fit neatly under one tactical variant as such but still allowed the investigation into the effectiveness of such a community based programme. The community leaders became active participants in the process of research. This became a learning tool for them.

3.6 ASSESSMENT OF THE COMMUNITY-BASED PROJECT'S IMPACT ON MALARIA PARASITE PREVALENCE AND ANTIMALARIAL ANTIBODIES

3.6.1 Introduction

Making primary health care services universally available through community based distribution had proved successful in various communities (Kols and Wawer, 1982). In these programmes, essential services and supplies for important health problems were provided in the community by residents (often volunteers) who had been locally trained in an effort to enable the community to handle their own health problems more effectively.

Malaria is a priority health problem in many parts of tropical Africa. Community-based distribution of chloroquine phosphate for treating malaria was considered an effective control measure as it would reduce mortality caused by the disease. This section describes the Saradidi community-based malaria control project. The project utilised volunteer community health workers to supply antimalarials for chemotherapy and chemoprophylaxis. This section presents the impact of the programme on parasitaemia rates and on antimalarial bodies.

3.6.2 Materials and methods

a) Background

Malaria is holoendemic in Saradidi (Roberts, 1974). The principal species is Plasmodium falciparum. Earlier studies in the area suggested that malaria contributed to at least 38% of infant mortality; when fenitrothion was used as a residual insecticide to reduce the transmission of malaria by reducing the population of Anopheles mosquitoes, malaria prevalence decreased significantly, and infant mortality rates fell from 157 to 98 per 1000 live births (Payne et al., 1984).

Through dialogue with the community, they decided to try to prevent deaths and disease due to malaria as one of their main actions to improve their health status (Kaseje and Spencer, 1987).

b) Objective

The objective of malaria control in Saradidi was to provide prompt, appropriate treatment in each village to persons with malaria, through CHWs.

c) Diagnosis and treatment

The CHWs gave chloroquine phosphate to every person who came for treatment saying they had malaria. The CHW did not screen the symptoms and attempt to decide if the person had malaria or not. If a person returned the next day, the same treatment was repeated. However, if symptoms persisted on the third day, the patient was referred to the Saradidi clinic. Seriously ill patients including those who could not take oral medication because of vomiting were also referred for parenteral treatment. Blood films were not collected. Information taken by the CHWs from each person and how drugs were given has been described and reported by Spencer et al, (1987).

d) Chloroquine supply

Chloroquine was purchased from commercial sources using funds supplied by the World Health Organization (WHO) and taken to a community-built clinic at the Saradidi programme centre. From there, chloroquine was distributed to

the CHWs. Each CHW received a tin of 1,000 tablets of 150 mg chloroquine base and 500 ml of syrup of 50 mg chloroquine base per ml. To replenish their supply, the CHWs had to return to the clinic with their record book. Malaria treatment was also available from Saradidi community clinic, two Ministry of Health dispensaries in the area and a mission hospital. However, more than half of the population lived at least 5 km from these facilities. Chloroquine could also be purchsed in small shops. Before the programme was initiated, the shops were the major source of antimalarial drugs. Advice on what drug and what dose to take were often given by shopkeepers (Mburu et al., 1987).

The community-based distribution was first initiated in Areas A and B and if successful it was to be extended to Area C which served as a control area (see Chapter 1). The epidemiology of malaria and the demographic, economic and cultural composition of the population were virtually identical in the three areas (Spencer et al., 1987).

The CHWs rapidly became the principal source for treatment of malaria in these villages (Mburu et al., 1987).

e) Chemoprophylaxis

In Area A, malaria chemoprophylaxis with chloroquine was provided to all pregnant women who wished to receive it. The dosage was chloroquine phosphate 300 mg base weekly. The CHW visited the household every 2 to 4 weeks. One weekly dose was to be taken in the presence of the CHW, then a sufficient supply until the next visit was left with the women.

f) Training

The CHWs were trained in treatment of malaria and the points stressed were, who to treat, who to refer to the clinic, how much drug to give, how to keep records, and how to get more drugs.

Several workshops were held at various times to keep the CHWs up to date on diagnosis, treatment, record keeping and referral. The CHWs also reported any problems encountered so that modifications could be made and communicated at these workshops.

g) Parasitological surveys

Parasite prevalence surveys were carried out in the study areas by field teams consisting of four technicians. Twenty-five villages (9 in Area A, 10 in Area B and 6 in Area C) were randomly selected in the first two surveys. Subsequently, the same villages were revisited; two were visited five times, eleven four times, six three times, one twice and five once. School children aged 6 to 14 years from the school in the village were examined in the morning. Persons of other ages from the village were examined in the afternoon by asking them to come to a central place. Surveys of two weeks each were done in November 1980, March 1981, May 1982, November 1982, March 1983, and November 1983. Two of these surveys (May 1981 and May 1982) were done within 4-6 weeks after the long rains began when, malaria transmission was at its peak. The others were carried out in the dry season or at the beginning of the short rains. Three surveys (November 1980, March 1981 and May 1981) were carried out before the control programme began. All persons surveyed were treated with chloroquine phosphate base 10 mg base per kg.

Thick blood smears made from fingerprick were stained with Giemsa and examined by trained microscopists. At least 100 fields were examined before a slide was called negative. Mixed infections were noted. For the first four surveys, all positives and 10% of the negatives were checked by a supervisor. This process was discontinued because the proportion of detected errors was less than 0.5% and so the exercise was not considered cost effective.

h) Serological tests

Beginning in May 1981, blood specimens for antimalarial antibodies were collected by fingerpricks as well as the blood slide. The samples were placed on filter papers as previously described (Collins et al., 1987). Filter paper specimens were stored at -20°centigrade until examined. The number of parasitological and serological specimens differed because a serological specimen was not taken from every person and a number of filter papers were destroyed when a freezer in the laboratory failed.

Specimens collected in May 1981 (before intervention) were eluted from filter papers and examined by the indirect fluorescent antibody test (IFA) as previously described (Sulzer, 1969; World Health Organization 1974; Collins et al., 1977). The reaction to three antigens (P.falciparum, P.malariae and P.ovale) were tested. Each specimen was examined at dilutions of 1:80, 1:320, 1:1280 and 1:5120. A reciprocal titre of 80 or more was considered positive.

Specimens collected in March 1982, May 1982, November 1982, March 1983 and November 1983 were examined with the enzyme-linked immunosorbent assay (ELISA) as previously described (Collins et al., 1977). P.falciparum parasites from continuous in vitro cultures were used as antigen. All samples were tested at a dilution for population samples based on comparative studies in the laboratory. An absorbance of 0.3 or greater was considered positive. Values greater than 1.0 were considered strongly positive with confidence limits >95%.

i) Cohort

A cohort of children 0 to 9 years of age was followed prospectively with parasitological and serological specimens. Two villages in Area B and one village in Area C were randomly selected and approximately 35 children in each village chosen by going from household to household. Specimens were examined at two dilutions, 1:100 and 1:1000 in the ELISA. Each child in the cohort was treated with chloroquine phosphate 10 mg base per kg each time they were sampled.

3.6.3 Results

a) Parasitaemia

Since results from Areas A and B were very similar, the data from these areas were combined. Provision of antimalarial treatment in each village of Areas A and B did not alter parasitaemia rates. The parasitological prevalence by age after the control programme began in areas A and B combined were very similar to those in Area C (Table 3.3). Parasitaemia rates were high even during the dry season and particularly among children (Figures 3.3 and 3.4).

The prevalence of malaria infections was significantly higher in Areas A and B before the control programme began in both the rainy (Chi square = 350, $p<.0000001$) and dry season (Chi square = 363, $p<.000001$) than after the control programme began. However, the prevalence of parasitaemia in Areas A and B after beginning control measures was not statistically significantly different ($p>0.5$) from that in Area C in either the rainy or dry seasons. The most likely explanation for the differences in Areas A and B is that a change in environmental conditions occurred, resulting in lower levels of transmission; a drought began in 1982 which continued for most of the two years.

Parasitaemia rates in all age groups were significantly higher during the rainy season (Table 3.3). In general, children 1-14 years of age had the highest prevalence of parasitaemia, although infants less than 1 year old also had high rates (Figure 3.4).

b) Species

P.falciparum was the most common species (Table 3.4). It was present in 98.2% of 8105 positive slides alone or in combination with P.malariae and/or P.ovale.

c) IFA tests

Results from the IFA tests done in the May 1981 survey confirmed the high proportion of infections with P.falciparum. Only 31 (1.5%) of 2,040 samples had a reciprocal titre to P.falciparum of less than 80. In contrast 19.2% and 22.6% of the 2040 samples had reciprocal titres less than 80 to P.malariae and P.ovale respectively. The geometric mean reciprocal titre was 1066.2 to P.falciparum, 113.0 to P.malariae and 91.4 to P.ovale.

In only 25 (1.2%) samples were reciprocal titres to P.malariae and/or P.ovale higher than those to P.falciparum: in 23 of these 25 the recriprocal titre to P.falciparum was 80 or more (but less than that of the other species). High rates of seropositivity to P.falciparum were obsered in all age groups (Table 3.5).

d) ELISA tests

Seropositivity rates to P.falciparum in the ELISA were high and increased with age (Table 3.6b). When the results from Areas A and B combined were compared with those from Area C in surveys done after the malaria control programme began. no statistically significant difference was found in the proportion of seropositive samples in any age group (Figure 3.2).

e) Cohort studies

Parasitaemia rates were high in the cohort of children followed in each survey (Table 3.7). Most serum samples from the cohort children were positive for antibodies to P.falciparum when examined by the IFAT and were highly positive by the ELISA at a dilution of 1:100 (Table 3.6a). Seropositivity rates in the ELISA at 1:1000 dilution were lower. The higher rates of parasitaemia and seropositivity observed in Area C were most likely because the entire cohort of 33 children from Area C were from one village with very high transmission. Figure 3.5 presents monthly absorbance value for ELISA tests on pregnant women in 1986.

Many people lived long distances from any dispensary, clinic or hospital; thus facilities for malaria diagnosis and treatment were not readily accessible. During discussions at meetings before the malaria control programme was initiated, the community developed the confidence that by working together they could do something about their health problems.

The CHWs were selected, trained and supervised. Referral capability was available for patients who failed to respond to treatment or who were acutely ill.

The parasitological and serological results demonstrate that malaria is hyper to holo-endemic in Saradidi. P.falciparum is the predominant species. Despite the high levels of transmission, seasonal variations occurred in all age groups and parasitaemia rates increased significantly in the surveys done during 4-6 weeks after the beginning of the rainy season, when transmission levels were highest.

Malaria control is often equated with reduction in prevalence. In fact morbidity and mortality rates caused by malaria can decline, significantly improving the health of the population, in the absence of any decrease in parasitaemia rates (Molineaux and Gramiccia, 1980). Having reductions in prevalence as an objective of malaria control programmes that originally have the sole aim of providing prompt diagnosis and appropriate treatment to persons with malaria is unrealistic. Measuring mortality rates to evaluate a programme is also difficult. Specific cause of death is usually impossible to determine since pathological examination cannot be carried out and death may have resulted from many causes, and also mortality rates are already decreasing in many areas as health services and general living conditions improve. Similar problems exist with the definition and measurement of morbidity. For these reasons, indicators such as the accessibility to treatment, utilisation of services, the referral capability of seriously ill patients and treatment failures and status of drug supply may be of greater usefulness in monitoring progress of malaria control activities such as those carried out in Saradidi than more classical indicators such as morbidity and mortality. Community-based malaria control activities in Saradidi depended upon active community participation; the involvement of volunteer community health workers who received training, support and supervision; the presence of referral capability; a recognised health problem; the confidence that something could be done by the community and the availability of antimalarial drugs in adequate quantities. One important problem for Saradidi was how to maintain the supply of drugs when external resources were no longer available. The solutions considered included recovering of costs from users of the community-based drug supply system, which was already operational in Areas A and B.

3.7 DISCUSSION

A community-based malaria control programme was initiated in Saradidi,

Kenya. The problem of malaria was identified by the community through those who participated in community meetings. They became committed to developing activities that would reduce malaria as a problem as this was believed to be responsible for many deaths in the community particularly of children (Kaseje and Spencer, 1987).

A deeper understanding into the problem of malaria brought to the surface the fact that the community had to organise itself to address the problem, they had to rely on available resources, they had to use the most appropriate technology available to and affordable by them and that everyone in the community needed to have equal access to the technology.

The result of this community initiative was a comprehensive integrated health care system that had malaria as a main focus but was not limited to malaria (Kaseje et al.. 1987d).

The capability of the community to participate in these activities was increased by the training of community health workers who were selected and supported by the community to be local resource people to facilitate health development. The community health workers (CHWs) were technically supervised by health personnel from the Saradidi Clinic.

The results of the study on malaria transmission confirmed that malaria is holo-endemic in Saradidi and is transmitted intensely all the year round but with peaks during the rainy season of April-August and October-November. This is in accordance with the findings of other workers (Fontaine, 1978).

The main mosquito species found in Saradidi were An.gambiae sensu strictu, An.arabiensis and An.funestus confirming earlier findings by Fontaine and his coworkers in the same general area (Fontaine et al.. 1978).

The transmission fluctuations demonstrated in this study was reflected in the seasonal variations which occurred in all age groups. Parasite rates increased significantly in the surveys done during four to six weeks after the beginning of the rainy season, when transmission levels were also highest.

The malaria control activities did not decrease parsitaemia rates or the prevalence of antimalarial antibodies. Nontheless, the programme was successful in providing antimalarial treatment. As demonstrated in Chapter 2 the average number of treatments given by the CHW per person per year was 1.24. More than 75% of persons who suspected they had malaria went to the CHWs first for treatment (Kaseje et al.. 1987e). The CHW quickly became the source of antimalarial treatment in the area (Mburu et al., 1987).

Malaria control is not to be necessarily equated with reduction of the prevalence of parasiteamia. It was demonstrated in Garki, Nigeria that both morbidity and mortality can be reduced significantly, thus improving the health status of the community, through antimalarial activities but without decreasing the parasitaemia rates (Molineaux and Granmicia. 1980). The measurement of morbidity and mortality rates proved difficult and very expensive. Finding out causes of death in Saradidi was impossible apart from very rough verbal autopsies. Deaths in a community where there are so many conditions that impact on the health of the child, usually have multiple causes. Thus the impact of a control programme like Saradidi may be impossible to evaluate in terms of morbidity and mortality. For these reasons. indicators such as accessibility to treatment, utilisation of services, the referral capability for seriously ill patients and treatment failures and status of drug supply were of greater usefulness to us and could be the same for the health services in monitoring progress of malaria control activities. Service indicators rather than indicators of definitive outcomes should receive greater attention (Spencer, 1987).

The community-based malaria control activities in Saradidi depended upon active community participation, active trained and supported CHWs, referral capability, a recognised priority health problem and availability of resources to address the problem. The level of activities and their scope must be such that they can be continued indefinitely. The success of the project will only be determined many years later if it is sustained and managed completely by the

community themselves without external inputs other than professional skills and guidance.

Table 3.1 Rainfall distribution in Saradidi in mm by month, 1986

Month	No. days recorded	Location A rain-fall	Location A daily average	Location B rain-fall	Location B daily average	Saradidi rain-fall	Saradidi daily average
January	31	1.26	0.040	0.79	0.025	1.025	0.033
February	28	2.85	0.102	2.33	0.083	2.590	0.093
March	31	5.25	0.169	5.12	0.165	5.185	0.167
April	30	5.13	0.171	6.33	0.211	5.730	0.191
May	31	9.82	0.317	9.84	0.317	9.830	0.317
June	30	5.77	0.192	5.14	0.171	5.455	0.182
July	31	6.60	0.212	5.06	0.163	5.830	0.188
August	31	2.15	0.069	2.77	0.089	2.460	0.079
September	30	1.39	0.023	2.30	0.077	1.845	0.062
October	31	3.49	0.113	4.89	0.158	4.190	0.135
November	30	4.21	0.140	4.18	0.139	4.195	0.140
December	31	0.61	0.020	0.79	0.039	0.700	0.023
Total	365	48.52	0.133	49.97	0.137	49.085	0.134

Table 3.2 Incident of malaria and anopheles biting rate in Saradidi by month, 1986

	Jan	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec
Incidence of infection (%)	10	6	8	65	71	68	67	69	10	9	15	11
No anopheles bites per person	0.8	0.25	0.65	8	15	15.4	14.8	3	0.6	0.8	1.4	0.8

Table 3.3 Prevalence of malaria parasitaemia before and after beginning of the control programme by area, age and season, Saradidi, Kenya, 1980-1983

Age (years)	Before Control Programme: Dry Season* Areas A and B No. slides examined	% positive**	After Control Programme began: Dry Season+ Areas A and B No. slides examined	% positive	Area C No. slides examined	% positive
<1	145	53.1	213	48.4	99	49.5
1-5	758	68.2	1078	66.5	486	64.8
6-14	1256	72.5	2583	50.4	1117	48.1
15-29	267	30.0	972	16.2	445	20.0
>30	95	12.6	988	9.1	624	11.2
	2521	63.3	5834	40.6	2771	38.4

	Rainy Season*** No. slides	% positive	No. slides	% positive	Rainy Season++ No.slides	% positive
<1	59	79.7	43	60.55	18	66.7
1-5	527	89.0	318	77.7	141	76.6
6-14	1200	88.7	772	78.4	145	84.8
15-29	182	61.0	145	44.1	47	44.7
>30	181	45.9	216	38.9	35	42.9
	2149	82.5	1494	68.7	386	72.3

* November 1980 - March 1981 **Positive for any malaria species ***May 1981 +November 1982 - March 1983 ++November 1983

Table 3.4 Parasitaemia by Plasmodium species in 8105 positive slides, Saradidi, Kenya, 1980-1983

Species	Percent
P.falciparum (PF)	87.1
P.malariae (PM)	1.6
P.ovale (PO)	0.4
PF + PM	8.5
PF + PO	1.8
PF + PM + PO	0.7

Table 3.5 Results of indirect fluorescent antibody (IFA) test by age Saradidi, Kenya, May 1981

Age (years)	No. examined	% positive*
<1	56	85.7
1-5	489	96.7
6-14	1160	99.5
15-29	167	99.4
>30	168	100.0
	2040	98.5

* Reciprocal IFA titre to P.falciparum 80 or more

Table 3.6a Antibodies to P.falciparum by indirect fluorescent antibody test (IFA) and enzyme-tested immunosorbent assay (ELISA) in a cohort of children by test. area and date. Saradidi. Kenya 1982-1983

		May 1982		November 1982		March 1982		November 1982	
Area	Test	No. examined	% positive*	No. examined	% positive	No. examined	% positive	No. examined	% positive
A and B	IFA	45	100	130	96.9	130	100	70	100
combined	ELISA 1:100	28	100	130	99.2	124	83.9	66	81.8
	ELISA 1:1000	46	50.0	130	38.5	124	5.6	66	6.1
C	IFA	-	-	34	100	34	100	-	-
	ELISA 1:100	-	-	22	100	32	96.9	-	-
	ELISA	-	-	34	52.9	33	48.5	-	-

* IFA reciporcal tire 80 or more; ELISA absorbance more than 1.0 (absorbance >0.3 considered positive > 1.0 highly positive)

Table 3.6b Results by area and age of enzyme-linked immunosorbent assay (ELISA) in surveys* done after inception of malaria control programme, Saradidi, Kenya 1982 1983

Age (years)	Areas A and B combined			Area C		
	No. examined	% positive	% highly positive**	No. examined	% positive	% highly positive
Less than 1	176	97.2	21.0	63	96.8	22.2
1 to 5	963	99.3	57.8	420	100.0	56.6
6 to 14	2435	100.0	80.5	858	100.0	73.8
15 to 29	840	100.0	89.0	249	100.0	89.2
more than 30	874	100.0	95.9	357	100.0	96.1
	5288	99.8	78.3	1947	99.9	74.9

* Serological surveys combined - May 1982, November 1982, March 1983 and November 1983. All were done after the malaria control programme.

** Optical density (O.D.)

Table 3.7 Parasitologic prevalence of malaria in cohort of children examined at invervals by area, age and date Saradidi, Kenya, 1981-1982

Area	Age Group (years)	No. in age group at beginning	May 1981 No.* examined	%+	May 1982 No. examined	%+	November 1982 No. examined	%+	March 1983 No. examined	%+	November 1983 No. examined	%+
A and B	0-5	47	17	100	10	100	30	80.0	30	46.7	20	75.0
combined	6-9	83	23	78.3	39	84.6	92	65.2	92	39.1	88	62.5
	10 or more	-	-	-	4	75.0	8	50.0	8	50.0	22	63.6
Total		130	40	87.5	53	86.8	130	67.7	130	41.5	130	64.6
C	0-5	10	-	-	-	-	10	90.0	10	80.0	-	-
	6-9	22	-	-	-	-	22	77.3	22	50.0	31	90.3
	10 or more	2	-	-	-	-	2	100	2	50.0	3	66.7

* Age group at time of specimen. Birth date estimated by assuming all children at mid-point of year when first examined

Fig 3.1a Monthly rainfall in Saradidi,1986

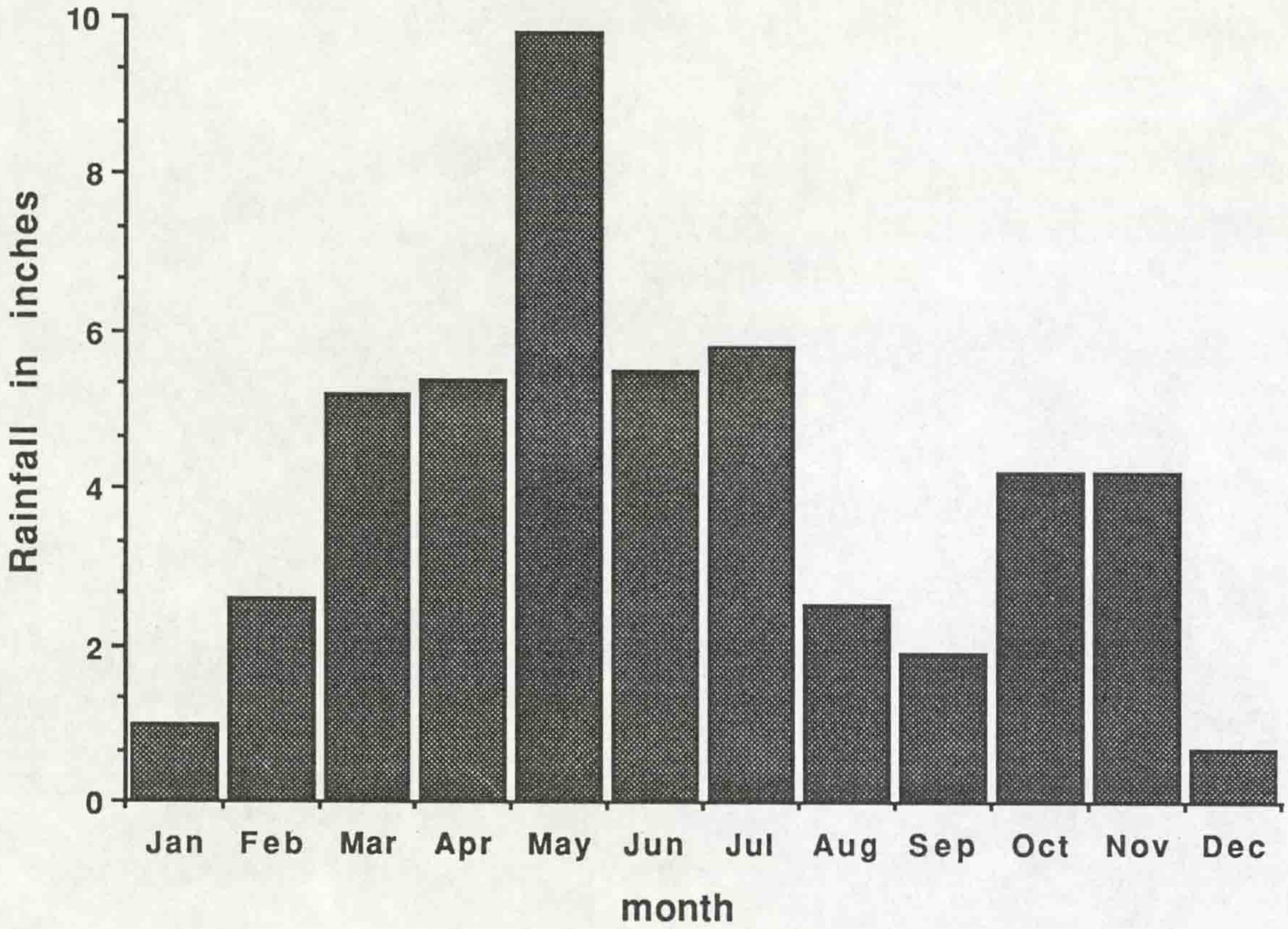


Fig 3.1b Monthly mosquito collections in Saradidi, 1986

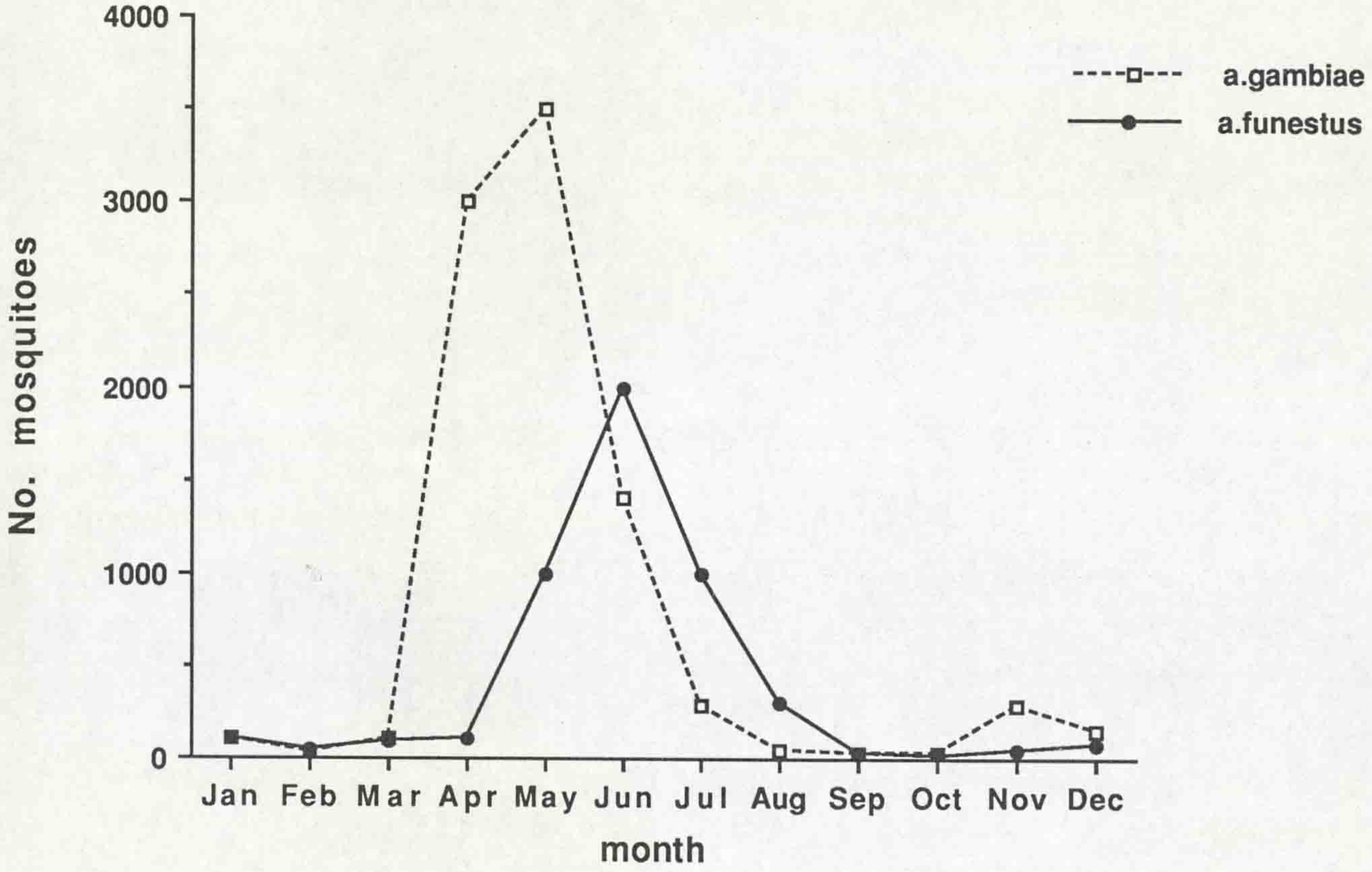


Fig 3.1c Malaria incidence in Saradidi by month,1986

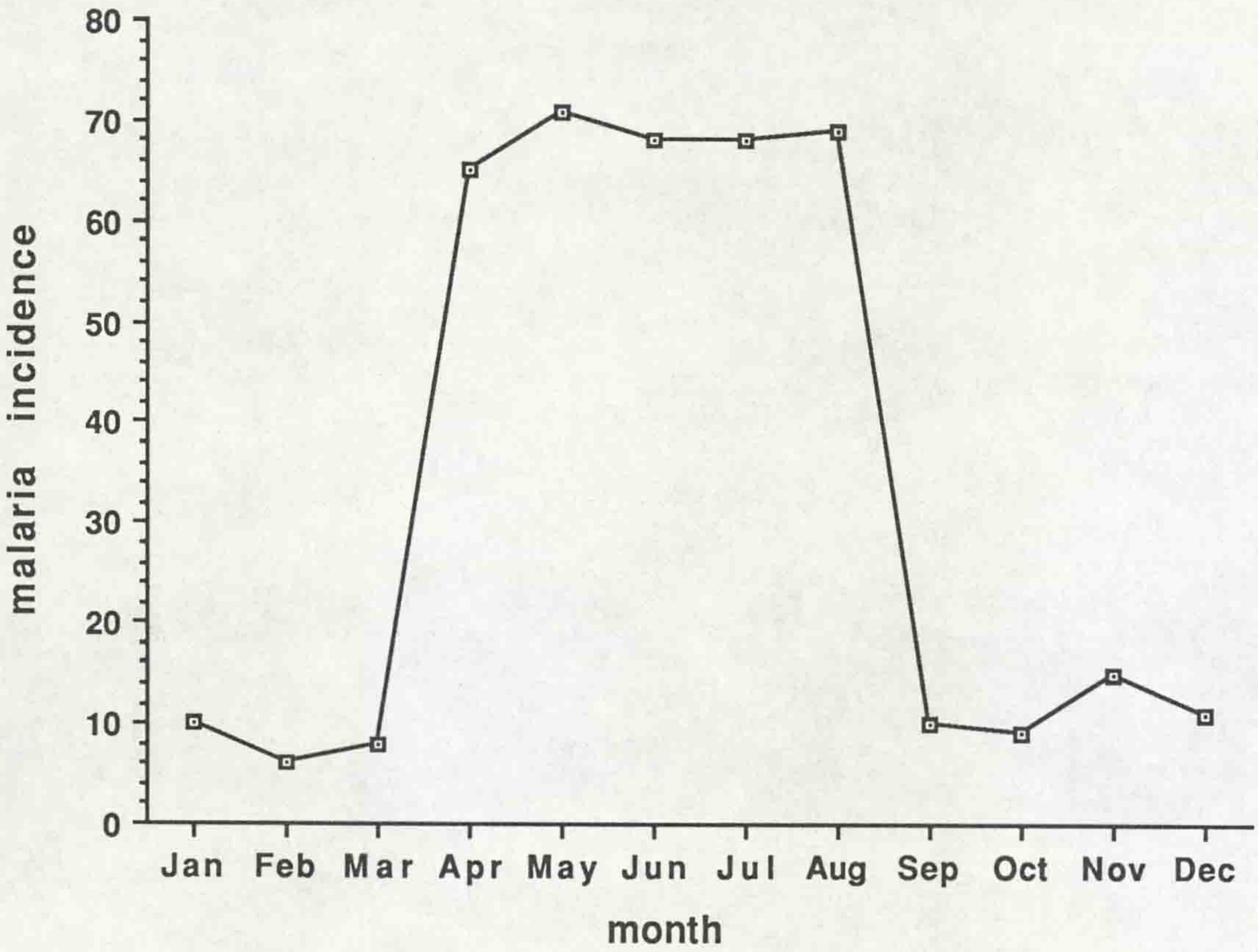


Fig 3.1d Monthly mosquito biting rate in Saradidi, 1986

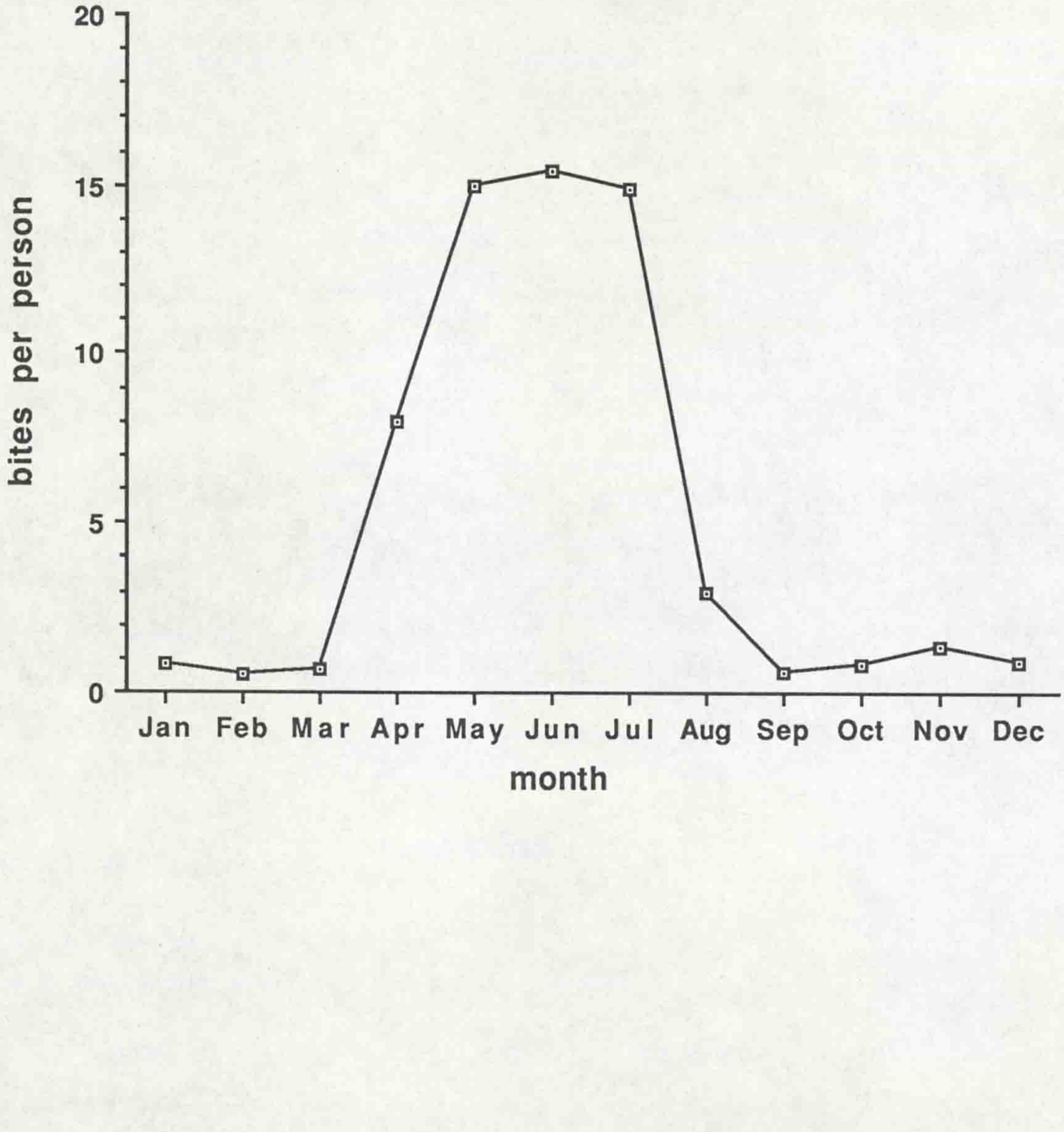


Fig 3.2 Percent population strongly positive to ELISA test by age and area

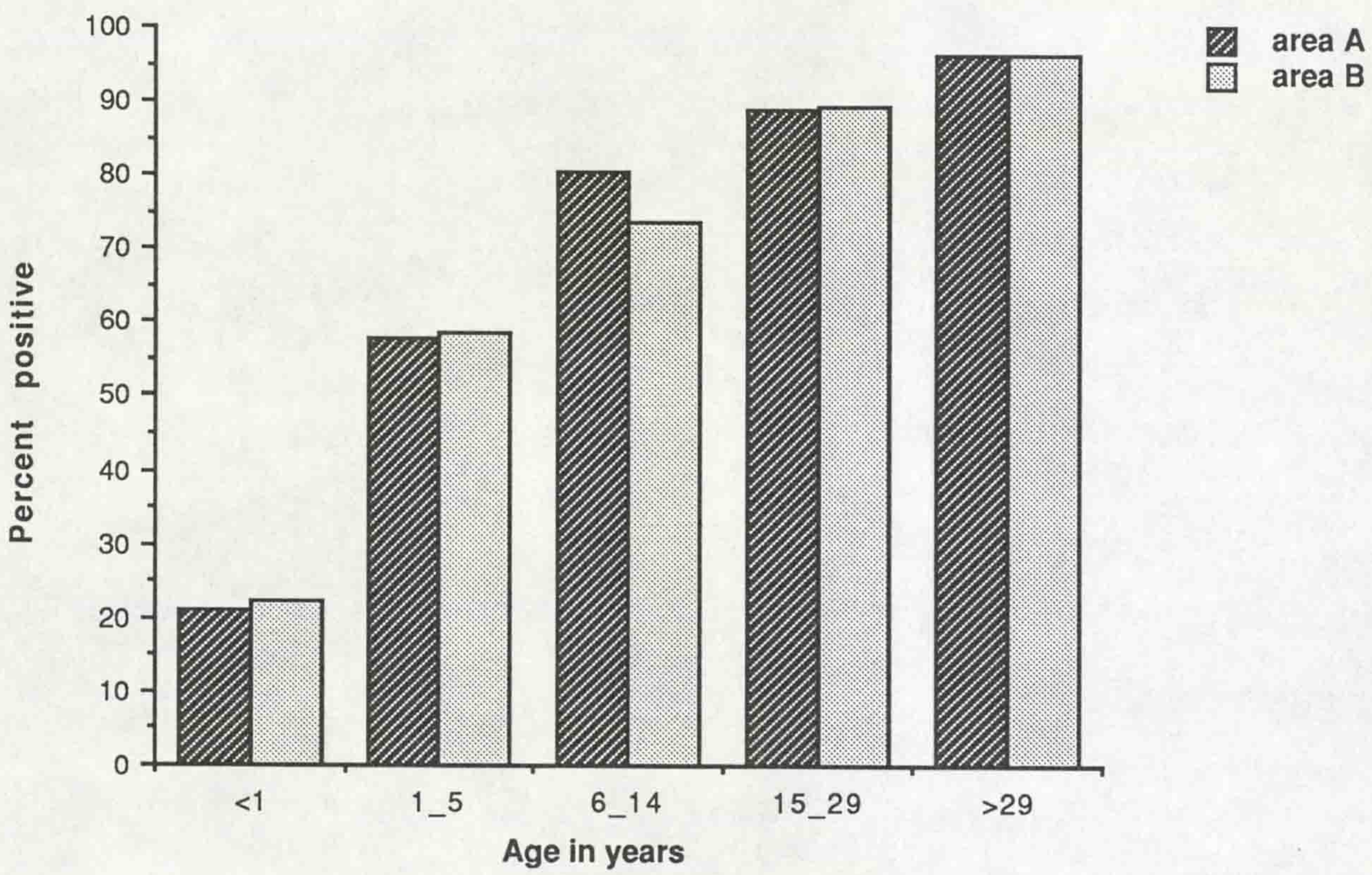


Fig 3.3 Malaria prevalence in Saradidi by age, area and season

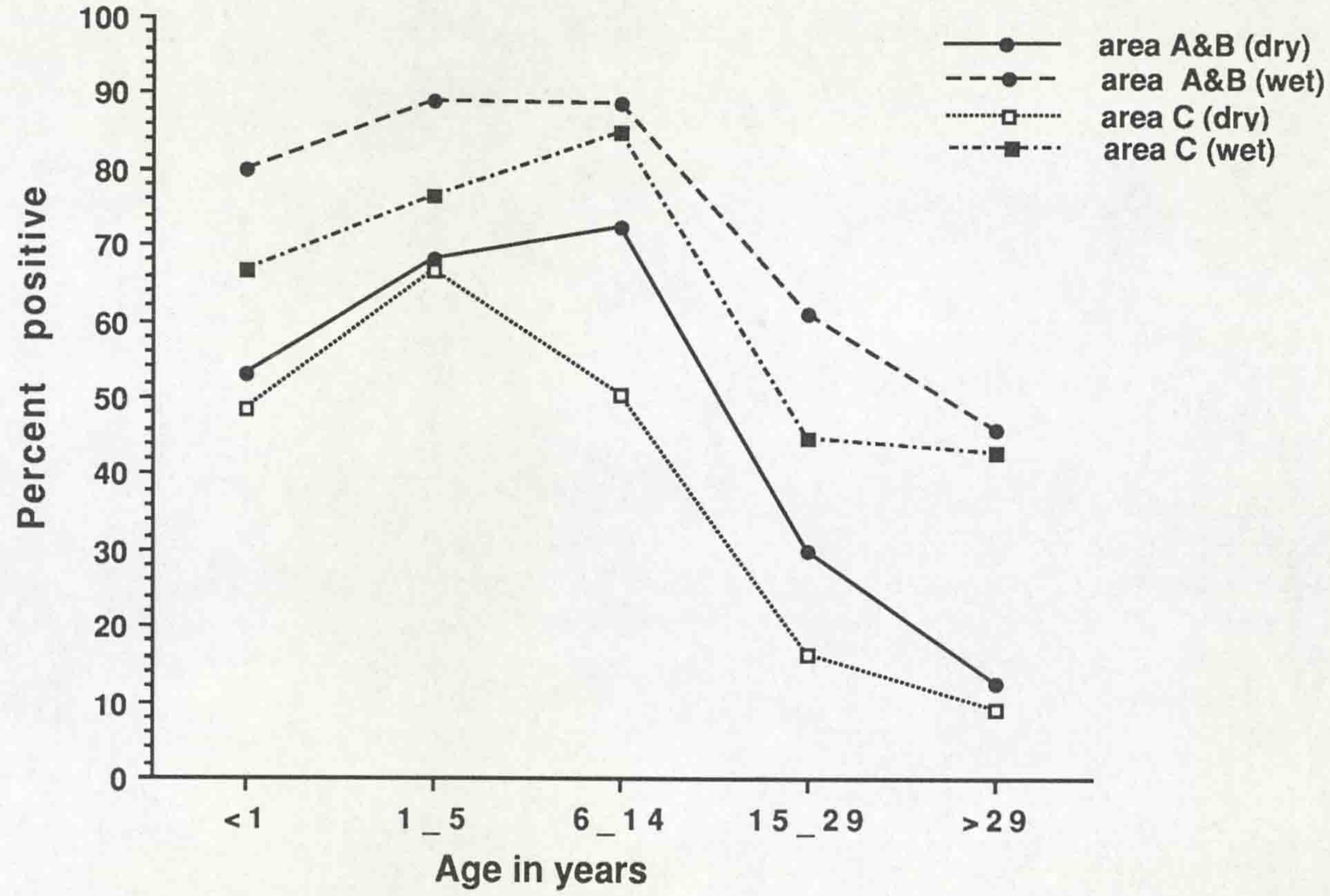


Fig 3.4 Percent parasite prevalence in pregnant women and infants by month 1986

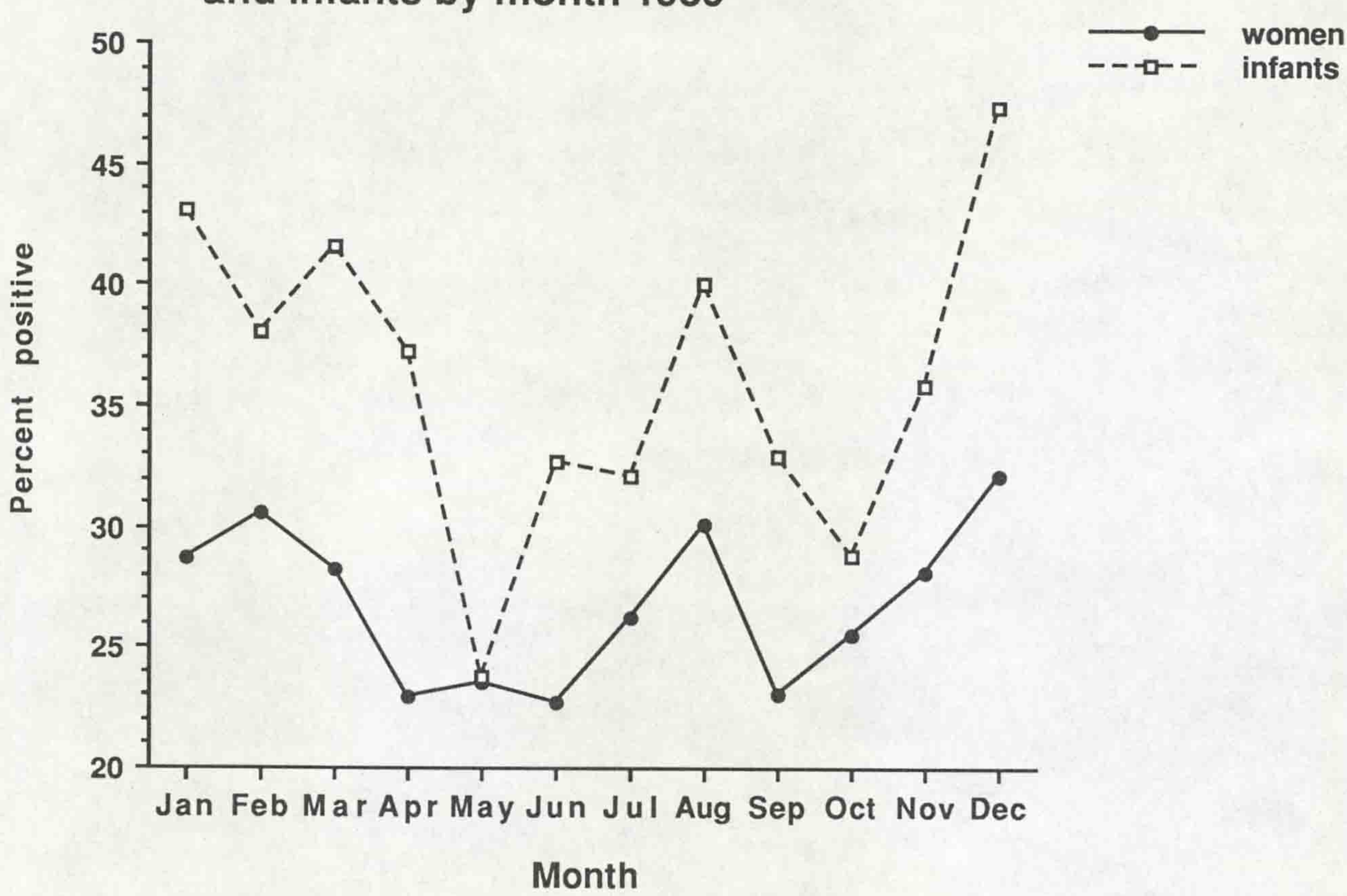
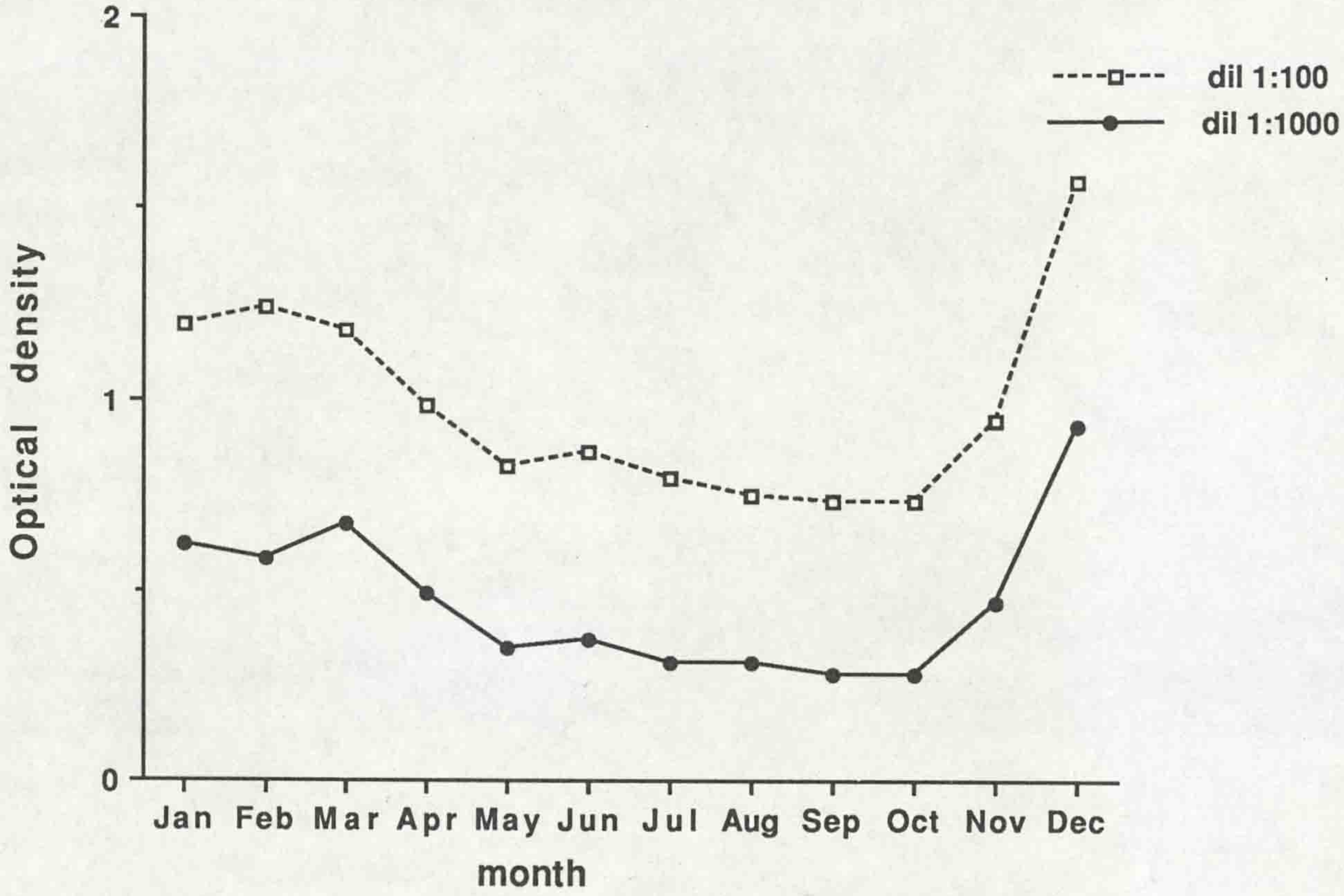


Fig 3.5 Mean monthly absorbance value of ELISA test on pregnant women by month 1986



CHAPTER 4

CHANGING RESPONSE OF PLASMODIUM FALCIPARUM IN SARADIDI TO CHLOROQUINE AND AMODIAQUINE IN VARIOUS TARGET GROUPS 1980-1987

4.1 INTRODUCTION

Drug-resistant Plasmodium falciparum malaria continues to spread in Africa and other parts of the world (World Health Organization, 1984; Spencer, 1985). Accumulated epidemiological evidence suggests the factors involved are not well understood (Peters, 1984; Onori, 1984). It was therefore important to monitor the sensitivity of P.falciparum in Saradidi over a period of time with the installation of a community-based chemotherapy and chemoprophylaxis project (Spencer et al., 1987). The primary objectives of this surveillance was to detect early any change in the sensitivity of the parasite to chloroquine and to determine the relationship of resistance to drug use by comparing drug response patterns in the areas where chloroquine was readily available in each village with the area where it was not. This information would enable us:

1. to recommend changes in the choice and dosage of drugs to be used in primary health care for treatment of malaria;
2. to determine the drugs appropriate for prophylaxis among high risk groups like pregnant women and visitors from non-malarious areas.

This surveillance was particularly important for choosing the drug and dosage that would be used in the pregnancy study that was designed to determine the most effective regimen in primary health care. Results of the investigations done in 1981 and 1982 prior to beginning drug distribution have been reported (Spencer et al., 1983a). In this section is presented the findings of the in vivo and in vitro studies completed through November 1987. The earlier data are presented for comparison. Chloroquine resistant P.falciparum malaria was first documented in 1982 in Kenyans living in malarious areas in western Kenya (Spencer et al., 1983b) and occurs in as many as 30% or more of P.falciparum infections in Kenyans in some areas (Watkins et al., 1984).

4.2 MATERIALS AND METHODS

4.2.1 In vivo tests

Both modified and standard 7-day WHO in vivo tests for the sensitivity of P.falciparum to chloroquine were carried out (World Health Organization, 1973). The details of materials and methods used at Saradidi have been reported by Spencer and colleagues (1987).

In October 1987 children in nursery to standard 4 at three schools in Saradidi were screened with thick blood smears for malaria infection. Those with 500 or more P.falciparum parasites per cubic mm of blood, without other malaria parasites seen and with a negative Dill Glazko test for 4-aminoquinolines in the urine were enrolled into the study. Children were assigned sequentially to receive either chloroquine or amodiaquine in a total dose of 25 mg base per kg body weight given over 3 days. In a sample of the children, venous or finger-prick blood was obtained for in vitro drug sensitivity testing. Children were seen daily for the first week and again at day 14 after starting treatment and thick blood smears were made at each visit.

A total of 146 children were enrolled into the study, with the same number of males and females. Of these, 145 were followed up until day 15; one child was excluded for receiving the wrong drug.

4.2.2 In vitro tests

Rieckmann macro in vitro tests were done as previously described. Rieckmann et al. (1968) and Spencer et al. (1987) and the Rieckmann micro test was done as previously reported by Rieckmann et al. (1978) and described for the Saradidi study by Spencer et al. (1987).

In the November study, in vitro micro tests were performed by the method of Rieckmann using predosed plates from WHO. Briefly, parasitised blood was diluted 1 to 10 in culture medium, 50 µl was placed into wells containing different concentrations of drug and the parasites were incubated

for 24 hours. Results were read as the concentration of drug needed to inhibit schizont development.

4.2.3 Chloroquine levels

To document that adequate whole blood levels of chloroquine were achieved, we collected 100 µl blood samples by finger prick on days 1 or 2 and transferred them to a 25 x 75 mm piece of RAPACO No. 1024-0.38 filter paper (Rochester Paper Co., Rochester, Minn., USA). Specimens selected were eluted and levels of chloroquine and desethylchloroquine were determined by high performance liquid chromatography (Patchem et al., 1983).

4.2.4 Areas

Tests were done in the 3 study areas which had been designated A, B and C as previously described (Spencer et al., 1987). In Areas A and B antimalarial treatment with chloroquine phosphate provided by volunteer village health workers was available free of charge in each village (widespread drug use). Area C served as a control and did not have free chloroquine treatment available in each village. In all 3 areas, chloroquine phosphate could be purchased at local shops and was also available from the Ministry of Health dispensaries (Mburu et al., 1987).

4.3 RESULTS

4.3.1 In vivo tests

a) In Saradidi area

Results of the 7 day in vivo test are presented in Table 4.1. Failure of P.falciparum infections treated with chloroquine base 10 mg/kg was not detected until June 1983. From June 1983 until February 1984 the proportion of children with resistant infections did not change significantly. In February 1984 a higher proportion of infections treated with 10 mg base per kg were

resistant in Area A as compared with the control area C; this difference was statistically significant (P = 0.03 Fisher's exact test, 2-tailed). The reverse had been true in September 1983 but the difference was not statistically significant (P > 0.05). Before September 1984, resistance to treatment with chloroquine base 25 mg/kg had been seen in only 2 infected children. In September 1984 only chloroquine 25 mg/kg base was administered. At 7 days, 9 (10.2%) of the 88 infected children had resistant patterns, including one child who had only a 58% decrease in parasitaemia at 48 hours and did not clear subsequently (RII-RIII resistance). Because 5 (5.7%) children had an RII pattern (reduction but no clearance of parasitaemia), follow up was extended to day 14, at which time an additional 13 (14.8%) children had recrudescent parasitaemia (RI). Therefore, at 2 weeks 22 (25%) P.falciparum infections were resistant in vivo. The proportion of resistant isolates in Areas A and B were not significantly different from those in Area C.

In November 1987, a similar study was carried out in a cohort of children enrolled in an on-going project. These children, aged 2 to 18 months, were screened for malaria infection at routine follow-up visits. Those with P.falciparum infections were sequentially assigned to receive either chloroquine or amodiaquine syrup in a total dose of 25 mg/kg given over 3 days, as in the previous study. All drugs were given under observation, and thick smears were taken on days 0, 2, 7 and 14. Urines were tested for 4-aminoquinolines. Finger-prick blood was obtained from a sample of the children for in vitro drug sensitivity testing.

Of the 152 children enrolled into second study, 142 or 93% were followed to day 7, and 139 or 91% to day 14. Table 4.3 shows the findings at day 7 and day 14. Only 20% of children receiving chloroquine and 49% of those receiving amodiaquine had sensitive infections. Of children given chloroquine, 50% did not have a 75% or greater decrease in parasitaemia, indicating higher level (RIII) resistance. Among amodiaquine recipients, 11% had RIII resistance. At

day 14, the proportion of children with parasites had increased from 80 to 89% in the chloroquine group and from 51 to 76% in the amodiaquine group.

Table 4.4 summarises the in vivo sensitivity of P.falciparum to chloroquine in Saradidi based on studies done on school age children since 1980. It demonstrates that the level of parasite sensitivity to treatment has decreased since 1983, though most of the resistance is at the RI and RII levels.

b) Nyakach area to compare chloroquine and amodiaquine

The mean age of children studied in Nyakach of Nyanza Province in November 1987 was 7 years, with a range of 2 to 13. The response to chloroquine and amodiaquine at day 7 is presented in Table 4.2. Only 68% of children given chloroquine cleared their infections, compared with 96% given amodiaquine. These were considered sensitive infections by WHO criteria. 14% of children receiving chloroquine cleared their parasites but had a recrudescence by day 7, which is classified as RI resistance. By contrst, only 4% of amodiaquine recipients had RI resistance. An additional 18% of children given chloroquine failed to clear their parasites, though they had at least a 75% decrease in the number of parasites. These are considered RII resistant infections. There was no RII resistance in the amodiquine group. By day 14 the number of children with recurrent or persistent infections increased from 32 to 59% among chloroquine recipients and from 4 to 19% among amodiaquine recipients.

4.3.2 In vitro tests

In 1981, all isolates in the macro test had an MIC of 1.5 x 10_{-6} mol/l blood or less (Table 4.5). In contrast, 1 (8.3%) isolate in 1982 and 3 (10.7%) isolates in 1983 had an MIC greater than 1.5 x 10_{-6} mol/l. From 1981 to 1983 the proportion of isolates inhibited at every chloroquine concentration decreased. Similar results were found with the Rieckmann micro test (Table 4.5). By 1984 12 (31.6%) isolates had an MIC greater than 1.6 x 10_{-6} mol/l blood.

Effective chloroquine concentrations in the two in vitro tests for 50% (EC50) and 99% (EC99) inhibition of schizont development as determined by probit analysis are shown in Table 4.6. A decrease in the sensitivity of P.falciparum to chloroquine over time, peaking in 1984. is evidenced by the increasing EC50s and EC99s.

In October-November 1987 the in vitro test was attempted with 133 isolates and were successful with 112 or 84%. Table 4.7 shows the number of isolates whose growth was inhibited at each concentration of drug tested. Normally, parasites are considered sensitive if they are inhibited by chloroquine 114 nmol/L or less and by amodiaquine 80 nmol/L or less. As can be seen, only 15% of parasites appeared to be sensitive to chloroquine and 13% to amodiaquine. However, a higher proportion of isolates were not inhibited by higher concentrations of chloroquine, 28% continuing to grow at even the highest concentration tested.

Table 4.8 shows the proportion of isolates from Saradidi, Nyanza Province since 1982 which have been sensitive and resistant to chloroquine. Those tested in 1987 appear to have a much higher proportion resistant. Table 4.9 shows an even more dramatic change with amodiaquine.

4.3.3 Chloroquine levels

Filter paper specimens collected on day 1 from 10 of 12 children with chloroquine resistant infections in September 1983 were examined for chloroquine and desethylchloroquine. The mean chloroquine level was 270 µg/ml (range 147-412 µg/ml) and the mean desethylchloroquine level was 115 µg/ml (range 22-218 µg/ml). These concentrations were within the expected range of normal drug absorption (Patchem et al., 1983).

In summary an increase in in vivo resistance among school children in Saradidi has been observed with 32% of infections being resistant to chloroquine. A higher level of resistance was seen in children 2 to 18 months

old, with 80% of infections resistant to chloroquine and 51% resistant to amodiaquine. The higher resistance in the younger children may be due to lower levels of acquired immunity. In vitro resistance in Saradidi appears to have increased significantly in 1987.

Before 1983, all 71 infections treated with chloroquine were sensitive in vivo; parasitaemia cleared by day 3 and remained absent through day 7. In June 1983, 23.1% of 26 infections treated with chloroquine base 10 mg/kg either recrudesced in 7 days (RI resistance, 5 infections) or decreased but failed to clear (RII resistance, 1 infection). In September 1983, 16.2% of 68 and in February 1984 13.2% of 53 infections were resistant in vivo after treatment with chloroquine base 10 mg/kg. A course of chloroquine base 25 mg/kg over 3 days remained effective; only 2 (1.6%) of 129 infections examined were resistant in vivo; in both, parasitaemia cleared then recurred (RI). In September 1984, however, 9 (10.2%) infections were resistant after treatment with chloroquine base 25 mg/kg; in 4 of these parasitaemia decreased but never cleared (RII).

Similar results were observed in vitro. In the Rieckmann macro in vitro test, 63.3% of 30 P.falciparum isolates tested were resistant to chloroquine (minimal inhibitory concentration (MIC) >1.25 x 10_{-6} mol/l blood) in June 1983, as were 61.8% of 34 isolates in the Rickmann micro test (MIC .1.14 x 10_{-6} mol/l blood). The effective drug concentrations to chloroquine for 99% inhibition (EC50) increased in the macro in vitro test from 0.617 x 10_{-6} mol/l blood in May 1981 to 1.917 x 10_{-6} mol/l in June 1983 and in the micro test from 0.929 x 10_{-6} mol/l blood in May 1982 to 3.458 x 10_{-6} mol/l in September 1984. It was not possible to associate increased drug pressure with the development of resistance since resistance occurred in areas, including Area C, with varying degrees of drug use. The results demonstrated a changing pattern of the response of P.falciparum to chloroquine in Saradidi, but that the resistance levels were such that chloroquine could still be used as the drug of first choice

for treatment of clinical malaria at the dose of 25 mg/kg and for chemoprophylaxis in Saradidi.

Finally, a more rapid progression in the proportion of resistant isolates and the degree of resistance than in Saradidi was noted at the Kenya coast (Sixsmith et al., 1983; Watkins et al., 1984). It appears from the data presented that Saradidi rates have more than caught up with the resistance rates of the coast.

In malarious areas like Saradidi, the decision to change recommendations for initial treatment of malaria should be based not only upon the pressure of drug resistance but also upon the frequency and severity of resistance, its geographical distribution, the epidemiology of malaria, including the level of immunity by age in the population, the severity of the illness, the cost and the toxicity of alternative drugs and the accessibility of a referral system and its ability to cope with severely ill patients and those who have failed to respond to initial antimalarial therapy. Even when resistance is present, clinical symptoms may be ameliorated and mortality or significant morbidity prevented. Thus it is impossible to make uniform recommendations for changing initial treatment based on the occurrence at some previously decided level since each situation will vary according to local conditions (Spencer et al.. 1987).

This study has demonstrated a decrease in in vivo sensitivity of Plasmodium falciparum in Saradidi from 99% in 1983 to 79% in 1985; an increase in in vitro resistance in Saradidi from 18% in 1983 to 32% in 1987; and a greater sensitivity of P.falciparum to amodiaquine than to chloroquine both in vivo and in vitro; while over 85% of infections are still sensitive to a single dose of pyrimethamine/sulfadoxine (Fansidar).

The response to chloroquine and amodiaquine at day 7 in the 1987 study showed that 68% of children given chloroquine cleared their infections, compared with 96% given amodiaquine. 14% of children receiving chloroquine cleared their parasites but had a recurrence by day 7. (RI resistance). By

contrast, only 4% of amodiaquine recipients had RI resistance. An additional 18% of children given chloroquine failed to clear their parasites, though they had at least 75% decrease in the number of parasites (RII). There was no RII resistance in the amodiaquine group. The number of children with recurrent or persistent infections increased from 39 to 59% among chloroquine recipients and from 4 to 19% among amodiaquine recipients.

4.4 IN-VIVO TESTING OF PLASMODIUM FALCIPARUM RESPONSE TO CHLOROQUINE PHOSPHATE IN PREGNANT AND NULLIPAROUS WOMEN

4.4.1 Introduction

In areas of high Plasmodium falciparum malaria endemicity, pregnant women are at risk of developing adverse effects from malaria infection including delivering low birth weight babies. It is therefore recommended that they use chloroquine in a suppressive dose of 300 mg weeky (approximately 5 mg/kg) throughout pregnancy and during the first two months postpartum.

Weekly suppressive doses of chloroquine in school age children have been effective in suppressing peripheral parasitaemia in areas with chloroquine sensitive parasites. However, large scale chemoprophylaxis programmes in young children have been difficult to maintain; and attempts at community based malaria chemoprophylaxis even only among pregnant women have achieved discouragingly low rates of coverage. Thus with a less than optimally effective drug and difficulty in maintaining a chemoprophylaxis delivery system, the strategy requires reassessment and hence the main objective of this thesis.

Available information indicates that primiparous women and to a lesser extent women in their second pregnancy are at particular risk when compared to multigravid women (McGregor, 1984).

There is more than a 20-fold increased risk of mortality in babies born with birth weight under 2500 gms compared to those with birth weight over 3000 gms (McCormick, 1985); there is also a 5-fold increase in the risk of post-neonatal mortality among low birth weight babies as compared to their counterparts with normal birth weight (McGregor, 1984). An effective antimalarial drug should be able to clear peripheral parasitaemia. Evaluation of the delivery of weekly suppressive doses of chloroquine in school age children has shown that it is effective in suppressing peripheral parasitaemia in areas with chloroquine sensitive parasites. However, large scale chemoprophylaxis programmes in young children have been very difficult to maintain. Likewise, we were only able to reach 30% of pregnant women in Saradidi with chloroquine and only a few times during pregnancy (Kaseje et al., 1987). We concluded that the weekly prophylaxis for pregnant women is impossible to maintain except under special or research conditions. It is because of this that a study was designed to try out alternative methods which would be manageable in providing chemoprophylaxis and would be provided at antenatal clinics (ANC).

This section presents the results of an investigation which was designed to evaluate in vivo parasite sensitivity to chloroquine at therapeutic doses (chloroquine at 25 mg/kg) and suppressive doses (chloroquine at 5 mg/kg) in pregnant women and a group of non-pregnant women of similar age and gravid status in Saradidi, an area with high levels of transmission of P.falciparum and known levels of chloroquine resistant P.falciparum.

Thus both regimens would be evaluated for their ability to clear peripheral parasitaemia. Chloroquine at 5 mg/kg would be evaluated over 4 weeks and chloroquine 25 mg/kg would be evaluated over 7 days for the ability to clear peripheral parasitaemia. This would be useful in determining the appropriate monthly dosage of chloroquine.

The birth weight of infants delivered in Siaya District Hospital, which is the referral base for Saradidi programme, was examined and the prevalence of

P.falciparum infection in pregnant women attending antenatal clinics at the same hospital was determined. The in vivo parasite response to chloroquine at 25 mg/kg given over three days (in women with parasite densities >1500/mm^3 blood) and to chloroquine at 5 mg/kg weekly (in women with parasite densities <1500/mm^3 blood) over 28 days in pregnant and never-pregnant women were compared.

4.4.2 Methods

a) Record review

April-June, 1986 Delivery Ward records for consecutive live-born singleton deliveries were reviewed by maternal age, parity and the newborn gender and birth weight.

b) Parasitaemia screening

Pregnant women attending antenatal clinics in Saradidi on 13th, 16th and 19th June 1986 were screened using a Giemsa stained thick blood smear for P.falciparum parasite infection. Parasite densities per mm^3 of blood were estimated by counting parasites in fields with 300 leucocytes and using a value of 8000 leucocytes/mm^3 blood to calculate parasite density. Never-pregnant women at a local school were screened in the same way.

c) Study groups

- Pregnant primiparous women
- Pregnant multiparous women
- Non-pregnant nulliparous women
- Non-pregnant multigravid women

d) Eligibility for the study

Criteria for eligibility for the investigation included:

- Pregnant women (first or subsequent pregnancy)
- Non-pregnant women (gravida 0 or 1+)
- Blood slide positive for P.falciparum parasitaemia <500 parasites/mm^3, as a pure infection

- No history of antimalarial drug use in the last 2 weeks
- Residents of the study area
- Willingness to participate in the study

Criteria for exclusion:

- Unknown or unsure reproductive history
- P.falciparum parasitaemia <500 parasites/mm^3
- Mixed parasite infection
- Antimalarial drug use in the last 2 weeks
- Non-residents of the study area
- Unwillingness to participate in the study

Enrolment into the study:

- Pregnant women meeting the eligibility criteria were sought from antenatal clinics and non-pregnant women from the surrounding villages and schools and were enrolled into the study if they met the eligibility criteria.
- Women with parasitaemias between 500-2000/mm^3 were enrolled into the chloroquine 5 mg/kg weekly dosage group.
- Women with parasitaemia >2000/mm^3 were enrolled into the chloroquine 25 mg/kg therapeutic treatment group.

e) In vivo drug study

Women screened as above who were infected with P.falciparum only and had no recent history of antimalarial drug use and negative results on urine testing for chloroquine were enrolled and followed up on days 2, 7, 14, 21 and 28. Pregnant and never-pregnant women with screening parasite density <1500/mm^3 were treated with weekly chloroquine phosphate at 5 mg/kg; those with >1500 parasites/mm^3 were treated with chloroquine 25 mg/kg divided over three days: 10, 10 and 5 mg/kg body weight.

Information collected at enrolment included name, age, reproductive history, history of fever and antimalarial drug use in the previous seven days.

History of fever and antimalarial drug ingestion was obtained on each follow-up visit.

At enrolment the following specimens were obtained on all study subjects:

- Giemsa stained thick and thin blood film
- urine specimen for specific gravity and modified Haskins testing for chloroquine

Specimens collected at follow-up visits on days 2, 7, 14, 21 and 28 included all of the above except serum and RBC clot which were collected only on day 28. Urine testing for chloroquine levels was done using a modified Haskin's procedure. Non-pregnancy status was confirmed by sensi-slide (Roche Pharmaceuticals) B-specific urine test for human chorionic gonadotropin.

4.4.3 Results

a) Newborn birth weights

The mean birth weight of first borns was 2772 grams, 21% weighed less than 2500 grams; all newborns that were not first borns had a mean birth weight of 3000 grams (Table 4.10).

b) Malaria parasite screeening

A total of 342 pregnant women were screened and 145 (42.4%) with P.falciparum infection were identified (Table 4.11), 68% of 66 first pregnancy women were parasitaemic compared to 33% of 178 women in subsequent pregnancies (X^2 = 25.01, df = 1, P <0.001). The geometric mean parasite density was significantly higher in first pregnancy women than in subsequent pregnancies (P <0.01) 50% of 98 never-pregnant women were parasitaemic. This was lower than the prevalence in primiparous women (X^2 = 55.33, 1 df, P <0.001)) and higher than the prevalence in older women in subsequent pregnancies (X^2 = 8.08, 1 df, P <0.01).

c) In vivo studies

75 pregnant women and 41 never-pregnant women were enrolled and completed at least 7 days of follow-up. Their distribution into chloroquine 5 mg/kg/weekly and chloroquine 25 mg/kg treatment groups and proportion clearing their peripheral parasitaemia by day 7, 14 and 21 is shown in Table 4.12. 20 of the 29 pregnant women in the chloroquine 25 mg/kg group were in their first pregnancy and accounted for seven of the eight women who were parasitaemic on day 7. This failure to clear parasites by day 7 in the chloroquine 25 mg/kg group was more common in primiparous women (7 of 20) compared to never pregnant (0 of 17), (X^2 = 6.45 2 df, $P < 0.05$) and women in subsequent pregnancies (1 of 9). Only 9 of the 46 women with low parasitaemias on chloroquine 5 mg/kg weekly were primiparous. Pregnant women were more likely to clear their parasitaemia on the chloroquine 5 mg/kg/weeky regimen than the younger never-pregnant women and did not require intervention with a therapeutic dose of chloroquine or amodiaquine. The need for therapeutic intervention when it occurred among the pregnant women was more common in primiparous women, although the difference was not significant.

A higher proportion of pregnant women failed to respond to chloroquine 25 mg/kg at each of day 7 and 14 compared to never-pregnant women. However, by day 21, new infection or recrudescence was observed commonly in the pregnant group. This was coincident with low levels of chloroquine as assessed by urinary excretion with 5 ppm of chloroquine and its metabolites detected.

Amodiaquine at a dose of 25 mg/kg was used to treat 19 women who failed to clear their parasites after chloroquine use and 16 (84%) of these women were aparasitaemic on day 7 after starting treatment. The three failures were among the 11 primiparous women.

d) The problem with drug administration

Vomiting after chloroquine administration was reported by 11 (15%) of the 73 pregnant women but was, except for 2 women, 4 hours after drug administration. Vomiting was reported in one of the non-pregnant women. Pruritus associated with chloroquine administration was reported by 41 (56%) of the pregnant women and by 6 (14%) of the non-pregnant (younger) women.

Antihistamines (chlorphenaramine) were used to counteract pruritus. but systematic evaluation of its efficacy was not performed.

As an overview of the data collected, women who received chloroquine 25 mg/kg had higher trends of 4-aminoquinolines excretion in the urine on day 14 than the women on chloroquine 5 mg/kg weekly. but had lower levels by day 21. All women on chloroquine 25 mg/kg regardless of parasitological response, exhibited absorption of chloroquine and typically had urine levels of 8 ppm by day 7.

4.5 DISCUSSION

This series of studies demonstrates a changing pattern in the sensitivity of P.falciparum to chloroquine in the Saradidi community. Failure of chloroquine treatment at 10 mg base per kg was first noted in June 1983. The rate of failure of chloroquine treatment has increased steadily until by November 1987 only 68% of cases studied cleared their infections at the dosage of 25 mg/kg. This has been shown by data from both in vivo and in vitro studies.

The data also show that the changes in the in vitro pattern began before drug intervention (Spencer et al., 1983b). Of interest is the fact that probit analysis predicted the in vivo response since the EC50s and EC99s were increasing over time in both the macro and micro in vitro tests. A similar but less striking trend was noted in the MICs. From June 1983 to September 1984, the proportion of isolates resistant in vivo and the degree of resistance in vivo

(RI to RII as defined by WHO, World Health Organization, 1973) increased. This increase was found to be even more dramatic by 1987.

It is difficult to ascribe the changing pattern of chloroquine sensitivity to the widespread use of chloroquine in Areas A and B. Changes were noted in the in vitro response before the beginning of intervention. In June 1983 (P = 0.14. Fishers exact test, two tailed) and in February 1984 (P = 0.03. Fishers exact test, two tailed), the proportion of resistant infections treated with 10 mg/kg was greater in Areas A and B (drug intervention) compared with Area C (control). However, in September 1983 (X^2 = 3.05, P <0.10) eight of 33 infections were resistant in Area C compared to three of 35 in Area A. Significant levels of resistance to chloroquine base at 25 mg/kg were first noted in September 1984. More rapid progression in the proportion of resistant isolates and in the degree of resistance has been noted at the Kenyan coast (Sixsmith et al.. 1983; Watkins et al.. 1984), however the levels of resistance at Saradidi are now just as high as at the coast. The level seems to depend on the immune status of the cases. Much higher levels and rates of resistance occurred in the cohort of children 6-18 months examined.

Thus, no striking differences in proportion of resistant infection were found in the areas where chloroquine phosphate was available in a village (Areas A and B) when compared with the control (Area C). Thus the studies do not implicate drug pressure as the major cause of the development of chloroquine resistance in Saradidi.

These results emphasise the importance of monitoring the response of P.falciparum to antimalarial drugs. The consequences of high level resistance are very grave especially among children. Initial indications of drug failure should come from an increase in the proportion of persons who do not respond to treatment in the peripheral facilities. In a place like Saradidi, community health workers should play an important role in the identification and reporting of problems of treatment failure. Such reports would need to be investigated and necessary action taken.

The levels of resistance existing in Saradidi may make chloroquine a rather useless drug even for the purposes of prophylaxis among the high risk groups of pregnant women and particularly those pregnant for the first time. In the selection of an alternative drug to chloroquine the decision should take into account: the frequency and severity of drug resistance, its geographical distribution, the epidemiology of malaria including the level of immunity by age in the population, the severity of illness, and the cost, availability and toxicity of the alternative drugs.

It is also important to consider the quality and accessibility of the referral system and its ability to cope with the severely ill patients and those who have failed to respond to initial antimalarial therapy. All these factors must eventually influence the policy decision and guidance provided by the Ministry of Health regarding the choice and dosage of antimalarials to be used for chemoprophylaxis and chemotherapy to various target community members.

The proportion of primiparous women treated with therapeutic doses of chloroquine (25 mg/kg) who failed to clear P.falciparum parasites from their peripheral blood was significantly higher compared to the proportion in multiparous and never-pregnant women. The response of the parasite in primiparae was similar to the response observed in children aged 5 years and under in previous studies in this area of western Kenya. Similarly, the prevalence and parasite density of P.falciparum infections were higher in primiparous women as compared to multiparous women.

In women with initial parasite densities 1500/mm^3, clearance or maintenance of asymptomatic low density infections was not significantly different between pregnant and never-pregnant women. However, primiparous women were again more likely to develop increasing parasitaemia with symptoms requiring therapeutic intervention. As peripheral parasitaemia and placental parasite infections do not equate, we cannot ensure that clearance of peripheral parasites will be accompanied by the clearance of placental

infection, but it is arguable that where parasites are sensitive, the administration of an initial therapeutic dose of chloroquine at 25 mg/kg body weight should precede the use of suppressive treatment, if placental parasites are to be reduced.

Primiparous women require special attention due to their higher rates of delivering low birth weight babies. which is perhaps due to their higher prevalence and density of malaria parasitaemia experienced by them. In areas with high levels of transmission of P.falciparum. it would appear that these women notably derive the greatest benefit from chemoprophylactic treatment. It would also seem that a therapeutic dose of 25 mg/kg over 3 days is required to treat symptomatic malaria infections. Alternative drugs should be used only in patients with identified chloroquine-resistant parasites. If chemoprophylaxis is to be used in asymptomatic primiparous women, a therapeutic dose of chloroquine is necessary initially to clear the possible asymptomatic parasitaemia and a monthly regimen of chloroquine (25 mg/kg/week) should be used thereafter. If symptomatic malaria infection intervenes while on this regimen, then an alternative antimalarial drug like Fansidar at therapeutic doses is required. Despite the theoretical benefits of regular chemoprophylaxis during pregnancy, weekly administration of chloroquine may be difficult to maintain, especially in areas where chloroquine associated pruritus is common and in a rural community in the context of primary health care.

Regardless of the intervention chosen. it appears that whatever the local or systemic immune alteration is that makes the primiparous women more susceptible to malaria and the deleterious effects on the foetus and newborn, this alteration will also make her less likely to respond to antimalarial treatment. These results tend to support the use of monthly chemotherapeutic chloroquine (25 kg/mg) instead of the weekly currently recommended regimen of 5 mg/kg.

In conclusion, we have observed a modest increase in in vivo resistance

among school children, with 32% of infections resistant to chloroquine in 1987 as opposed to none in 1982. There is a much higher level of resistance among young children 2 to 18 months old who had 80% infections resistant to chloroquine and 50% resistant to amodiaquine in 1987.

The rate of chloroquine-resistance in vivo is also higher among women pregnant for the first time. These vulnerable groups need special consideration in the selection of drugs and protection against malaria and its consequences.

Table 4.1 Sensitivity of Plasmodium falciparum to chloroquine in vivo Saradidi. 1979-1987

Date	Area	Dose chloroquine base[1]	No. infections treated	No. (%) resistant	No resistant at level[2] RI	RII	RIII
May 1981	A and B	10 mg/kg	26	0	-	-	-
		25 mg/kg	15	0	-	-	-
May 1982	A and B	10 mg/kg	20	0	-	-	-
June 1983	A and B	10 mg/kg	26	6(23.1)	5	1	-
	C	10 mg/kg	9	0	-	-	-
Sept. 1983	A	10 mg/kg	35	3(8.6)	2	1	-
	C	10 mg/kg	33	8(24.2)	4	4	-
	A	25 mg/kg	32	1(3.1)	1	-	-
	C	25 mg/kg	34	0	-	-	-
Feb. 1984	A	10 mg/kg	24	6(25.0)	2	4	-
	C	10 mg/kg	29	1(3.4)	-	-	-
	A	25 mg/kg	24	1(4.2)	1	-	-
Sept. 1984	A and B	25 mg/kg	51	5(9.8)	2	2	-
	C	25 mg/kg	37	4(10.8	2	2	-
Oct. 1987	A, B and C	25 mg/kg	47	7(21.0)	5	3	1

1 Chloroquine base: 10 mg/kg in single dose or 25 mg/kg given over 3 days

2 RI = clearance followed with recrudescence by day 7

RII = decrease in parasitaemia but no clearance

RIII = no decrease in parasitaemia

3 Area A and B compared with area C: p = .14, Fisher's exact test

4 Area A compared with area C at 10 mg/kg chisquare = 3.05, p >.05

5 Area A compared with area C at 10 mg/kg, p = .03, Fisher's exact test

Table 4.2 In vivo response at Day 7 to chloroquine and amodiaquine, Kenya 1983-1987

		Chloroquine		Amodiaquine	
Date	Location	No.	% Resistant	No.	% Resistant
+ May 1983	Coast	69	12	60	0
+ June 1983	Coast	21	14	21	5
* Oct. 1985	Nyanza	42	21	42	0
** Nov. 1987	Nyanza	72	32	73	4
*** Nov. 1987	Nyanza	70	80	72	51

\+ Primary school children in Kilifi, Coat Province
* Primary school children in Saradidi, Nyanza Province
** Nursery and primary school children, Nyakach location in Nyanza Province
*** Children aged 2-18 months in Saradidi, Nyanza Province

Table 4.3 In vivo response at day 7 and day 14 in children 2-18 months old, Nyanza Province, November 1987

	CHLOROQUINE			AMODIAQUINE		
Follow-up	Number	Parasitaemia No.	%	Number	Parasiaemia No.	%
Day 7	70	56	80	72	37	51
Day 14	72	64	89	67	51	76

Table 4.4 In vivo sensitivity of P.falciparum to chloroquine 25 mg/kg among school children in Saradidi, Kenya. 1980 1987

Year	Number examined	Resistance percent pattern			
		Sensitivity*	RI*	RII	RIII
1980	125	100	0	0	0
1981	122	100	0	0	0
1983	119	98	2	0	0
1984	222	92	4	3	1
1985	42	79	12	7	2
1986	389	82	13	5	0
1987	72	68	14	18	0

* at day 7

Table 4.5 Response of Plasmodium falciparum to chloroquine in the Rieckmann Macro Test, Saradidi. Kenya. 1981-1983

Concentration chloroquine 10^{-6} mol/l blood	May 1981		May 1982		June 1983	
	No. isolates*	% inhibited**	No. isolates*	% inhibited**	No. isolates*	% inhibited**
0.25	17	5.7	14	0	30	0
0.5	16	31.3	14	14.3	29	0
0.75	14	71.4	13	53.8	27	33.3
1.0	17	76.5	13	53.8	30	36.7
1.25	13	92.3	11	90.9	20	60.0
1.5	1	100	14	85.7	29	75.9
2.0	15	100	12	91.7	28	89.3
3.0	12	100	12	91.7	24	91.7

* Not all isolates examined at every concentration
** Schizont development inhibited >99% of drug free controls.

Table 4.6 Effective drug concentrations[1] for 50% (EC_{50}) and 99% (EC_{99}) inhibition of schizont development in the Rieckmann Macro and Micro in vitro tests for sensitivity of P.falciparum to chloroquine, Saradidi, Kenya 1981-1983

	EC_{50} (10^{-6} mol l^{-1} blood)		EC_{99} (10^{-6} mol l^{-1} blood)	
Date	Macro test	Micro test	Macro test	Micro test
May 1981	0.202	-	0.617	-
May 1982	0.153	0.309	0.796	0.929
June 1983	0.269	0.395	1.917	1.411
September 1984	-	0.609	-	3.458

[1] Grab, B. and Wernsdorder, W.H. Unpublished. World Health Organization. Geneva. WHO/MAL/88.990

Table 4.7 Minimal inhibitory concentrations (MIC) of chloroquine and amodiaquine in the micro test, at Saradidi. Kenya (October-November 1987)

	CHLOROQUINE		AMODIAQUINE	
MIC nmol/L	Number inhibited	Cumulative % inhibited	Number inhibited	Cumulative % inhibited
<80	9	8	15	13
114	8	15	NA	-
160	9	23	58	65
320	27	47	27	89
640	28	72	NA	-
No. tested	112		112	

Table 4.8 In vitro sensitivity of P.falciparum to chloroquine by the Micro Test. Nyanza Province. Kenya. 1982-1987

Year	Number	Sensitive* (%)	Resistant (%)
1982	31	96	4
1983	34	82	18
1984	38	68	32
1985	57	68	32
1986	64	64	36
1987	12	15	85

* Schizont development inhibited at ≤114 nmol/L

Table 4.9 In vitro sensitivity of P.falciparum to amodiaquine by the Micro Test, Nyanza Province, Kenya, 1984-1987

Year	Number	Sensitive* (%)	Resistant (%)
1984	37	100	0
1985	57	100	0
1986	73	90	10
1987	112	13	87

* Schizont development inhibited at ≤80 nmol/L

Table 4.10 Maternal age and birth weight distribution by pregnancy number among consecutive live singleton births. Siaya District Hospital, Kenya, April - June 1986

Pregnancy No.	No.	Mean age yrs (± SD)	Mean birth weight (±SD)	Birth weight <2500 gms No. (%)
1	191	18.1 (2.6)	2772 (499)	41 (21)
2	85	20.9 (2.2)	3028 (536)	15 (18)
3	57	22.7 (3.1)	3111 (473)	3 (5.3)
4	46	24.5 (2.8)	3191 (457)	2 (4.4)
5	30	25.9 (2.8)	3168 (310)	1 (3.3)
6	41	29.5 (3.5)	3164 (567)	5 (1.2)
>7	45	33.0 (5.6)	3110 (55.4)	4 (3.9)

Table 4.11 Prevalence and density of Plasmodium falciparum infections in women in western Kenya

Parity	Number tested	Positive N (%)	Geometric mean Parasite density per mm^3/blood
non-pregnant	98	43 (50)	462
0	66	45 (68)	1538
1-3	77	26 (34)	336
4-5	52	15 (29)	299
>6	49	16 (33)	330
Total	342	145 (42.4)	593

Table 4.12 Characteristic of parous and nulliparous women with Plasmodium falciparum infection and day 7, 14 and 21 follow-up after chloroquine treatment

Treatment Group	Pregnancy status	Number	Mean Pregnancy Number	Mean Age (yrs)	Initial geometric mean parasite density/ mm^3 n	Number aparasitaemic Day 7 n	Day 7 %	Day 14 n	Day 14 %	Day 21 n	Day 21 %
Chloroquine 25 mg/kg (over 3 days)	Nulliparous	27	-	14.3	2537	17	100	12	71	5	29
	Parous	29	1.8	19.9	4343	21	72	12/28	43	8/27	30
	Primiparous	20	1	17.9	4828	13	65	7	35	3/19	16
	Multiparous	9	32	24.6	3311	8	89	5/8	63	5/8	63

21 day Follow-up Chloroquine 5 mg/kg/weekly						Aparasitaemia		Low Parasitaemia		Required Tx	
	Nulliparous	24	-	14.5	131	9	38	13	54	2	8
	Parous	46	3.7	23.8	227	26/4	60	10/43	23	7/4	16
	Primiparous	9	1	19.1	692	5	56	1	11	3	33
	Multiparous	37	4.3	25.0	173	21/34	60	9/34	26	4/34	12

CHAPTER 5

EFFECT OF MALARIA CHEMOPROPHYLAXIS TO PREGNANT WOMEN PROVIDED BY COMMUNITY HEALTH WORKERS ON PARASITAEMIA, HAEMOGLOBIN LEVELS AND ANTIMALARIAL ANTIBODIES.

5.1 INTRODUCTION

The consequences of malaria in pregnant women living in holoendemic malarious areas such as occurs in many areas of sub-Saharan Africa differ markedly from those occurring in pregnant women in areas of low endemicity (Brabin, 1983; McGregor, 1984). In tropical Africa, pathologic effects from malaria are significantly more frequent in women pregnant for the first time (primigravidae). Many workers have shown that parasite density and the prevalence of anaemia are greater among them (Bruce-Chwatt, 1952; Archibald, 1956; Cannon, 1958; Gilles et al., 1969; Bray and Anderson, 1979; Brabin, 1983; McGregor, 1984) Mean birth weights are lower, particularly in first born children in association with dense placental parasitisation which leads to higher perinatal and neonatal mortality. Maternal deaths are rare despite placental parasitaemia prevalence rates of 20%-34%. with rates as high as 74% reported (McGregor, 1984). In Africa, no study has quantified a clear and significant relationship between malaria and foetal wastage (abortions and still births). Congenital malaria infections are rare, probably due to protective antibodies acquired transplacentally (Covell, 1950; Bruce-Chwatt, 1952).

To reduce mortality and morbidity due to malaria, chemoprophylaxis is recommended for pregnant women living in malarious areas (World Health Organization, 1984). In some situations, community health workers (CHWs) may be involved in providing antimalarial chemoprophylaxis to pregnant women as part of malaria control activities.

In the community-based malaria control project initiated in May 1982 in Saradidi (Kaseje and Spencer, 1987; Spencer et al., 1987) chloroquine phosphate

chemoprophylaxis for malaria was made available to pregnant women. The drug was provided by volunteer community health workers living in each village. This chapter reports the results of investigations done on the pregnant women attending antenatal clinics in Saradidi between January 1983 and March 1984 and their infants.

5.2 MATERIALS AND METHODS

Saradidi was divided into three sections, desginated Areas A, B and C based on the degree and duration of community organisation (Kaseje and Spencer, 1987). These areas were homogeneous with regard to geography, people, economy and malaria transmission. More than 98% of malaria infections were caused by Plasmodium falciparum alone or mixed with P.malariae and/or P.ovale (Spencer et al., 1987).

5.2.1 Chemoprophylaxis

Antimalarial chemoprophylaxis with chloroquine phosphate base 300 mg weekly was made available to pregnant women living in Area A. Tablets were provided by the World Health Organization. The drug was given by community health workers (CHWs) living in each village. CHWs were volunteers chosen and supported by the community (Kaseje et al., 1987). The characteristics of 126 CHWs in Saradidi were as follows: 96.8% were women, 99.2% were married, 75.4% were between 25 and 39 years of age and 80.2% had had at least 5 years of formal education (only 7% had had none). On her routine visit to the household, the CHW explained the programme and encouraged pregnant women to take chemoprophylaxis.

If the women agreed, a weekly dose was taken then and a sufficient amount of drug left until the next home visit by the CHW. In addition to information from the CHW, pregnant women were informed about chemoprophylaxis at antenatal clinics sponsored by the Saradidi Rural Health and

Development Programme (SRHDP) and at the community meetings. They could then go to the CHW and request chemoprophylaxis.

In Areas A and B, the CHW also provided treatment to persons with malaria (Spencer et al., 1987). Chloroquine phosphate base at 10 mg/kg was given. Thus pregnant women who were not on chemoprophylaxis or those who received it but developed the symtoms of malaria could go to the CHWs and be treated.

In Area C the CHWs were trained and were able to give information and education to the community about malaria control and treatment as was being done in Areas A and B, but had no drugs to supply, either for chemoprophylaxis or chemotherapy. They could, however, refer people to the shops or clinics to obtain chloroquine if they needed it.

5.2.2 Ante-natal clinics

The SRHDP operated maternal and child health clinics at the programme centre in Area A each week on Tuesday and Friday. Each Wednesday a mobile maternal and child health clinic was conducted at one of four sites in Areas B and C. Pregnant women were encouraged to attend these clinics once each month. Each woman was seen by a community health nurse, medical student or physician. Although these clinics did not provide chemoprophylaxis for malaria, all women from Area A who attended the clinics were told about the CHWs chemoprophylaxis programme.

5.2.3 Study design

Between January 1983 and March 1984 every pregnant woman attending an antenatal clinic in Saradidi was entered into the study. At the initial visit, age, parity, village, household and personal numbers and whether the woman said she was taking chemoprophylaxis was recorded. On each visit (usually monthly) until delivery, a urine sample, a thick blood film and a finger prick sample for

haemoglobin level were collected. Thick blood films were collected from the infants of these women at the first visit after birth and monthly as long as the child was brought to the clinic.

The study was carried out by male technicians who had been working in Saradidi since August 1980; they lived in the area during the week and spoke Luo, the language of the Saradidi people. They were trained for two weeks to standardise their techniques before the study began. Supervision of data collection and record keeping was done monthly by the principal investigator.

The urine was tested for the presence of 4-aminoquinolines using the Dill-Glazko test (Lelijveld and Kortmann, 1970). The thick blood film was stained with Giemsa and examined for malaria parasites. At least 100 fields were examined before a slide was diagnosed as negative. Haemoglobin level was determined immediately using a Spencer's haemoglobinometer.

5.3 RESULTS

5.3.1 Study population

A total of 930 pregnant women were examined at least once: 357 from Area A, 373 from Area B and 200 from Area C. This represents 45.9% of the 2027 live births registered in Saradidi between 1st September 1982 and 31st August 1983 (Spencer et al., 1987). Only 104 (29.1%) of the 357 women from Area A said they were taking antimalarial chemoprophylaxis (women from Areas B and C were not offered chemoprophylaxis). The difference between women who were taking chemoprophylaxis from those who were not by age and parity was not statistically significant. The number of samples taken from each woman ranged from 1 to 6 (mean 2.2 for women taking prophylaxis and 2.1 for other women). In Area A, the proportion of women on chemoprophylaxis significantly increased with age ($p<.0005$ - see Table 5.1).

5.3.2 Parasitaemia in pregnant women

As shown in Table 5.2, parasitaemia was significantly more frequent in blood samples from women not taking chemoprophylaxis ($p<.005$). P.falciparum alone or as a mixed infection with P.malariae and/or P.ovale was present in 98.3% of positive slides. Among those with two or more samples, the proportion with all samples negative for parasites was significantly higher ($p<0.005$) in those taking chemoprophylaxis (Table 5.3). The prevalence of parasitaemia by month showed little variation and ranged from 22.7% of sample collected in June 1983 to 32.1% in those taken in December 1983; the differences by month were not statistically significant.

5.3.3 Haemoglobin levels

Haemoglobin levels in women in all three areas who said they were taking antimalarial chemoprophylaxis were significantly higher than those from women who were not on prophylaxis (Table 5.4). No significant differences in haemoglobin levels were found by area among samples from women not on chemoprophylaxis.

The mean haemoglobin level of 127 samples from women taking chemoprophylaxis was 9.95 g/ml (range 6-13.5 g/ml) compared with 9.62 gmdl (range 5.0-13.0 g/ml) in 1111 samples from women not on antimalarial chemoprophylaxis (Student's t test = 2.3483, df = 482, P 0.0193).

5.3.4 Dill-Glazko tests

Urinary samples for 4-aminoquinolines were significantly more often positive in women on chemoprophyalxis ($P <.0005$ - see Table 5.2).

5.3.5 Infant parasitaemia

From May 1983 until March 1984 a total of 1047 blood slides from 317 infants were examined for malaria parasites: 391 (37.3%) were positive (Table

5.5). Parasitaemia rates increased rapidly by age; 49.6% of 135 samples from infants 4 months of age were positive for parasites.

Although samples from infants whose mothers said they were taking weekly chemoprophylaxis with chloroquine had a lower prevalence of parasitaemia (32.6% of 190) when compared with those infants whose mothers gave no history of antimalarial chemoprophyalxis (38.4% of 857), the difference was not statistically significant.

Although parasitaemia rates by month ranged from 23.8% of samples collected in May 1983 to 46.9% of samples taken in December 1983, the differences were not statistically significant (P >0.05).

5.3.6 Maternal parasitaemia prior to delivery

Among the 314 mothers whose infants were followed, those taking chemoprophylaxis had a significantly lower proportion of thick blood films positive for malaria (<0.05) at 0 to 2 months before delivery but not at 3 or 5 months before (P <05 - see Table 5.6).

5.3.7 Serological tests

1) Pregnant women

At a serum dilution of 1:100, virtually all (96.9%) of the 1677 samples from pregnant women were positive (absorbance $\geq$0.3) in ELISA and a high proportion (42.4%) were strongly positive (absorbance more than 1.0); the mean absorbance was 0.96 (Table 5.7). Even at serum dilution of 1:1000 59.5% of the samples were positive and 7.1% were highly positive; the mean absorbance was 0.45. The percentage of samples which were positive or highly positive varied for different months (Table 5.7), being lowest in September and highest in December. The mean absorbance value declined from the December peak to low values during August through October. There was little variation in parasitaemia rates during the 12 months study period (Table 5.7). No significant difference

was found in mean ELISA absorbance values between samples with or without parasitaemia or in relation to chemoprophylaxis (Table 5.8).

2) Infants

Antibodies to P.falciparum as measured by the IFA test were common: 81.6% of the 938 samples had a positive reciprocal titre of 80 or more (Table 5.9). 86.1% of 36 samples from children less than one month of age were serologically positive. Seropositivity decreased after one month then increased presumably due to the decay of the passively acquired maternal antibodies (see Figure 5.1).

Samples were taken sequentially during the course of infant life. The infants experienced parasitaemia very early in their development. There was a drop in seropositivity after the first month of life, but this was followed up by a gradual increase so that by four months of age 88.2% of 118 samples were seropositive. This early increase in seropositivity following an initial drop reflected the infants early experience with malaria parasites.

Similar results were obtained with ELISA. Serum dilutions of 1:100 and 1:1000 gave similar patterns as IFA. Only a small proportion of samples gave an absorbance greater than 8.2% of the 1025 samples at a serum dilution of 1:100 and none at a dilution of 1:1000.

There was no significant differences in the ELISA seropositivity rates found at either serum dilution when samples from infants whose mothers took chloroquine chemoprophylaxis were compared with those infants whose mothers were not taking chemoprophylaxis.

Serological end points (IFA) and absorbance values (ELISA) were determined for paired samples taken during the first and second months of life and subsequent samples taken thereafter from three to eight months after birth. Table 5.10 shows the results of the paired samples.

The ELISA test indicated an increase in response between the first and

second months and a continued increase up to seven months of age. With the IFA test, changes between the first and second months and between less than two months and the third and fourth months were minimal. The difference then steadily increased up to the eighth month samples. Examination of the paired samples gave no evidence of a drop in either IFA titres or the ELISA absorbance values between the initial and the succeeding samples.

There was no statistical difference between parasitaemia rates by month between May and December although the rates ranged from 23.8% (May) to 47.3% (December) ($P < 0.2$ - see Table 5.11).

The effect of chloroquine prophylaxis and the presence of parasitaemia in the mother at the time the maternal samples were taken on the paired antibody response were examined (Table 5.12). With ELISA no significant differences were detected between the paired antibody response in the mother and infant, whether the mother reported taking chloroquine for prophylaxis or not, nor whether the mother had detectable parasitaemia or not.

In the IFA test there was no significant difference in the antibody responses of the mothers. However, samples from infants of mothers taking prophylaxis had significantly lower mean titres when compared with samples from the other infants ($P < 05$). No significant difference was found in the antibody responses of mothers or infants with regard to detectable parasitaemias in the mothers.

5.4 DISCUSSION

This study of the efficacy of weekly chloroquine for antimalarial chemoprophylaxis to pregnant women provided by volunteer CHWs demonstrated significantly fewer blood smears positive for malaria parasites and higher haemoglobin levels among pregnant women on prophylaxis when compared with those not receiving weekly drugs. In addition, women taking chemoprophylaxis were significantly more likely to have blood specimens negative for parasitaemia

than the other women. Although the definition of chemoprophylaxis was based solely on history, pregnant women on chemoprophylaxis also had significantly more urine samples containing 4-aminoquinolines as evidenced by a positive Dill-Glazko test.

Parasitaemia rates in infants were indicative of an intense level of transmission. Infection was present in some infants less than one month of age and almost half of the samples from infants 4 months of age and older were positive for malaria parasites. The relatively linear rate of acquisition of infection observed in the first 4 to 5 months of life suggests that in Saradidi passive acquisition of maternal antibodies from the placenta does not seem to influence the development of parasitaemia as seen in West Africa. These results are similar to those found earlier in Kisumu (Fontaine. 1978). Parasitaemia rates in the first 5 to 6 months of life were similar to those found in Nigeria, but in Saradidi samples from older infants (6 months and older) had a similar proportion of positivity, while in Nigeria parasitaemia rates continued to increase up to 80% or 90% (Bruce-Chwatt, 1952; Gilles, 1968; Molineux and Gramiccia, 1980). The reason for these two different patterns is unknown. Peak transmission is probably less intense in Saradidi and Kisumu than in parts of Nigeria and antimalarial drugs may be more available and accessible in Saradidi.

In this study 29.1% of 357 pregnant women from the area where antimalarial chemoprophylaxis was provided by community health workers, said they were taking it. This result is both encouraging and disappointing. Another study in the same area demonstrated that more than half of women not on chemoprophylaxis were in fact unaware of the programme (Kaseje et al., 1987b). Thus, in Saradidi, community health workers were not effective in providing this service. In areas where chemoprophylaxis is to be given to pregnant women, this experience in Saradidi suggests that it could be provided as part of primary health care services at antenatal clinics and not depending on CHWs to supply.

Given the many responsibilities of the CHWs in Saradidi (Kaseje et al., 1987a), it is probable that asking them to provide chemoprophylaxis to pregnant women may have overloaded many of them. The decision to ask CHWs to provide prophylaxis must be weighed against how much this task would interfere with other responsibilities they are performing. It has been shown that more than 80% of pregnant women in Saradidi attend an antenatal clinic at least once during pregnancy and more than 60% attend more than three times (Bennet, UNICEF Report, 1987). We recommend that chemoprophylaxis might better be provided there than by CHWs in each village. The advantages are obvious. The CHWs could concentrate more on education and treatment, and chemoprophylaxis provided at the clinic would be part of the comprehensive antenatal care. This recommendation also implies that the weekly regimen would be impossible, since pregnant women could not possibly attend antenatal clinics weekly and are normally seen monthly at the antenatal clinics. A monthly regimen should be developed and tested for effectiveness. This has been done at Saradidi and results are presented in Chapters 8-11.

This study on pregnant women provided an opportunity to examine parasitaemia and antimalarial antibodies in pregnant women and to study the acquisition of infection and the inheritance of antiplasmodial antibodies by infants and to measure changes in antibody responses during the early months. The effect of antimalarial chemoprophylaxis was also studied. Antimalarial antibodies were examined from May 1983 until March 1984 in monthly samples taken from the 930 pregnant women attending antenatal clinics in Saradidi, and 317 of their infants; as has been described by Collins et al. (1987). Seropositive rates in pregnant women were uniformly high, and mean enzyme-linked immunosorbent assay (ELISA) absorbance values were not related to the presence of parasitaemia or history of chemoprophylaxis. Parasitaemia was present in 26.5% of 1677 slides from pregnant women and there was little variation by month of sample. Mean ELISA absorbance value did vary by month of sample.

Seropositivity rates in infants were high as measured in both the indirect fluorescent antibody test (IFAT) (81.6% of 938) and the ELISA at 1:100 (83.8% of 1025) and 1:1000 (34.8% of 1025) serum dilutions. Seropositivity rates decreased slightly after birth but by 4 months of age rates were again high.

Parasitaemia was present in 26.5% of 1677 slides from pregnant women. Paired comparisons were made on maternal samples collected less than 2 months before parturition and samples from the infants collected within 2 months after birth. The paired antibody response by IFAT or ELISA was not dependent on the presence of detectable parasitaemia in the mother. Infants from mothers with a history of antimalarial chemoprophylaxis had significantly lower IFAT titres ($P < .05$) than other infants.

Measuring the absorbance of a 1:100 serum dilution by the ELISA appeared to be an excellent method with which to measure longitudinal serologic changes in a population. Detailed results have been reported by Collins et al. (1987).

It has been shown that children inherit certain malaria antibodies from their mothers and that antibody responses increase in children with increased malaria experience (McGregor et al., 1965; Collins et al., 1977; Campbell et al., 1980). Previous studies using the IFAT have indicated a 'decay' in the antibody response and a subsequent increase in response which begins with exposure. The high levels of passive antibody acquisition from the mother via the placenta declines after birth which is rapidly followed by a rapid acquisition of active immunity in response to parasitaemic challenge.

Parasitologic examinations in this population have indicated that malaria infection is present in some infants before 1 month of age and that by the fifth month, about 50 percent of the infants had parasitaemia (Spencer et al., 1987a). This intense level of transmission is no doubt responsible for the very rapid increases in both the IFAT and the ELISA responses, with slight evidence of an early decrease in mean antibody responses. No evidence was found that presence

of patent parasitaemia in the mother nor her participation in the chloroquine prophylaxis programme either markedly increased or decreased the maternal antibody responses. These factors also apparently had only minimal effect on the acquisition of antibodies by the infants before delivery.

The serologic responses of the pregnant women at Saradidi showed a marked seasonal variation as indicated by the mean absorbance values and the high and very high ELISA responses. Thus, at Saradidi, pregnant women were most likely to have increased serologic titres starting in November, which peaked in December, and slowly declined during January to March. The period of lowest level serologic responses was August to October. These seasonal changes were not reflected by the presence or absence of detectable infections with P.falciparum.

The use of the two different serologic tests offered a chance to determine the test most applicable in determining maternal-infant antibody response levels and changes in the antibody responses of the infants with increase in age. The results were essentially the same with both tests. The only variation was between the antibody responses of infants born of mothers taking chloroquine for chemoprophylaxis.

The ELISA results indicated no significant difference whereas with the IFA test. infants of mothers taking prophylaxis had significantly ($P < .05$) lower geometric mean IFAT titres in samples collected before the age of 2 months. In addition, the infants appeared to show increases in their ELISA responses at an earlier age than in their IFA responses.

The ELISA has the advantages of being non-subjective in its reading and uses only one or two dilutions of the samples (very large numbers can be examined in a short time). The ELISA appeared to indicate increases in titres at an earlier age, whereas the IFAT was able to indicate a difference in the effect produced by chloroquine prophylaxis. The studies confirm the fact that the two tests may, in fact, be measuring similar but sometimes dissimilar antibodies. In

this situation, however, the IFAT appeared to offer few advantages over the ELISA. Measurement of the absorbance of a 1:100 serum dilution by the ELISA appears sufficient to identify positive responders, high-level responders and seasonal changes. This method could be used to quantitate longitudinal serological changes in the population.

Table 5.1 Proportion of pregnant women from Area A taking malaria chemoprophylaxis by age, Saradidi, 1983-1984

Age (years)	No. Women	% Taking chemoprophylaxis
15 - 29	271	25.1
30 - 44	82	43.9
Unknown	4	0
Total	357	29.1

Chi square = 10.7, df = 1, P <.005 (excludes those of unknown age)

Table 5.2 Relationship between parasitaemia, urine Dill-Glazko test for 4-aminoquinolines and antimalarial chemoprophylaxis in pregnant women, Saradidi, 1983-1984

Women on chemoprophylaxis	Number samples*	% with parasitaemia	No. urine samples	% Dill-Glazko test positive
Yes	265	17.7	255	15.7
No	1700	26.2	1656	8.3
	Chi square = 8.702		Chi square = 14.45	
	df = 1		df = 1	
	P <.005		P <.005	

* Number of samples, not number of pregnant women

Table 5.3 Relationship of antimalarial chemoprophylaxis and proportion of pregnant women with two or more parasitologic samples where all samples were negative for parasites, Saradidi, 1983-1984

	Number of women with two or more parasitologic samples	% of women with all samples negative
Chemoprophylaxis	79	69.6
No chemoprophylaxis	516	51.6

Chi square = 10.66, df = 1, P <.005

Table 5.4 Relationship between haemoglobin level and antimalarial chemoprophylaxis in pregnant women, Saradidi. 1983-1984

Haemoglobin level (g/ml)	Chemoprophylaxis Yes (% samples)	Chemoprophylaxis No (% samples)
5.0 - 7.4	0.8	6.0
7.5 - 9.9	40.2	44.3
10.0 - 13.5	59.1	49.7
Total	127	1111

Chi square = 6.730, df = 2, P <0.5

Table 5.5 Relationship between age of infant, presence of parasitaemia in infant and chemoprophylaxis status of mother Saradidi, 1983-1984

Mother on Chemoprophylaxis	Yes		No		Total	
Age of infant in months	Number of samples	% positive*	Number of samples	% positive	Number of samples	% positive
less than 1	6	0	37	2.7	43	2.3
1	26	7.7	131	13.0	157	12.1
2	24	20.8	146	24.7	170	24.1
3	27	37.0	132	37.9	159	37.7
4	27	44.4	108	50.9	135	49.6
5	26	38.5	95	53.7	121	50.4
6	13	38.5	67	55.2	80	52.5
7	17	41.2	47	53.2	64	50.0
8	9	44.4	38	55.3	47	53.2
9 or more	15	46.7	56	64.3	71	60.6
Total	190	32.6	857	38.4	1047	37.3

* Percent of thick blood films positive for malaria parasites.

Table 5.6 Parasitaemia in relation to antimalarial chemoprophylaxis by month of sample before delivery in 314 pregnant women whose infants were followed, Saradidi, 1983-1984

Months before delivery	Chemoprophylaxis No. of samples	% positive*	No Chemoprophylaxis No. of samples	% positive
0 - 2 +	115	17.4	447	26.4+
3 - 5 ++	33	21.2	113	25.7++
6 - 9	-	-	8	37.5
Total**	148	18.2	568	26.4

* Percent thick blood films positive for malaria parasites

** Chi square = 4.21, df = 1, P <.05. Percent of positive slides in pregnant women on chemoprophylaxis compared with those from women not taking prophylaxis

\+ Comparison of two groups at 0-2 months. Chi square = 4.01, P <.05

++ Comparison of two groups at 3-5 months. Chi square = 0.27, not significant

Table 5.7 Absorbance value distributions in the enzyme-linked immunosorbent assay (ELISA) and positive malaria parasitaemias in pregnant women, Saradidi

Month*	Total samples	Parasitaemia % positive	1:100 serum dilution Absorbance (O.D.) % <0.30	% >1.00	Mean	SD**	1:1000 serum dilution Absorbance (O.D.) % <0.30	%>1.00	Mean	SD
March	152	28.2	5.3	65.1	1.18	0.51	23.0	21.7	0.67	0.42
April	131	22.9	1.6	48.0	0.98	0.36	31.2	9.1	0.49	0.31
May	157	23.5	0.0	28.0	0.82	0.32	50.9	1.9	0.35	0.24
June	167	22.7	5.4	32.3	0.86	0.40	58.5	7.1	0.37	0.31
July	179	26.2	4.5	25.1	0.79	0.31	55.8	1.1	0.31	0.22
August	169	30.1	3.6	19.5	0.75	0.31	61.5	0.0	0.31	0.23
September	152	23.0	3.3	17.7	0.73	0.27	62.5	0.0	0.28	0.17
October	86	25.5	7.0	17.4	0.73	0.28	62.7	0.0	0.28	0.18
November	142	28.1	4.3	35.2	0.94	0.48	42.9	9.1	0.47	0.35
December	87	32.1	3.5	96.5	1.57	0.36	0.0	34.4	0.93	0.26
January	160	28.7	0.0	75.6	1.19	0.30	8.1	8.7	0.62	0.27
February	95	30.5	0.0	81.0	1.23	0.26	5.2	1.0	0.58	0.18
Totals	1677	26.5	3.1	42.4	0.96		40.5	7.1	0.45	

* March 1983-February 1984, ** Standard deviation

Table 5.8 Mean ELISA absorbance values of samples from pregnant women with or without Plasmodium falciparum infections and who did or did not participate in chemotherapy programme

	Maternal Chemoprophylaxis (+)			Maternal Chemoprophylaxis (-)		
	Mean	SD	N	Mean	SD	N
Parasitaemia* (+)						
ELISA 1:100	0.87	0.40	41	0.98	0.43	405
ELISA 1:1000	0.40	0.26	41	0.49	0.33	405
Parasitaemia (-)						
ELISA 1:100	0.94	0.40	191	0.94	9.42	1040
ELISA 1:1000	0.42	0.32	191	0.45	0.33	1040

* At time serum specimen collected

Table 5.9 Antibodies to Plasmodium falciparum in the enzyme-linked immunosorbent assay (ESLIA) by age of infant

Month of age	No. samples examined	% with absorbance greater than 0.3 at serum dilution 1:100	1:1000
less than 1	41	80.5	34.1
1	139	73.4	18.0
2	158	71.5	19.0
3	165	72.1	25.5
4	127	81.9	34.6
5	116	90.5	44.8
6	84	100	48.8
7	73	100	47.9
8	45	100	60.0
9 or more	74	100	59.5
Total	1025	83.8	34.8

Table 5.10 Changes in mean ELISA absorbance* and geometric mean IFAT** titre for paired samples from infants taken before two months of age and during succeding months.

Age of paired samples	Mean ELISA absorbance				Geometric mean IFAT			
	No.	Initial	Secondary	Diff.++	No.	Initial	Secondary	Diff.+++
1-2 months	51	0.41	0.46	+0.05	42	2.29	2.33	+0.04
2-3 months	89	0.37	0.47	+0.10	81	2.22	2.25	+0.03
2-4 months	72	0,.04	0.56	+0.15	69	2.21	2.24	+0.03
2-5 months	63	0.34	0.61	+0.27	65	2.13	2.31	+0.18
2-6 months	55	0.29	0.64	+0.35	52	1.93	2.41	+0.48
2-7 months	28	0.26	0.83	+0.57	32	1.96	2.52	+0.56
2-8 months	20	0.23	0.77	+0.54	18	1.73	2.53	+0.80

* Mean absorbance at 1:100 dilution enzyme-linked immunosorbent assay

** Indirect fluorescent antibody

\+ Age in months between paired samples. First sample collected less than 2 months after birth. Subsequent samples taken from 3 to 8 months after birth.

++ Mean difference between mean ELISA absorbance values of paired samples from infants taken less than 2 months after birth and subsequent samples taken later expressed as absorbance value.

+++ Geometric mean difference between IFA titres of paired samples

Table 5.11 Malaria parasitaemia in infants by month of sample, Saradidi,

Months*	Number examined	% positive
May	21	23.8
June	55	32.7
July	87	32.1
August	80	40.0
September	94	32.9
October	118	28.8
November	162	35.8
Decemember	131	47.3
January	132	43.1
February	118	38.1
March	36	41.6
	1034	37.2

* May 1983 - March 1984

Table 5.12 The effect of chloroquine prophylaxis or patent parasitaemia on mother-infant antibody relationship*

		ELISA**			IFA*** test		
		No.	Mother	Infant	No.	Mother	Infant
Chloroquine prophylaxis	Yes	30	0.94+	0.38	27	3.53++	2.30
	No	157	0.90	0.42	154	3.48	2.35
	p-value^{y}		0.64	0.45		0.45	0.04
Patent parasitaemia in mother	Yes	43	0.97	0.38	42	3.54	2.25
	No	144	0.89	0.43	139	3.47	2.31
	p-value		0.24	0.27		0.17	0.63

* Paired maternal-infant samples. Maternal samples collected within 2 months of parturition and infant samples collected less than 2 months after birth.

** Enzyme-linked immunosorbent assay

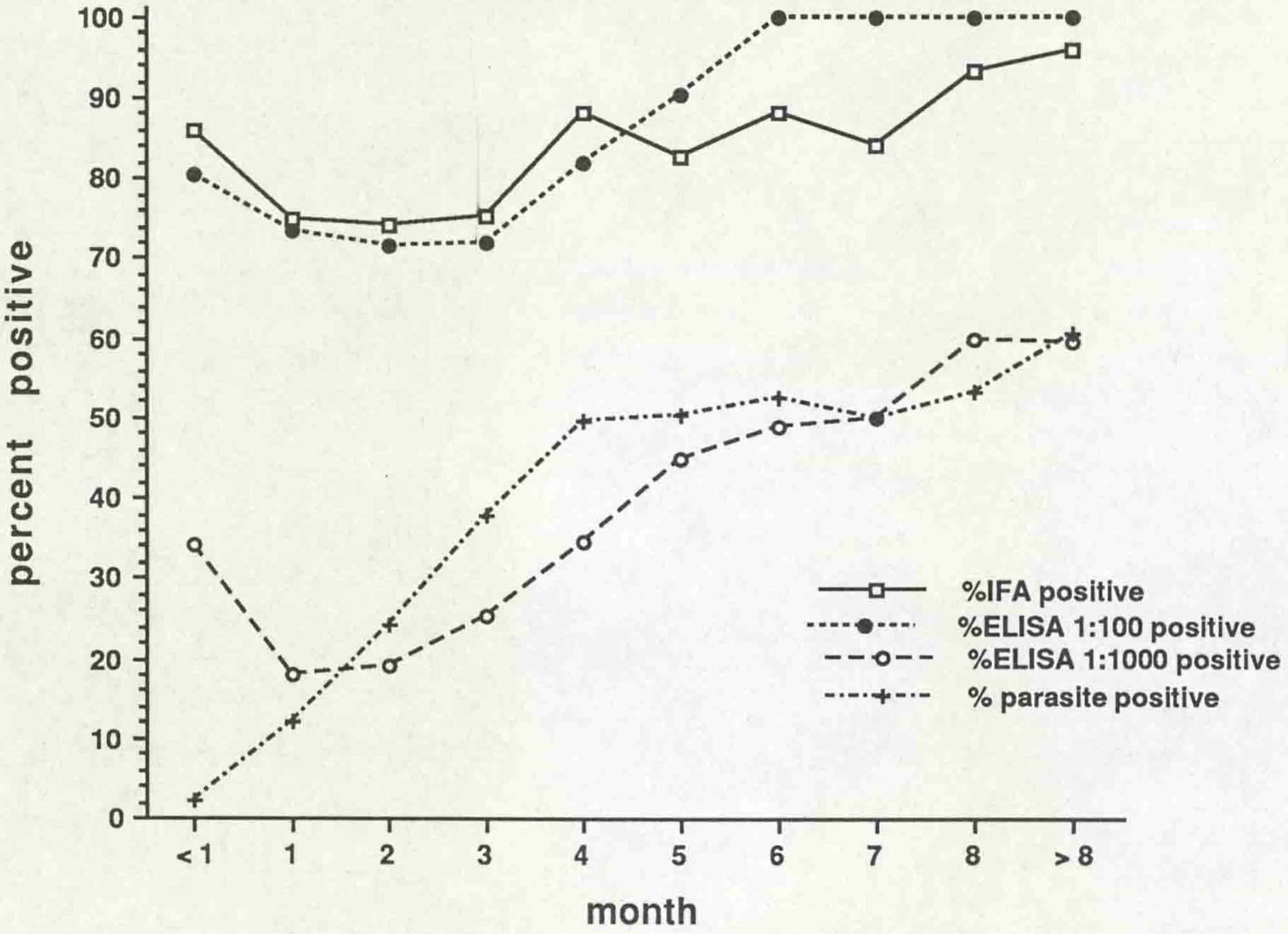
*** Indirect fluorecent antibody test

\+ Mean absorbance at 1:100 dilution

++ Geometric mean titre

y Student's test

Fig 5.1 Percent infants with parasites & positive to IFA and ELISA tests for P. falciparum antibodies



CHAPTER 6

POPULATION STRUCTURE, MORTALITY AND MORBIDITY TRENDS IN SARADIDI, 1980-1987

6.1 BACKGROUND

Saradidi is a rural area in Western Kenya near Lake Victoria, inhabited by the Luo tribe. The area is characterised by high infant mortality rates, a high rate of population growth, acute land shortage, and poverty (Central Bureau of Statistics, 1981; Kaseje & Spencer, 1987: Sindiga, 1985). As part of the health development programme, the people of Saradidi organised themselves into 3 areas (designated A, B and C) corresponding to the degree and length of community organisation. These areas were subdivided into villages based on geographical and administrative boundaries but also taking kinship ties and church affiliations into consideration. One or more Volunteer Community Health Workers (CHWs) were chosen and supported by each village (Kaseje & Spencer, 1987).

In May, 1982, a malaria control programme was initiated (Spencer et al., 1987). Chloroquine phosphate to treat malaria was made available in each village in Areas A and B. Treatment was provided by the CHWs. In area A CHWs also provided chloroquine chemoprophylaxis for pregnant women. Area C served as a control area; CHWs were selected and trained but they did not have antimalarial drugs.

The community decided that a census and registration of vital events would be important to them for planning and evaluation. It was also considered necessary to collect baseline information about the population in order to measure the impact of the intervention (providing chloroquine phosphate for malaria in each village). In addition, a census was carried out to determine if the demographic aspects of the three areas were comparable before the control programme began and to provide information for planning health services for

the community.

6.2 METHODS OF DATA COLLECTION

An attempt was made to establish a system of data collection on fertility morbidity and mortality at the family level. Each household had a form written in the local language on which they would record this information. In the case of mortality they needed to record date and cause of death, name, and sex of the dead, age at death.

For morbidity they were to record date, age, sex and the illness. This card was pinned behind the door in each household. The morbidity data presented here is drawn from these records for the year 1986 on 20% of households in the project area.

These data were improved by the regular visits to the households by a specialised and well trained team.

The teams obtained the information from the mother, father, spouse or any other adult and responsible member of the household. This information could be checked by looking into the family register but this was only useful for mortality data, and only for the deaths of those who were already in the family register. The visits were made at least every six months.

6.2.1 Population listing and count - 'census'

Between 1980 - 81, a team of 6 people who had received training visited each household in the project area, gave it a number and wrote down the names, sex and age of everybody who lived in the household. People who had not stayed at least 3 months were considered visitors. For women 15-49 information was obtained on the number of children ever born, children still living, date of last delivery and breastfeeding status. For all adults (15 years and above) education level, source of income and marital status were also obtained. Each individual was given an identification number which included:

- area number

- village number
- household number and
- personal number in the household.

A carbon copy of the list was left behind in the household. The reverse side of this list had space where mortality and morbidity experiences of the households were to be recorded.

The counting and listing provided information on the population structure which would also make it possible for the demographic trends to be followed up. It also provided a sampling frame for sample surveys.

6.2.2 'Census' Update - (Six monthly household surveillance)

Following the completion of the 'Census', the field team then visited each household once every six months to monitor change in the population. The team used both the carbon copy of the form in the household and the Community Health Workers register to enquire about changes. They obtained information on births, deaths and migrations.

a) Births

Date of birth, names, sex, number born, fate at birth, place of birth, person assisting the delivery, birth weight, name of mother, age of mother, parity, date of previous delivery were recorded and an identity number was given to the child.

b) Deaths

Name of the dead, date of birth, date of death, age and sex of the dead symptoms preceeding the death and the cause of death; (this information was obtained from an adult relative of the deceased) persons consulted, and place of death, were recorded.

c) Migrations

Name, sex, age, identity number, whether migrating in or out, reason for migrating. The data of the field team were validated by another independent

team of two senior technologists who randomly picked 10% of the households and gathered the same information for comparison.

6.2.3 Other sources of data

Baseline and Periodic sample surveys were carried out:-

a) Sample Surveys

Sample surveys were carried out beginning with the baseline one in 1979. In these surveys, information was gathered on a number of factors affecting the health status of the community in addition to morbidity and mortality.

b) Morbidity Surveys

Morbidity data were gathered by:

(i) asking whether a member of the household had been ill in the previous one month, who had suffered (age and sex) and what they had suffered from, and for how long they had been unable to do their normal activities, and what they had done about the illness;.

(ii) examining children under the age of five years;

(iii) collecting specimens to determine occurance of infections during the survey by laboratory examination on specimens.

A one year retrospective survey

This was carried out at baseline and follow up surveys on 10% of the households. Births in the households in the past one-year (12 months) and deaths by age and cause in the same period were recorded.

6.3 RESULTS

6.3.1 Baseline sample survey

A total of 1365 households were visited. There were 5767 people living in these households giving a mean household size of 4.2 persons per household. The age and sex distribution of the population is given on Table 6.1 and figure 6.1. 369 births occurred in the past one year in the project area, giving a

Crude Birth Rate (CBR) of 59 per 1000 mid-year population, 102 deaths were reported to have occurred in the past 12 months giving an annual Crude Death Rate (CDR) of 16.3 per 1000 mid year population. Infant Mortality Rate (IMR) was 181 per 1000 children born alive during the same period of time. Childhood mortality was 22.1 per thousand children aged 1-4 years.

Table 6.2 compares these figures with the National figures. Table 6.3 summarises the main causes of death in the area. Most deaths were due to diarrhoea and vomiting, acute respiratory infections and measles.

6.3.2 Census and Updates

a) Population structure

The population composition by age and sex at census is presented in Table 6.4. The population was 42,755; 17.1% of these were below age five, 47% was below 15 and 20% were women in the reproductive age. These proportions compare well with 20%, 52% and 22.5% respectively, from the baseline sample survey.

Under the age of 20 there were approximately equal numbers of males and females (Table 6.4, Figure 6.2). However, there were more females after the age of 20. The overall sex ratio was 86 males to 100 females. At census the 42,755 people made up 10,778 households giving a mean number of persons per household of 4.

The singulate mean age of marriage for men was 27 years and 19.9 years for women. In every five-year age group fewer women than men had never been married. Only 0.1% of women aged 50 to 59 had never been married. More than one third of women 50-59 were widowed. Men were significantly more likely than women to be married to more than one spouse and divorced and separated were higher among men. A significant fraction of men remained unmarried until the age of 40 (0.8%). The proportion of polygamous marriage was higher in older men.

Levels of formal education were higher for men at every age and the young people of both sexes had more years of education than older people. For those of age 60 years and above 73.1% of men and 96.1% of women had never attended school while only 2.7 of men and 18.5% of women aged 20-29 had never attended school.

(b) Fertility

The proportion of women without children was high, 8.6% of women 40 years and over had never had a live birth. The crude birth rate for the study was 40.5 per 1000 mid-year population. The mean number of children born by age of mother was 3.57. The total fertility was 6.2. The highest age specific fertility rate was 0.313 in women 20 to 24 years old the general fertility rate was 203.6 per thousand women 15-44 years.

Breast feeding and duration of amenorrhoea following pregnancy in women aged 15 to 44 were assessed in the census. The estimated median duration of breast feeding was 20 months and of amenorrhoea was 10 months. 73.5% of the women 15-44 years who had delivered 13 to 18 months earlier were still breast feeding and menstruation had returned in 71.4% of the same women.

c) Mortality Rates

The mortality rates reported here were measured by different methods. Table 7.5, 7.6 and 7.7 summarise the mortality rates in Saradidi by year of study, method of estimation, and age.

Although different methods of estimation have been used, the mortality decline in Saradidi over the last seven years is impressive.

The 1980 rate of IMR was based on an indirect estimation developed by Brass 1975 based on children ever born, and children now living; and calculated according to the method of United Nations (1983) and using regional life tables developed by Coale and Demeny (1966). This IMR was 155 per thousand live births was comparable to 181 which was the finding of the sample survey.

Project activities for intervention started in 1981 - 1982. According to the 'Census' and updates we saw a remarkable decline in infant mortality rate. Infant mortality is very sensitive both to material living conditions and to direct or indrect interventions. Making MCH services available and accessible to children under 5 years and pregnant women within the context of Primary Health Care (PHC) and with adequate community participation could explain this remarkable impact. The impact on infant mortality and peri-natal mortality is summaried on tables 6.8 and 6.9.

After 1984, free chloroquine for the treatment of malaria which was being provided by CHWs ceased but it continued to be provided at cost within easy reach of the community at the village level, as described in Chapter 2.

Throughout the years when updates were carried out, measles was always identified as among the leading killers of children under 5 years. In 1981 measles was responsible for 35.7% of 224 reported deaths in infants up to 12 months of age and 40.9% deaths in children one to four years. In 1986 measles was responsible for 34.5% of 306 deaths 1-4 years, figure 6.3 shows this relationship.

The major causes of death and their proportional contribution to total mortality are presented in Table 6.10.

The information in this section was obtained from answers to open-ended questions asked to the parent in the case of children, or other adult relatives of the deceased. The data obtained in this way may not have been reliable because, of the local specification of symptoms do not always coincide with recognised medical diagnoses, but it was the only way to obtain the information in the circumstances of the community programme.

The interviewers tended to give the responses their own interpretation which could have been often biased. Thus the recorded cause of death could be different from the real cause. This information was particularly difficult to gather as it was sensitive and emotional. People in Saradidi did not feel

comfortable talking about their dead. In this way much of the relevant information could have been consciously or unconsciously suppressed.

Often a death is caused by a multiple of conditions and the primary cause of death may be very difficult to discern. A chronic illness may be difficult to differentiate from the terminal illness which ends in death. It is also important to recognise that in communities like Saradidi, the majority of childhood deaths (excluding the perinatal and neonatal deaths) result from the cummulative burden of recurrent deases and malnutrition. Thus to assign one cause to a death of an infant or young child could be misleading. It is important to note that this finding was consistent, irrespective of the method of data collection used and inspite of the above mentioned problems

c) Morbidity (From forms kept in households)

Data on morbidity presented here were from the forms left in the households as described earlier. The findings were:

- low compliance and
- bias towards adult morbidity as data for children 1-4 were virtually non-existent.

The data obtained and presented in the chapter showed a peak rate of morbidity in the period July to September. The rise starts in the months of May and persists through September. The main rainy season (long rains) as presented in Chapter 3 starts in April and finishes in July. There is increased mosquito activity during this period which persists for some time, even after the rainy period until the surface water is substantially reduced (see figure 3.1).

Malaria begins to rise in April. Acute Respiratory Infection (ARI) rises in May and diarrhoea and vomiting shows more variation with peaks in May and September. However, there was little data for the months of February, November and December which makes it difficult to draw valid conclusions.

Community based data gathering attempted in Saradidi was considered an innovative method of collecting data on the total population with community participation. It proved unreliable because of low compliance. The quality of data was also probably compromised by the large number of people gathering and recording the information without adequate training and or supervision. Thus the recording may not have been adequately standardised apart from the record form itself. While this information can be useful for local purposes of planning with the community it may be inadequate for use beyond the programme for external comparisons.

d) Discernable Trends during the study period

The most obvious trend has been the reduction in mortality rates. The downward trend occurs in all age groups. At the start, the infant mortality rates were quite high as compared to the national average (Table 6.2). The mortality rates presented on Table 6.5, 6.6 and 6.7 show the downward trend although IMR for 1986 was higher than that of 1983-84. It was still much lower than the baseline rates. The PHC activities at Saradidi continued effectively even after the cessation of external financial inputs for research activities in 1984, and the community has been responsible for all the programme activities since then. Tables 6.8 and 6.9 present the infant and perinatal mortality rates by study area and study period with regard to intervention period. Measles was the leading cause of mortality in the area (See Table 6.10) and outbreaks occurred every two years, the first one during the study period occurred in 1982.

Another measles, outbreak occurred in 1986 but the reported measles mortality was less than in 1982. (Figure 6.3). It is possible that the programme interventions in terms of education and immunisation could have prevented the outbreak expected in 1984 and modified the 1986 outbreak.

6.4 DISCUSSION

6.4.1 Age and sex composition

The population pyramid and sex ratios observed in Saradidi are similar to others reported from tropical Africa and are similar to others reported from tropical Africa and Kenya (Bradley et al, 1982; Brass et al, 1968; Molineaux and Gramiccia, 1980; Sai, 1984; United Nations, 1979; van Ginnekin et al. 1984). Compared with many western countries the population was characterised by a high proportion of children under 15 years, a low proportion of adults 65 years and older a high proportion of women 15 to 44 years (Sai, 1984; Population Reference Bureau, 1983). As expected, the population structures of the 3 areas were remarkably similar.

The observed excess of females to males at all ages (except less than 1 year), particularly over age 20, and the differences from expected values in the age pyramid are probably due to differential male migration rates by age and sex. Many adult males migrate from Saradidi to seek salaried employment in urban areas but leave their families behind. Misreporting of age is a common problem to demographic surveys done in tropical Africa (Brass et al, 1968), but the vast differences in the proportion of males and females in the working age group cannot be attributed to differential age misreporting.

The results are supported by the 1969 and 1979 National Censuses in which the sex ratios from Siaya District were almost identical to the findings presented in this chapter (Ominde, 1975). In an analysis of migration patterns from the 1969 census, almost 10% of the population of the Nyanza Province were living in other provinces (Ominde 1975). The sex ratio in urban areas of Kenya is very high reflecting the net in-migration of males.

6.4.2 Marital status and education

There was no attempt to separate distinctions in marital status. Thus it is likely that multiple marital categories such as free union, consensual union,

customary marriage, religious and civil marriage were all included. As in most African cultures marriage has a definite meaning in Saradidi though difficult to define in general terms. The people easily distinguish a 'single' from a 'married' person.

Almost everyone in Saradidi gets married. When compared with females, males were less likely to be married in any age category but were more likely to have more than 1 spouse. The decreasing proportion of men with multiple spouses in younger age groups could be due to the fact that polygamy is decreasing or to the fact that men only take a second wife when they are older, and ready for a second one. Only 18.2% of women 40 to 49 years were widowed, divorced or separated. This low proportion is similar to those found in other tropical African societies where polygamy is high (Brass et al, 1968). This high rate of polygamy may be a contributing factor to the low sex ratio since many men may need to go elsewhere for wives as well as to the fact that a significant fraction of men remain unmarried until the age of 40. It is erroneous to calculate the number of married women per 100 married men because only men living in Saradidi were included.

The calculated singulate mean age of marriage for men and women were similar to those found in other areas of Africa (Brass et al, 1968). The method is affected by unreliable age reporting. In addition, it can only be used in a population where the proportions married at specific ages have not been appreciably affected by migration. In general, data from Africa displays marked under reporting of females in their teens and an over reporting of females over 20. Although there may have been unreliable age reporting in Saradidi, there is no evidence to suggest that migration has affected the proportion married in any age group.

In Saradidi, males have had more years of formal education than females. In both sexes the younger have had more education than those older. In Kenya today all children are required to complete 8 years of school. However,

these data suggest that the compulsory education requirement may be enforced more with males than with females at the present time. In 1983, Kenya and Mauritius were the only countries in the African region to have approached the 100 per cent school enrolment target (Sai. 1984). The 0.3% of women 40 to 49 years old who said they were students could have been attending training programmes sponsored by the Saradidi community development programme.

6.4.3 Childlessness

The observed rate of primary infertility observed was high but similar to other African countries (Belsey, 1976). This is consistent with a study of infertility in Kenya based on world fertility data which noted that there was a higher proportion of childlessness in the coastal areas both in eastern and western Kenya (Hebubm, 1979). This seemed to be correlated with polygamy and presumably is related to high rates of venereal disease.

6.4.4 Fertility

There was no significant differences by area in crude birth rates and general fertility rates. Crude birth rates were high although lower than the 45.7 reported from Machakos, Kenya (van Ginnekin & Muller, 1984) or the 53 recently reported for the whole of Kenya. The reported total fertility from the year of 6.2 is less than the values of about 8.1 previously reported from Kenya and less than those of 7.6 reported in the same district in 1969 and 1979 (Anker & Knowles, 1982); Central Bureau of Statistics, 1980). The number of children born during the past year was calculated from the census question which asked women 15 years of age and older the date of the last pregnancy. It is possible that there was under reporting of births occurring in the previous year either because the date of birth was misclassified or because the birth was not recorded (in some households this information was provided by someone other

than the mother). There is no reason to suspect that ferility in Saradidi has been decreasing significantly. The high rate of polygamy, the relatively high frequency of infertility and the prolonged period of breast-feeding and amenorrhoea observed probably contributed to fertility rates being somewhat lower than the national average.

6.4.5 Mortality

In the census, education level of the mother was a powerful determinant of reported mortality rates in children this confirming earlier studies in Kenya (Mosley, 1985). Data from Kenya suggest that child mortality differentiates between regions of Kenya can largely be explained by differences in maternal education and level of household poverty. The infant mortality rates estimated from the census were higher than those in Machakos of Kenya as a whole (van Ginneken et al., 1984; Heinin et al., 1979; Central Bureau of Statistics, 1980). The infant mortality rates calculated in this study were very similar to those found previously in Siaya District and emphasised that this district has one of the highest infant mortality rates in Kenya.

6.4.6 Malaria Mortality

There are three main ways of attempting to measure mortality due to a disease such as malaria; from clinical records and looking at recorded cause of death; from observing the rise in mortality during malaria epidemics and determining the fall in mortality when malaria is brought under control and by attempting a verbal autopsy following deaths in a study population.

The scarcity of postmortem series in Africa, means that what is usually viewed as the best possible source of accurate data is not useful for assessing malarial mortality in Africa. Clinical records of cause of death are equally unsatisfactory in most of Africa as most people die outside the hospitals. Add to this the scarcity of laboratory facilities in many peripheral care centres

which means that most diagnoses of malaria are not definitive then hospital data and even death certificates is a poor guide to malaria problems in the community. Nevertheless it is the case that malaria is the commonest cause of admission and of death in children under 5 years of age in most of tropical Africa. Perhaps the most informative picture for Kenya comes from comparing the mortality rates of the least and most malarious districts. The malarious districts have much higher mortality rates than the lower malarious districts (Kibet et al. 1981). The under two year mortality rate in the highly malarious districts was found to be consistently double that seen in less malarious districts even when the other factors like mothers education was taken into account.

The best African data on malarial mortality derive from local field reseach projects that have attempted to stop malaria transmissions, usually by means of residual insecticides combined with chemotherapy and chemoprophylaxis. Of particular interest are the Kisumu Fenitrothion Project 1973-1976, and the Garki Project, 1971 to 1974.

The Kisumu study in Western Kenya used Fenitrothion to achieve a 96% reduction in malaria transmission. This was accompanied by a fall in the infant mortality rate of 40%, from 157 to 93 per thousand. The malarial effect was greatest between 3 and 10 months of age (Payne et al., 1976).

The second study was in Garki area of northern Nigeria 1971-1973, with extremely intense seasonal malaria transmission. Intervention was by residual insecticide with propoxur, backed up by mass drug administration for part of the area, which had a very substantial effect in reducing transmission. Infants were not given drugs unless and until they were found to be infected (Russell et al., 1963 and Spencer et al., 1987). The most dramatic findings were a large fall in the infant mortality rate from 255 to 55 and 102. Comparable unprotected villages had an IMR greater by 80 and 90 during the two intervention years. The death rate of children aged 1-4 years was less than half that in

unprotected villages and so also was the crude death rate. Moreover, in the absence of protection there was a close seasonal parallel between the IMR and the rate of conversion of infants to parasite positivity, with the IMR about 10% of this rate of incidence measure. Under protection the IMR both fell and lost its seasonal peaks (Molineaux and Gramicia, 1980).

Our finding in Saradidi however was that community based chemotherapy and chemoprophylaxis for pregnant women did not seem to have an added effect on mortality as there was no significant difference between villages with and villages without community based chemotherapy.

Molineaux (1985) in an exceptionally careful and imaginative analysis of the data on malaria mortality, confronts two problems. The first is that much greater reduction in mortality is observed after malaria control operations than the fall in the number of deaths ascribed specifically to malaria would suggest. The reason for this has not been fully worked out. Even in the Kisumu project where malaria is holoendemic the crude death rate after insecticide spraying fell more than could be accounted for by infant and early childhood mortality decreases. Experimental work on interaction of other infections with malaria in laboratory animals is consistent with a synergistic effect on mortality under some circumstances. The scale of this has not been precisely assessed although the data available suggests its relevance.

A converse effect appears to prevail in two West African studies, where the removal or massive reduction in deaths from malaria had led to a much smaller fall than expected in infant and young child mortality. This was seen in Garki, Nigeria, where malaria control removed the seasonal peaks of malaria deaths but mortality remained high overall, and in the Gambia when a measles epidemic shifted the peak season of infant mortality from the malaria without massive effects on total mortality. The possible hypothesis to explain these results is that of competing risks: a certain number of children are postulated as likely to die anyway, possibly with low birth weights and for other ultimate

reasons, and the immediate cause of death may be malaria if present and some other infection if malaria is absent. Malaria, measles and diarrhoea and vomiting are sufficient causes of death and only one of them is necessary. Thus, deaths averted by malaria control are not equal in number to deaths due to malaria.

The studies are all compatible with heavy infant and childhood mortality in uncontrolled holoendemic malaria areas being greatly reduced by the control of the transmission of malaria while access to chemotherapy through CHWs alone does not seem sufficient to cause significant mortality decline.

In earlier studies, in Saradidi area Payne et al., 1976 appeared to show malaria as significant cause of mortality. This was supported only partly by our findings. The overall reduction in the mortality between the years reflected in the census and updates described by Spencer et al., 1987 was more likely due to the integrated approach to PHC intervention and not due to any single purpose vertical approach. Any vertical approach may not be effective in reducing mortality in a population like Saradidi in which competing mortality risks are numerous. A child that does not die of malaria in Saradidi may easily die of measles, diarrhoea and vomiting or malnutrition. Thus, intervening against any one of these may not save a child who was equally susceptible to the other infections. It is only where a specific disease has a high prevalence coupled with a high fatality rate among otherwise healthy people that a disease specific intervention could have any demographic impact. This has been demonstrated by Mosley, 1985.

In conclusion, the more accurate methods of data gathering for the measurement of mortality and morbidity are usually too expensive to be used routinely by non-research non-sentinel activity/areas. The community based data gathering, depending on members of the community to collect or record information may be too unreliable to justify any effort in compiling and analysing the data gathered.

The most reliable but economic way of data gathering is repeated surveys and using the appropriate questions and methods of analysis and interpretation of such data. For this to be useful, the questionnaires must be well designed and administered by a well trained and well supervised team of interviewers. There should be validity check-ups, instituted to ensure accuracy and minimise errors.

The most frequent errors are from the omission of deaths of the very young and of illnesses not considered important or life-threatening and also mis-statement of age and timing of the events.

Proxy measures of health status like the assessment of nutrition status of children under 5 years of age may be useful in providing indirect morbidity measures and can be used in impact evaluation. Anthropometric measures are not without problems, but they are cheap and relatively easy to carry out by unsophisticated staff in unsophisticated settings. These measures provide reasonable proxy measures not only for morbidity but also for mortality risk.

Well selected service statistics may be useful in indicating morbidity trends. Their usefulness is enhanced by rapid processing and feed-back to those who collect the information indicating clearly from the results how they are applied in their daily work.

Table 6.1 Age and sex distribution at Saradidi from baseline sample survey in 1979.

Age (Yrs)	Male	Female	Total
Under 1	174	221	368
1 - 4	372	457	799
5 - 9	443	484	927
10 - 14	437	476	913
15 - 19	303	339	642
20 - 24	277	333	610
25 - 29	201	243	441
30 - 34	160	172	326
35 - 39	73	124	197
40 - 44	79	78	157
45 - 49	67	84	151
50 - 54	43	61	104
55 - 59	11	15	26
60 - 64	11	17	28
65 +	13	24	28
Total	2664	3111	5717

Table 6.2 Vital rates at Saradidi from baseline sample survey (1979) as compared to national rates.

Rate	Saradidi	Kenya
Crude birth rate	59	55
Crude death rate	16.3	14
Infant mortality rate	181	80
Child mortality rate	22.1	13

Table 6.3 Main causes of death from baseline sample survey

Cause	Infants (< 1 Yr)		Children (1-4 yrs)	
	No.	%	No.	%
Diarrhoea and Vomiting	18	28.6	9	23.7
Measles	9	14.3	11	28.9
Malaria	3	4.8	2	5.3
ARI	12	19.0	3	7.9
Other	21	33.3	13	34.2
Total	63	100	38	100

Table 6.4 Population of Saradidi, Kenya by age and sex

Age in years	Males	Females	Total
Less than 1	795	790	1585
1 - 4	2806	2902	5708
5 - 9	3213	3402	6615
10 - 14	3080	3049	6129
15 - 19	2575	2421	4996
20 - 24	1466	1654	3120
25 - 29	868	1462	2330
30 - 34	736	1022	1758
35 - 39	558	914	1472
40 - 44	581	932	1513
45 - 49	548	961	1509
50 - 54	547	777	1324
55 - 59	520	705	1225
60 - 64	80	964	1444
65 or more	981	1046	2027
Total	19754	23001	42755

Obtained by census in 1980 - 1981

Table 6.5 Infant mortality rate by year and method of estimation at Saradidi.

Year	Level	Method
1979	181/1000	Annual retrospective sample survey
1980	155/1000	Indirect Estimation from Census
1982	73/1000	Direct estimation from 6 monthly updates
*1983-84	67/1000	Direct estimation from 6 monthly updates
1986	83/1000	Direct estimation from 6 monthly updates

* During the intervention with community based chemotherapy for everybody and chemoprophylaxis for pregnant women.

Table 6.6 Child (1-4) mortality rate by year of estimation based on 6 monthly updates of 'census' at Saradidi.

Year of estimation	Child mortality rate
1982	25.5/1000
* 1983 - 84	18.2/1000
1986	11.9/1000

*During the community based chemotherapy for all and chemoprophylaxis for pregnant woman.

Table 6.7 Crude death rate by year of estimation and method at Saradidi.

CRUDE DEATH RATE			
Year	Level		Method
1979	17.3/1000	-	Retrospective sample survey
1982	13.1/1000	-	Direct estimation from 6 monthly updates
*1983 - 84	12.3/1000	-	Direct estimation from 6 monthly updates

* Years of antimalarial intervention

Table 6.8 Infant mortality rate by area and period, Saradidi.

Area	Pre-intervention		During invervention		Post-intervention	
	No.	Rate*	No.	Rate*	No.	Rate*
A	128	168	104	115	115	99.7
B	79	160.6	52	91.9	66	106.3
C	101	158	78	103.9	96	110.1
All areas	308	162.5	234	105.5	277	104.6

* IMR = No. of deaths of children under the age of 1 year during the period per 1000 live births in the same period.

Table 6.9 Perinatal mortality rates* by area and period in Saradidi.

Area	+Pre-intervention		++During invervention		+++Post-intervention	
	No.	Rate	No.	Rate	No.	Rate
A	46	60.4	79	87.6	82	71.1
B	40	81.3	45	79.5	49	78.8
C	49	76.8	73	97.2	82	94.3
All areas	135	71.2	197	88.8	213	80.5

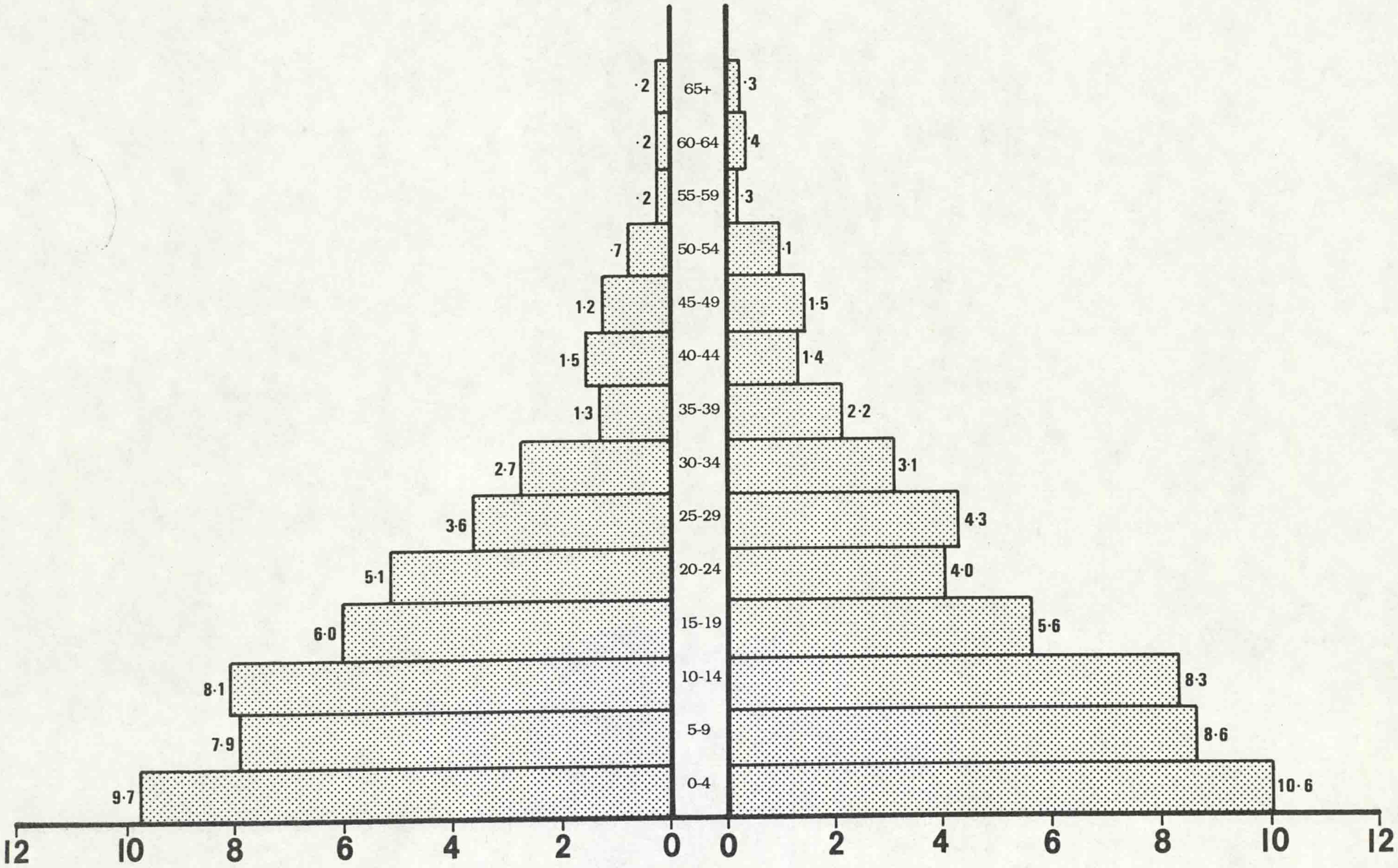
\+ Pre-intervention 1st May, 1981 to 30th April, 1982.
++ During intervention 1st September, 1982 to 31st August, 1983
+++ Post intervention 1st September, 1985 to 31st August, 1986.

Table 6.10 Causes of death in 1985 and 1986 as percent of total deaths in Saradidi.

CAUSES	% OF TOTAL DEATHS	
	1985 (N=306)	1986 (N=253)
	%	%
Malaria	13.6	6
Measles	37	34.5
Diarrhoea & Vomiting	14.6	10.6
Malnutrition	6.9	4.8
Anaemia	1.9	-
ARI	15.3	13.6
Unknown/unspecified	11	30.9

Figure 6.1

Saradidi Baseline cross section Demographic Survey (1979) Population Distribution By Age and Sex



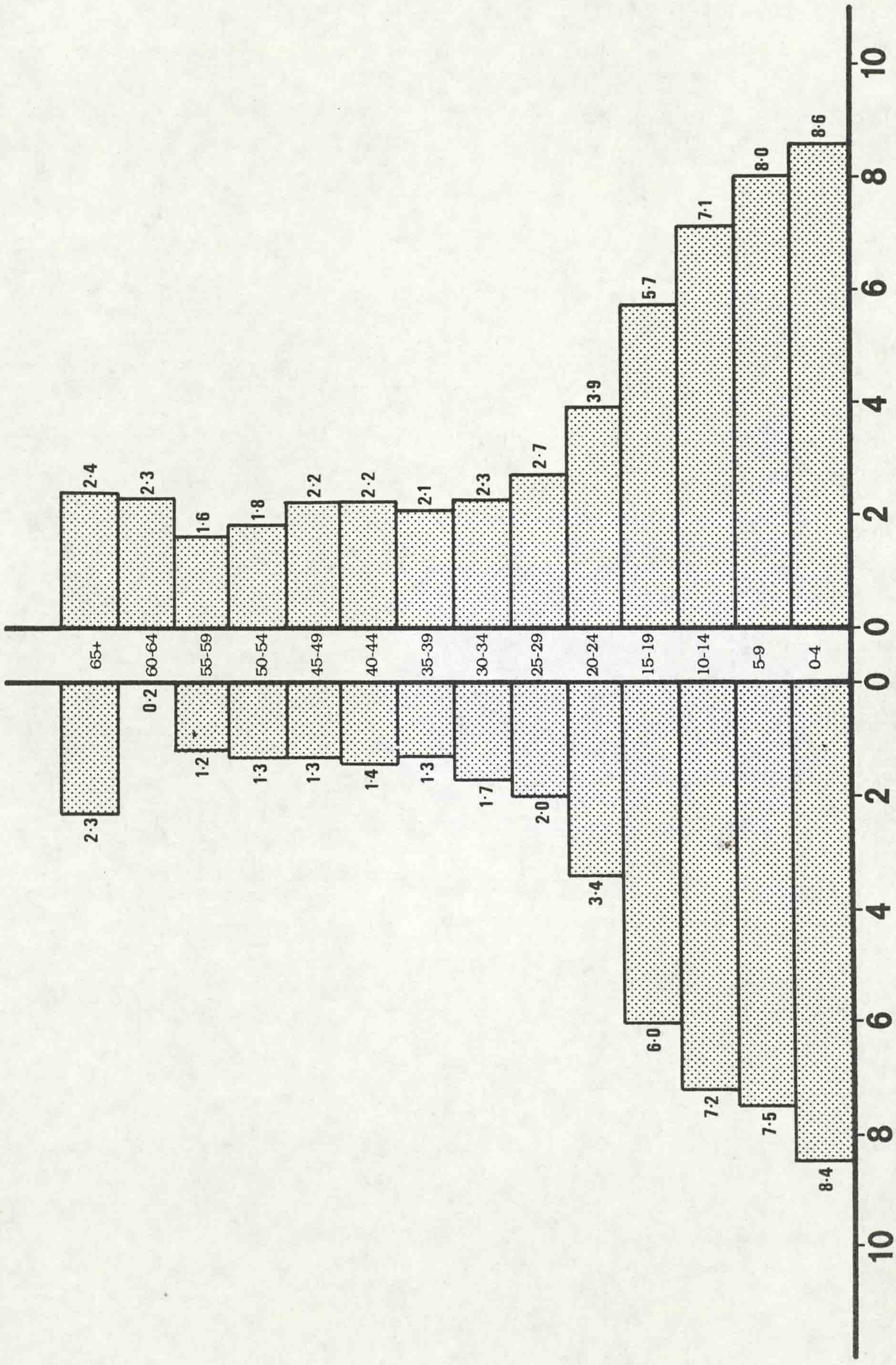
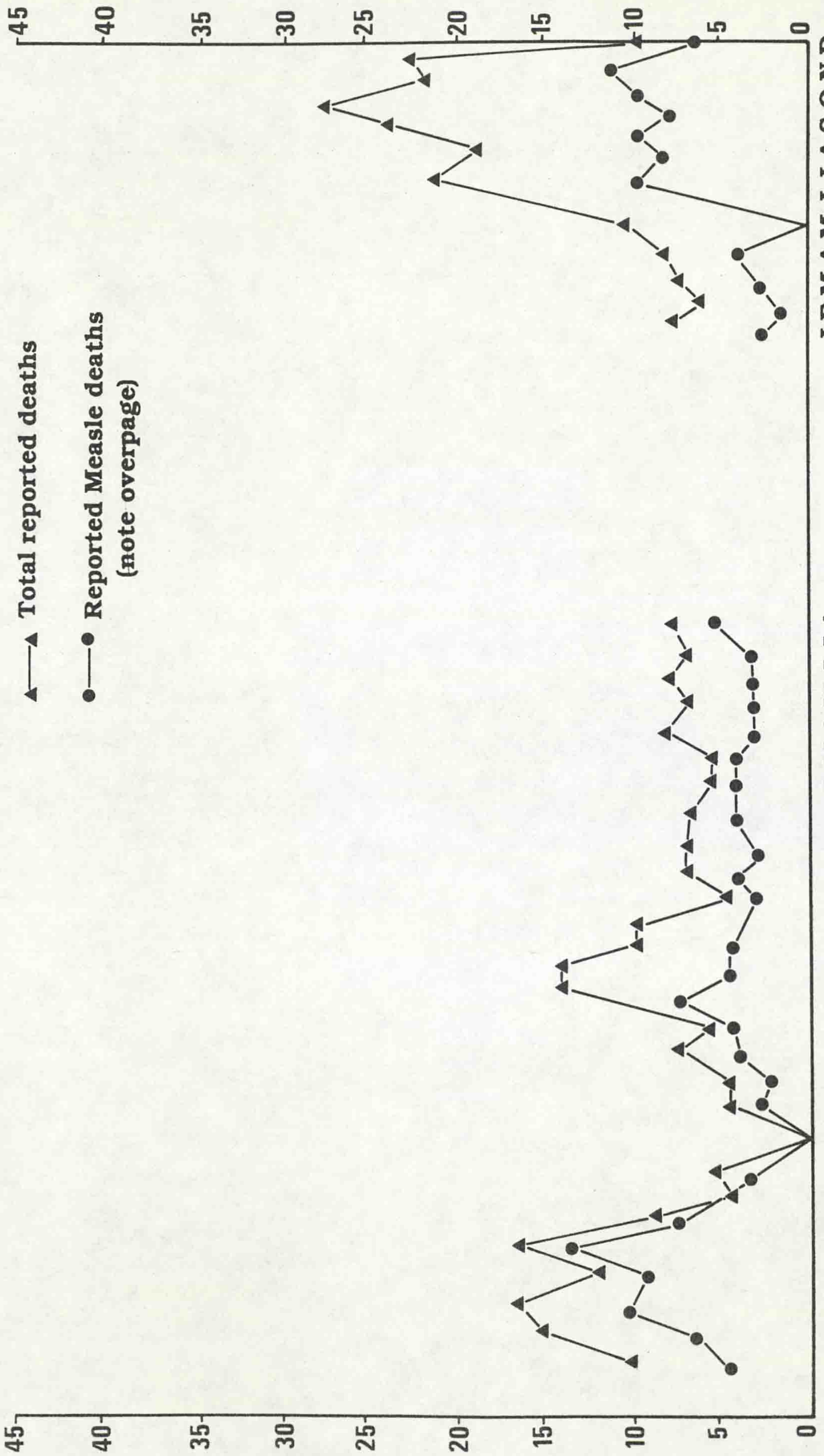

Population Distribution in Saradidi
by Age and Sex
65+ 2·4 2·3
60-64 2·3 0·2
55-59 1·6 1·2
50-54 1·8 1·3
45-49 2·2 1·3
40-44 2·2 1·4
35-39 2·1 1·3
30-34 2·3 1·7
25-29 2·7 2·0
20-24 3·9 3·4
15-19 5·7 6·0
10-14 7·1 7·2
5-9 8·0 7·5
0-4 8·6 8·4
0 2 4 6 8 10
Obtained by Census 1980-81

Figure 6.2

Figure 6.3 **Reported Total and Measles Deaths by Period - SARADIDI, KENYA**



CHAPTER 7

DEMOGRAPHIC, MALARIOMETRIC AND NUTRITION CROSS-SECTION SURVEY IN SARADIDI, 1985.

7.1 INTRODUCTION

Primary Health Care (PHC) aims at making essential health care universally available and accessible to the population served (World Health Organisation and United Nations Children's Fund, 1978). Accessibility in this sense implies that health care is geographically, financially, culturally and functionally within easy reach of the people. The Saradidi Rural Health and Development Programme (SRHDP) was conceived and developed by the people of Saradidi to bring health care and other services within their reach.

The research activities in Saradidi were designed, with the community respresentatives, to evaluate the effectiveness of PHC strategies in Saradidi as applied to malaria control. Some of the findings from the studies have been presented in Chapters 1-6. This chapter presents the results of a cross-sectional survey whose main objective was to find out whether the programme in general but the community based malaria control project in particular, was making any impact on the health status of the community.

Although measuring the impact of one component of a comprehensive multi-intervention programme like SRHDP is very difficult, the attempt was considered worthwhile for programme planning and for documenting lessons that could be shared with the national government and with others involved in similar programmes. The demographic data presented in Chapter 6 indicated encouraging mortality trends in Saradidi but the declining rates could not be attributed to the community based malaria chemotherapy and chemoprophylaxis, since a similar fall in rates was also observed in the control area.

The survey presented in this Chapter was an attempt to find out whether the health status of the children born during the programme who were living in Areas A and B which had community based chemotherapy with chloroquine

phosphate provided at the village level by Community Health Workers (CHWs) was better than the health status of similar children living in Area C, the control area.

Mortality levels, nutrition status, the rate of splenic enlargement, prevalence of parasitaemia and haematocrit levels were used as indicators of health status.

7.2 MATERIALS AND METHODS

7.2.1 The target group and indicators

The survey focused on children under the age of 5 years. These are the children born during the life of SRHDP. The following indicators were used in the survey:

a) Infant, early childhood (1-4 years) and crude mortality rates by a one-year retrospective survey.

b) Infant and early childhood mortality rates by indirect methods developed by Brass et al. (1968) and Brass (1975).

c) Malariometric indices as measured by parasite rates and the rate of spleen enlargement.

d) Haematological indices, only haematocrit was done.

e) Nutritional status of the children under the age of five years based on weight for age, weight for height and height for weight.

7.2.2 The sample

The cross-sectional survey covered 10% of the total population of Saradidi in such a way that all the three study Areas A, B and C were represented on the sample proportionately to their population sizes. Due to resources available for the survey, a cluster sampling technique was used. A total of 110 clusters were selected for the survey: 40 in Area A, 40 in Area C and 30 in Area B. Each cluster was to have an average of 14 households (see Table 7.1). Index households

were selected from the household listing compiled during the census and updated every six months. Having selected index households using random number tables, the index households were interviewed and then a cluster of households around each index household was interviewed by going to the next nearest household until the required cluster size of 14 households was reached.

7.2.3 Data collection

A structured questionnaire was used for interviewing (see Appendix) in addition to anthropometric measurements, and finger prick blood samples for malaria parasites and haematocrit estimation. The questionnaire was administered by trained laboratory technicians who were also given training for this survey in order to standardise their techniques and their approach. They were used to the area as they had worked there since 1980 and so they were aware of most of the common field problems.

The questionnaire and other data collection instruments were pre-tested and the results of the pre-test were discussed with the interviewers. No major problems came out from the pre-test. Data gathering took a period of 25 days.

7.3 RESULTS

7.3.1 The study population

A total of 6,625 people were included in the survey, 2,579 (38.9%) from Area A, 1,652 (24.9%) from Area B and 2,394 (36.1%) from Area C. The age and sex composition of the population from the three areas was the same. The distribution (Table 7.2 and Figure 7.1) was similar to that of the baseline survey population sample (Table 7.3 and Figure 6.1) and to that of the total population obtained by complete population listing described in Chapter 6 (Figure 6.2).

7.3.2 Vital rates by a one year retrospective survey

There were 339 births in the twelve months preceding the survey in the

population of 6,625. The Crude Birth Rate (CBR) was therefore 49.8 per thousand sample population, and was slightly lower than the baseline obtained in 1979, and the national rates (see Table 7.4 which presents the vital rates by year and study area).

The overall total fertility rate was 7.61. This rate was 8.2 for Area A, 7.3 for Area B and 8.0 for Area C.

There were a total of 116 deaths in the 12 months preceding the survey, of which 37 occurred in Area A, 31 in B and 48 in C. Table 7.5 presents the distribution of deaths by area and age.

The Crude Death Rate (CDR) was 17.5. A total of 39 infant deaths were recorded giving an Infant Mortality Rate (IMR) of 118.2 per thousand live births in the survey in the past 12 months. The IMR was still higher than the national average but was lower than the baseline rate (see Table 7.4 for the rate by study area). In Area A the IMR had dropped by 67.1 per thousand as compared to Areas B and C where it had dropped by 64.9 and 48.4 per thousand live births respectively. The IMR was also highest in Area C and lowest in Area A. This suggests a possible impact of the malaria control programme, since Area A was the longest exposed to the malaria control activities as discussed in Chapter 1.

Childhood Mortality Rate (CMR) had gone up substantially in 1985. This might be explained, at least partly, by the possibility of a measles outbreak. Table 7.6 on deaths by cause indicates that measles was responsible for 33% of all the deaths in the sample, and 48% of all childhood deaths. Measles outbreaks occurred in this community every two years. Since there had been an outbreak in 1982, another outbreak was expected in 1984. This may explain the high CMR figures as compared to 1979 which might have been a non-measles outbreak year.

Other major causes of death in Saradidi were diarrhoea and vomiting (12.2%), malaria (12.2%), ARI (10.4%), all of which are preventable by well known simple interventions. The data presented do not show any impact of the programme on the leading killer diseases in the area.

7.3.3 Early childhood mortality by the indirect methods of estimation

a) The indirect method

Questions on child survivorship were included on the survey questionnaires to obtain the number of children ever-born by sex, the number who had died by sex and hence the number that were living, also by sex. These questions were asked to each woman in the child bearing age. The information thus obtained was used for the calculation of the proportion of children ever-born who have died. This proportion was used to calculate the probability of death for the children born in Saradidi community from 1983-1984 and then to estimate the mortality rate.

This and other methods of indirect demographic estimates were developed and described by Brass, 1975; Feeney, 1976; Sullivan, 1975; Hill, 1980 and Trussel, 1982.

The data presented in this chapter were obtained through computer analysis using a programme developed for the estimation of mortality through the indirect methods, the computer programmes for demographic analysis, 1976. In theory the computation starts off with the calculation of the average parity of the women in the sample. This is done by dividing the total number of children ever borne with the total number of women in the sample. It is noted that parity is a measure of retrospective fertility (Brass, 1975). The second step is to calculate the proportion of children dead for each five year age group of the mothers. This is done by dividing the number of children reported dead in each age group of mothers by the number of children reported ever born in the same age group. This gives the proportion of children dead by each age group of mother.

The third step involves the calculation of multipliers which are requried to adjust the reported proportion of children dead by age group of mother as explained above. The multipliers are calculated from the ratios of the mean parity of one age group to the mean parity of the next age group and using a

table of coefficients for each age group which has already been established in the method. The probability of dying is then obtained by multiplying the obtained multiplier by the corresponding proportions of dead children by age groups of mother. A different coefficient is provided for each of the four families of model life tables in the Coale-Demeny system.

Finally, the period to which the resulting probability figure refers is calculated since it is an estimate of a number of years before the survey date. The reference year is obtained from the multiplier already calculated.

It is important to note that the estimated rate refers to two or more years prior to the actual survey. The mortality rates are therefore retrospective not current.

b) The mortality estimates

The infant mortality rate estimated by the indirect method for 1984 was 167 per thousand live births. This shows an increase from 1982 and 1983 when it was 105 and 93 per thousand live births respectively, using the North Coale-Demeny model (Table 7.7). The 1-4 childhood mortality rates for the same years were 138, 65 and 76 per 1000 children in that age group in the survey for 1984, 1983 and 1982 respectively (Table 7.8). Thus mortality rate in 1984 has increased considerably. The early childhood mortality rates were lowest in 1983 during community based chemotherapy and selective chemoprophylaxis.

Life expectancy at birth varies correspondingly to the mortality rates above; 38.8 years, 53.3 years and 50.7 years respectively (Table 7.9). This is lower than the Kenyan expectation of life at birth figure which was 54 at the time of this survey.

7.3.4 Nutritional status

1,366 children aged under five years were examined. All the five month age groups were well represented in the sample (Table 7.10). Each child had weight and height measured and recorded. The age was also noted as carefully

as possible, confirming the mother's response with the household's family record prepared during the census and updated every six months (see chatper 6) and also with the CHWs register.

There are three ways that are commonly used to assess the nutritional status of groups of children. The first of this is by using a classification based on a deficit in weight or height for age, originally proposed by Gomez and modified by Jelliffe (Bengoa, 1974; Gomez et al., 1956; Jelliffe, 1966). Thus the measurements of weight and height by age and sex are used to place an individual child being assessed, in the appropriate centile of weight for height, height for age and weight for age. Thus placement is either calculated or done according to graphs that have been developed for that purpose, examples being those prepared by the National Centre for Health Statistics/CDS reference population reported by Waterlaw et al. (1977). This method is more appropriate relatively to well nourished children with few children in the extremes of the distribution of measurements.

The second way is by comparing the measurements obtained to the median of the reference population and expressing them according to percentages of the median of that reference population. In this case the grades of deficit (mild, moderate and severe) are determined by establishing arbitrary cut off points.

The third way of assessing nutrition status, which was used in the analysis of data presented in this chapter, is based on standard deviation (z) scores, instead of centile distributions of height, weight and weight for height or percentage deviation from the median of the reference population. This method is particularly useful for populations where many children lie outside the extreme centiles of the reference population. The weight for height and height for age are expressed as multiples of the standard deviation of the reference population rather than percentages of the median. This allows a more precise analysis and presentation of the data and avoids the problem of arbitrary cut off points and groupings which may not always be appropriate.

a) Height for age

The height for age z score showed that the children were short for their age as seen in the negative z scores in Tables 7.10. This was found in all three areas of the study and there was no statistically significant difference in height for age scores by area of residence (Table 7.11).

The distribution of height for age z scores by age groups, showed a definite trend where the group 0-4 months showed the least difference from the expected international standard. The deviation from the standard increases with age and is greatest among the 10-24 months age group. Table 7.11 and Figure 7.4 present the mean z scores by 5 month age groups. It appears from these data that nutritional status of children in Saradidi is worse during the second year of life. The children tend to catch up from the third year upwards.

b) Weight for age

In general the children were also lighter than is expected for their age. The mean weight for age z score was -1.1943 $\pm$ 1.2533. Table 7.12 presents the mean z scores by area. There is a statistically significant difference in the mean z scores, Area A appearing to be better in weight for age z score than Areas B and C ($F = 4.43$, $df = 2$, $P < 0.05$).

Looking at the weight for age z scores by age groups it was observed that the trend was similar to that of height for age presented above. Table 7.13 and Figure 7.2 present the distribution of weight for age z scores by sex. The only age group with the expected weight for their age was 0-4 months whose weight for age was closest to the international standard. Generally, children under 12 months had better nutritional status than older children.

c) Weight for height

This is considered the most important index of nutritional status since, unlike weight for age and height for age, it is thought to be reasonably independent of age. In a retrospective survey one expects many errors in the statement of age particularly if the information is based solely on the mothers

memory. For this reason a greater emphasis is put on this index than on the first. The children were found to have normal weight for their height indicating that their nutritional deprivation indicated by height for age and weight for age were either chronic or genetically determined. The international standard may not be exactly the right standard for the children in the Saradidi community. On the other hand the observed stunting may be a result of prolonged underfeeding coupled with frequent infections or infestations. Area C children were generally worse off than the children from Areas A and B (Table 7.14) but the observed difference was not statistically significant ($F = 2.93$, $df = 2$, $P < 0.050$). The weight for height z scores also demonstrated the difference between age groups with the second year of life being the most affected (Table 7.15, Figure 7.3). The age group 0-9 months compared well with the international standard for their weight for height. The deviation from the normal was greatest in the group 20-29 months. Beyond 35 months weight for height improved again and continued to improve with age (see Table 7.15 and Figure 7.3).

Both the males and females start off with very good weight for height z scores in early infancy (0-9 months). After that age the males drop below the international standard and tend to remain so up to the end of early childhood (59 months) (Figure 7.5). The females remain above or about the international standard for most of the same period and only fall very slightly below the standard during the age 15-29 months (see Table 7.16 and Figure 7.6).

The nutrition status of the female children in Saradidi was bettter than for the male children throughout early childhood (0-59 months). The reasons for this might be found in the traditional child rearing practices and would have to be investigated.

7.4 SPLENOMEGALY, MALARIA PARASITES AND HAEMATOCRIT LEVELS

7.4.1 Splenomegaly

Of 1315 children examined for splenic enlargement, 428 (32.5%) had

enlarged spleens. There was marked difference between the study areas. Area A had the lowest spleen rate (28.8%) followed by Area C (34.3%), while area B had the highest rate (48.9%). The difference was statistically significant (X^2 = 73.2, df = 2, P <0.0001).

7.4.2 Malaria parasites

Malaria parasite prevalence rates were very high in all three areas. Of 1307 children examined for malaria parasites, 414 (81.8%) were positive in Area A, 266 (81.3% in Area B and 408 (86.4%) in Area C. In all 83.5% of the examined blood slides were positive. The difference between groups was not statistically significant.

7.4.3 Haematocrit levels

These were mostly normal as 91.8% of the samples were above 20% of packed cells. The cut off point was perhaps too low but the findings still show that less than 10% of the children were anaemic. There was very little difference in haematocrit level between the study areas (P >0.050).

7.5 DISCUSSION

The Saradidi Rural Health and Development Programme was initiated, planned, managed and evaluated by the community with necessary back up from resource professionals to ensure that reliable data were gathered for objective assessment of the project. The cross-section survey presented in this chapter was one of several attempts to assess the effectiveness of the programme in improving the health status of the community and particularly the most vulnerable members of the community, the children under the age of 5 in this case.

As explained in Chapter 1, the community was divided into three areas, A B and C, to try these different interventions to malaria control. After two years

of antimalaria activities there was no clear cut difference among the three study areas attributable to community based chemotherapy with or without chemoprophylaxis for pregnant women provided by CHWs in A and B respectively. The survey reported here was another attempt to find out whether the community based drug supply system had had any benefits to the villages who participated in it.

The indicators of measurement selected included mortality trends, nutritional status of children under the age of five years, malaria parasitaemia and splenomegaly rates in the same children.

The population structure of Saradidi was similar to that demonstrated by the baseline survey in 1979 and was similar to those reported by other workers in tropical Africa (Bradley et al., 1982a; Molineaux and Gramiccia, 1980; Sin, 1984). The population was characterised by a high proportion of children under 15 years, low proportion of adults 65 years and older and high proportion of women 15 to 44 years.

The crude birth rate was slightly lower than the national average (49.8 per 1000 survey population). The total fertility rate was still quite high (7.6).

The crude mortality rate was unacceptably high, 17.5 as compared to the Kenya national figure of 13.5 per thousand mid-year population. Infant mortality rate had dropped appreciably from 181 at baseline to 118 per thousand live births. The rate of mortality among children 1-4 was astounding. The CMR at follow-up survey was double the rate reported at baseline. The reason for this was difficult to pinpoint. The fact that 1984-85 was a measles outbreak year and the fact that measles accounted for nearly half of all the reported childhood deaths may offer a clue.

It is to be noted that the infant mortality dropped by 67.1 per thousand in area A as compared to a drop of 48.4 per thousand live births in area C. Thus, although the IMR was dropping in all three areas, the magnitude of drop was highest in area A suggesting an added impact that might be attributed to

community based supply of chloroquine for chemotherapy and chemoprophylaxis for pregnant women.

The little gain in IMR decline was insignificant when compared to the extraordinarily high rise in childhood mortality rate. This is of special concern considering the fact that the leading causes of mortality in Saradidi are all preventable. It would be difficult to justify PHC efforts such as those made in Saradidi if they would not have a significant impact on early childhood mortality through the prevention and control of preventable diseases and improving the nutrition status of the children.

Other findings in the same general area in Saradidi demonstrated the effectiveness of malaria control in reducing infant and childhood mortality (Payne et al., 1978). The difference between our intervention and theirs was lack of effective vector control in ours. It appears that vector control may be the key to mortality reduction contrary to what is suggested in WHO's Tactical Variants in Malaria Control (WHO, 1979). There is no study that has reported the effectiveness of chemotherapy alone in reducing mortality rates. Malaria still remains an important factor in the causation of mortality in holoendemic areas and should be tackled by the most appropriate methods within primary health care interventions. The findings reported in this and other chapters of this thesis would suggest that a single purpose antimalarial intervention can only work if it includes major integrated vector control (IVC) activities. Otherwise it must be coupled with the control of other endemic diseases like diarrhoea and vomiting, measles and acute respiratory infections.

The unusually high CMR reported in this chapter could also have been a result of an error in the study. It is still strange that only CMR was out of the expected range. Both IMR and CDR were within an acceptable range. It is the high CMR that also contributed to a rather high CDR.

Various studies have demonstrated the inadequacy of retrospective surveys and national census enumerations in producing reliable demographic

information (VanGinneken, 1984b). It has also been shown that multiround surveys can only produce reliable data if they are very well supervised. This is also true of a continuous surveillance system in which each household is visited fortnightly by a trained team to record demographic events. These highly imposing and expensive methods of gathering and processing massive amounts of data may be possible only in very few circumstances and yet they are also not without problems and deficiencies.

The methods are too expensive and hence unaffordable except as research projects; they may be quite inconveniencing to the study communities and may in themselves influence the rate of vital events that they are set up to measure. Thus one ends up measuring the results of demographic research interventions rather than measuring the demographic trends as they really are.

Due to limited resources available, the methods used in our system must not only be reliable but also sustainable, replicable and usable in informing decision making processes for more efficient delivery of services to communities in accordance with needs.

Continuous surveillance is reliable but can only be done as research on very small populations like Malumfashi (Bradley and Pugh, 1976), Danfa project in Ghana (Kpedekp et al., 1975) and Machakos project in Kenya (van Ginneken, 1984). For larger populations this method is impossible. In Saradidi two methods were used:

1) Repeated demographic updates following an initial complete population listing of the whole community in 1980-1982.

2) Cross-section retrospective surveys on 10-15% samples drawn from the total population every 3 years, as the one reported in this chapter and the initial baseline survey in 1979.

The purpose for which demographic data is being collected determines the degree of reliability needed. This should determine the method employed and hence how much of the resources available could be directed to demographic information gathering.

For many years the assessment of nutrition status of groups of children has been done using a classification based on a deficit in weight for age originally proposed by Gomez and later modified by Jellife (Gomez et al., 1956; Jelliffe, 1966; Bengoa, 1974; Waterlow, 1977). The need to distinguish between chronic and acute, or present and past malnutrition was highlighted by the WHO/FAO Expert Committee on Nutrition (WHO/FAO, 1971). Several authors have suggested methods for the classification of nutrition status, based on weight and height, which would bring out the distinction between chronic and acute malnutrition in a group of children assessed (Seoane and Latham, 1971; McLaren, 1972; Waterlow and Rutishauser, 1974). In the cross-sectional nutrition survey reported in this chapter, height for age and weight for height have been used as primary indictors for nutritional status of Saradidi children in accordance with the recommendation of FAO/UNICEF/WHO Expert Committee on Nutrition Surveillance (WHO, 1976).

The findings based on these two indicators were essentially consistent with each other. The height for age z scores showed the children to be below the expected height standard for their age. This indicated that the children being assessed were either suffering chronic malnutrition or had suffered malnutrition in the past. This indicator also reveals the age when the height deficit became most obvious. The children seemed to grow well while only breast feeding (0-4 months) but to slow down as soon as weaning started after the fourth month.

Unfortunately, the onset of weaning also coincides with the period in the infants life when the levels of maternal antibodies are no longer adequate to protect the infant from various infections. Thus a combination of poor weaning and increasing burden of disease expresses itself in height for age deficit. The growth deficit tended to be consistently worse for male children, a finding that could not be adequately explained from the available data.

The weight for height indicator confirmed that the children tended to be the expected weight for their height. They were not suffering acute

malnutrition. The slight weight deficit experienced was among the male children.

The weight for age z score values confirmed the finding that nutritional status worsens in the second year of life but improves in the third and later years. It also agreed with the finding that the nutrition status of male children was poorer than that for female children.

The use of weight for height for nutritional assessment has the distinct advantage that it is independent of age and so it is of even greater value when assessing children whose ages may not be acurately known (Kpedekpo, 1971; Habicht, 1974; Wray, 1975). The children assessed in this survey had reasonably accurate ages since they were born during the life of the programme and were therefore included on the family and CHWs registers soon after birth. The survey team always checked these registers to confirm the age and hence the height for age and weight for age findings in the survey could be taken as reliable.

The spleen rate was lower in Areas A and C. The expected prevalence of at least 50% in children living in holoendemic areas (Roberts, 1974). Malaria parasite rates were very high in all the three areas and so was the proportion of children with normal haematocrit values.

In conclusion this survey failed to demonstrate that community based chemotherapy and chemoprophylaxis for pregnant women had any added beneficial effect to other community based general PHC activities detectable by the methods and indictors used in the survey. It was possible that IMR reduction in Area A could be attributed to the availability and accessibility of chloroquine at the village level. If this was so then CMR should also have responded. The use of chemotherapy and limited chemoprophylaxis for malaria control appears to be inadequate in bringing about a measurable impact in the health status of the recipient community.

Table 7.1 The number of individuals in the survey by households, clusters and study area in Saradidi, 1985

Study area	A	B	C
No. of clusters	40	30	40
No. of households (14 per cluster)	616	401	598
No. of individuals	2579	1652	2394

Table 7.2 Population structure of the sample by age and sex in Saradidi, 1985

Age	Male		Female			
	No.	%	No.	%	% Female	Total
0-4 years	600	9.1	646	9.8	49.5	1306
5-9 years	555	8.4	535	8.1	49.1	1090
10-14 years	515	7.8	504	7.6	49.5	1019
15-19 years	328	5.0	301	4.5	47.9	629
20-24 years	177	2.7	244	3.4	58.0	421
25-29 years	95	1.4	212	3.2	69.1	307
30-34 years	94	1.4	214	3.2	69.5	308
35-39 years	94	1.4	153	2.3	61.9	247
40-44 years	79	1.2	148	2.2	65.2	1039
Unknown age and sex						32
TOTAL	3013		3580		54.3	6625

Table 7.3 Age and sex distribution by area (Baseline survey, 1979)

Age Group (years)	M	F	Total	M	F	Total	M		F		Total	
							No	%	No	%	No.	%
Under 1	98	130	200	77	91	168	87	3.1	92	3.3	179	6.4
1-4	209	234	443	163	193	356	186	6.6	204	7.3	390	13.9
5-9	257	293	550	186	191	377	221	7.9	242	8.6	463	16.5
10-14	293	302	595	144	174	318	228	8.1	233	8.3	461	16.4
15-19	174	183	357	129	156	285	169	6.0	156	5.6	325	11.6
20-24	184	201	385	93	132	225	144	5.1	111	4.0	255	9.1
25-29	113	137	250	88	103	191	101	3.6	120	4.3	221	7.9
30-34	71	81	152	83	91	174	77	2.7	86	3.1	163	5.8
35-39	34	50	84	39	74	113	36	1.3	62	2.2	98	3.5
40-44	42	36	78	37	423	79	42	1.5	39	1.4	81	2.9
45-49	40	43	83	27	41	68	34	1.2	42	1.5	76	2.7
50-54	23	28	51	20	33	53	21	0.7	29	1.0	50	1.8
55-59	8	10	18	3	5	8	5	0.2	8	0.3	13	0.5
60-64	7	12	19	4	5	9	6	0.2	11	0.4	17	0.6
65+	8	11	19	5	4	9	7	0.2	8	0.3	15	0.5
Not stated	13	15	28	5	9	14	9		14		23	
TOTAL	1573	1739	3312	1104	1344	2448	1373		1457		2830	

Table 7.4 Vital rates by area and year of survey as compared to the current national rates

Vital rate	STUDY AREA/YEAR								
	A		B		C		ALL AREAS		Kenya
	1979	1985	1979	1985	1979	1985	1979	1985	1985
Crude birth rate*	56.2	56.2	58.4	46.6	55.3	45.1	56.6	49.8	55
Crude death rate**	16.3	14.3	19.7	18.8	17.4	20.1	17.8	17.5	14
Infant mortality rate***	177.4	110.3	181.8	116.9	178.0	129.6	179.1	118.2	80
Child mortality rate****	22.1	26.9	25.3	29.1	20.1	46.6	22.5	34.2	13

* Calculated as number of births in the survey per thousand survey population

** Calculated as number of deaths in the survey per thousand survey population

*** Calculated as the number of deaths in the survey under the age of one year per thousand live births in the survey

**** Calculated as the number of deaths of children aged 1-4 years per thousand population of children 1-4 in the survey including the dead children

Table 7.5 The number of deaths in the survey population by age and area, in Saradidi, 1985

Age (years)	AREA A	B	C	Total No.	%
<1	16	9	14	39	33.6
1-4	13	9	18	40	34.5
>4	8	13	16	37	31.9
Total	37	31	48	116	100

Table 7.6 Reported causes of death in Saradidi by area, 1985

CAUSE	AREA A No.	%	B No.	%	C No.	%	ALL AREAS No.	%
Measles	13	36.1	8	25.8	17	35.4	38	33
Diarrhoea and vomiting	5	13.9	5	16.1	4	8.3	14	12.2
Acute respiratory infections	5	13.9	5	16.1	2	4.2	12	10.4
Malaria	3	8.1	7	22.6	4	8.3	14	12.2
Unknown and others	10	27.8	6	19.4	21	43.8	37	32.2
Total	36	100	31	100	48	100	115	100

Table 7.7 Infant mortality rate estimated indirectly by area and reference date, Saradidi, 1985

Reference Date	AREA A	B	C	ALL AREAS
1979	153	136	89	126
1981	143	114	89	105.3
1983	105	77	108	93.7
1984	144	118	246	167.3

Table 7.8 No of deaths per thousand children 1-4 years in the reference population, Saradidi, 1985

Reference Date	AREA/CHILD MORTALITY RATE A	B	C	ALL AREAS
1978	90	108	61	86.3
1981	85	85	61	76
1983	62	49	78	65
1984	114	90	213	138

Table 7.9 Life expectancy at birth by Area and reference date

Reference Date	AREA/LIFE EXPECTANCY IN YEARS A	B	C	ALL AREAS
1978	47.8	46.9	55.3	50.0
1981	48.7	51.5	56.0	50.7
1983	53.9	59.5	53.9	53.3
1984	43.1	53.6	37.2	38.8

Table 7.10 Nutrition status based on height for age z score by age in Saradidi, 1985

Age group (months)	No.	Mean score	S.D.
0-4	92	-1.33	1.78
5-9	119	-1.88	1.50
10-14	125	-2.38	1.92
15-19	112	-2.11	1.86
20-24	106	-2.52	1.47
25-29	106	-1.89	1.67
30-34	103	-1.97	1.72
35-39	140	-1.82	1.57
40-44	93	-1.99	1.58
45-49	125	-1.89	1.42
50-54	78	-1.99	1.27
55-59	82	-1.80	1.37
All children	1281	-1.97	1.78

Table 7.11 Nutrition status based on height for age z score by area in Saradidi, 1985.

Area	No.	Mean score	S.D.
A	500	-1.93	1.67
B	322	-2.03	1.56
C	459	-1.98	1.64
All areas	1281	-1.97	1.63

Table 7.12 Nutrition status based on weight for age z score by area in Saradidi, 1985

Area	No.	Mean z score	S.D.
A	499	-1.07	1.31
B	323	-1.24	1.19
C	460	-1.30	1.23
All areas	1282	-1.19	1.25

Table 7.13 Nutrition status based on weight for age z score by age in Saradidi, 1985

Age group (months)	No.	Mean score	S.D.
0-4	92	0.24	1.44
5-9	119	-1.04	1.21
10-24	126	-1.83	1.27
15-19	113	-1.40	1.25
20-24	106	-1.60	1.18
25-29	106	-1.42	1.15
30-34	103	-1.23	1.17
35-39	140	-1.12	1.19
40-44	92	-1.24	1.12
45-49	124	-1.32	1.03
50-54	78	-1.19	.850
55-59	83	-1.07	.94
All children	1282	-1.19	1.25

Table 7.14 Nutrition status of children in Saradidi based on weight for height z score by area, 1985

Area	No.	Mean z score	S.D.
A	492	.19	1.42
B	320	.15	1.16
C	456	.02	1.27
All areas	1268	0.08	1.30

Table 7.15 Nutrition status of children in Saradidi based on weight for height z score by age groups, 1985

Age group (months)	No.	Mean score	S.D.
0-4	83	1.44	1.51
5-9	119	0.60	1.49
10-14	124	-0.09	1.71
15-19	112	-0.10	1.37
20-24	106	-0.30	1.37
25-29	106	-0.33	0.94
30-34	103	-0.06	0.99
35-39	139	-0.13	1.00
40-44	92	-0.06	1.20
45-49	124	0.05	1.09
50-54	78	0.04	0.85
55-59	82	0.02	0.90
All children	1268	0.078	1.30

Table 7.16 Nutrition status of children in Saradidi based on weight for height z scores by age and sex, 1985.

Age (months)	SEX/Z SCORES FEMALE No.	Mean score	S.D.	MALE No.	Mean score	S.D.	MALE AND FEMALE No.	Mean	S.D.
0-4	41	1.82	1.50	42	1.08	1.45	83	1.44	1.51
5-9	57	0.69	1.66	62	0.52	1.32	119	0.60	1.49
10-14	66	0.05	1.36	58	-0.25	2.03		-0.09	1.71
15-19	53	-0.11	1.21	59	-9.95	1.42	112	-0.08	1.32
20-24	37	-0.18	1.15	69	-0.36	1.48	106	-0.30	1.37
25-29	55	-0.28	0.88	51	-0.39	1.01	106	-0.33	0.94
30-34	59	0.04	0.96	44	-0.20	1.02	103	-0.06	0.99
35-39	65	0.01	0.93	74	-0.04	1.06	139	-0.01	1.00
40-44	36	0.32	0.99	56	-0.31	1.27	92	-0.06	1.20
45-49	68	0.11	1.13	56	-0.03	1.04	124	0.05	1.09
50-54	39	-0.13	0.81	39	0.21	0.88	78	0.04	0.86
55-59	43	0.18	0.97	39	0.16	0.79	82	0.02	0.90

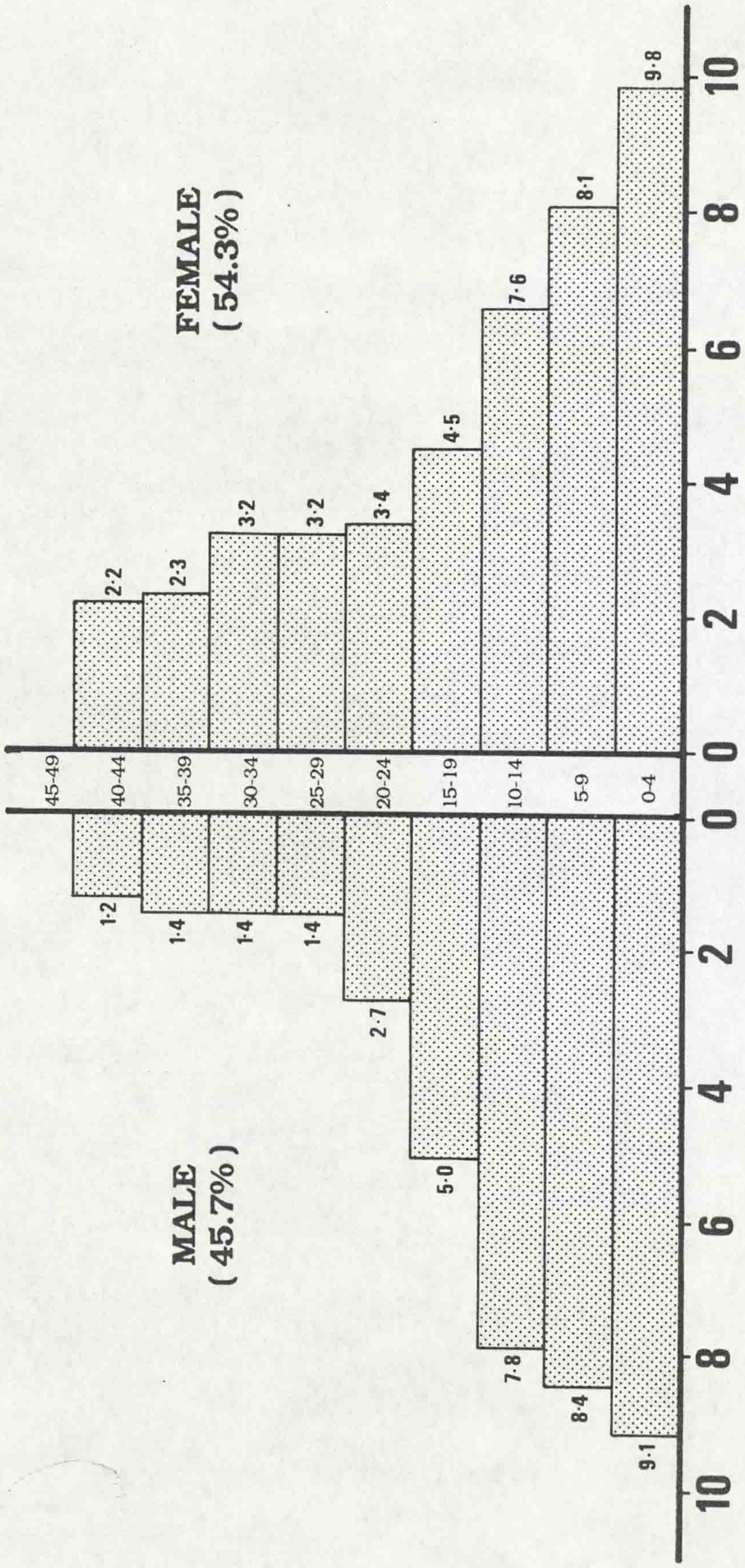


Fig. 7.1 **Saradidi Follow- up cross- section Demographic Survey, (1985) Population Distribution by Age and Sex.**

Fig 7.2 Saradidi Weight for Age by sex

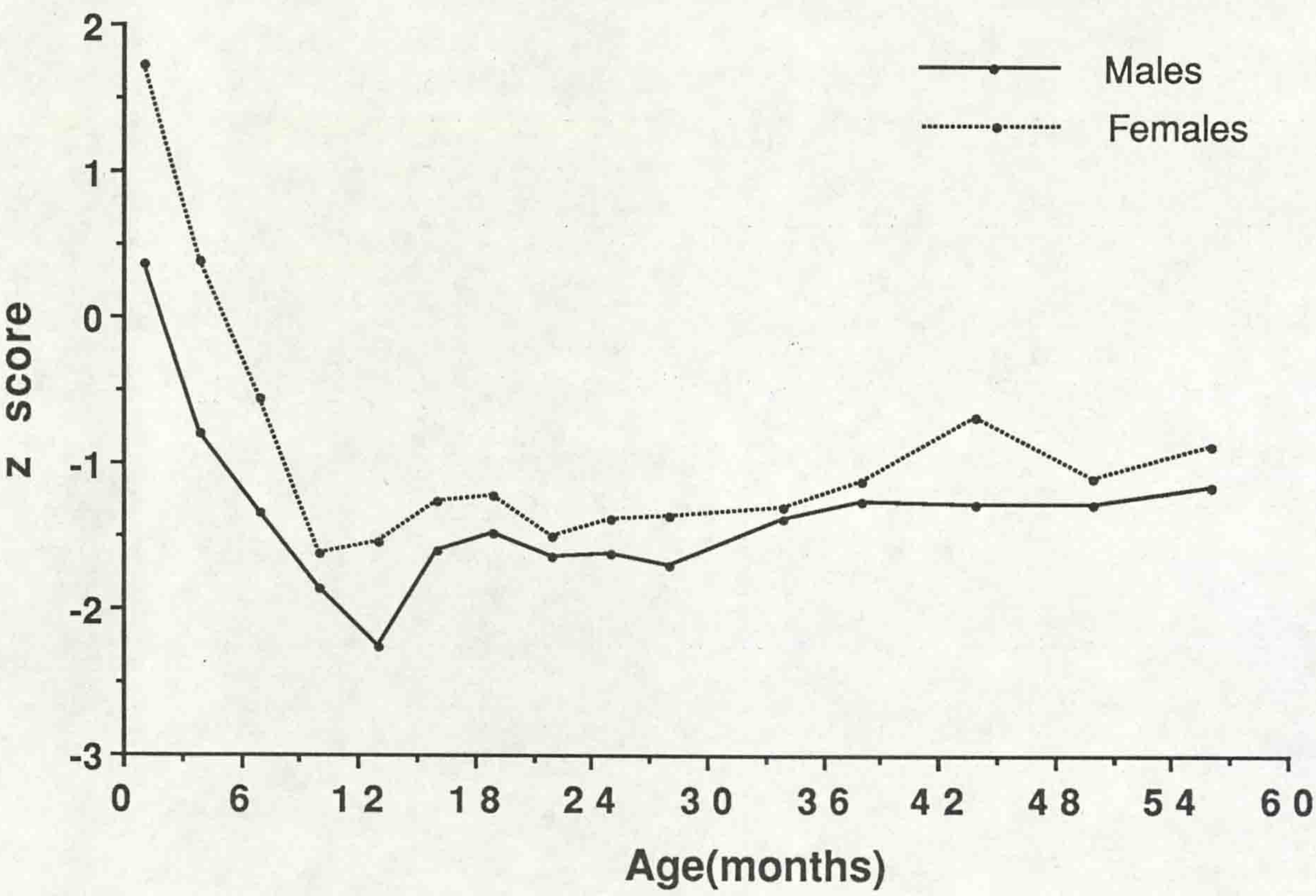


Fig 7.3 Saradidi Weight for Height by sex

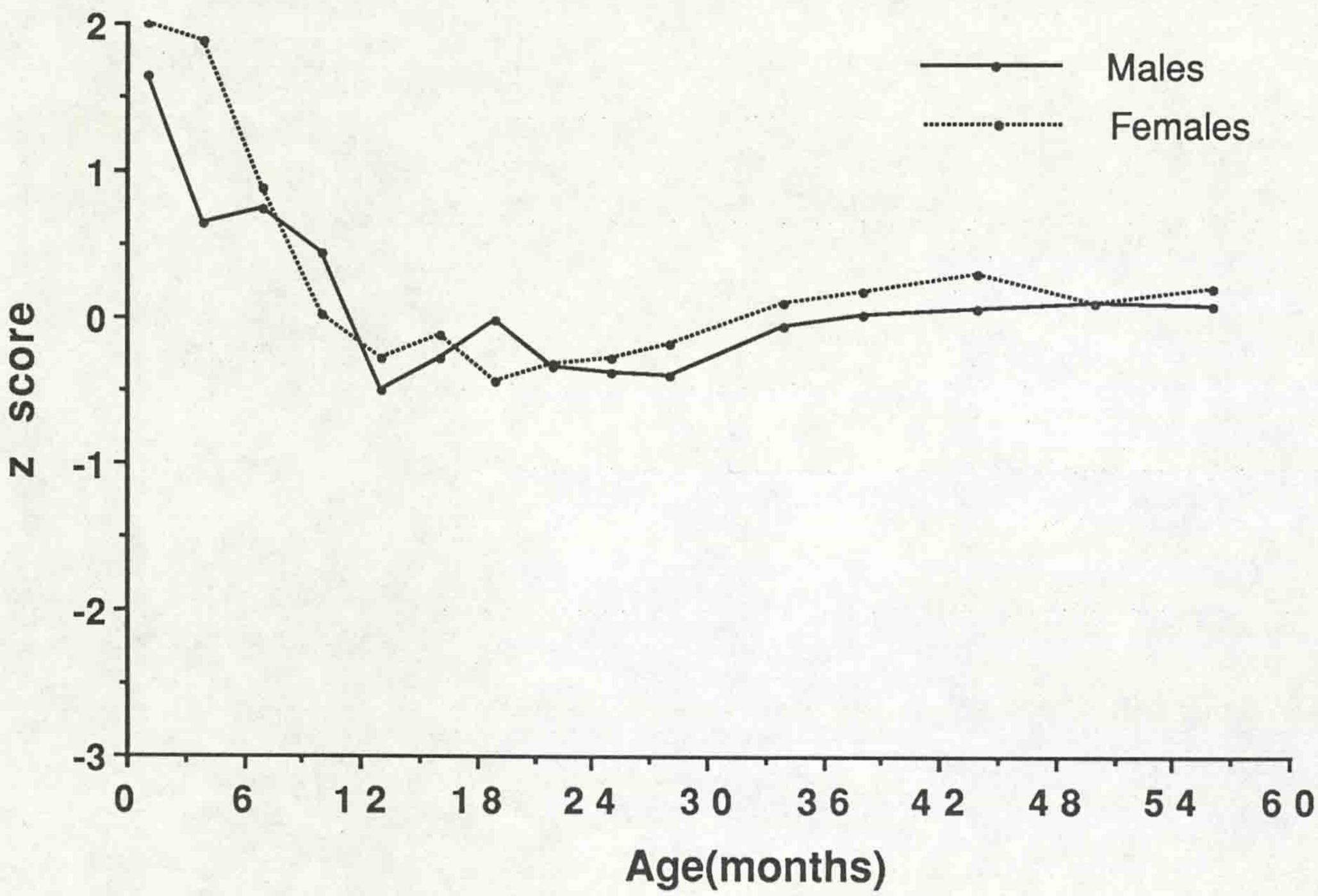


Fig 7.4 Saradidi Height for Age by sex

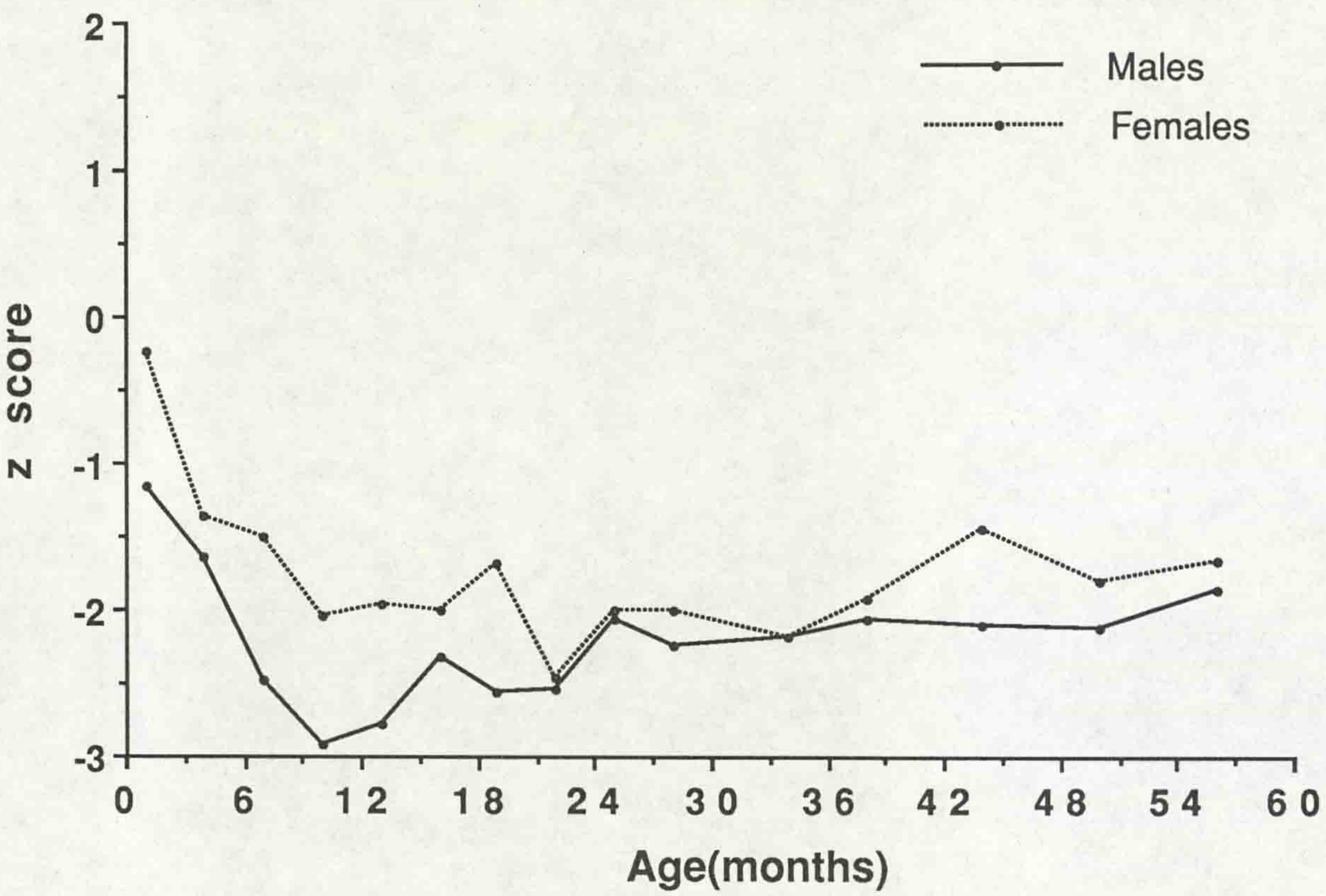


Fig 7.5 Nutrition status of male children in Saradidi

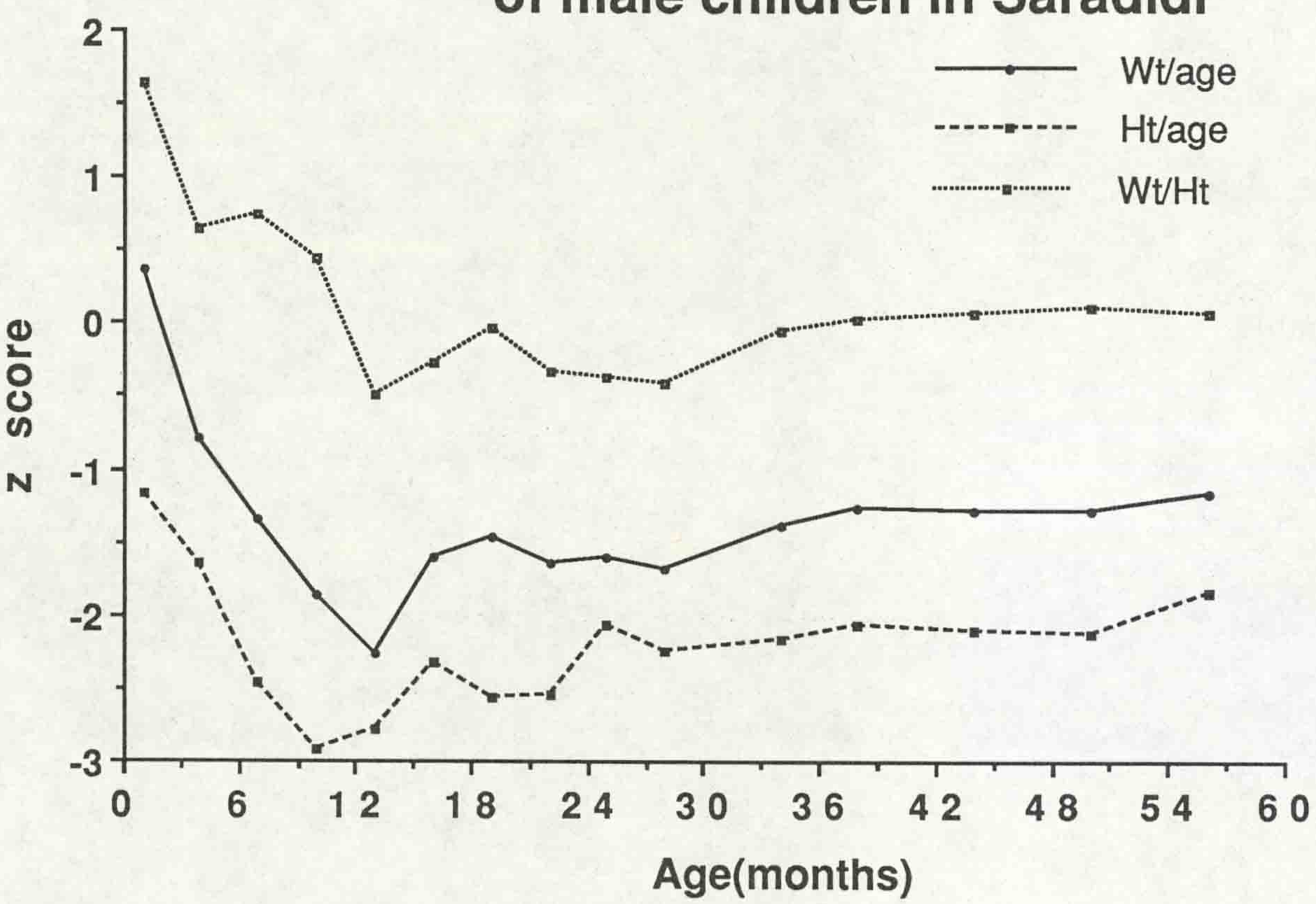
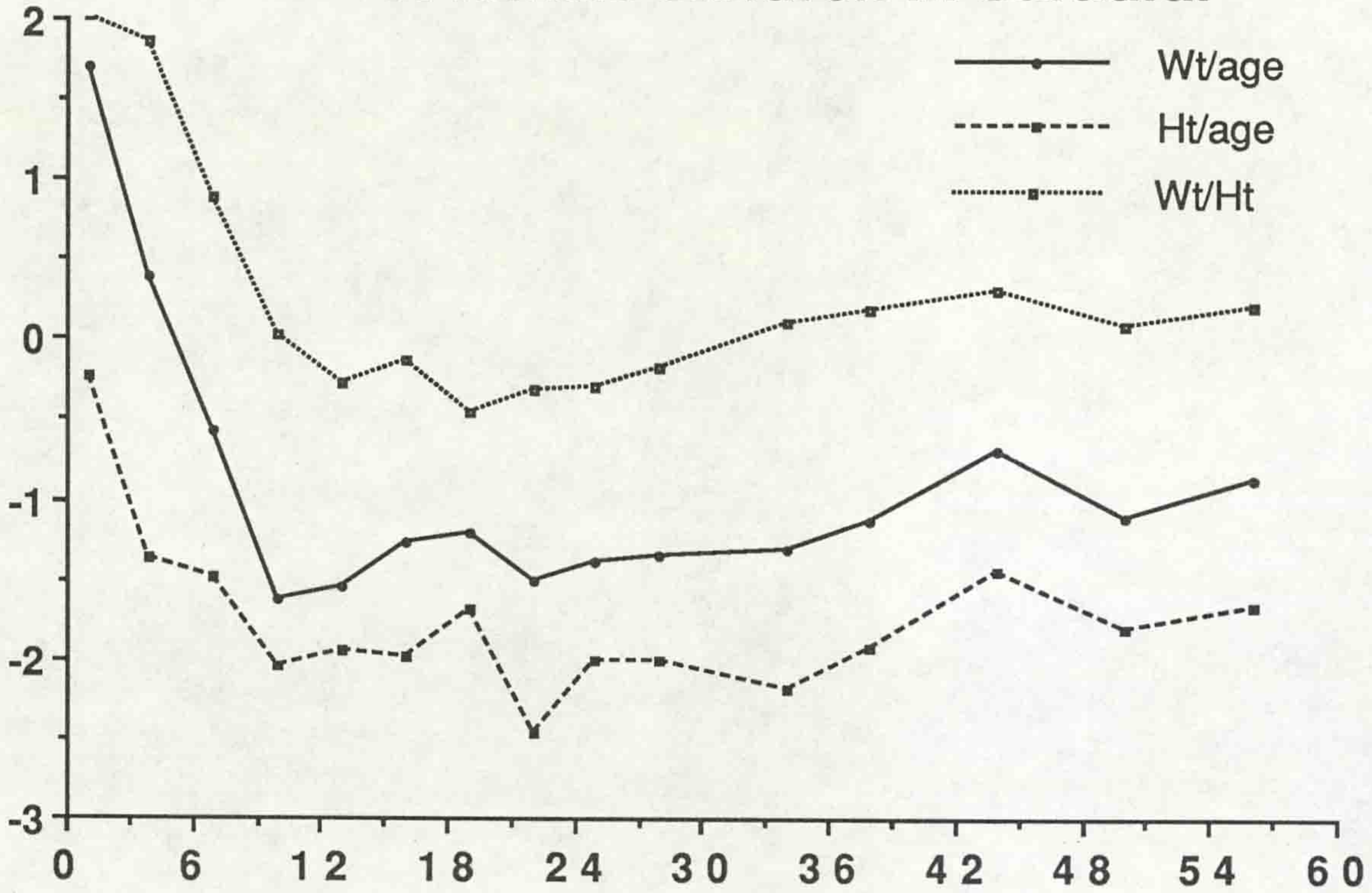


Fig 7.6 Nutrition status of female children in Saradidi



CHAPTER 8

EFFECTIVENESS OF MONTHLY ANTIMALARIAL CHEMOPROPHYLAXIS: THE STUDY DESIGN, METHOD, MATERIALS AND SUBJECTS

8.1 BACKGROUND INFORMATION AND RATIONALE FOR THE STUDY

In establishing four levels of malaria control intervention (Tactical variants 1-4), the World Health Organization (WHO, 1979) has identified pregnant women as a target group for antimalarial chemoprophylaxis in an effort to prevent the deleterious effects of this disease on the mother, foetus and newborn.

Numerous adverse effects of malaria on the pregnant woman and the developing foetus and newborn have been described and include maternal morbidity and mortality (Gilles et al., 1969; Kortman, 1972; Reinhardt et al., 1978; Herd et al., 1981; McGregor, 1984; Strang et al., 1984), spontaneous abortions (Wikaramasuriya, 1937; Kortman, 1972), still births (Wikaramasuriya, 1937; Kortman, 1972); prematurity and low birth weights (Wikaramasuriya 1937; Spitz, 1959; Archibold, 1964; Jelliffe, 1968; Kortman, 1972; Reinhardt, 1978; McGregor, 1983; McGregor, 1984) congential malaria (Kortman, 1982; McGregor, 1984) and neonatal mortality (Marton, 1972; Herd et al., 1981). Primiparous and younger women appear to be at an increased risk for both malaria infection (McGregor et al., 1959; Archibold, 1964; Jelliffe, 1968) and delivering low birth weight babies (Kortman 1972; Bray and Anderson, 1972; Brabin, 1983; McGregor, 1983). Low birth weight has been shown to be one of the most important predictors of neonatal mortality and infant mortality (McCormick, 1985). In the United States, birth weights below 2500 gm have been associated with a 40-fold increased risk of neonatal death and a 5-fold increase in post-neonatal (1-11 months) death (McCormick, 1985). In the developing world, low birth weight infants may have even higher risk of dying, however, additional information on birth weight specific mortality rates is unavailable. Thus the primary concern regarding maternal malaria is that it causes low birth weight and predisposes to

higher risks of perinatal, neonatal and post-neonatal mortality.

There are a number of maternal factors which are known to have a significant affect on birth weight. These factors fall into two broad categories:

1) those for which interventions would be difficult, long term or unavailable and

2) those for which interventions are relatively effective, and short term.

The first group includes factors such as altitude, socioeconomic status, maternal age, parity, maternal height and pregravid weight. The second group includes nutritional status of the pregnant women during her pregnancy, anaemia, physically demanding work during the third trimester of pregnancy, maternal health during pregnancy, birth interval, tobacco smoking or chewing and alcohol consumption.

The important factors in the category of maternal health include hypotension, hypertension with or without pre-eclampsia/eclampsia; maternal infections including malaria, urinary tract infections and amniotic fluid infections.

Babies born to primiparous women with malaria infected placentae have a mean birth weight of approximately 150 grams lower than babies born to primiparous women with no placental infection (McGregor, 1983). The decrease in birth weight associated with malaria infection in subsequent pregnancies is smaller (Kortman, 1972; McGregor, 1984; Brabin, 1983) and significant differences after the second pregnancy have not been observed. Malaria placental infection is most highly correlated with low birth weight. Maternal blood parasitaemia, although associated with placental parasite infection, is not necessarily a good predictor of placental infection or low birth weight (McGregor, 1983).

Newborn infants are thought to be protected for the first 3-6 months of life by passively transferred maternal antimalarial antibody. Mothers with higher levels of antibody appear to transfer more antibody to the infant (Collins

et al., 1985; Bruce-Chwatt, 1985). The degree to which this passively transferred antibody is protective against malaria infection or clinical illness is not well understood. However, in endemic areas, young infants appear to be relatively spared from both infection and severe clinical illness during the first 6 months of life. Antimalarial chemoprophylaxis of pregnant women can decrease the exposure to malarial antigen and, theoretically, lower the antimalarial antibody titers, thereby decreasing the amount of antibody which is transferred to the foetus (Collins et al., 1985). Thus, despite the expected benefits of malaria chemoprophylaxis in increasing birth weight, there is a potentially detrimental effect of chemoprophylaxis in decreasing passively transferred antibody to the newborn and thereby reducing protection against malaria in the first months of infancy.

The present recommendations for antimalaria chemoprophylaxis use in pregnant women from the WHO include the use of weekly chloroquine at a dosage of 5 mg/kg or 300 mg (WHO, 1984). A recent WHO Expert Group (November, 1985) recommended that pregnant women also receive a curative treatment dose of antimalarial drug at the time of their first pre-natal visit.

The delivery of weekly chemoprophylaxis to pregnant women presents difficult logistic problems in many developing countries and the cost of such programmes may be prohibitive due to limited resources, under developed infrastructures and competing priorities. Chemoprophylaxis programmes in school-aged children have been shown to be difficult to administer because of problems of drug supply, distribution, communication, staffing, community participation and side effects from the drug (MacCormack and Lwihula, 1983).

Our study reported in Chapter 5 showed a compliance rate of only 30% when CHWs provided chloroquine phosphate for chemoprophylaxis for pregnant women. There is a general agreement that malaria infection in the pregnant woman adversely affects the health of the mother and the developing foetus. The present recommendations for antimalarial chemoprophylaxis, although

considered an appropriate intervention strategy by experts in the field, have not been evaluated in terms of expected benefits of the intervention. As a result of high programme costs, most countries have either not adopted, or have not placed high priority on antimalarial chemoprophylaxis programmes.

This investigation was intended to respond to these needs by evaluating a strategy of monthly chemoprophylaxis that could be delivered at antenatal clinics. This, if sufficiently effective, would be affordable and sustainable by many developing countries since antenatal services are relatively well utilized (Bennett, 1987).

8.2 OBJECTIVES OF THE STUDY

The main objective of this study was to find out the most effective and practical approach to reduce the impact of malaria in pregnancy for women living in an area of stable malaria.

8.2.1 Specific objectives

1. To find out whether antimalarial chemoprophylaxis using chloroquine phosphate at the dose of 10 mg/kg given monthly to pregnant women who have access to chemotherapy at the village level is more effective than villaged based chemotherapy alone.
2. To find out whether antimalarial chemoprophylaxis using chloroquine phosphate at the dose of 5 mg/kg given weekly to pregnant women who have access to chemotherapy at the village level is more effective than village based chemotherapy alone.
3. To find out whether antimalarial chemoprophylaxis using chloroquine phosphate at the dose of 5 mg/kg weekly is more effective than 10 mg/kg monthly when given to pregnant women who have access to chemotherapy at the village level.
4. To relate the effectiveness of antimalarials in pregnancy to parity, village/area of residence and type of haemoglobin.

8.3 BASIC ASSUMPTIONS AND HYPOTHESES

In hyper- and holo-endemic areas of malaria, malaria mortality and morbidity is concentrated among children (under five years) and pregnant mothers. Malaria is a very important cause of mortality and morbidity in such areas.

Early chemotherapy of clinical malaria with chloroquine phosphate at a dosage of at least 25 mg/kg body weight is effective, in the study population, in preventing the death of that person from the clinical episode; this is therefore the highest operational priority.

Chemoprophylaxis in addition to chemotherapy is effective in reducing morbidity particularly among pregnant women, the greatest impact being among primigravidae.

Mass chemoprophylaxis cannot maintain high coverage indefinitely at an acceptable cost. Regular chemoprophylaxis delays the development of antimalaria immunity and the magnitude of the delay depends on the regularity of administration (Voller et al., 1964).

The use of antimalarials in large quantities in an area may favour the selection of resistant parasites; the selection is enhanced by high coverage and low dosage. Chloroquine is still an effective antimalarial drug for treatment and chemosuppression of malaria infections in the study community (Spencer et al., 1987).

The hypothesis tested was whether chemoprophylaxis had a beneficial effect to pregnant women who had access to chemotherapy when given at the dose of 10 mg/kg monthly (instead of 5 mg/kg weekly) within a community based PHC system. The hypothesis was tested by investigating the effect of monthly chemoprophylaxis at the level of 10 mg/kg on:

1) mean birth weight
2) percentage of new borns less than 2500 grams at birth
3) placental parasite infection measured at the time of delivery

4) percentage of new borns with congenital parasitaemia

5) mean weight of the placenta

6) percentage of children born premature (less than 36 weeks gestation)

7) percentage of abortions and still births

8) the survival of the infant in the perinatal, neonatal and post-neonatal periods.

Additional assessment was carried out on the mother from recruitment up to delivery and six weeks after delivery. The variables assessed included:

1) weight gain during pregnancy

2) prevalence and density of parasitaemia during antenatal and post-natal visits

3) complaint of febrile illness during two weeks preceeding the ante-natal visit

4) mean haematocrit levels during antenatal and post-natal visits.

8.4 METHODOLOGY

8.4.1 Recruitment of pregnant women into the study

An attempt was made to register all pregnant women as soon as their pregnancy status was suspected. This was done in the following ways:

a) Through the CHWs: The CHW included all the women in her area who were most at risk of becoming pregnant (based on age and marital status) and visited them every month to find out their pregnancy status based on last menstrual period (LMP).

b) During the 1985 cross sectional survey: A question concerning LMP was asked to all women in the fertile age group (15-49).

c) During the census updates 1985 and 1986: The census team enquired about LMP of all the women 15-49 years of age.

d) During other contacts with project staff: Project staff asked about LMP of all female patients and clients aged 15-49 years.

All women suspected of being pregnant were entered into a provisional pregnancy list, if they had missed two menstrual periods, pending confirmation by clinical examination at 12-16 weeks. As soon as they were confirmed pregnant, they were entered into a confirmed pregnancy list.

Primigravidae and multigravidae were entered into the study and were assigned to any one of the three study groups in such a way that each individual had an equal probability of entering into any of the three treatment groups as previously described (Peto et al., 1986). The numbers were balanced after every sixth entry into the study to ensure equal numbers into the groups at all stages of study as described by Peto et al. (1976).

8.4.2 Selection of subjects

Women and their subsequent newborns would be included in the study if they met the following criteria:

1. residents of areas A, B and C of Saradidi rural health programme
2. they were contacted prior to the 16th week of gestation
3. they gave consent to participate in the study.

All women who met the study subject criteria were entered into the study at the time of their first antenatal care visit. They were then followed up at 20 weeks, 36 weeks and were admitted for delivery at the project's health centre. They were last seen at 6 weeks post partum. The infants were examined at six months and at 12 months.

At entry into the study each subject was interviewed and examined to determine their:

1. history of febrile illness in the previous two weeks
2. parity
3. residence
4. risk status
5. gestation

6. weight
7. height
8. haematocrit
9. parasitaemia status (prevalence and density)
10. haemoglobin type

8.5 SAMPLE SIZE AND TREATMENT GROUPS

Three hundred and sixty one women were entered into the study, at least half of them were primigravidae. Each of the subjects were assigned to different study groups as follows:

Group 1 — 120 women were entered into this group, 70 of them were primigravidae. This group were given the monthly chemoprophylaxis using 10 mg/kg body weight using chloroquine phosphate. This was provided at the antenatal clinics by nurses and community health workers.

Group 2 — 121 women, 70 of whom were primigravidae had access to village based chemotherapy based on 25 mg/kg body weight when ill and a weekly chemoprophylaxis using chloroquine phosphate based on 5 mg/kg body weight.

Group 3 — 120 women, 70 of whom were primigravidae were the comparison group and they had access to village based chemotherapy based on 25 mg/kg weight when ill like the other two groups but without any regular chemoprophylaxis.

The list of confirmed pregnant women which was prepared by the Clinical Officer was then transferred to the principal investigator who assigned them to the groups using a table of random numbers and random permuted blocks as described by Pockock (1983). The investigator could not predict ahead of time which treatment group the next case would go to. This minimised the use of judgement or systematic bias and hence provided a basis for statistical

analysis. Thus bias in subject selection was minimised at assignment and in handling during antenatal visits and postnatal visits.

The midwife also delivered the babies and collected specimens at delivery and the laboratory technicians did not know what groups the subjects belonged to. The subjects knew their treatment groups. For this reason the study was not double blind for practical purposes but the measurements of weights and lengths and parasitological examinations could not have been biased since they were carried out by people who did not know the treatment groups of the subjects.

The subjects in group 3 who were not on prophylaxis had free access to treatment whenever they wanted it either from the CHW in their own village, at the antenatal clinics or at any health centre in the area. They were therefore not disadvantaged by not being on prophylaxis.

The subjects on chemoprophylaxis were not unduly pressurised to take the antimalarials regularly, as prescribed since the emphasis of the investigation was not to assess the biological effectiveness of chloroquine given 5 mg/kg weekly as compared to 10 mg/kg monthly and as compared to 25 mg/kg whenever ill with malaria, but to evaluate a chemoprophylactic regime as it would operate in actual PHC service delivery system. Thus any irregularities that would be found in a normal health care delivery system in a developing country like Kenya were anticipated but no undue efforts were made to avoid them.

Sample size

Using statistical tables for the determination of the sizes of each of the study groups (i.e. based on the level of expected differences in parasite rates, density and birth weight among the groups and the confidence level), the sizes of the most important cells of the above selected parameters were in excess of the minimum required sample sizes. Also taken into account were the practicalities of finding enough study subjects of each study group given a fixed reference population of 60,000 people. Each treatment group was required to have at least 100 subjects, particularly for the assessment of birth weight.

8.6 DATA COLLECTION

The study women were given cards at the first visit as they are entered into the study on which the details of first and subsequent visits were recorded. They were then seen monthly for routine ANC check-up except at 12, 20 and 36 weeks of gestation, at delivery and six weeks after delivery when the following examinations were undertaken, in addition to the routine assessment:

a) specimen of urine for the presence of chloroquine by Haskin's test,

b) blood slides for malaria parasites (prevalence and density),

c) blood specimen in heparinised capillary tubes for the measurement of haematocrit, and

d) 5 ml of blood obtained by venepuncture, during the first visit only, for haemoglobin electrophoresis.

Haemoglobin electrophoresis and measurement of height, were carried out only once at recruitment.

At the end of the third stage of labour, the placenta was washed and left with a 6 cm length of cord stump. The placenta was weighed with the membranes intact within two hours of the delivery of the placenta.

A cotyledon on the maternal side was identified, cleaned and incised and the blood from it was used to prepare a thin and thick blood smear. The slides were dried and stained with Giemsa and were then examined for malaria parasites and parasite count.

The slides were examined by a trained microscopist who examined them under oil immersion. One hundred fields were examined. The investigator checked 10% of the slides and compared results.

Blood slides were made from cord blood and examined in the same way as described for placental specimens.

Immediately after birth, the baby was weighed using a weighing scale for newborns. The same scale was used, as much as was possible for weighing all the newborns in the study. The weight was taken by the midwife trained in weighing

skills and was checked once a month by a Medical Officer or the investigator to ensure the use of standard techniques in weighing. The arm, head and chest circumference, and height were measured and recorded on the information sheets. The infants were examined for haematocrit and parasitaemia.

The measurements above were repeated at 6 and 12 months. Blood was obtained from a heel-prick for the preparation of blood slides for malaria parasites, identification and count at 6 and 12 months.

Abortions, still births and premature deliveries were recorded by date and maternal characteristics.

Deaths of infants were recorded by date of death and from these records, deaths in the first 7 days, the first 28 days and in the first year of life were obtained.

Thus the three groups were compared in terms of:

1) Mother - haematocrit, parasitaemia and parasite density, at every visit, delivery and six weeks post-partum

2) Pregnancy outcome

3) Placenta - weight, parasite density, malaria pigment

4) Newborn - birth weight, parasitaemia and parasite density, serrogate measurements at birth, at six months and at 12 months, infant survival through the first year of life, infant nutrition by anthropometric measures, age at first parasitaemia and frequency of febrile illnesses.

All the study women were advised to deliver at the SRHP Centre to allow standardised anthropometric measurements and collection of specimens for laboratory investigations.

All measurements were done by the same person after careful training and were checked at least once every month by the investigator.

Haskin's test which had been shown to be more accurate than the Dill Glazko test (Rombo, 1985) was used for the detection of urine chloroquine.

Laboratory examination/procedures

The blood slides were obtained by finger or heel pricks in adults and infants respectively, and thin and thick smears were made on the same slide for the same subject, air dried, labelled appropriately and stained with Giemsa. The slides were then examined for infection and identification of species and for definitive parasite density under oil immersion. The counting was done against 300 leucocytes and recorded. From this the number of parasites per mm^3 of blood was worked out using the following formula:

$$\frac{\text{parasite count} \times 8{,}000}{300} = \text{parasites/mm}^3 \text{ of blood}$$

(assuming average total WBC count of 8,000/mm^3)

8.7 DATA ANALYSIS

The outcome variables of this investigation included:

Maternal morbidity: haematocrit

episodes of parasitaemia

placenta parasitaemia

spontaneous abortion (born dead before 28 weeks) or

still births (born dead at 28 weeks and afterwards) ;

Premature births (born alive <36 weeks gestation)

Birth weight (mean and percent <2500 grams)

Congential malaria parasitaemia

Neonatal mortality

Perinatal mortality

Post-neonatal mortality.

Explanatory variables measured included maternal age, parity, prophylactic regimen, and area of study. Maternal parasitaemia, placental parasitisation and cord parasites were both explanatory and dependent variables (see Figure 8.1).

The information was analysed in the following general categories:

1) Descriptive information for each variable in the study population.

2) Single variable analysis for each treatment group using outcome variables in order of priority: mean birth weight, proportion of newborns under 2500 grams, placental parasitaemia rates, still birth rates.

3) Single variable analysis of maternal characteristics and outcome variables in order of priority: mean birth weight, and proportion of newborns under 2500 grams, placental parasitaemia rates, still birth rates.

4) Single variable analysis of maternal parasitaemia and placental parasitaemia as indicator variables and outcome variables in order of priority: mean birth weight, proportion of newborns under 2500 grams, still birth rates.

5) Multivariate analysis using indicator variables that were shown to be significant in single variable analysis.

As indicated above, there are some variables which were both predictor variables and outcome variables (i.e. maternal blood parasitaemia and placental parasitaemia) and this situation was conducive to path analysis which assessed the importance of a given variable as a predictor of outcome both by itself and as a member of a causal pathway which includes other variables. For example, maternal age has a direct effect on birth weight and also has an effect on maternal malaria parasitaemia which, likewise, has an effect on placental parasitaemia, both of which affect birth weight. The results of data analysis are described briefly in this chapter but are presented in detail in Chapters 9-11.

8.8 RECRUITMENT AND THE CHARACTERISTICS OF THE STUDY POPULATION

8.8.1 Recruitment Dates

The recruitment of study subjects started in March 1985 and continued

until September 1986. A total of 361 women were registered and assigned to the study groups as already explained. The study ended up with 112 women (31%) in group 1 (receiving monthly chloroquine at the dose of 10 mg/kg); 121 (33.5%) in the second group, receiving the weekly chemoprophylaxis at the dose of 5 mg per kg body weight and 116 (32.1%) of the women were in the third group, those who were treated by the CHWs in their villages whenever they were unwell and felt that they needed treatment for malaria. For 12 study subjects the group assignment was not indicated on the forms and were treated as missing cases at analysis.

Most of the subjects (200) entered the study in 1985 and the remaining 149 subjects entered in 1986 bringing the total number to 361 which included the 12 cases for whom neither the treatment group nor the date of entry was indicated.

The seasons in Saradidi are as follows: the wettest season is April-July followed by August-November. Malaria transmission remains very high during these two seasons (see malaria transmission study presented in Chapter 3). The rest of the months, December-March, are relatively dry months, although the extent and intensity tend to vary from year to year. This seasonal pattern in Saradidi is also described in Chapter 3.

The recruitment of subjects by month is summarised in Table 8.1. Half of the subjects were recruited during the short rains, one third during the dry season and the rest (16%) were recruited during the long rains, the season of peak malaria transmission.

8.8.2 Recruitment areas

The recruitment areas were the same as areas A, B and C discussed in Chapter 1 which was based on the 1980-1984 study in which area A received village based malaria chemotherapy and chemoprophylaxis for pregnant women provided by the CHWs; area B received village based chemotherapy alone while

area C did not receive any chloroquine supplies from the study project, and served as the control area. From 1985, the beginning of the study described in this section, all the three areas were treated the same. The three areas could have differed enough to affect chloroquine use in such a way that the area of study could have been a factor influencing some of the outcomes measured in the current study. It is for this reason that the study population was examined by the areas they came from to ensure that all the three areas were equally represented in the study groups. Table 8.2 shows that subjects from each of the study areas A, B and C were equally distributed in the study groups. Nearly half (142) of the women came from area A while 123 or a third of the women came from area B. These were the areas with experienced CHWs and were closer to the study centre. the Saradidi Health Centre. These factors were beneficial operationally, but it is to be noted that these were also the areas with the longest contact with antimalarials and might not have been representative of any high malaria endemicity in Kenya.

8.8.3 Age at recruitment

Age is a factor which could affect the results of the study either directly or indirectly through its relationship with parity. The mean age of the women in the study was 21.7 $\pm$ 5.6 but ranging from 15 to 46 years (Figure 8.2). The mean age increased with increasing parity as would be expected. There was no significant difference in the age distribution among the study groups and among the study areas. These mean ages were 22.6 $\pm$ 11.4 years for the monthly chemoprophylaxis group; 22.4 $\pm$ 6.1 for the weeky chemoprophylaxis group and 22.9 $\pm$ 11.6 for the group receiving treatment only when ill. These mean ages were not significantly different.

The distribution of the mean age of the study cases by study areas also showed no difference; the mean age of the women from areas A, B and C being 25.9 $\pm$ 17.6, 25.3 $\pm$ 17.6 and 22.8 $\pm$ 12.0 respectively.

There was no reason to expect any difference in age distribution either by age or by treatment group but it was necessary to ascertain that there was no difference as age could be a significant confounding variable. It was concluded that the treatment groups were comparable in terms of age distribution.

8.8.4 Haemoglobin types

Of the 361 cases, 252 were examined for haemoglobin type as it is a factor that could determine the malaria experience of the subjects and could cause a difference and thus interfere with study results. Twenty-eight (11.1%) were found with the abnormal haemoglobin type AS.

8.8.5 Abortions

The history of abortions in individual subjects could be a factor influencing outcome of the current pregnancy, gestational age and hence birth anthropometric measurements. Therefore the women were asked at recruitment if they had had any abortions in their obstetric history. Of the 361 women 17 (4.8%) had had abortions; 14 of them had had only one abortion.

8.8.6 Height and weight

Anthropometric measurements of the mother could influence birth anthropometric measurements which were the key indicators of effectiveness in this study. It was necessary to determine these carefully and to ensure that their distribution in the three treatment groups was as uniform as possible. The height of the woman was also used to determine the risk status of the women as any primigravida who was less than 150 cm had to be excluded from the study and referred to centres better equipped to perform operative deliveries.

The mean height of the study population was 160.5 $\pm$ 6.5 cm with a range of 139 to 179 cm (Figure 8.3). Among the treatment groups the mean heights were 160.2 $\pm$ 6.7, 160.4 $\pm$ 5.9, and 160 $\pm$ 6.2 in treatment groups receiving

monthly chemoprophylaxis, those receiving weekly chemoprophylaxis and those receiving treatment when ill, respectively (F = 24, df = 2,345. P<0.05).

However, looking at the heights of the women by study areas, the women from area C had a significantly higher mean height of 162.5 ± 7.2 as compared to 160.4 ± 5.6 and 159.7 ± 6.4 in areas A and B respectively (F = 3.93, df = 2,345, P <0.05).

The mean recruitment weights were significantly different neither by treatment group nor by study area, see Table 8.3.

8.8.7 Parity

Parity is another factor that was expected to affect the outcome indicators in this study. It was therefore necessary to ensure that the distribution of women in the three study groups was uniform with regard to parity. The most important parity group is the primigravidae. The analysis of the treatment groups indicated that the differences among the treatment groups were very small 36 (31.9) in the groups receiving chloroquine treatment when ill (Table 8.4).

8.8.8 Urine chloroquine

The excretion of chloroquine and chloroquine metabolites in the urine was examined at recruitment and at every visit to the clinic for the purposes of the study. This was originally designed as a check for compliance. The Haskin's test was used which has been shown by Rombo (1985) as more sensitive and more specific than the Dill Glazko.

It was also important to demonstrate that chloroquine consumption pattern at the time of entry into the study was similar among the study groups. Of 291 subjects tested at recruitment 48 (16.5%) had chloroquine in their urine. There was no difference in the rate of positivity by treatment group.

8.8.9 Febrile illness at recruitment

Most of the 331 women asked at recruitment whether they had had a febrile illness within two weeks preceding the date of recruitment answered positively. There was no difference in response to this question by treatment group nor by study area (Table 8.5). About 80% of all the women had had fever within the two weeks preceding their recruitment.

There was also no difference in the history of febrile illness between the women with the normal (AA) haemoglobin type and those with the abnormal (AS) haemoglobin type.

The women with a positive history of febrile illness were more likely to have chloroquine in their urine than those who did not have such a history. This implies that the women in the study were no more likely than the general population to use chloroquine routinely for the treatment of fever at the time of recruitment.

8.8.10 Parasitaemia at recruitment

The study subjects were examined to see whether they had malaria parasites as they entered the study. Of 230 subjects examined, 36 (35%), 37 (33.9%) and 37 (37%) of them were positive in the monthly chemoprophylactic, weekly chemoprophylactic and chemotherapy when ill groups respectively (see Table 8.6 and Figure 8.4).

Considering the parasite rates by haemoglobin type, again no difference was found as 67 (32.8) of those with normal haemoglobin had parasites as compared to 10 (38.5%) of those with abnormal haemoglobin.

The mean parasite counts were different neither by study area nor by treatment groups. Area A had a mean value of 25.2 $\pm$ 61.9, area B 32.8 $\pm$ 60.1 and area C 23.3 $\pm$ 85.0. The monthly chemoprophylaxis group had a mean value of 25.4 $\pm$ 54, weekly chemoprophylaxis group had 24.1 $\pm$ 67 and the treatment when ill group had 33.4 $\pm$ 74 parasites per 300 white blood cells.

Parasite rates were different by parity, primigravidae had 59/113 (57.2%) and 20/68 (29.4%) for para 1 and 30/123 (24.4%) for parity of 2 and over. This difference was strongly significant (X^2 = 20.01, df = 4, P <0.001). When all the parities were considered together then there was no difference in parasite rates among the treatment groups (Table 8.6).

The difference in parasite rates by parity was also reflected in parasite counts which were higher among the primigravidae and decreased as parity increased. The parasite counts were 45.55 ± 79.5. 25.1 ± 54.2. 10.86 ± 26.8 and 15.65 ± 62.9 for para 0, para 1 and para 2 and para 3 or more. (F = 3.34, df = 4,290. P <0.01). These parasite counts per 300 WBCs were significantly higher for primigravidae then the other parity groups.

The mean parasite counts did not differ by haemoglobin types; 27.1 ± 69.8 in normal haemoglobin subjects as compared to 31.9 ± 60 among those with haemoglobin AS type.

8.8.11 Haematocrit levels at recruitment

The haematocrit levels at recruitment were normal and remained so throughout the study. The levels showed no difference by treatment groups, study areas, parity or haemoglobin type (Table 8.7).

8.9 DISCUSSION

There was need to undertake this study in order to determine the effectivness of a monthly chemoprophylaxis given to pregnant women at the antenatal clinics at the rate of 10 mg/kg body weight using chloroquine phosphate. This need had been identified by several workers (MacCormack and Luihula, 1983; Kaseje et al., 1987) who showed that the weekly prophylactic regimen is impossible to administer in most of rural tropical Africa.

The importance of prophylaxis has been highlighted by several workers who have shown that chemoprophylaxis will:

a) improve maternal health during pregnancy (Gilles et al., 1969; Reinhardt et al. 1978);

b) reduce foetal wastage (Kortman, 1972);

c) increase mean birth weight (Jelliffe, 1968);

d) reduce the rate of congenital malaria (McGregor, 1984);

e) reduce neonatal mortality (McCormick, 1985);

f) reduce the rate of low birth weight babies (Brabin, 1983).

The focus of this study was to look particularly at the birth weight as one of the most affected outcomes of pregnancy as a result of malaria in pregnancy. It is also a factor that appears to determine the survival of the new-borns in the perinatal and neonatal period and also in the post-neonatal period. This is where the benefits of a successful chemoprophyaxis programme would be maximal and could thus be most easily observed.

Although the sample size was calculated as described, reaching the required number was quite difficult. Up to one third of the study subjects did not have all the investigations carried out due to operational and social difficulties. The patients who had to be referred were often also lost to the study as the referral facilities and personnel were not committed to the study, although they had expressed willingness to participate in it.

Equal numbers were recruited into the three treatment groups. The analysis of their basic characteristics presented in this chapter shows that the three groups were comparable in terms of area of residence. parity, haemoglobin type, height, weight, and their morbidity and parasite rates at recruitment.

The differences found were mainly with regard to parasitaemia rates and parasite counts which were both higher in the primigravidae. This was expected as it had previously been shown by McGregor (1959). Jelliffe (1968) and Brabin (1983).

Most subjects were recruited during the wet months and less than one third of the cases were recruited during the dry months of the year.

It can be concluded that the study groups were comparable at recruitment in terms of basic characteristics and morbidity. The differences noted were limited to parity and were to be expected. Effects of the three different antimalarial interventions could be observed.

Table 8.1 The number of study subjects by month of recruitment

Season	Month	Recruitment	
		No.	%
Long rains (very wet)	April	22	6.1
	May	8	2.3
	June	6	1.7
	July	21	5.8
	Subtotal	57	15.9
Short rains (moderately wet)	August	39	10.8
	September	52	14.4
	October	52	14.4
	November	34	9.4
	Subtotal	177	49.0
Dry season	December	37	10.2
	January	30	8.3
	February	21	5.8
	March	27	7.5
	Subtotal	115	31.8
Unknown		12	3.3
GRAND TOTAL		361	100

Table 8.2 Distribution of study subjects by study area and treatment groups

Treatment group	Study Area							
	A		B		C		Total	
	No.	%	No.	%	No.	%	No.	%
Monthly chemoprophylaxis	47	29.9	44	32.4	21	37.5	112	32.0
Weekly chemoprophylaxis	58	36.9	47	34.6	16	28.6	121	34.7
Treatment when ill	52	33.2	45	33.0	19	33.9	116	34.2
Total	157	100	136	100	56	100	349	100

X^2 = 1.59, Df 4, p >0.05

Table 8.3 The mean weight of the study population by area and by treatment group

Area	Mean weight	F. value	Df	P. value
A	56.5 ± 6.4			
B	56.9 ± 7.2	0.44	2,334	>0.05
C	55.9 ± 7.1			
Treatment group				
Monthly chemoprophylaxis	56.4 ± 6.3			
Weekly chemoprophylaxis	56.8 ± 6.4	0.20	2,330	>0.05
Treatment when ill	56.2 ± 7.7			

Table 8.4 Distribution of study subjects by parity and treatment groups

Treatment group	Parity							
	0		1		2+		Total	
	No.	%	No	%	No.	%	No.	%
Monthly chemoprophylaxis	36	31.9	24	35.2	48	36.9	108	34.7
Weekly chemoprophylaxis	41	36.2	21	31.0	44	33.8	106	34.1
Chemotherapy when ill	36	31.9	23	33.8	38	29.3	97	31.2
Total	113	100	68	100	130	100	311	100

X^2 = 1.15. Df = 4. p >0.05

Table 8.5 Study subjects reporting history within two weeks of recruitment by area of study and by treatment groups

N = 331

Treatment group	Area			
	A	B	C	Total
Monthly chemoprophylaxis	38 (88.4)*	32 (76.2)	17 (85)	87 (82.9)
Weekly chemoprophylaxis	41 (71.9)	38 (88.4)	12 (80)	91 (79.1)
Treatment when ill	42 (85.7)	36 (83.7)	15 (79)	93 (83.8)
Total	121	106	44	271
Unknown				60

X^2 = 1.42. Df = 4. p >0.05
* = Percent with history of febrile illness

Table 8.6 The rate of parasitaemia at recruitment by parity and treatment group

Treatment group	Parity							
	0		1		2+		Total	
	No.	%	No	%	No.	%	No.	%
Monthly chemoprophylaxis	17/36	47.2*	12/24	50	7/41	16.7	36/101	35
Weekly chemoprophylaxis	21/41	51.2	2/21	9.5	13/44	29.5	36/106	34
Treatment when ill	21/36	58.3	6/23	26.1	10/38	26.3	37/97	38.1
All treatment groups	59/113	52.2	20/68	29.4	30/123	24.4	109/304	35.9

X^2 = 10.01. Df = 4, p <0.05

* = Percent with positive blood smear

Table 8.7 Mean haematocrit levels by study area, treatment group, parity and haemoglobin type

	Mean haematocrit level	
Area A	33.7 ± 4	(F = 1.05. df = 2,319
Area B	33.0 ± 4.3	p >0.05)
Area C	33.5 ± 3.7	
TREATMENT GROUP		
Monthly chemoprophylaxis	33.4 ± 4.2	
Weekly chemoprophylaxis	32.8 ± 3.9	(F = 0.48, df = 2,272
Treatment when ill	34.0 ± 4.1	p >0.05)
PARITY		
0	32.8	
1	33.9	(F = 0.12, df = 2,183
2	33.9	p >0.05)
3 and over	33.7	
Unknown parity	32.0	
HAEMOGLOBIN TYPE		
AA	33.7 + 4.1	-
AS	34.1 ± 4.0	

N = 295

Figure 8.1 The main independent and dependent variables in Saradidi pregnancy study

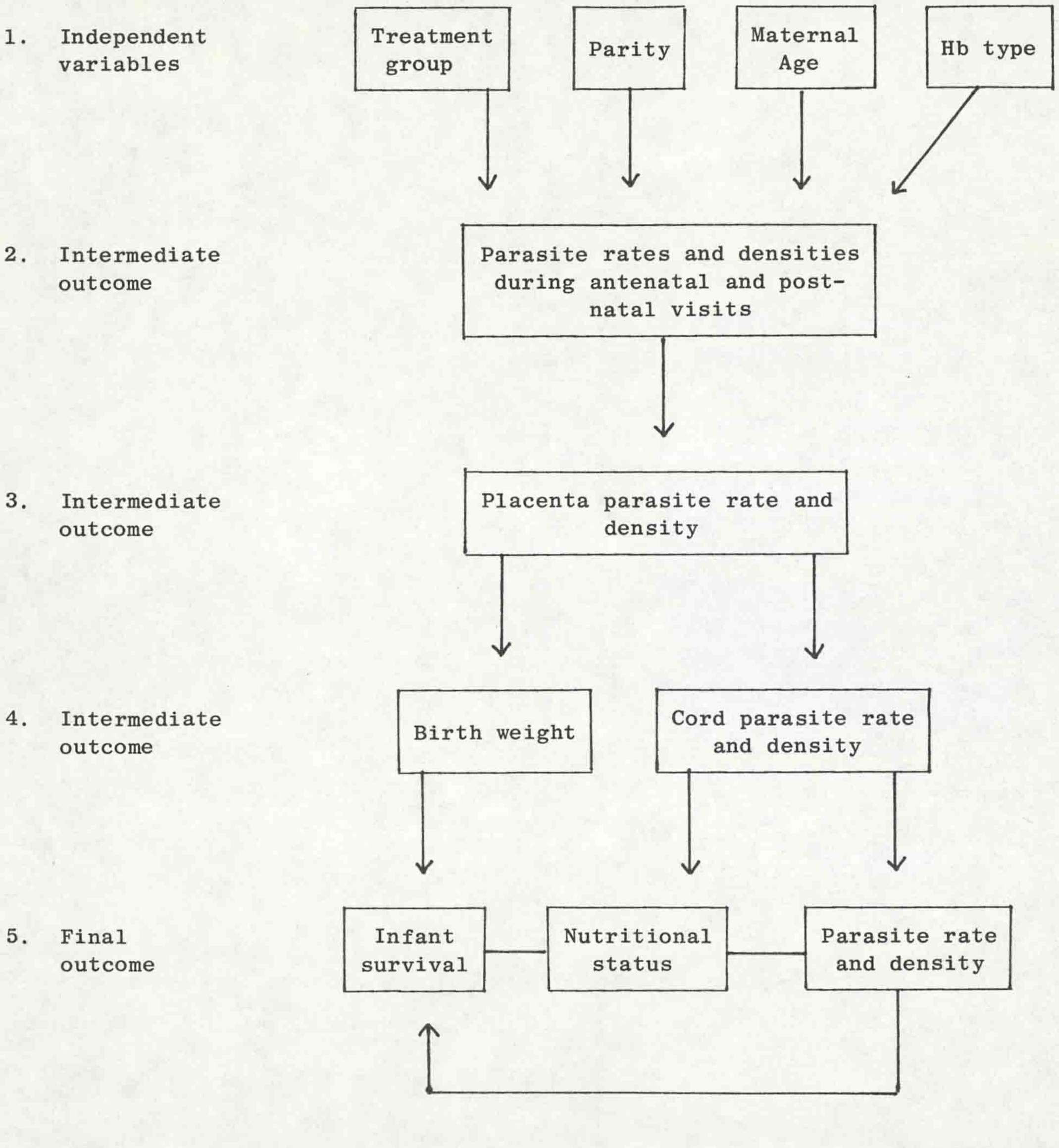


Fig. 8.2 Distribution of mothers by age

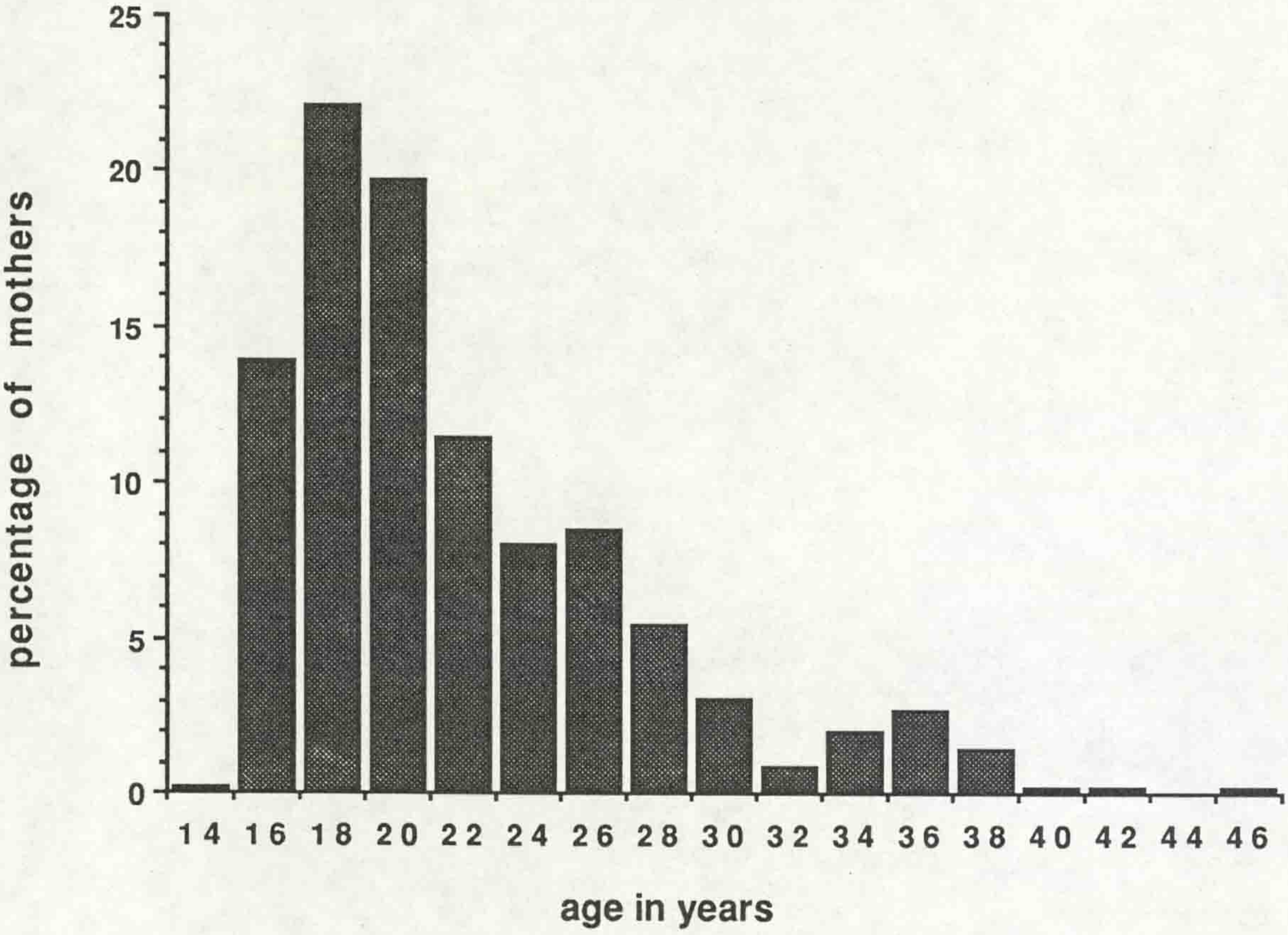


Fig 8.3 Distribution of study subjects by height

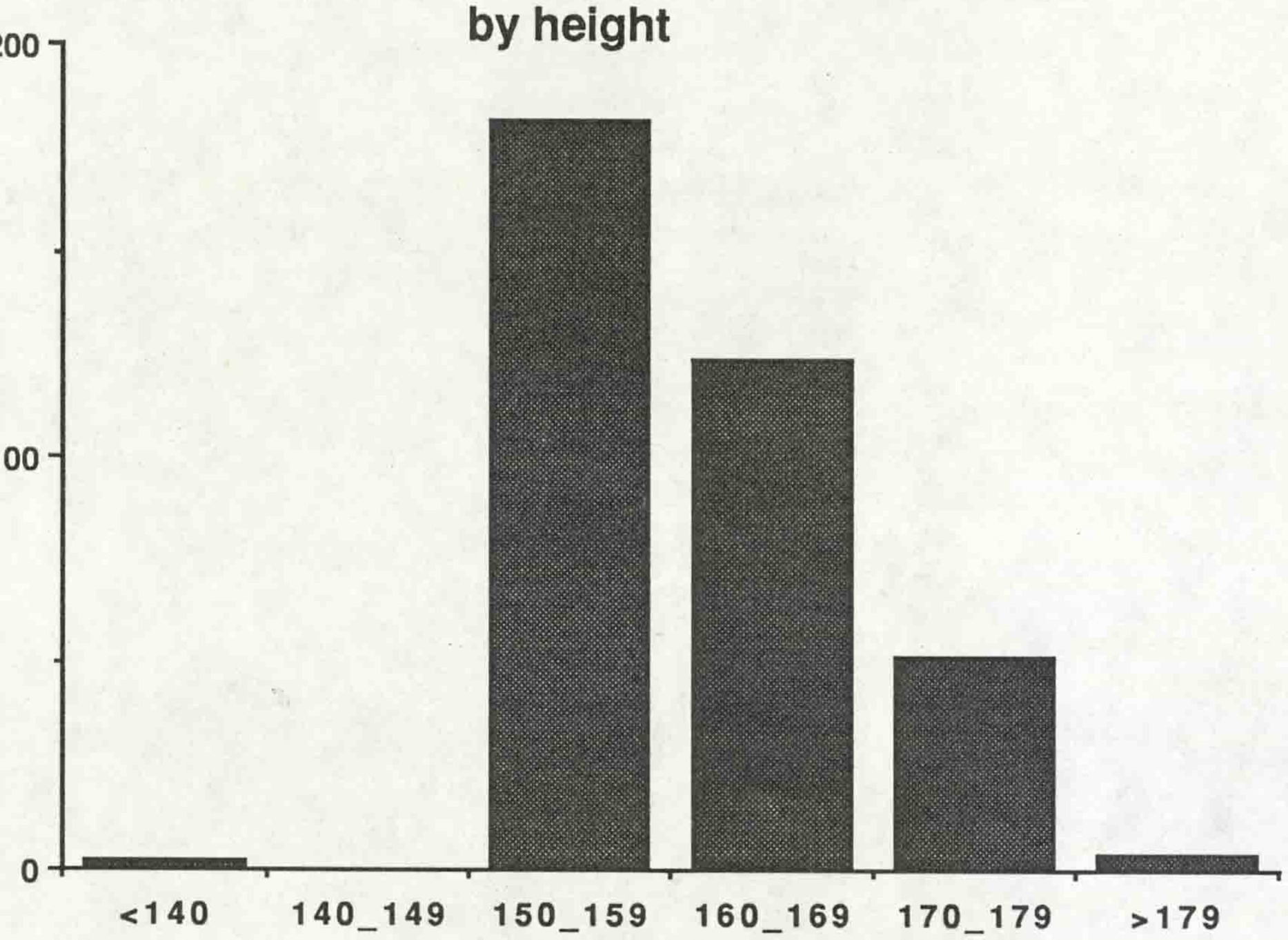


Fig 8.4 Distribution of mothers by mean log^e parasite density at recruitment

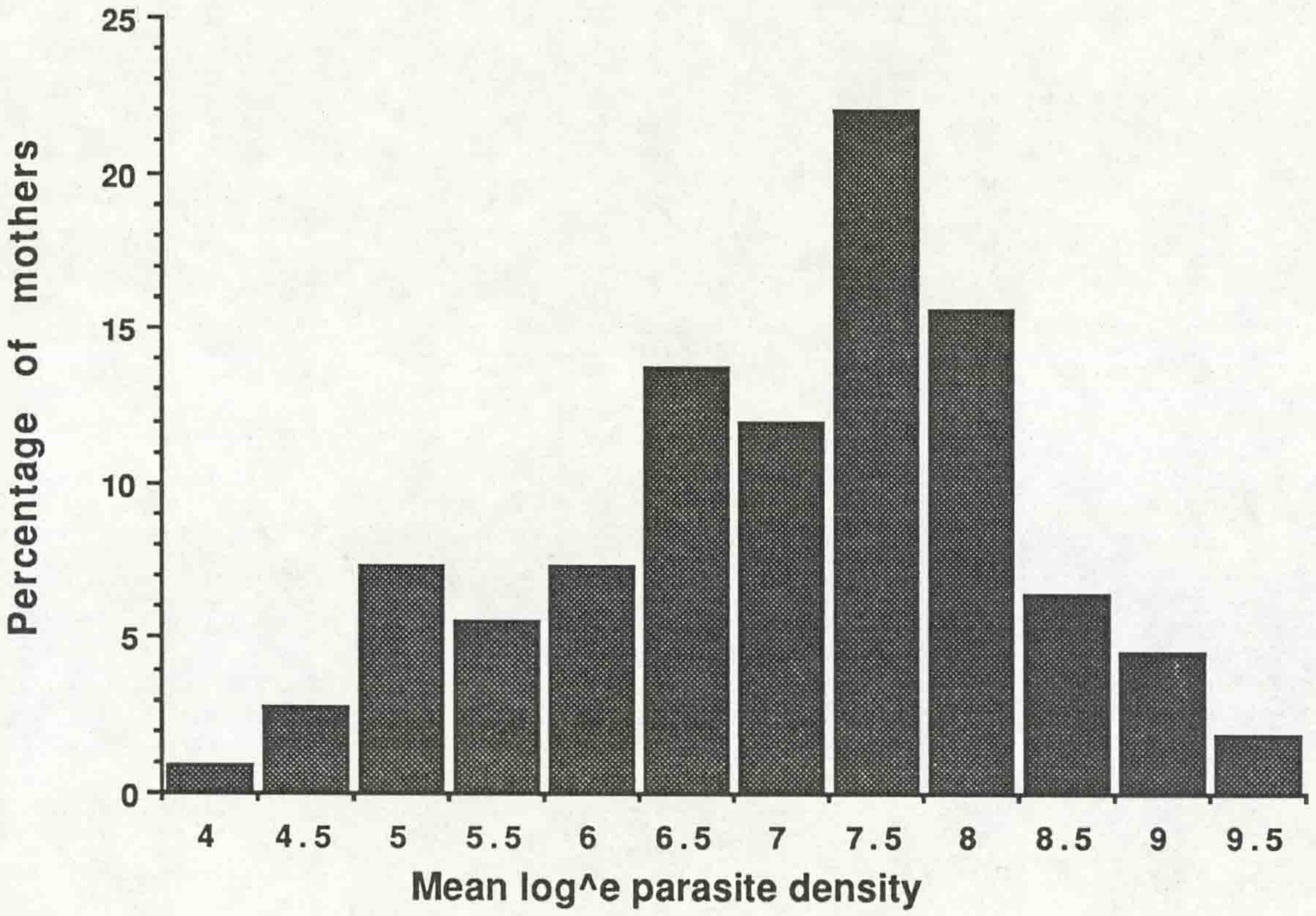
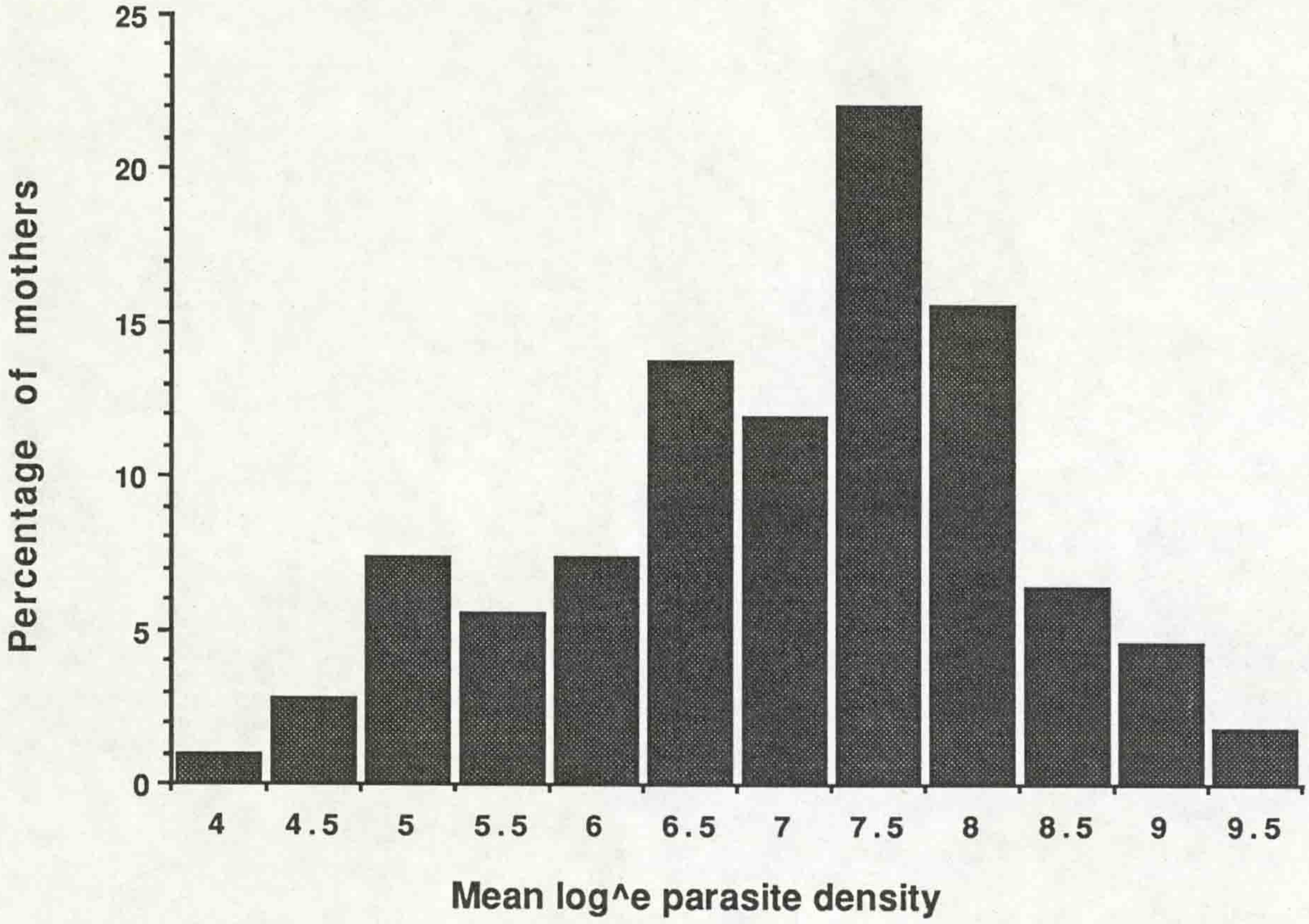


Fig 8.5 Distribution of mothers by mean log^e parasite density at recruitment



CHAPTER 9

EFFECTIVENESS OF MONTHLY ANTIMALARIAL CHEMOPROPHYLAXIS IN THE PREGNANT WOMAN DURING ANTENATAL AND POSTNATAL PERIODS

9.1 INTRODUCTION

The characteristics of the study groups and methodology of the study have been described in detail in Chapter 8. In this chapter the findings during the antenatal and postnatal periods are presented and discussed.

Particular attention is paid to parasite prevalence and density during pregnancy: at recruitment (12-14 weeks), at 20 weeks, 36 weeks, delivery and six weeks after delivery. Haematocrit levels and occurrence of febrile illness are presented as an indicator of maternal morbidity and urine chloroquine as an indicator of chloroquine consumption.

Explanatory variables considered include area of residence, parity, age of the mother, and treatment group.

9.2 RESULTS

9.2.1 Febrile illness

a) By visit:

A very high proportion of women had a history of febrile illness during the two weeks period preceding their antenatal visit. The proportion seemed to be highest during the first visit when 82.1% had a positive history of fever as compared to 66.8% during the third visit (Table 9.1).

The proportion with fever could have been reduced as shown in subsequent visits, because of treatment and chemoprophylaxis given to the women or it may be that febrile illnesses are less frequent during the third trimester. The drop in the proportion of women reporting fever during the second visit can be linked to treatment received at recruitment. The time difference between the first two visits was often only four weeks or less.

b) By area

In general the frequency of febrile illness did not vary significantly with area of residence (Table 9.2).

The decline in prevalence rate of reported febrile illness corresponded with the decline in the prevalence rate of malaria parasites among the women attending antenatal clinics (Table 9.3).

c) By parity

Although febrile illness had high frequency in all visits, except visit 2, and in all parities, women of para 0 and 1 had higher frequency of febrile illness than those of parity 2 and over (Table 9.1). This was most marked in visit 2 (see Table 9.4). The difference during this visit was statistically significant (X^2 = 23.4, df = 2, P <<0.001), while the differences observed during the other visits were not statistically significant.

d) By treatment group

The women who were treated only when ill did not have a higher prevalence of reported febrile illness except at their second visit. Otherwise the differences between treatment groups and by gestation were not statistically significant.

This supports the view that the observed decline noted in Sections 9.2 a) and b) was due to the advancing gestation and not due to the administration of drugs, Table 9.5.

e) By age

The effect of age, like that of parity, was seen most clearly during the second antenatal visit. The differences during other visits were not statistically significant. The slight decline in the prevalence of febrile illness by gestation was consistent with the findings reported above.

9.2.2 Parasitaemia during antenatal and postnatal periods

a) By visit

Peripheral parasite prevalence varied considerably during the period that

each individual was in the study. Of 316 cases who had blood slides done for malaria parasites at first visit, 111 (35.1%) were positive. The prevalence rate dropped consistently in successive visits to a minimum at delivery and postnatal visits (Table 9.7).

Density of parasites at first visit was generally high. About half of those who had parasites had three thousand parasites per cc of blood or higher. The parasite density at third visit was lower than second and first visits. Most were lower than three thousand per cc of blood. At the fourth visit, the parasite density tended to be similar to the third visit and the fifth visit. In short, the density of parasites was high in the second trimester but it dropped in the third trimester and the postnatal period.

The density of placental parasites was higher than that of peripheral parasites and parasite density was lowest in the cord blood.

a) By area of residence

The difference in parasite prevalence by area of residence was not statistically significant. The drop in parasite prevalence according to the stage of the pregnancy was more marked in areas A and B which were higher in the second trimester and lower in the third trimester than area C (Table 9.7).

The mean log of parasite densities varied by area of residence, but the difference was not statistically significant (Table 9.8). At delivery, area C had a higher mean parasite count than areas A and B, a difference which was statistically significant ($F = 3.67$, $df = 2,192$, $P < 0.05$).

c) By age

Mothers' age did not significantly affect parasite density. The women aged 15-24 years had the highest value (Table 9.8). The same results obtained for all the five visits 1, 2, 3, 4, 5 and 6.

The log of density during the antenatal period and at delivery was higher among the younger women but the difference was not statistically significant. This was true also of the findings at the postnatal visit. It was noted that there

was also no significant difference in placental and cord parasite densities by age of mothers.

The women who were seen for the first time during delivery and had not been followed up during the antenatal period showed a marked difference in parasite prevalence by age. 64% of women who were aged below 25 years had parasitaemia compared to 43.5% of those 25-34 years and 26.3% for those who were 35 years and older. The difference was statistically significant ($X^2 = 8.7$, df = 2, P <0.05)..

d) By parity

There was a marked difference between parities with regard to prevalence of parasitaemia. At first visit the primigravidae had more than twice as high parasite prevalence as multiparous women (Table 9.9). The parasite prevalence declined with gestational age in all parities but again was more marked among primigravid women. After delivery the prevalence of parasitaemia was low in all parity groups and the difference between parity groups was no longer significant. For the women seen for the first time in labour, the difference of parasite prevalence by parity is even greater: 56.3% for primiparious, 54.4% para ones, and 33.3% para twos and over. This difference was statistically significant ($X^2 = 13.1$, df = 2, P <0.01).

It is also to be noted that the overall prevalence rate of parasitaemia at delivery was much higher for this group of women compared to the women who were followed up during the antenatal period.

The density of parasites was much higher among primigravidae at all visits and they remained constantly high until the time of delivery when the density dropped considerably and there was no longer any difference between the primigravidae and multigravidae. The difference in mean counts between primigravidae and multigravidae at first visit was statistically significant (F = 3.34, df = 10/283, P <0.005). The same difference was seen during the second visit (F = 3.5, df = 10/236, P <0.005), third visit (F = 2.09, df = 10/227, P <0.025)

and fourth visit (F = 2.55, df = 10/243, P <0.005), but not at the fifth (post natal) visit (F = 1.45, df = 3/283, P >0.05). The parasite counts decreased with increasing parity and were highest in primiparous women and lowest in grandmultiparae. But the difference was not statistically significant in any visit. The parasite densities also decreased with increasing gestation, reaching the lowest value at the postnatal visit.

The placental and cord parasite densities did not differ significantly with parity either.

e) By treatment group

Parasite prevalence rates at all visits did not differ significantly by treatment groups, but declined with increasing gestational age (Table 9.11).

It is clear from Table 9.11 that although the group that was only being treated when ill had slightly higher parasitaemia prevalence rates the difference was not statistically significant.

The mean parasite count reflected a similar result as for the prevalence in that there was a decided decline in mean parasite counts as pregnancy progressed, but at each antenatal visit the difference between the groups was not statistically significant (Table 9.12). Although these differences were not statistically significant, the group that received drugs at antenatal clinics had the lowest mean parasite count at every visit, while the group that was given treatment ony when ill had the highest counts at every antenatal visit.

Table 9.12 also shows that the parasite counts were higher during second trimester than in third trimester.

The impact of the treatment groups was more obvious on placental and cord parasite counts which were lowest for the monthly prophylaxis group and highest for the group treated only when ill (F = 3.64, df 2,259, P <0.050 and F = 3.98, df = 2,280, P <0.025 respectively).

f) By febrile illness

The women who had a history of febrile illness were much more likely to

have parasites in their blood than those who did not (X^2 = 6.17, df = 1, P <0.025). This difference was less during the second, third and fourth visits and was just significant at the fifth visit.

g) By haemoglobin type

Haemoglobin type also did not seem to influence prevalence of parasitaemia (Table 9.13), neither did it influence the mean parasite count which was 722.67 ± 169.8 for AA and 826.67 ± 60.0 for AS.

9.2.3 Haematocrit level

The mean haematocrit level was high throughout the study period. Very few women had haematrocrit levels below 30% at any time during the study. Table 9.14 presents the mean haematocrit levels by area and clinic visit. The differences by area of residence were small and not statistically significant at all antenatal and postnatal visits.

As pregnancy progressed, there was a definite trend for haematocrit values to rise in all the areas.

The difference by parity was very small. The primiparous women had the lowest value at all the visits but the difference was not statistically significant at any of the clinic visits (Tabe 9.15).

The mean haematocrit levels after delivery were much higher than any time during pregnancy.

The proportion of women with haematocrit values below 30% decreased with gestation. It was highest at the first visit (12-16 weeks) and lowest at the fifth visit which was postnatal (see Table 9.16).

The difference in mean haematocrit levels by haemoglobin type was small and not statistically significant.

9.2.4 Weight and weight gain in pregnancy

The average weight of the women at the end of the first trimester when

they were recruited into the study was 56.6 kg. The mean weight gained during the second and third trimester was 2.5 kg per trimester. At delivery the women lost a mean of 6 kg and gained 0.2 kg by the end of the puerperium. Table 9.17 presents the mean weights at 12 and 36 weeks by treatment group. The difference in weight gain during pregnancy by treatment group, parity, age and area of residence were small and not statistically significant (Table 9.18).

9.2.5 Chloroquine in the urine

Two hundred and eleven cases were examined for urine chloroquine at their first visit using the Haskin's test as previously described. Of these, 18 (8.2%) were positive. This percent positivity increased a little during subsequent visits but not as much as would have been expected. This can be explained, at least partly, by the fact that for group one the examination took palce at least 4 weeks after chloroquine intake, whereas group two might have been regular with their weekly intake of chloroquine. Many women would not have been tested at the appropriate time since the Haskin's test can only detect chloroquine up to 10 days after intake (Rombo, 1985). Chloroquine in urine remained rather low throughout the pregnancy. The test used was supposed to be more sensitive than the one that had been used in the study before, the Dill Glazko test which was shown by Lars Rombo to be less sensitive than the Haskin's test (Rombo, 1985).

The pattern of positivity shown in Table 9.19 indicates that the frequency of positive urines started moderately low (8.2%) and increased somewhat to 14.1% and 12.1% at visits 2 and 3 respectively and dropped again to 8.1% and 7.3% at delivery and at the end of the postnatal period. This pattern was consistent in all the three areas. Women from area B had generally higher urine positivity rates as compared to areas A and C. Area A tended to show the lowest rate of chloroquine positivity and the rate dropped to very low levels after delivery. This may reflect the pattern of chloroquine consumption in the different areas.

9.3 DISCUSSION

This chapter presents the experience of the study population during the progress of pregnancy from its recognition at 12-16 weeks to its termination at 40 weeks and at the complete physiological return to normal apart from lactation. The main outcomes presented include:

- history of febrile illness
- prevalence and intensity of parasitaemia
- weight gains in pregnancy
- occurrence of anaemia through haematocrit estimation
- excretion of chloroquine in the urine.

These outcomes were examined in the light of independent variables that were expected to affect these outcomes:

- maternal age
- area of residence
- parity
- period of amenorrhoea
- chemoprophylaxis status, and
- haemoglobin types.

The key findings presented are that prevalence of febrile illness and parasite prevalence and density are higher in primigravid women and decline with increasing parity and that these outcomes also decline as pregnancy progresses.

Age, haemoglobin type and the treatment group showed effects on the same outcomes but the effects were too small to produce statistically significant differences in values manifested.

The findings presented in this chapter confirmed earlier findings in that malaria parasite prevalence was much higher among primigravid women and decreased with increasing parity and the drop in the prevalence rate with increasing gestation was most dramatic among the primigravidae. The density of

parasite decreased only slightly during pregnancy but dropped dramatically after delivery.

Several workers have shown that the effects of malaria in pregnancy are most marked in primigravidae (Cannon, 1958; 1978; Reinhardt, 1978. Bray. 1979). Our findings were in agreement with these findings.

Younger women tended to have higher prevalence rates and higher intensity of infection than older ones. These differences by age were not statistically significant. The main factors influencing these variables appeared to be parity and gestation.

The findings of Blacklock and Gordon (1925) suggested no relationship with the age of the mother. His findings were supported by Cannon (1958) and Bray (1959). The study by Reinhardt (1978) was different as it showed decreasing incidence of parasitaemia with increasing age of the pregnant woman.

No clear cut difference was obtained in the rate of parasitaemia and intensity of malaria infection between the various treatment groups. There was no significant difference between the women who obtained monthly chemoprophylaxis from antenatal clinics as compared to those who received it weekly from CHWs or as compared to those who obtained chemotherapy from CHWs whenever they felt ill. This was a consistent observation throughout pregnancy. Thus, the rates still declined with increasing gestational age and were higher in primigravidae. No statistically significant difference from one treatment group to another was detectable throughout pregnancy or at the postnatal visit.

The group of women from outside the Saradidi community who were studied in the same way as the study group, when they came in labour to deliver at nearby health facilities, manifested much higher prevalence rates and itensity of infection at the time of delivery than the study population. This finding suggested that there was a difference between the study population and the general population of pregnant women in Saradidi and its surrounding

communities. This difference may be explained by the fact that the study women were seen at antenatal visits every month and treated for any complaints they had, they also had access to chloroquine for chemotherapy at the village level provided by CHWs and they were also from a community that had been exposed to intensive education and information regarding malaria control through a community based health care programme for at least five years.

Nevertheless, the intensity of infection, as indicated by parasite counts, was lowest for those receiving monthly chemoprophylaxis and highest among those treated only when ill, although the difference was not statistically significant throughout pregnancy and pueperium. It should be noted that the response of P.falciparum in Saradidi had decreased considerably between 1980 and 1987 (see Chapter 4). Thus, the lack of clear difference between the treatment groups could also be explained by the fact that the drug used was not adequately effective, and the monthly dosage of 10 mg per kg might have been too low.

Urine chloroquine was not found as frequently as was expected at the study design stage. The results could be taken to mean lack of compliance, irregularity of intake, inappropriate timing of the tests or combinations of these. Given the fact that the Haskin's test can only detect chloroquine in urine for up to ten days (Rombo, 1985) most of the women on monthly chemoprophylaxis would not have been tested at a suitable time for them to have chloroquine or its by-products in their urine. Irregularity of the weekly intake would also be a valid explanation which further supports the view that the weekly regimen for pregnant women in holoendemic areas is an impractical strategy, not feasible in situations of greatest need.

Lars Rombo in 1985 compared the sensitivity of Haskin's test, Wilson Ederson test and Dill-Glazko test. He showed conclusively that Haskin's test was the most sensitive of the three tests. An intake of 5 mg/kg of chloroquine could be detected in urine up to ten days. The urine chloroquine positivity was

consistently low because most of the subjects, except the weekly group, would have been visiting the Clinic long after 10 days following chloroquine intake. The group that received their monthly chloroquine at antenatal clinics for example was always examined after 30 days or more. The weekly prophylaxis group did not take the drug under supervision. It was hoped that the information and education given by the CHWs would have been adequate in encouraging them to take their drug weekly. It is clear from these results that although the women in the weekly group received their drugs regularly, they did not take them as regularly as was expected of them.

This negative finding was important in that it showed clearly that outside of a research setting it is very difficult to ensure that drugs are taken regularly especially when the woman is not ill. For this reason weekly chemoprophylaxis for pregnant women, although theoretically important, is impossible to implement operationally in rural communities like Saradidi.

MacCormack and Luihula (1983) described a chemosuppression programme under ideal conditions but which still failed because of logistic, administrative and social problems. Among the reasons for not taking the drugs was that the target community were not convinced that regular chloroquine intake prevented malaria and kept it instead for the treatment of febrile illnesses. This may partly explain why our weekly group performed more poorly than the monthly group who took the drugs under supervision at antenatal clinics. Chloroquine is an expensive drug. A course for treatment of malaria costs $0.80 which many families in Saradidi cannot afford. The temptation of keeping the drugs given for chemoprophylaxis of pregnant women for the treatment of anyone in the household whenever they suffered febrile illness may have been too great as the women saw this use of the drugs as more important than chemoprophylaxis.

The problem of itching was reported by MacCormack and Luihula (1983) as a common cause of noncompliance in children in the Tanzanian study. A study

by Spencer et al. in Saradidi (1987) showed that chloroquine-induced pruritus occurred in 20.3% of adults who were treated with chloroquine and followed up for two days. This study is briefly presented in Chapter 2. This problem could have contributed to poor compliance particularly in the weekly group.

MacCormack and Luihula (1983) concluded that the failure of chemosuppression was due not only to the failure of the distribution system but also a growing unwillingness of the target population to swallow the drug.

In an earlier study in Saradidi, discussed in Chapter 5, Kaseje et al. (1987) reported that only 30% of the targeted pregnant women participated in the chemoprophylaxis for pregnant women. The reasons for not taking it included lack of information about the availability of the service (53% of the women interviewed), chloroquine-induced itching (8.4%) and that they saw no reason for taking any drugs when not sick (14%).

Many people in Saradidi also associated abortions and stillbirths with chloroquine intake (Kaseje et al., 1987), although there is no evidence to substantiate that chloroquine in usual doses for treatment or chemoprophylaxis causes foetal loss (Bruce-Chwatt, 1986). However, this belief is strong in Saradidi and is difficult to change since unmarried, pregnant girls are known to use overdosage of chloroquine to induce abortions. In addition abortions occurring as a result of malaria infection may be blamed on chloroquine if chloroquine or chloroquine-like tablets were taken at the same time for treatment of the illness.

All the measures of morbidity: febrile illness, haematocrit value, prevalence of parasitaemia and parasite counts improved as pregnancy progressed from second trimester to the third. The frequency of reported febrile illness, parasitaemia rates and parasite density were highest early in pregnancy and were lowest at delivery and postnatally. Haematocrit levels remained high throughout pregnancy but also showed an upward trend with increasing gestational age to a maximum value at the postnatal visit.

Similar results have been obtained by other workers. Several workers have studied malaria in pregnancy (Clark, 1915; Blacklock and Gordon, 1925: Bruce-Chwatt, 1952; Cannon, 1958), but they did not give a clear insight into the prevalence of malaria in pregnancy and its relation to parity, age, and gestation.

Clark, 1915; Grantham, 1938; Torpin, 1941; Bruce-Chwatt, 1952; Cannon, 1958; Mennon, 1972; Lewis, 1973 and Reinhardt, 1978 all tended to stress the relationship between malaria and late pregnancy but without defining the timing of late pregnancy.

A study by Campbell in 1980 working in central America did not show a difference during the different trimesters.

Some studies have suggested different results; for example, Kortman, working in East Africa (1972) showed increased parasite rates before 25 weeks and after 36 weeks. Grantham (1938) reported increased parasitaemia rate during puerperium, a finding that has not been confirmed by other workers. The Kortman results are consistent with ours up to 36 weeks. It is interesting that in his study (1972) where he compared the prevalence of malaria parasitaemia in the same group of subjects during pregnancy and the subsequent puerperal period and the malaria prevalence in these puerperal women with that of non-pregnant women, he did not show any increase of malaria in the postpartum period. This study was repeated by Bray in 1979 who came up with comparable results.

Pingond (1969) reported highest parasite rates between 16 and 28 weeks, followed by a steady decline to the lowest level at term. Bray (1979) reported high parasite rates and lowered IFA titres of malaria antibodies before the 24th week. He concluded that immune response to malaria parasitisation inhibited in early pregnancy but improved as pregnancy progressed.

Pringond (1969), Gilles (1969) and Bray (1979) have all reported increased occurrence of clinical malaria from the end of the first timester. Brabin (1983) concludes that the peak prevalence of infection occurs at 13-16 weeks. It is suggested that the observed manifestations of malaria in pregnancy may be due

to temporary immunosupression (Brabin, 1983) or a switch to a less sensitive effector mechanism (Erling, 1982b). Humoral immunity is believed not to be reduced during pregnancy (Loke, 1978; Carter and Dresser, 1983). Cell mediated immunity appears to be more suppressed during pregnancy (Glapsys and Clark, 1983; Loke, 1978).

The results obtained in this study did not show a clear cut difference between the women who had homozygous AA haemoglobin type and those who had the heterozygous (AS) haemoglobin type. Parasite rates and density were lower and haematocrit levels were higher among the women with AS haemoglobin but the difference was never statistically significant.

Studies elsewhere have shown that people with AS haemoglobin are at an advantage over those with AA in high malarial endemicity areas. The studies show that in aerobic conditions (18% oxygen), P.falciparum invades at the same rate in AA and AS but in relative anaerobic conditions (5% oxygen) as is found in the vasculature of deep organs, changes occur in the AS cells which appear to diminish penetration by parasites and retard the growth of intracellular parasites (Pasvol et al., 1978). The AS cells also show an early tendency to sickle when parasitised and develop rheological characteristics which predispose to elimination of cells and, with them, parasites by phagocytosis (Roth et al., 1978). The integrity of the cell membrane is also impaired allowing potassium ions to leak out making the intracellular environment metabolically unacceptable to the parasites (Friedman et al., 1979). Therefore occurrence of high parasite prevalence is more common among people with normal (AA) haemoglobin types.

There is therefore relative resistance of AS red blood cells to infection by P.falciparum which derives from a combined effect of several mechanisms. This effect was not shown in our results mainly because of the sample size. There were very few women with AS haemoglobin and hence valid comparisons were limited. It is also possible that the physiological and biochemical changes occu ring during pregnancy overwhelm the other factors that enhance the

resistance of the pregnant women to malarial infection.

In his review paper based on his studies in the Gambia, McGregor (1984) reports that malaria is an important cause of anaemia in malarious primigravidae which can be assessed on the basis of haematocrit levels as an indicator of the problem. Anaemia also decreases with increasing parity and gestation. It was also observed that anaemia could occur without a febrile illness. The results presented in this chapter were surprising in showing that anaemia was very infrequent in the study population, perhaps due to easy access to antimalarial drugs and information as already discussed. The mean levels of haematocrit remained high throughout pregnancy and tended to increase with gestation. This is consistent with McGregor's findings in 1984.

Primigravidae are more prone to anaemia (Kortman. 1972; van Dongen and van Hoof, 1982), but Kortman could not explain this due to lack of correlation between parasite density and severity of anaemia.

Gilles (1969) could not demonstrate the relationship between parasitaemia and anaemia. The study of red blood cells showed that haemolysis continued even two weeks after the disappearance of parasites. He concluded that the onset of anaemia was in the second trimester (16-24th week), but that it improved in the third trimester. Lawson (1967) put the onset at 20-28 weeks, while Kortman did not observe a predilection gestation period.

The anaemia observed was haemolytic (Gilles, 1969; Kortman, 1972). The demand for increased haemopoiesis and foetal growth may lead to folic acid deficiency and megaloblastic anaemia.

The rupture of parasitised cells and their removal from circulation by phagocytosis cannot explain the degree of anaemia observed in malaria especially in low grade infections. The destruction of both the parasitised and non-parasitised red blood cells occur (Maegratih, 1948). This is perhaps due to an autoimmune reaction (Facer, 1980).

Parasite density in placentae correlated well with the anaemic state of

the mother (Jilly, 1969; Reinhardt, 1978). It may be that the placenta plays a role in the immunopathological process leading to anaemia in pregnancy.

Malaria-induced anaemia is more severe in primigravidae and is highest around 18 weeks which is a little earlier than the onset of haemodilution (Koller, 1982). It follows the peak period of prevalence of clinical malaria (12-18 weeks). The anaemia can be explained on the basis of parasitisation of red blood cells or on the basis of humoral immunological reactions directed against the surface of erythrocytes either as autoantibodies or directed against parasite antigens associated with the surface of erythrocytes (Maegraith, 1948; Facer, 1980).

Such an apparent immunologically provoked haemolytic anaemia develops after peak infection but is not correlated to its magnitude (Gilles, 1969; Kortman, 1972). Thus anaemia occurs mainly in early pregnancy and decreases with increasing amenorrhoea such that there is insignificant difference between malarious and non-malarious women by delivery time.

Heaviest stress on haemoglobin occurs at 16-26 weeks of amenorrhoea after the peak of malaria prevalence but before the onset of the physiological anaemia which starts at the end of the second trimester.

From the presented and discussed findings, it appears that susceptibility of the pregnant woman changes with the period of amenorrhoea as measured by prevalence and density of parasitaemia which decreased with increasing amenorrhoea period and haematocrit which tended to increase. The data suggest that the pregnant woman tends to regain some control over malaria in later stages of pregnancy.

The pregnancy weight and weight gain during pregnancy reported in this chapter would be considered normal for a third world community like Saradidi. The weight gain was affected neither by parity nor chemoprophylaxis.

It was important to monitor pregnancy weight and hence weight gain since these two maternal weight parameters have been shown to correlate well with birthweight (Arroyaave, 1975; Lechtig et al., 1975). The two can predict

the birth weight quite well, a fact which has been reported on by various authors (Beilly and Kurland. 1945; Love and Kinch. 1965; O'Sullivan et al.. 1965, Thompson et al., 1968; Eastman and Jackson, 1968; Niswander et al., 1969; Ademowore et al., 1972).

Several research workers have also shown the impact of maternal food supplementation on birth weight (Habischit et al.. 1974; Jacobson. 1974; Lechtig et al., 1975). In 1975 Simpson et al. demonstrated a decrease in the prevalence of low birthweight babies as pregnancy weight and weight gain increased. He demonstrated a linear correlation between birthweight and weight gain in pregnancy.

In this study the weight gain was measured from around 16 weeks of amenorrhoea up to 36 weeks of gestation The mean weight gain was 4.9 kg. Thus the total weight gain should have included 1 kg in first trimester and another 1 kg during the last month when the growth rate is about 200 grams per day. The corrected mean weight gain should be 6.9 kg which is within the normal range of weight gain in pregnancy (Falkner and Tanner, 1978). This is consistent with the mostly normal birthweights presented in Chapter 10.

The results presented in this chapter suggest that:

a) there was no significant difference between outcome variables in the women who received regular chemoprophylaxis and those who were treated only when ill with fever;

b) there was a statistically significant difference between primigravid women and multigravid women in terms of febrile illness and parasite prevalence and density;

c) the effect of malaria was most evident during the second trimester but decreased during the third trimester and was lowest in the postnatal period.

These factors should be taken into account in planning antimalarial action for pregnant women, and particularly in situations of scarce resources,

poor infrastructure and inadequate logistical support. If an effective drug is used it can be targeted at the most vulnerable population (the primigravidae) at the most vulnerable period (during second trimester or as soon as pregnancy is confirmed until the end of the third trimester) and through the most feasible and cost-effective means (monthly antenatal clinics). The effective drug should be made available and accessible, at the village level, to the rest of the population. Effective information and education on self-diagnosis, chemotherapy, prevention and control of malaria are important components of the package.

Table 9.1 Percent of women reporting febrile illness by vist and parity

Visit	N	PARITY/PERCENT FEBRILE ILLNESS				
		0 %	1 %	2 %	>2 %	All Parities %
1	329	77.1	82.7	87.5	83.5	81.8
2	262	44.7	35.0	16.7	13.0	29.8
3	205	67.2	71.4	56.0	63.5	65.9
4	210	78.5	69.1	56.5	69.7	70.5
5	146	68.2	77.8	78.6	62.0	69.9

Table 9.2 Percent of women with history of febrile illness by area of residence and visit

Visit	N	AREA			ALL AREAS
		A	B	C	
1	335	81.6% (124)	83.0% (107)	81.5% (44)	82.0% (275)
2	268	30.6% (37)	29.3% (31)	24.4% (10)	28.1% (78)
3	211	59.6% (56)	70.4% (57)	77.8% (28)	69.3% (141)
4	217	69.0% (69)	67.1% (59)	82.8% (24)	73.0% (152)
5	153	72.5% (50)	67.2% (43)	68.4% (13)	69.4% (106)

Table 9.3 Prevalence of febrile illness and malaria parasites by gestation

Gestation (weeks)	N	Proportion reporting fever	N	Proportion with positive slides
12-16	335	275 (82.1%)	315	111 (35.1%)
20	295	230 (78.0%)	268	78 (29.1%)
40	237	152 (70.0%)	212	35 (16.5%)
6 post partum	152	106 (69.7%)	151	25 (16.6%)

Table 9.4 Percent of women reporting febrile illness by visit and parity

Visit	PARITY/PERCENT FEBRILE							
	0		1		2		>2	
	No.	%	No.	%	No.	%	No.	%
1	91/118	77.1	58/70	82.9	35/40	87.5	81/97	83.5
2	42/94	44.7	21/60	35.0	5/30	16.7	10/77	13.0
3	45/67	67.2	3549	71.4	14/25	56.0	40/63	63.5
4	51/65	78.5	38/55	69.1	13/23	56.5	46/66	69.7
5	30/44	68.2	38/36	77.8	11/14	78.6	31/50	62.0

Table 9.5 Percent of women with febrile illness by clinic vist and treatment group

Visit	N	Monthly prophylaxis		Weekly prophylaxis		Treatment when ill		All groups	
1	331	87	82.9%	91	79.1%	93	83.8%	271	81.9%
2	267	25	28.4%	24	24.5%	30	35.3%	78	29.2%
3	210	43	63.2%	45	67.2%	52	69.3%	140	66.7%
4	216	49	70.0%	49	64.5%	54	77.1%	152	70.4%
5	150	39	72.2%	35	68.6%	40	66.7%	104	69.3%

Table 9.6 Percet of women reporting febrile illness by visit and age

Visit	N	AGE/PERCENT FEBRILE						All ages	
		15-19 years		20-24 years		25-44+ years			
1	327	115	78.23	83	83.0%	70	87.5%	268	81.96%
2	263	51	42.14%	21	36.84%	4	4.7%	76	28.90%
4	214	66	73.33%	44	64.70%	40	71.42%	150	70.09%
5	149	40	67.80%	32	72.73%	31	67.39%	103	69.13%

Table 9.7 Malaria parasite prevalence by clinic visit and area of residence

Visit	N	AREA AND POSITIVITY OF SLIDES							
		A		B		C		ALL	
		No.	%	No.	%	No.	%	No.	%
1	316	45/143	31.5	55/124	44.4	11/49	22.5	111/316	35.1
3	199	24/89	27.0	13/79	16.5	6/31	19.4	43	21.6
4	212	16/100	16.0	12/85	14.1	7/27	25.9	35	16.5
5	151	11/66	16.7	10/65	15.4	4/20	20.0	15/151	16.6

Table 9.8 Log of mean density of parasites by clinic visit and area of residence

Visit	N	AREA			SIGNIFICANCE		
		A	B	C	F	df	P
1	301	6.77 ± 1.4	6.43 ± 1.7	8.9 ± 1.9	0.57	2294	> .05
2	253	5.94 ± 1.4	8.25 ± 2.2	8.99 ± 2.1	0.55	2251	> .05
3	190	5.12 ± 1.5	7.98 ± 0.8	5.49 ± 1.5	0.79	2187	> .05
4	195	6.56 ± 0.8	3.5 ± 1.2	7.86 ± 2.6	1.77	2191	> .05
5	150	3.39 ± 1.3	1.15 ± 0.8	1.89 ± 0.7	1.52	2177	> .05

Table 9.9 Percentage of women with positive blood slides by visit and parity

Visit	PARITY/POSITIVE SLIDES									
	0		1		2		>2		ALL	
	No.	%	No.	%	No.	%	No.	%	No.	%
1	59/113	52.2	20/68	22.4	10/37	27.0	20/88	22.7	110/310	35.5
3	21/60	35.0	11/48	22.9	3/24	12.5	7/60	11.7	43/193	22.3
4	15/60	25.0	10/51	19.2	4/34	13.0	5/65	7.6	34/205	16.6
5	8/45	19.6	7/36	18.2	2/14	13.3	8/50	16.0	25/145	17.2

Table 9.10 Geometric mean parasite density by parity and visit

Visit	No.	PARITY/GEOMETRIC MEAN PARASITE DENSITY 0	1	2	>2	ALL
1	291	1214.7	669.3	289.6	417.3	647.7
2	247	1557.6	1053.3	421.3	270.9	825.8
3	183	752.3	501.3	536.0	11.5	480.3
4	187	1035.7	401.6	367.7	39.5	461.1
5	142	235.7	174.7	184.8	289.1	221.1

Table 9.11 Percent women with positive blood slides by clinic visit and treatment group

Visit	N	TREATMENT GROUP/BLOOD SLIDE POSITIVITY Monthly prophylaxis		Weekly prophylaxis		Treatment when ill		All groups	
1	312	36	35.0%	37	33.9%	37	37.0%	110	35.3%
2	162	19	32.2%	19	34.6%	17	35.4%	55	33.95%
3	198	13	20.3%	14	21.2%	15	22.1%	42	21.2%
4	211	11	15.9%	11	15.3%	13	18.6%	35	16.6%
5	149	7	13.7%	11	22.0%	7	14.6%	25	16.8%

Table 9.12 Parasite density by clinic visit and treatment group

Visit	No.	TREATMENT GROUP/DENSITY Monthly prophylaxis	Weekly prophylaxis	Treatment when ill	All Women	Significance df = 1,284
1	297	677.3 ± 54.04	722.7 ± 67.01	889.9 ± 73.79	763.3 ± 343.57	0.57
2	253	593.9 ± 54.19	825.1 ± 83.37	898.9 ± 77.52	772.6 ± 210.55	0.55
3	185	512.0 ± 57.83	297.9 ± 29.79	549.3 ± 55.66	453.1 ± 401.69	0.68
4	194	256.0 ± 731.00	349.9 ± 11.81	786.4 ± 25.91	464.0 ± 351.77	1.77
5	148	120.8 ± 14.75	409.3 ± 44.31	147.7 ± 16.84	225.9 ± 512.16	2.16

Table 9.13 Percent women with positive blood slides by clinic visit and haemoglobin type

Visit	No.	HB TYPE/PARASITE POSITIVE SLIDES AAs		As		ALL				
		No.	%	No.	%	No.	%	X^2	df	P
1	230	67	32.8	10	38.5	77	33.5	0.31	1	>0.05
3	186	37	22.7	3	13.0	40	21.5	1.11	1	>0.05
4	192	27	16.1	3	12.5	30	15.6	0.44	1	>0.05
5	138	12	10.2	7	35.0	19	13.8	1.36	1	>0.05

Table 9.14 Mean haematocrit value by visit and residence area

Visit	No.	Area of residence A	B	C	ALL	F	P
1	322	33.7 ± 4.1	33.0 ± 2.8	33.5 ± 4.2	33.4 ± 4	.12	>0.05
2	275	33.8 ± 3.6	33.7 ± 3.6	33.2 ± 3.6	33.6 ± 4	.48	>0.05
3	186	34.8 ± 4.0	34.2 ± 4.5	34.6 ± 4.6	34.4 ± 4	.16	>0.05
4	198	36.8 ± 3.7	34.8 ± 3.9	35.2 ± 6.3	35.8 ± 5	2.23	>0.05
5	147	37.8 ± 4.5	36.1 ± 4.8	36.9 ± 3.7	37.0 ± 5	2.19	>0.05

Table 9.15 Mean haematocrit values by parity and clinic visit

Visit	No.	PARITY/HAEMATOCRIT VALUE (%) 0	1	2	>2	ALL	F	df	P
1	312	32	33.9	33.9	33.7	33.6	1.3	2304	>0.05
2	266	33.1	34.0	35.2	33.6	34.0	1.2	2256	>0.05
3	179	33.0	33.1	36.3	34.1	35.1	1.5	2168	>0.05
4	189	34.8	35.4	35.6	36.9	35.7	1.0	2179	>0.05
5	203	36.8	37.5	38.3	36.5	37.3	0.8	2130	>0.05

Table 9.16 The proportion of women with haematocrit value below 30% by clinic visit

Visit		1	2	3	4	5
Haematocrit below	No.	62	31	23	19	8
30 (%)	%	19.3	11.3	12.4	9.6	5.4
N		322	275	186	198	147

Table 9.17 The mean weight at 12 and 36 weeks and weight gain between the two visits by treatment group

Treatment group	No.	Mean weight at 12 weeks	Mean weight at 36 weeks	Weight gain
1	107	56.4 ± 6.4	61.4 ± 4.1	5.0 ± 2.1
2	116	56.8 + 6.5	61.4 + 6.8	4.6 + 1.3
3	110	56.2 ± 6.5	61.7 ± 3.5	5.5 ± 3.0
ALL	333	56.5 ± 6.5	61.4 ± 4.8	4.9 ± 2.1

Table 9.18 Mean weight gain at 36 weeks by area of residence, parity and treatment group

Factor Observed	Mean weight (kg)	F	Significance Df	P
Area A	4.5	1.16	2200	>0.05
Area B	5.4			
Area C	4.6			
Para 0	4.7			
Para 1	5.2			
Para 2	4.5	0.19	3185	<0.05
Para 3+	4.8			
Treatment group				
Monthly	4.0			
Weekly	4.9	2.31	2199	>0.05
When ill	5.5			

Table 9.19 Percent women with chloroquine in their urine at the time of their clinic visit by visit and area of residence

Visit	N	AREA/URINE POSITIVITY FOR CHLOROQUINE										
		A		B		C		ALL				
		No.	%	No.	%	No.	%	No.	%	X^2	df	P
1	211	8	8.8	7	7.9	3	8.0	18	8.2	1.7	2	>0.05
2	227	9	9.0	18	19.2	5	14.1	32	14.1	4.16	2	<0.05
3	173	6	7.7	10	15.2	5	12.1	21	12.1	3.9	2	<0.05
4	172	2	2.6	10	14.3	2	8.1	14	8.1	6.7	2	<0.05
5	137	4	6.2	5	9.3	1	5.6	10	7.3	1.51	2	>0.05

CHAPTER 10

PREGNANCY OUTCOME AT DELIVERY

10.1 INTRODUCTION

The study design, methods, materials and subjects have been described in Chapter 8. This chapter presents the results of the study on maternal health, pregnancy outcome and related parameters measured at the time of delivery.

Briefly, 361 women recruited into the study over a period of a year and a half. The women were randomised into three treatment groups as follows:

Group 1 received chloroquine phosphate monthly during ante-natal visits at the dosage of 10 mg/kg body weight;

Group 2 received from CHWs chloroquine phosphate at the recommended dose of 5 mg/kg body weight weekly but the intake was not supervised, and;

Group 3 were treated by CHWs at the village level whenever they had fever and they requested treatment. This was the control group.

In addition to the above three groups, another 200 women who were not in the study were examined in a similar way at delivery to test the possible influence that the study itself could have had on the control group.

The effectiveness of each of these regimens was evaluated using indicators described in Chapter 8. This chapter discusses the indicators assessed at the time of delivery. The main dependent variables observed were:

- birth weight
- peripheral parasitaemia at delivery
- placental parasitisation
- congenital malaria infection

Each one of these was assessed in terms of the main explanatory variables; the treatment group, parity, maternal age, haemoglobin type and area of residence. It is to be noted that the areas were defined according to the first phase of the

larger Saradidi Study which ended in 1984 and is summarised in Chapters 1-6. During that time area A received chloroquine for treatment at the village level and for chemoprophylaxis of pregnant women; area B received the village based chemotherapy alone but area C served as the comparison area. The communities in areas A and B had also been exposed and involved longest in the activities of the Primary Health Care Project and may have had much more awareness regarding health issues. This exposure could have affected the health seeking behaviour of the women from these three areas differently. This is why the effect of area of residence is considered for each variable.

10.2 RESULTS

10.2.1 Date of Delivery

Most of the deliveries occurred in 1986. Of the 361 women recruited into the main study 284 (78.7%) delivered in 1986, 20 (5.5%) delivered in 1985 and 30 (8.3%) delivered in 1987. The remaining 27 (7.5%) had unrecorded delivery dates.

Although malaria is holoendemic in Saradidi the intensity of transmission varies from season to season as described in Chapter 3. The season of delivery could influence both the experience of the study subjects with malaria infection and outcome of pregnancy. Figure 10.1 presents the number of deliveries by month of delivery. 135 (37.4%) of the women delivered during the dry months, 129 (35%) delivered during the wet months while 70 (19.4%) delivered during the moderately wet period. Thus the deliveries were all distributed throughout the year with fewer occurring in the months of October and November (Figure 10.1). Most of the babies were born full term (Figure 10.2).

10.2.2 Outcome of Deliveries

There were 14 (4%) non-live births. Out of 334 cases for whom the method of delivery was recorded, 329 (98.5%) were spontaneous vaginal

deliveries and only 5 (1.5%) were operative, three being forceps extraction and 2 caesarean sections. The result here cannot be considered as the rate of normal deliveries in the study area since the study sample was deliberately biased in favour of normal deliveries, given the facilities that were available for handling the deliveries. The number of non-live births was too small for analysis to find out the factors affecting it.

The mean height of the women in the study was 160.4 cm (Figure 8.3). Since the minimum height consistent with adequate pelvis is considered to be 150 cm or more, we found that nearly all of our subejcts should have had adequate pelves. Only one woman could have had cephalo-pelvic disproportion as an obstetric complication.

52.9% of the newborns in the study were male and the rest, 47.1%, were female.

10.2.3 The place of delivery

According to the design of the study, all the deliveries should have taken place at the Saradidi Clinic. Most of them did, 280 (77.6%). There was again a strong bias towards institutional delivery in the study since it was required to allow measurements, examinations and investigations required in the study to be undertaken. This is why 322 (89.2%) of the women had 'hospital' as their prior choice of place of delivery as they had to agree to this as a condition for entry into the study.

10.2.4 The rate of twinning

The rate of twinning was very low as only 2 sets out of 334 recorded deliveries were found. This was again a result that could not be generalised to the whole community as multiple pregnancies could have been lost from the study as they would have been referred to a hospital as soon as the diagnosis was made. A lot of the referred cases had severey inadequate information recorded

and were often lost from the study as it was impossible to set up a committed collaborating team at each of the referral centres.

10.2.5 Maternal Mortality

There was only one maternal death during the study. The woman was unable to get to the Saradidi Clinic during labour, delivered at home and died from post-partum haemorrhage. This gives a maternal mortality rate of 30 per 10,000 deliveries.

10.2.6 Birth Weight

The mean birth weight in the main study population was 3.13 $\pm$ 0.50 and the distribution is presented in Tables 10.1, 10.2 and 10.3. Only 25 of the 306 (8.2%) newborns whose birth weights were recorded could be classified as low birth weight babies as they weighed less than 2.5 kg (Figure 10.3). The distribution of the newborns by arm circumference was very similar to that of weight. The factors that could have affected birth weight are discussed in the sections that follow.

a) By mother's age

The mean birth weight increased very slightly with mother's age. The newborns of younger mothers aged less than 25 years had significantly lower mean birth weight than the newborns of older women ($F = 7.45$, $df = 2/302$, $P < 0.005$). The proportion of newborns with low birth weight also differed significantly by maternal age ($X^2 = 22.22$, $df = 2$, $P < 0.001$). For the newborns whose mothers were seen for the first time at the time of delivery; those who were less than 25 years of age had significantly lower mean birth weight than older ones ($F = 8.36$, $df = 2,198$, $P < 0.005$).

b) By parity

The mean birth weight of the first born newborns was significantly lower

(2.91 ± 0.47) than that of the other newborns 3.14 ± 0.46 (F = 8.72, df 4,301, P <0.005). Table 10.1 presents mean birth weights by parity. The primigravid women were followed by para ones. Thus birth weight tended to increase with parity.

10.7% of 112 newborns of primigravid women had low birth weight as compared to 6.4% of multiparous women. The difference was not statistically significant. This may be due to the sample size since most of the low birth weight babies were from primigravid women i.e. 15 out of 22 or 68.2%.

c) By chemoprophylaxis status

The mean birth weights were 3.08 ± 0.54 kg for the weekly chemoprophylaxis group and 3.07 ± 0.46 kg for the group receiving treatment only when ill. There was no significant difference in mean birth weights by the three treatment groups (see Table 10.2).

The percentage of newborns with low birth weight was 7.8% in the monthly group, 8.9% in the weekly group and 7.8% in those mothers treated when ill. The difference was again not statistically significant.

The other anthropometric measures were consistent with the birth weights (Table 10.3).

d) By area of residence

The mean birth weights did not differ significantly by area of residence although the means were slightly lower for area C. The percent of newborns with low birth weight did not differ by area of residence of the mothers.

e) By placental parasites

261 newborns were examined for birth weight and placental parasites. Of these, 22 (8.4%) were considered to be low birth weight babies because they were below 2500 grams. The rate of low birth weight babies was higher among mothers who had parasitised placentae (15.1%) as compared to mothers with unparasitised placentae (5.9%). The babies with parasitised placentae were more likely to be low birth weight babies than those with parasite free placentae. The difference was statistically significant (X^2 = 5.79, 1 df, P <0.025).

The mean birth weight of babies who had parasitised placentae was 2.91 ± 0.47 which was significantly lower than the mean birth weight of those with unparasitised placentae which was 3.11 ± 0.53 (F = 8.48, df = 1,257, P <0.005). High density of placental parasites was also associated with low birth weight as the difference between mean placental parasite density for low birth weight babies and normal birth weight babies was statistically significant (F = 6.35, df = 2,257, P <0.01 - see Table 10.10).

f) By peripheral parasitaemia

The mean birth weight was significantly lower for babies whose mothers had malaria positive blood slides at the time of delivery (F = 3.91, df = 1,202. P <0.05). Comparing mean parasite densities of women with normal birth weight babies with those with low birth weight babies, the mothers with low birth weight babies had significantly higher mean parasite counts at delivery (F = 5.2, df = 1,186, P <0.025). The mean parasite counts at the first two antenatal visits did not differ significantly between the women with low and those with normal birth weight babies (Table 10.8).

g) The implications of congenital malaria

Although the mean birth weight of babies who had cord parasitaemia did not differ significantly from those babies who had parasite free cord blood, 5/222 (22.7%) of newborns with low birth weight had cord parasitaemia compared to 19/265 (7.2%) newborns with no parasites in their cord blood. This difference was statistically significant (X^2 = 6.42, df = 1, P <0.025). This difference was also statistically significant for the women who were seen for the first time at delivery time (X^2 = 5.8, df = 1, P <0.025). The cord parasite density was also higher among low birth weight babies than normal birth weight babies (F = 7.06, df = 1,283, P <0.010 - Table 10.11).

h) By haemoglobin type

The mean birth weight did not differ significantly by haemoglobin type. The proportion of low birth weight babies was twice as high among normal

haemoglobin (AA) babies as compared to the newborns of mothers with sickle cell trait (AS).

i) By recruitment maternal parasitaemia

The mean birth weight was lower for newborns whose mothers were parasitaemic at recruitment. The difference in weight was statistically significant ($F = 4.37$, $df = 1,197$, $P < 0.05$).

Parity, age, placental parasites, and maternal peripheral parasites at delivery were considered in a regression model to predict birth weight. Once parity had been entered into the model, the other variables had little additional effect. It appeared that parity had the greatest influence over birth weight.

10.2.7 Maternal Peripheral Parasitaemia at the time of Delivery

The women were examined for malaria parasites at 12-20 weeks gestation, at 36 weeks, at the time of delivery nd asix weeks after delivery. In this section the maternal parasitaemia at the time of delivery is presented in the light of factors that could have affected it.

a) By maternal age

Eighty-four out of 185 (45.4%) women aged less than 25 years were parasitaemic at delivery as compared to 55/228 (24.1%) women 25 years and over. This difference was statistically significant ($X^2 = 22.1$, $df = 2$, $P < 0.001$).

For the supplementary study group who were only contacted and examined at delivery, the effect on maternal age on the prevalence of parasitaemia 64/100 women aged less than 25 years (64%) were parasitaemic as compared to 42/104 (56.5%) women over 25 ($X^2 = 13.23$, $df = 2$, $P < 0.01$).

The women under 25 years in the main study had significantly higher parasite counts than the women over 25 years at the time of delivery ($F = 7.3$, $df = 1,190$, $P < 0.01$). This is presented in Table 10.7. The difference in geometric mean density was higher for the younger women but the difference was not statistically significant.

b) By parity

In the main study, parasitaemia at delivery was much more common among primigravidae, 76/158 (48.1%) than for the multigravid women, 65/256 (25.4%) (X^2 26.6, df = 2, P <0.001).

For the supplementary study group seen for the first time at the time of delivery, 60:95 primigravidae (63.2%) were parasitaemic as compared to 22:39 (56.4%) para I and 25:71 (35.2%) para II and over. The multiparous with parity more than 1 had significantly lower prevalence of parasitaemia (X^2 = 13.06, df = 2, P <0.01).

There was no significant effect of parity on mean parasite counts in the main study.

c) By chemoprophylaxis status

The malaria prevalence was much higher among women who were not in the study until the time of delivery. Of 205 of these women, 107 (52.3%) were parasitaemic as compared to 16.0% of those who received monthly treatment, 15.9% of those who received weekly chemoprophylaxis and 18.6% of those who were only treated when ill. The difference was statistically significant (X^2 = 44.63, df = 3, P <0.001).

The mean peripheral parasite count at delivery for all groups was 464.3 $\pm$ 56.3 parasites per ml of blood. The women receiving monthly chloroquine chemoprophylaxis at 10 mg/kg body weight had the lowest mean density followed by those who were receiving a weekly dose of 5 mg/kg body weight. The women who were treated only when ill had the highest density (786.7 $\pm$ 97.2). The difference between the monthly and the weekly chemoprophylaxis groups was not statistically significant, but the ones receiving treatment only when ill had significantly higher parasite density than the monthly chemoprophylaxis group (F = 5.77, df = 2,191, P <0.005) (Table 10.8).

d) By area of residence

The geometric mean of maternal parasite density at the time of delivery

did not differ by area of residence. The mean log of parasite densities were: 6.824 ± 1.468 for area A, 7.090 ± 1.308 for area B and 7.521 ± 1.450 for area C.

Placental parasite presence was significantly associated with higher peripheral parasite density (F = 5.58, df = 1/261, P <0.05). This association was not found with cord parasitaemia.

10.2.8 The Placenta

The mean placental weight was 0.51 ± 0.08 and did not differ either by treatment group, parity group, area of residence or haemoglobin type.

Of the 268 placental specimens examined for malaria parasites, 74 (27.6%) were positive. Most of the infections were P.falciparum (95.9%) and the rest (4.1%) were P.malariae. These compared well with the results of maternal peripheral blood specimens.

The following factors were expected to influence placental parasites in terms of prevalence and intensity of infection.

a) Maternal age

The women who were followed up in the study during their antenatal period showed higher prevalence of parasitaemia among women aged below 25 years as 30.4% of them were parasitaemic as compared to 15.2% for women 25 years and over. This difference was statistically significant (X^2 = 5.5, df = 1, P <0.025) - see Table 10.7.

The geometric mean density of placental parasites was also higher for women aged below 25 years than those aged 25 and over (F = 8.99, df = 2,178, P <0.005).

b) Parity

Primigravidae had slightly higher prevalence rates of placental parasites (32.7%) as compared to para ones and twos (30%) but para threes and over had a lower prevalence rate which was not statistically significant (Table 10.4).

This placental parasite prevalence rate declined with increasing parity

but the difference in parasite counts by parity was not statistically significant. However, placental parsite density was higher among primiparae than multiparae. The difference was statistically significant ($F = 3.64$, $df = 3,197$, $P < 0.025$).

For women seen for the first time at delivery, the difference between primigravidae and those with three or more deliveries was even more significant. 47.5% of primigravidae were parasitaemic as compared to 33 (35.9%) para ones, 20 (44.4%) para twos and 36 (28.6%) women with three or more deliveries ($X^2 = 12.4$, $df = 3$, $P < 0.01$).

c) Chemoprophyalxis status (treatment groups)

The rate of placental parasites was higher in the primigravidae that were only treated whenever they were ill with malaria. Of 35 such women examined 16 (45.7%) had placental parasites as compared to 13/36 (36.1%) of primigravid women on weekly chemoprophylaxis and 4/30 (13.3%) who received monthly chemoprophylaxis at antenatal clinics. This difference was statistically significant ($X^2 = 8$, $df = 2$, $P < 0.025$) - see Table 10.4 and Figures 10.5, 10.6, 10.7 and 10.8. There was also a statistically significant difference when all the parities were considered together ($X^2 = 10.89$, $df = 2$, $P < 0.01$).

Thus there was a difference between treatment groups which was particularly marked in the primigravid women. The group receiving monthly chemoprophylaxis had the lowest placental parasite prevalence rate and those treated only when ill had the highest. The difference was observed among multiparious women although less markedly (Figures 10.6, 10.7 and 10.8), but still statistically significant.

For women seen for the first time at delivery, the placental parasite prevalence was significantly higher than the women who were followed up in the study ($X^2 = 46.4$, $df = 3$, $P < 0.001$) - see Table 10.7.

The monthly chemoprophylaxis group had the lowest density (213 ± 74) as

compared to 882 ± 2865 for the weekly group and 743 ± 1832 parasites per ml of blood for those on treatment only when ill. The monthly chemoprophylaxis group had significantly lower counts than the other two treatment groups (F = 5.9, df = 3369, P <0.005) the geometric mean densities of the parasites showed the same difference (Table 10.10).

d) Area of residence

The rate of placental parasite positivity in the main study differed from area to area. Of 127 blood films examined from area A, 35 (27.6%) were positive as comparted to 26/98 (26.5%) in area B and 13/43 (30.2%) in area C. The differences were not statistically significant, although the rate was slightly higher in area C.

e) Haemoglobin type

Of the 185 cases examined for haemoglobin type, only 22 (9.7%) had heterozygous (AS) type. The rest had AA haemoglobin type. Of the 22 women with AS haemoglobin, only 3 (13.6%) were parasitaemic at delivery as compared to 41:163 (25.2%) of women with normal haemoglobin.

The mean parasite density was also higher for the women with normal haemoglobin type but the difference was not statistically significant.

f) The effect of peripheral parasitaemia

The women who had peripheral parasitaemia were more likely to have placental parasites than those who did not. Of 30 women who had peripheral parasitaemia, 24 (64.9%) had placental parasites, as compared to 10% of those who were aparasitaemic. This difference was highly significant (X^2 = 75.23, df = 1, P <0.001).

It is to be noted that although most of the women with peripheral parasites had placental parasites, it was possible to have placental parasites with no peripheral parasites, as was found in ten percent of the women in the study.

The geometric mean densities of placental parasites did not differ between mothers who had peripheral parasites and those who were not parasitaemic at delivery time.

Among the women who were seen for the first time during labour and did not belong to the main study, 134 had peripheral parasites and of these 108 (80.6%) had placental parasites as compared to 39/239 (10.3%) who were aparasitaemic (X^2 = 148.6, df = 1, P <<0.0001).

The geometric mean counts of peripheral parasites were much higher among mothers who had placental parasites than for the mothers with parasite free placentae (F = 9.04, df = 1196, P <0.005).

10.2.9 Congenital Malaria

Cord parasitaemia was examined in 285 cases. Of these 22 (7.7%) were positive.

a) The effect of mother's age

It has already been shown that the younger women had higher occurrence of placental parasites and higher parasite densities. There was no apparent effect of age on cord parasites either in terms of prevalence or intensity of infection. The women aged 15-24 had 8%, 25-34 had 5.6% and 35 and over had 12.5% positive cord blood smears.

The geometric mean of the cord blood densities from newborns of mothers 35 years and over was slightly lower than the rest but the difference was not statistically significant (see Table 10.11).

b) The effect of parity

The effect of parity was not statistically significant. The numbers involved were too small for valid conclusions to be drawn. 8.7% of primigravidae, 5.2% of para ones and 10% of para twos and over had cord-blood parasitaemia. The difference was again not statistically significant (Table 10.5).

c) The effect of chemoprophylaxis

Of 94 women receiving monthly chemoprophylaxis 5 (5.3%) had cord parasites as compared to 3 of 95 women (3.2%) on weekly chemoprophylaxis and as compared with 14 of 96 (14.6%) women receiving treatment only when ill.

Thus those women on regular prophylaxis either monthly or weekly had significantly lower cord parasite rates (X^2 = 9.88, df = 2, P <0.01) - see Table 10.5.

The difference in geometric mean of cord parasite densities between treatment groups was also statistically significant, the monthly chemoprophylaxis group had the lowest mean density (F = 5.68, df = 2,280, P <0.01) - see Table 10.11).

d) The effect of haemoglobin types

All the cases who had cord blood parasitaemia were from mothers with the normal haemoglobin type (AA). Of the 24 women with the abnormal (AS) haemoglobin type (the sickle cell trait), none of their newborns had malaria parasites in their cord blood.

e) The effect of peripheral parasitaemia

The rate of cord blood positivity between mothers who were parasitaemic at delivery was much higher than those who were aparasitaemic (X^2 = 10.34, df = 1, P <0.01). The geometric mean peripheral parasite density was significantly higher for mothers with cord parasitaemia (F = 9.04, df = 1,196, P <0.025)

The women who were seen for the first time at delivery and were parasitaemic had even much higher cord parasite rates than the women who were on the main study. 51 of 138 (37%) as compared to 27:254 (10.6%), a difference which was statistically significant (X^2 = 38.88, df = 1, P <0.001).

f) The effect of placental parasitisation

Cord blood parasitaemia was associated with heavy placental parasitisation. The mean placental parasite density when cord blood was positive for malaria parasites was 2458 $\pm$ 5177 as compared to 502 $\pm$ 1500 for placental parasite count where cord blood was negative. The difference was statistically significant (F = 16.79, df = 1,252, P <<0.005).

Parasitised placenta was associated with a much higher rate of cord

parasitaemia, with 15/72 (20.0%) compared with unparasitised placenta 4/184 (2.17%), a difference which was highly significant ($X^2 = 26.22$, df = 1, $P < 0.001$).

The mean log of parasite density of the cord blood was 5.8416 $\pm$ 0.731 for the mothers with parasitised placenta and 5.955 $\pm$ 0.2254 for those with parasite free placenta. The difference was not statistically significant.

For the women seen for the first time at delivery, the association between cord and placental parasitaemia was even stronger. Seventy one (77%) of newborns with cord parasites had placental parasites as compared to 112 (30.7%) with cord blood negative slides. This difference was highly significant ($X^2 = 66.14$, df = 1, $P < 0.001$).

Placental parasite count was not higher among the cases whose newborns had parasites in their cord blood than those who did not have them.

10.3 DISCUSSION

In this section the results of the findings at the time of delivery are presented. Abortions and still births in the study were too few for analysis of causative factors and any probable impact of chemoprophylaxis on the rate of foetal wastage. Other studies have shown no impact of antimalarial chemopropylaxis on foetal wastage (Bruce-Chwatt, 1952; Kortman, 1972; and Reinhardt, 1978).

Only one maternal death occurred out of the 361 deliveries. McGregor (1984) also reported the fact that maternal deaths due to malaria are very rare even when placental malaria infection rate and intensity are both high.

Birth weight can be considered the most important pregnancy outcome, since it greatly contributes to the survival of the infant and to its health status in general. The impact of low birth weight in the health status and survival of the infants is discussed in Chapter 11. In this chapter the factors that influence the birth weight have been considered, since any influence on birth weight would also be reflected on infant mortality and quality or status of health.

Rooth (1980) makes the point that different populations have different frequency distributions of birth weights. However, we considered 2.5 kg as a reasonable cut off point for normal birth weights in our population. Only 8.2% of the newborns in this study fell below this cut off point. It is indeed agreed that making such a cut off point applicable to different communities is an over simplification of a very complex phenomenon (Wilcox, 1983).

The distribution of birth weight can be described as essentially Gaussian, but slightly peaked in the lower tail (Taback, 1951; MaKeown and Gibson, 1951). More precise descriptions of the birth weight distribution have been developed by Adams et al. (1968) and Ashford et al. (1968). The distibution of our birth weights was not expected to be normal.

Mean birth weights are lower, particularly in first-born children. This is often also associated with dense placental infections. In this study, parity had the most powerful influence on birth weight followed by peripheral and placental parasites. Low birth weight can be attributed to the impairment of placental function as a result of parasitisation as intravillous spaces are packed with macrophages and parasitised red blood cells. This may lead to foetal growth retardation and perinatal foetal loss due to antepartum and intrapartum asphyxia (Lawson, 1962). This leads to low birth wight (Reinhardt et al., 1978).

Other studies have looked at age, parity, maternal weight, sex of the baby and ethnic or socio-economic status (Jelliffe, 1968; Kortman. 1972 and Reinhardt, 1978) and found that parity, age and placental parasite tend to affect the birth weight of the newborns. Birth weight is low in the case of primigravidae, mothers with parasitised placenta, and those below 25 years of age. This was confirmed in the present study. No clear difference in birth weight by sex of the baby has been described. Those who have attempted (Reinhardt, 1978; McLaren and Ward, 1962) had opposite results, whilst Jelliffe (1968) saw no difference at all by sex of the baby. McGregor (1983) described an increase in birth weight with increasing parity, a finding which was confirmed in

this study. This finding demonstrates the decline in malaria impact as parity increases.

The mean singleton birth weight in this study was depressed by 202 grams in the presence of placental malaria infections. This was most pronounced among the first-borns and the difference diminished with parity.

There was no statistically significant relationship between placental parasite density and birth weight, although the lowest weights tended to occur in the categories of highest density. These findings were similar to those of McGregor et al (1983) in the Gambia. In their study the mean birth weight was depressed by 170 grams in the presence of parasitised placenta.

Other workers have found a mean difference of 55 to 310 g between children born of parasitised placentae and those of normal placentae (Bruce-Chwatt, 1952; Archibald, 1956; Cannon, 1958; Spitz, 1959; McLaren and Ward, 1962; Jelliffe, 1968; Kortman, 1972; and Reinhardt, 1978).

The occurrence of low birth weight babies born weighing less than 2.5 kg was more frequent with parasite infected placentae than non-malarious placentae. This association was also more marked among first-borns.

Low birth weight babies were found more frequently among primiparous (10.7%) than multiparous women (6.4%). This had also been described by Archibald, (1956); Cannon, (1958); Spitz, (1959) and Kortman, (1972). Birth weight was found to increase with increasing parity irrespective of the presence of placental malaria (McGregor, 1983).

The mechanism that results in a low birth weight baby is not clearly understood. However, some newborns may have low birth weight because they were born prematurely as malaria is known to precipitate prematurity (Archibold, 1964), but most are due to depressed placental function in foetal nutrition. Galbraith et al. (1980) have described histological changes in malarious placentae which are compatible with impaired materno-foetal exchange. McLaren and Ward (1962) found no evidence that malaria impaired

the transference of vitamin A from the mother to the foetus or the leaking of ergothioneine into foetal circulation.

The mean birth weight did not differ significantly by treatment groups. Other studies have shown this more conclusively (Spitz, 1959).

The dominance of P.falciparum infections in maternal peripheral blood, in placentae and in cord blood observed in this study to be over 95% of all infections has been reported by other workers (Spencer et al., 1987; Payne et al., 1976; Molineaux and Gramicia, 1980; Authonioz et al., 1979; Reinhardt et al., 1978; Logie et al., 1973 and Kortman, 1972).

In this study the prevalence rate of peripheral parasitaemia was higher than the prevalence of placental and cord parasitisation. This finding was consistent with those of other workers as shown in Table 10.9 (Bruce-Chwatt, 1952; Kortman, 1972, and Reinhardt, 1978). Other workers have found placental parasitisation to be more prevalent than peripheral parasitaemia (Blacklock, 1925; Jelliffe, 1968; Van Dougen and Van Hoof, 1982).

The rate of placental parasitisation was 27.6% and was consistent with rates found elsewhere in tropical Africa in general and Kenya in particular. The findings of other workers are summarised in Table 10.12.

The results reported in this chapter compared well with those of other workers like McGregor and his colleagues (1983) who found malaria infections in 1300 placentae (20%) of 6427 placentae examined. Like in their study, P.falciparum infection dominated (95.4%), followed by P.malaria (4.1%) and P.ovale (0.5%). Where McGregor et al.(1983) found a difference between urban and rural communities in terms of prevalence of placental infections, our communities in areas A and B which had Primary Health Care programme activities for four years prior to the commencement of this study, and area C which had PHC activities for only two years had rates of infection which were not statistically significantly different.

As in the Gambian study, our rate of placental infection was higher in

the primiparae (32.7%) than para ones (30%) and lowest in multiparous women (18.7%).

The placental parasite count was much higher among women who had peripheral parasitaemia ($F = 10.26$, $df = 1.108$, $P < 0.005$).

Placental parasite density was much higher among primigravidae than multiparous ($F = 7.98$, $df = 3,107$, $P < 0.005$) and also for women under 25 years compared to those over 25 yers ($F = 7.23$, $df = 2,107$, $P < 0.005$).

Maternal age seemed to affect the prevalence rate as the prevalence was significantly higher for women below 25 years of age as compared to those aged 25 years and over. This was also described by Kortman in 1972.

The influence of parity on placental parasite rates has been reported by several workers (Archibold, 1956; Cannon 1958; Spitz, 1959). Kortman (1972) working in Tanzania found no significant difference between primiparious and multiparous women. The primiparous in our study had higher rates (32.7%) than para ones and twos (30%) and para threes and over (18.7%). Thus the incidence of placental infection tended to decrease with increasing parity. Several studies have demonstrated higher placental parasites in primigravidae (Archibold, 1956). McGregor's Keneba (1983) data supported the importance of parity as a determining factor to parasitisation; the rate being highest in primigravidae and falling with increasing parity.

The implication and mechanism of placental parasitisation has not been well understood, but it is suggested that it may be due to local immunosuppression in the uterine area during pregnancy from some evidence from studies in mice. Blacklock and Gordon (1925) concluded that malaria exerts an important effect on foetal deaths in utero due to pathological alterations in the malaria infected placenta. Subsequent studies and the current one indicated no relationship between placental infection and still birth rates. Since still birth was a rare occurrence the sample size in our study was not large enough to demonstrate a relationship that may exist.

Blacklock (1925) in Sierra Leone found that parasites were more common in placentae than in maternal peripheral blood at the time of delivery. This was also reported by Clark in 1915 who found that parasites were twice as common in placentae than maternal peripheral blood. and could occur in the absence of any febrile illness. He expressed the view that pregnancy synergises malaria by lowering bodily resistance and furnishing an additional harbour for the development of the parasite.

Blacklock and Gordon (1925) found the incidence of placental infection to be 38% in Sierra Leone, 50% of whom were asymptomatic in spite of high density of infection.

The incidence of placental infection varies by study and country, some findings in several African countries are presented in Table 10.9 for comparison with the findings of this study.

More primigravidae had parasites in their placenta without peripheral blood parasitaemia and had higher parasite density than multigravidae (Lock, 1978).

Malaria parasite prevalence rates in peripheral blood and placenta are significantly higher in primiparae but multiparae had more malaria parasites in the placenta without peripheral blood parasites. The present study did not demonstrate the fact that placental parasite rate was higher than peripheral parasite rate.

Congenital malaria has always been considered a very rare occurrence. Measuring by the examination of cord blood samples. it was found that 7.7% of the examined samples were positive. The cord blood infection has been reported by several workers (Blacklock and Gordon. 1925; Bruce-Chwatt. 1952; Covell, 1950; Cannon 1958; Spitz, 1959). Clinical episodes of congenitally acquired malaria is known to be very rare (Covell. 1950). Many workers have reported that heavy placental parasitisation is not associated with cord parasitaemia (Blacklock and Gordon. 1925; Bruce-Chwatt. 1952; Covell. 1950; Cannon 1958;

Spitz, 1959). In this study there seemed to be a strong association between placental parasite presence with cord parasitaemia. but like in the above quoted studies, there was no significant relationship between placental parasite intensity and cord parasitaemia.

A similar relationship was observed between maternal peripheral parasitaemia at the time of delivery and cord parasitaemia; the association being mainly with parasite prevalence and not peripheral parasite density.

Whereas we found no significant variation in cord parasitaemia by age or parity of the mother, Kortman (1983) described a higher rate of parasitaemia among primiparous women. Working in Tanzania he observed an infection rate of 3.8% of newborns but like in the present study, the density of infection was always low Other workers have recorded rates as high as 21% in the Ivory Coast (Reinhardt, 1978) - see Table 10.9.

The women with parasitaemia at delivery were significantly more likely to have cord parasites than those who were parasite free 47/104 (45 2%) as compared to 25/94 (26.6%) (X^2 = 7.38/1, df = 1, P <0.001).

Blacklock and Gordon (1925) found cord parasitaemia rates as low as 0.03% in high endemicity areas. Covell (1950) reported higher prevalence in non-immune women living in hyperendemic areas. Kortman (1972) and Reinhardt (1980) found that cord parasitaemia was usually not accompanied by parasitaemia in the peripheral blood of the newborn. The parasites were probably rapidly eliminated from foetal circulation, perhaps by maternal immunoglobulins or other non-specific immune mechanisms. This was not looked for in the present study.

Campbell (1980) could not demonstrate protective effect of these passively acquired antibodies during the first half year of life in El-Salvador.

The mechanism of passing the placental barrier is unknown. Covell (1950) indicated the possibility of damage to the placenta during delivery as one possible mechanism. Wikramasuriya (1935, 1937) reported pathological changes

secondary to the infection, but this was not supported by concrete evidence.

In conclusion, the findings of the present study are consistent with those of similar studies elsewhere. Parity is the single most important factor determining birth weight, and peripheral, placental and cord-blood parasite positivity. The unique finding in this study is the effectiveness of monthly chemoprophylaxis in reducing both peripheral and placental parasite prevalence and intensity of infection. The comparison between the effectiveness of chloroquine phosphate used weekly, monthly or taken only during a febrile illness has not been done before.

Although the effect of monthly chloroquine on birth weight could not be demonstrated in the present study, its effect on placental parasites was impressive. Since placental parasites affected birth weight, it would seem that the effect of monthly chemoprophylaxis could be demonstrated in a larger sample or if a more effective drug than chloroquine was used.

The supplementary study group were recruited into the study in September and October 1987 to serve as a comparison group to the control group in the study population. This was to verify whether the antenatal care given to this group could have had a significant impact on their malaria parasite rates.

Table 10.7 shows that the supplementary group had higher parasite positivity rates. This difference could be attributed either to the regular antenatal care provided or to easy access to chloroquine provided by the CHW at the village level, or to information, education and communication available within the Saradidi community that could have modified the health seeking behaviour of pregnant women in Saradidi. This was not investigated in this study.

Operationally it would seem that pregnant women in a community with established community based health care activities which include the provision of education and drugs for antimalarial chemoprophylaxis have a significantly reduced chance of developing malaria infection. This would reduce maternal morbidity and improve outcome of pregnancy.

Thus if the resources for regular chemoprophylaxis are not available then a community based health care system with regular antenatal care could be a reasonable option for the care of pregnant women and particularly those that are pregnant for the first time.

Table 10.1 Mean birth weight by grouped parity

Parity	N	Mean birth weight
0	112	2.908 ± 0.47
1	62	3.119 ± 0.59
2	34	3.144 ± 0.46
3 and over	92	3.185 ± 0.50
Unknown parity	6	3.292 ± 0.48

Table 10.2 Mean birth weight by treatment group

Treatment group	N	Mean birth weight
Monthly chemoprophylaxis	102	3.03 ± 0.54
Weekly chemoprophylaxis	101	3.08 ± 0.54
Treatment when ill	103	3.07 ± 0.46
All women	306	3.06 ± 0.51

Table 10.3 Birth measurements by treatment group

MEASUREMENT	TREATMENT GROUP		
	WHEN ILL (n = 116	WEEKLY (n = 121)	MONTHLY (n = 112)
Birth weight	3.09 ± 0.73	3.10 ± 0.66	3.28 ± 0.45
Birth arm circumference	13.70 ± 0.78	10.92 ± 1.02	11.34 ± 0.91
Birth chest circumference	32.52 ± 2.97	33.13 ± 2.07	33.86 ± 1.63
Birth length	46.88 ± 3.01	46.85 ± 3.59	47.89 ± 2.87
Birth head circumference	34.05 ± 2.04	34.24 ± 1.86	34.35 ± 1.61

Table 10.4 Percent of placentae with malaria parasites by treatment group and parity

Treatment groups	PARITY				
	0	1	2	3+	Total
Monthly prophylaxis	4/30 (13.3%)	4/19 (21.1%)	4/13 (30.8%)	1/24 (4.2%)	13/85 (15.3%)
Weekly prophylaxis	13/36 (36.1%)	5/18 (27.8%)	1/6 (16.7%)	6/26 23.1%)	25/86 (29.1%)
Treatment when ill	16/35 (45.7%)	6/17 (27.8%)	4/9 (32.1%)	7/25 (18.7%)	33/86 (38.4%)
All groups	33/101 (32.7%)	15/54 (27.8%)	9/28 (32.1%)	14/75 (18.7%)	71/258 (27.5%)

Table 10.5 Percent cord blood smears positive by parity and treatment groups

Treatment Groups	PARITY 0	1	2+	All
Monthly prophylaxis (n = 94)	3.2	4.6	6.8	5.3
Weekly prophylaxis (n = 88)	2.9	-	5.1	3.3
Treatment when ill (n = 93)	18.4	11.8	15.1	10.8

Table 10.6 Placental and cord parasite positivity for women recruited in labour by age and parity

		% Positive	Significance of difference X^2	df	P
A) PLACENTAL PARASITE					
Age:	<25 years	57 (57.6%)			
	25-34 years	46 (56.1%)	2.8	2	>0.05
	35+ years	7 (36.8%)			
Parity	0	60 (63.2%)			
	1	18 (47.4%)	4.6	2	<0.05
	2	11 (64.7%)			
	3	22 (43.1%)			
B) CORD PARASITES					
Age:	<25 years	42 (42.4%)			
	25-34 years	25 (30.5%)	2.98	2	>0.05
	35+ years	6 (31.6%)			
Parity	0	43 (45.3%)			
	1	12 (31.6%)	6.51	2	<0.05
	2	3 (17.7%)			
	3	3 (29.4%)			

Table 10.7 Parasite prevalence in maternal peripheral blood and placenta by age, parity and study group

Age/Parity	Main study group	Supplementary group
Peripheral blood:		
Age <25	85 (45.4%)	64 (64.0%)
Age 25+	55 (24.1%)	42 (56.5%)
Parity 0	76 (48.1%)	60 (63.2%)
Parity 1+	65 (25.4%)	22 (56.4%)
Placental:		
Age <25 years	30.4%	93 (42.5%)
Age 25 years and over	15.2%	89 (31.3%)

Table 10.8 Mean log parasite density at recruitment by various experiences at delivery

Delivery factor	N	Mean density (± SD)	F	Significance df	P
Maternal age					
<25 years	64	3.77 (3.7)			
25-34 years	37	1.73 (3.06)	17.22	2,290	<0.05
35+ years	5	0.83 (2.08)			
Treatment group					
None	60	2.51 (3.46)			
When ill	22	2.44 (3.36)			
Weekly	6	2.73 (3.59)	0.19	2,294	>0.05
Monthly	19	2.37 (3.14)			
Birth weight group					
<2.5 kg	15	3.86 (3.35)			
2.5 + kg	89	2.55 (3.5)	2.85	1,269	>0.05
Placental parasite					
positive	84	2.48 (3.47)			
negative	20	3.41 (3.7)	3.14	1,228	>0.05
Cord parasite					
positive	47	2.73 (3.53)			>0.05
negative	57	2.22 (3.27)	0.35	1,249	

Table 10.9 Mean log of placental parasite density at second visit by various comparison factors at delivery

Comparison factor	N	Mean log density (± SD)	F	Significance df	P
Age of mother					
<25 years	76	1.97 (3.9)			
25-34 years	90	0.72 (2.11)	4.89	2,189	<0.01
>34 years	26	0.75 (2.15)			
Treatment group					
Monthly	62	1.30 (2.71)			
Weekly	65	1.07 (2.59)	0.34	2,191	>0.05
When ill	67	1.46 (3.06)			
Birth weight group					
<2.5 kg	16	0.97 (2.64)	0.18	1,186	>0.05
2.5 kg and over	172	1.27 (2.78)			
Placental parasite					
negative	123	0.35 (1.58)	117.8	1,155	<<0.005
positive	34	4.955 (3.65)			
Cord parasite					
negative	171	1.23 (2.79)	0.35	1,175	<0.05
positive	6	4.17 (3.37)			

Table 10.10 Mean log of placental parasite density by maternal age, treatment group and birth weight

Comparison factor	N	Mean log density (± SD)	Significance F	Significance df	Significance P
Age of mother					
<25 years	120	2.17 (3.40)			
25-34 years	113	1.83 (3.07)	1.84	2,257	>0.05
>34 years	27	0.89 (2.19)			
Treatment group					
Monthly	85	1.20 (2.46)			
Weekly	88	2.03 (3.32)	5.38	2,259	<.01
When ill	67	1.46 (3.06)			
Birth weight group					
<2.5 kg	22	3.55 (3.71)			
2.5 kg and over	261	0.36 (1.42)	6.35	2,257	<.01

Table 10.11 Mean log of cord parasite density by maternal age, treatment group and birth weight

Comparison factor	N	Mean log density (± SD)	F	Significance df	P
Age of mother					
>25 years	128	0.38 (1.49)			
25-34 years	120	0.49 (1.45)	0.16	2,278	>0.05
>34 years	33	0.47 (1.52)			
Treatment group					
Monthly	94	0.24 (1.14)			
Weekly	94	0.20 (1.11)	5.68	2,280	<0.01
When ill	95	0.87 (2.11)			
Birth weight					
<2.5 kg	24	1.23 (2.47)	7.08	1,283	<0.01
2.5 kg and over	261	0.36 (1.42)			

Table 10.12 Summary of findings on maternal parasitaemia, placental parasitisation, cord and infant peripheral parasitaemia by year and country of study.

Year	Author	Country of study	Maternal parasitaemia (%)	Placental parasites (%)	Cord parasites (%)	Child parasitaemia (%)
1915	Clark	Panama	2.0	4.7	0.25	-
1925	Blacklock and Gordon	Sierra Leone	8.0	38.2	0	0
1925	Van den Branden	Congo	56.3	1.8	-	0
1930	Butler	Ghana	23.1	18.0	2.0	-
1931	Lombart	Congo	56.0	50.0	2.0	0
1934	Swetz and Peel	Congo (Zaire)	76.0	74.0	6.0	2
1936	Dateens and Lavergue	Indochina	1.5	-	0.3	-
1947	Walton	Sierra Leone	-	-	-	0.3
1948	Peel and Van Hoof	Congo	60.2	37.6	-	0
1949	Garnham	Kenya	-	27	-	0.7
1952	Bruce-Chwatt	Nigeria	27.4	22.3	-	0.2
1956	Archibold	Nigeria	-	14.7	-	-
1958	Cannon	Nigeria	-	25.6	-	0
1958	Archibold	Nigeria	-	14.1	-	-
1959	Spitz	Nigeria	-	23.6	-	0
1962	McLaren and Ward	Tanzania	-	22.0	-	-
1968	Jelliffe	Uganda	5.6	16.1	-	0.2
1972	Kortman	Tanzania	23.2	19.7	3.8	-
1972	Logie et al	Gambia	-	31	-	-
1978	Reinhardt	Ivory Coast	39.4	33.7	21.7	-
1979	Authonioz	Senegal	-	21.0	-	-
1982	Van Dongen and Van Hoof	Zambia	8.4	18.1	5.9	-
1988	Kaseje (present study)	Kenya (Saradidi)	35.8	27.6	7.7	-

Fig 10.1 Number of deliveries by month

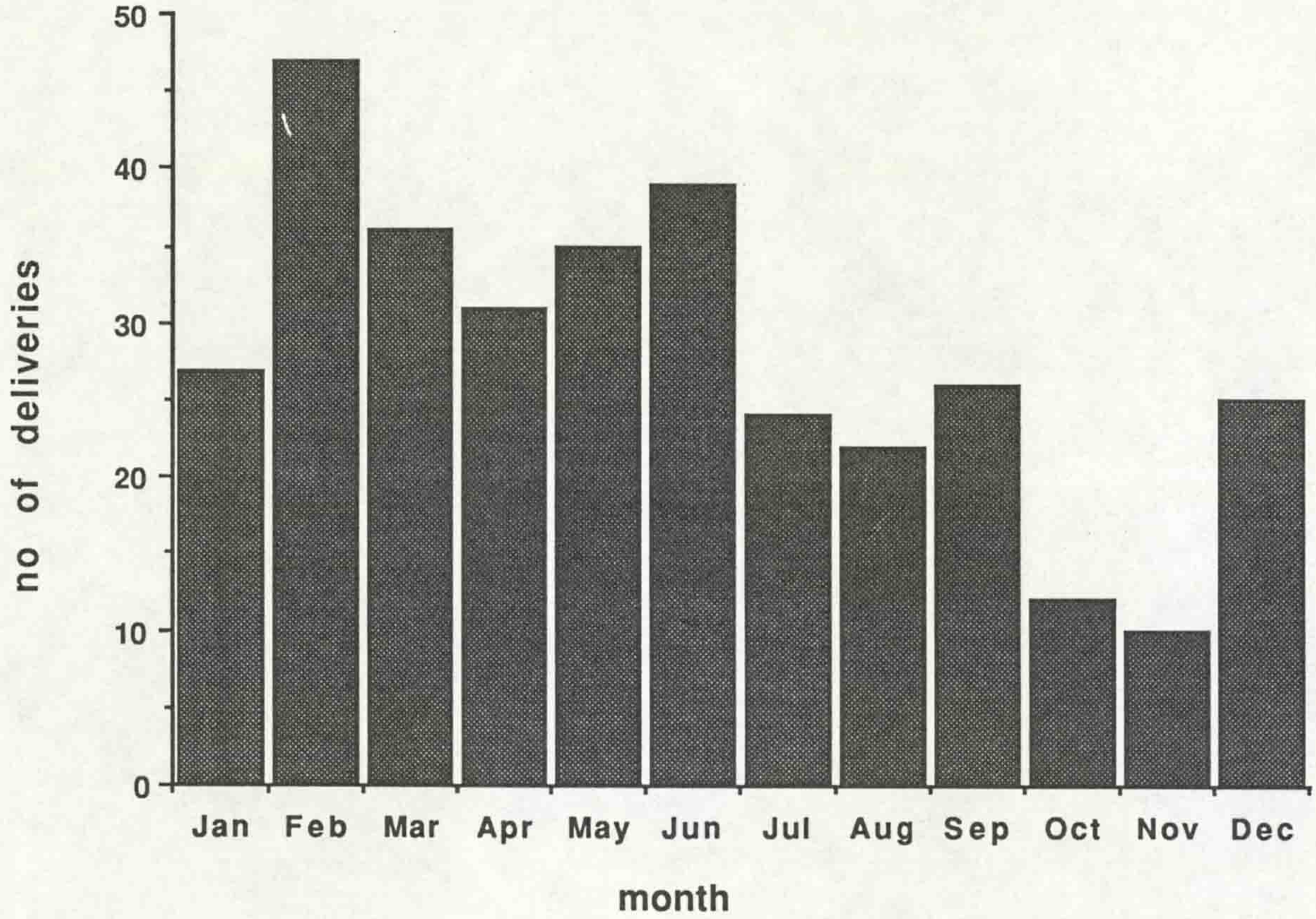


Fig 10.2 Distribution of births by gestational age

Fig 10.3 Distribution of newborns by birth weight.

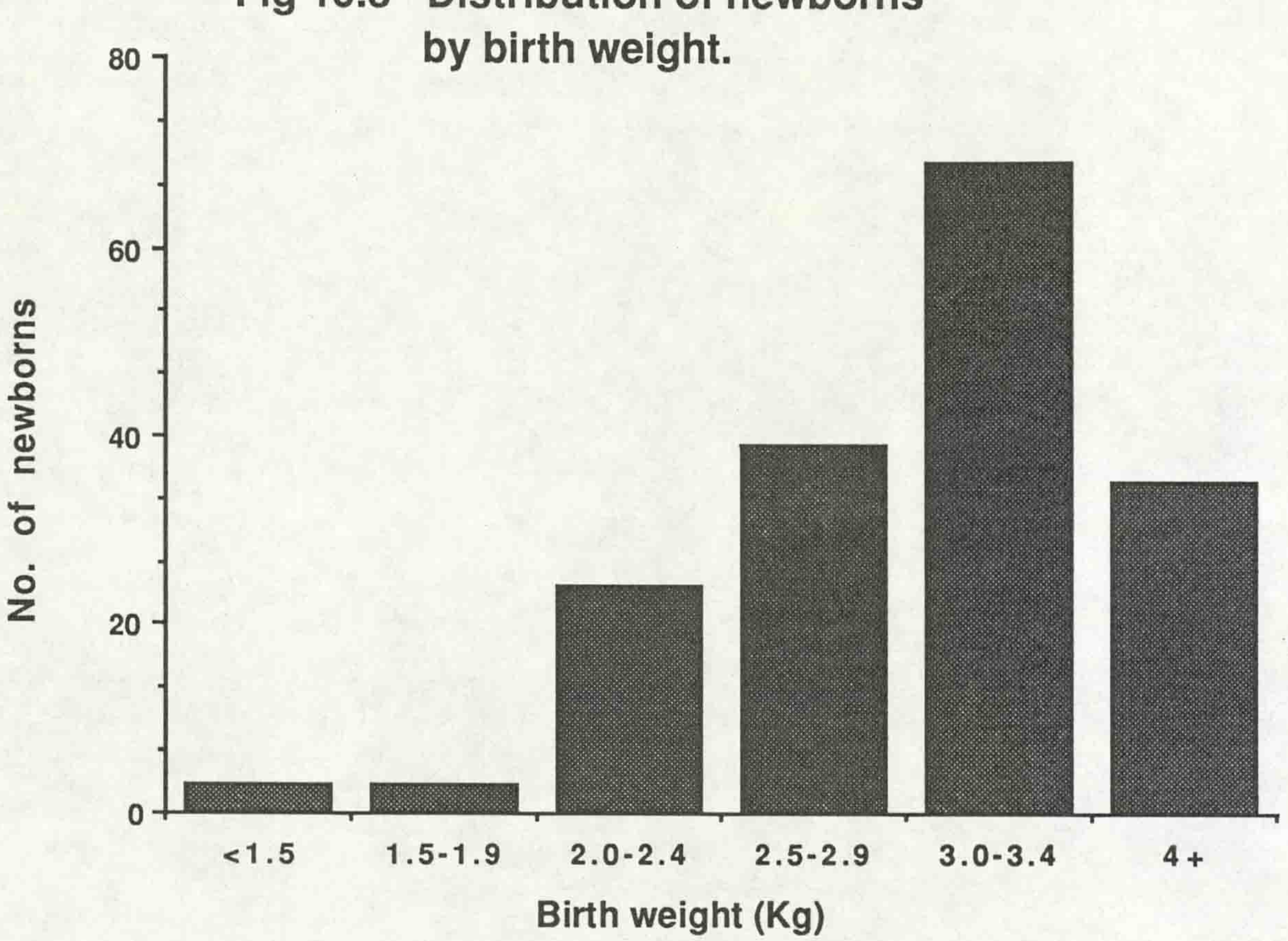


Fig 10.4 Mean birth weight by parity.

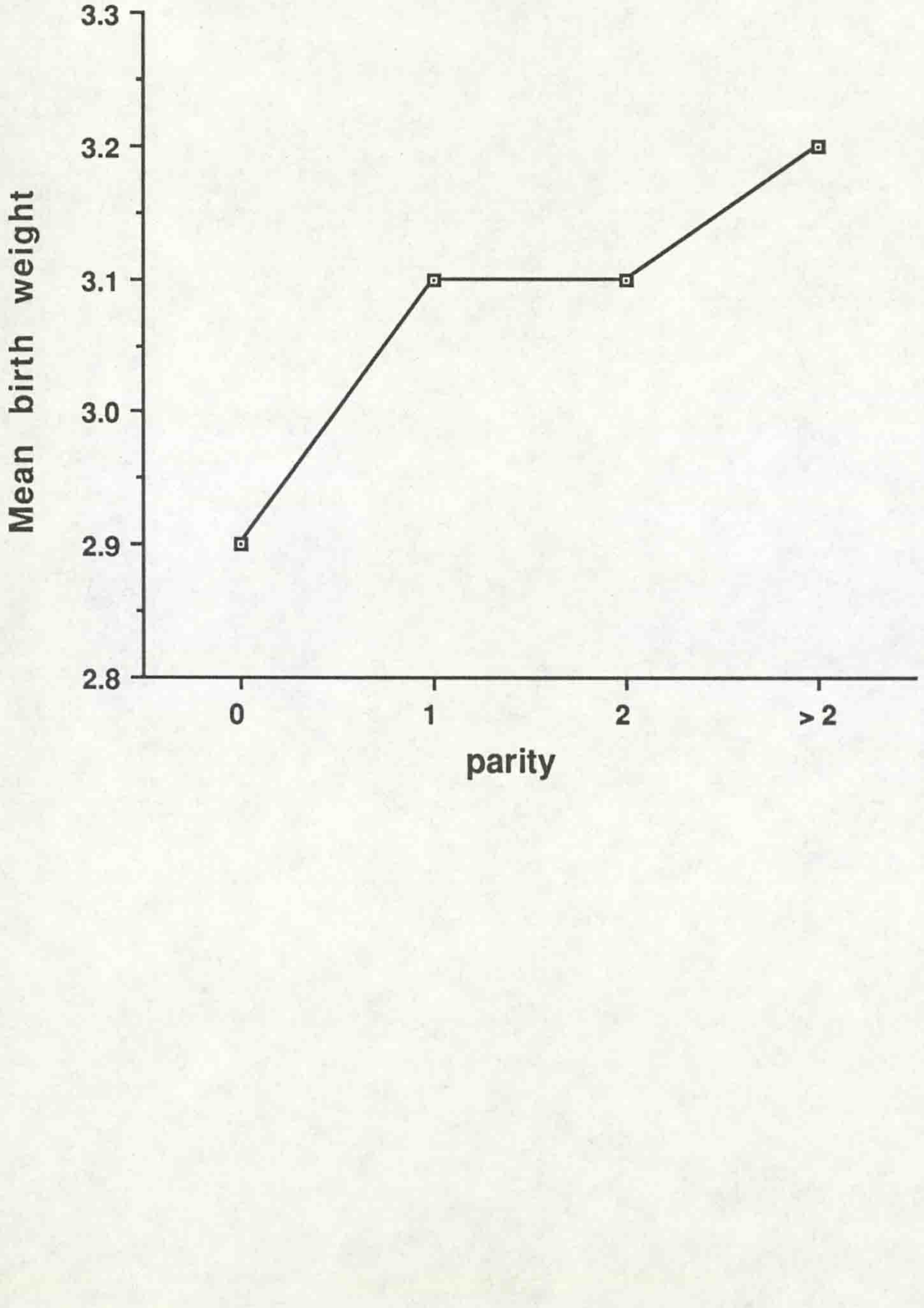


Fig 10.5 Percent parasite positive placental specimens by treatment group (para 0)

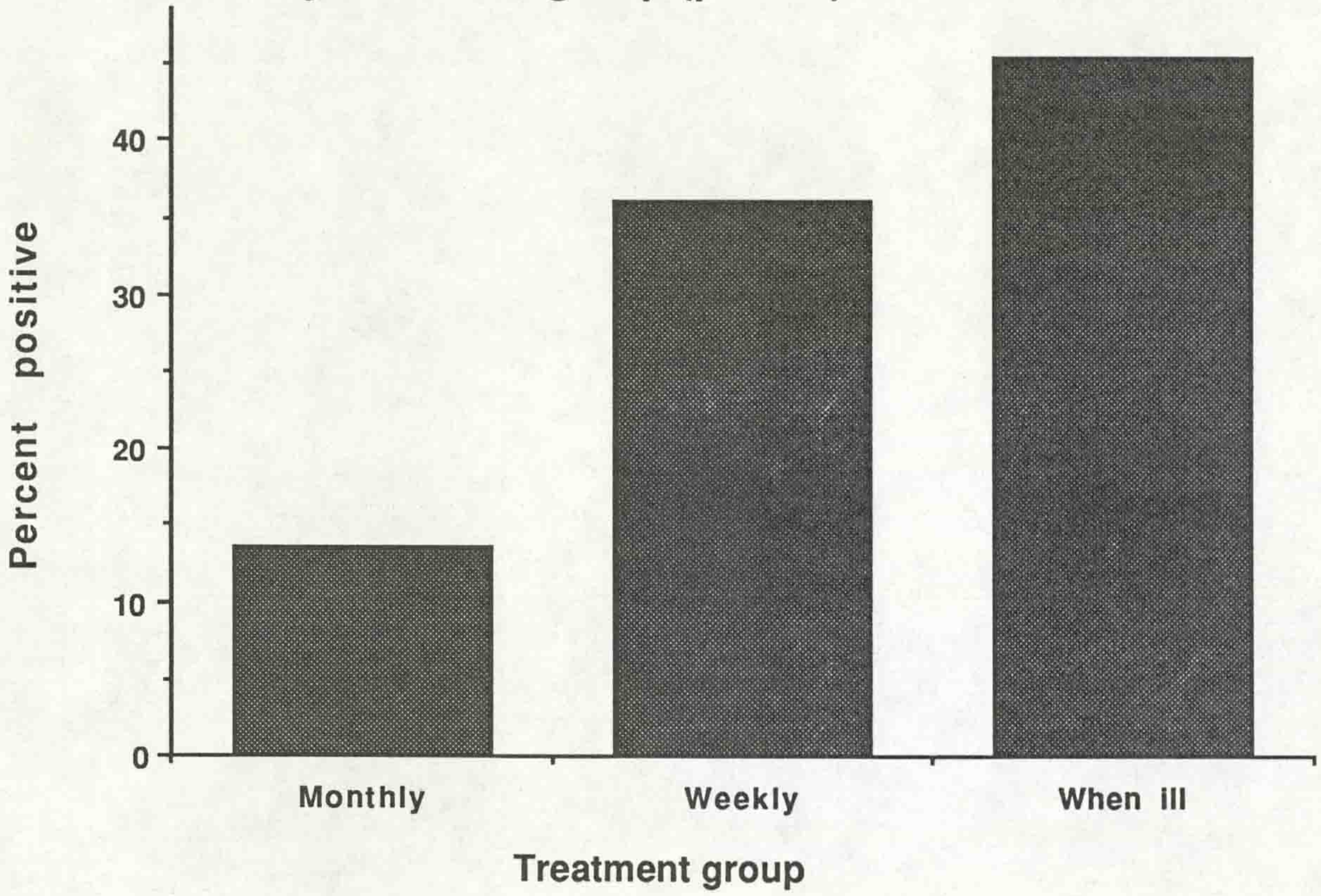


Fig. 10.6 Percent parasite positive specimens by treatment group (para 1)

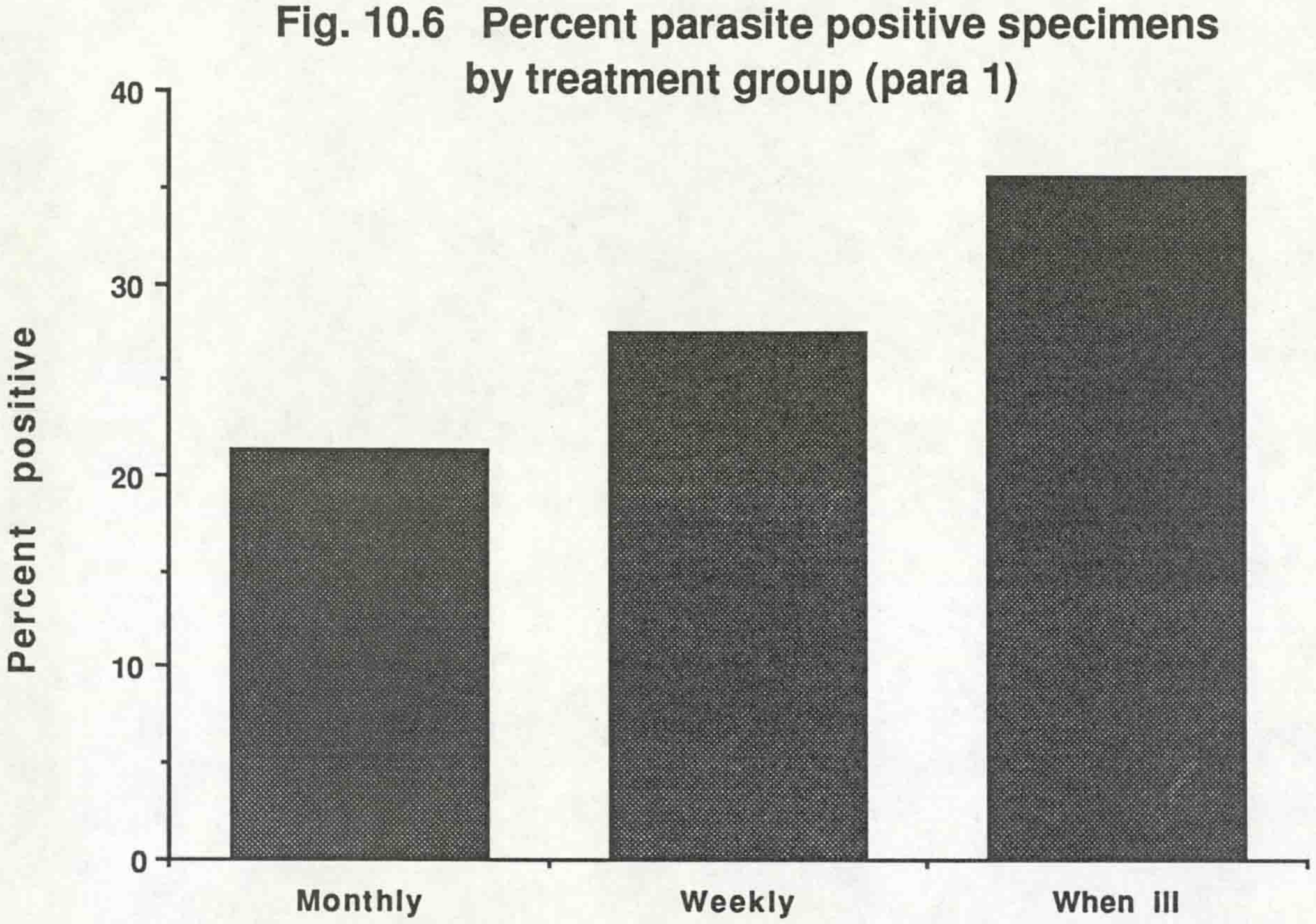


Fig 10.7 Percent parasite positive placental specimens by treatment group (para 1)

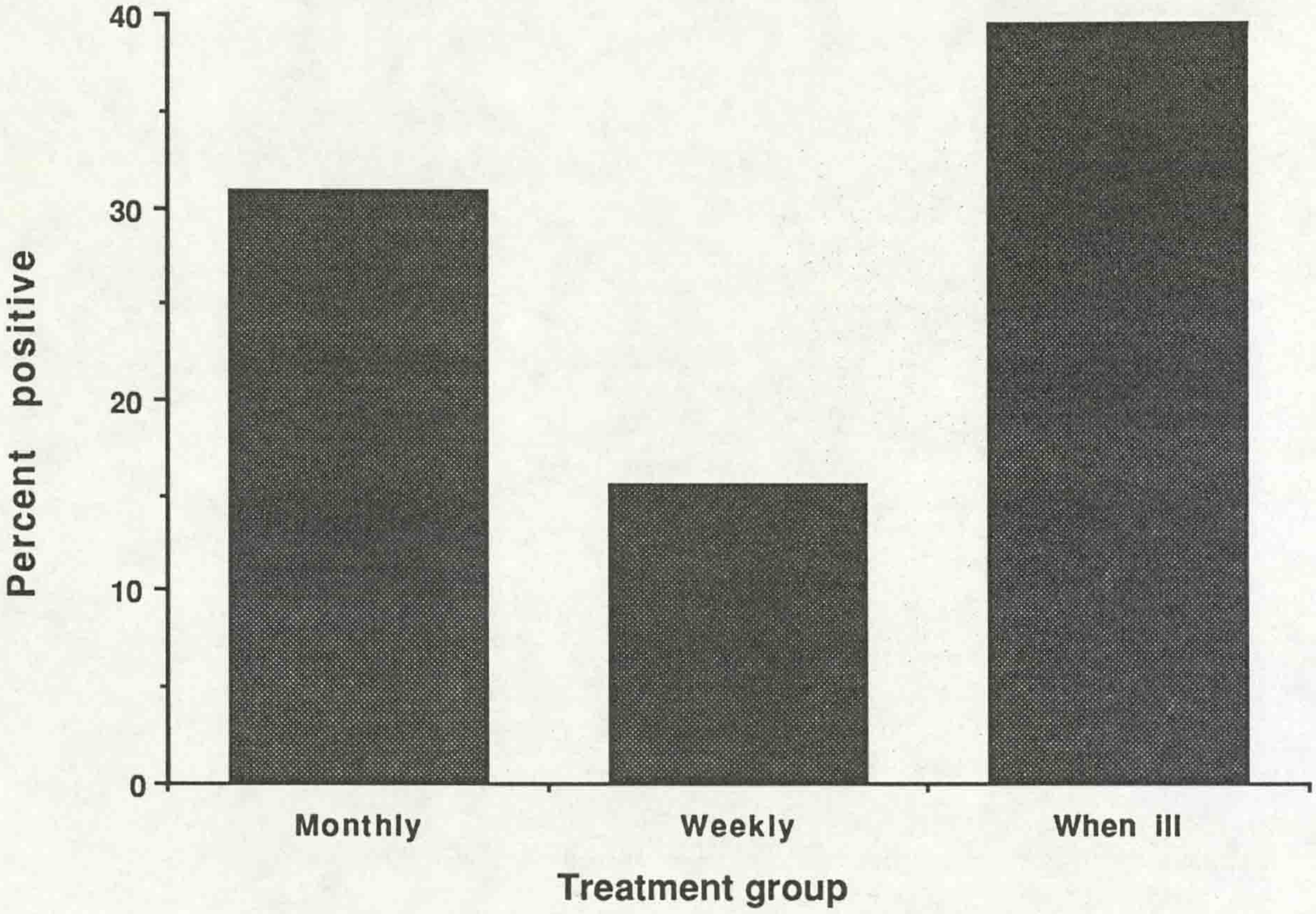
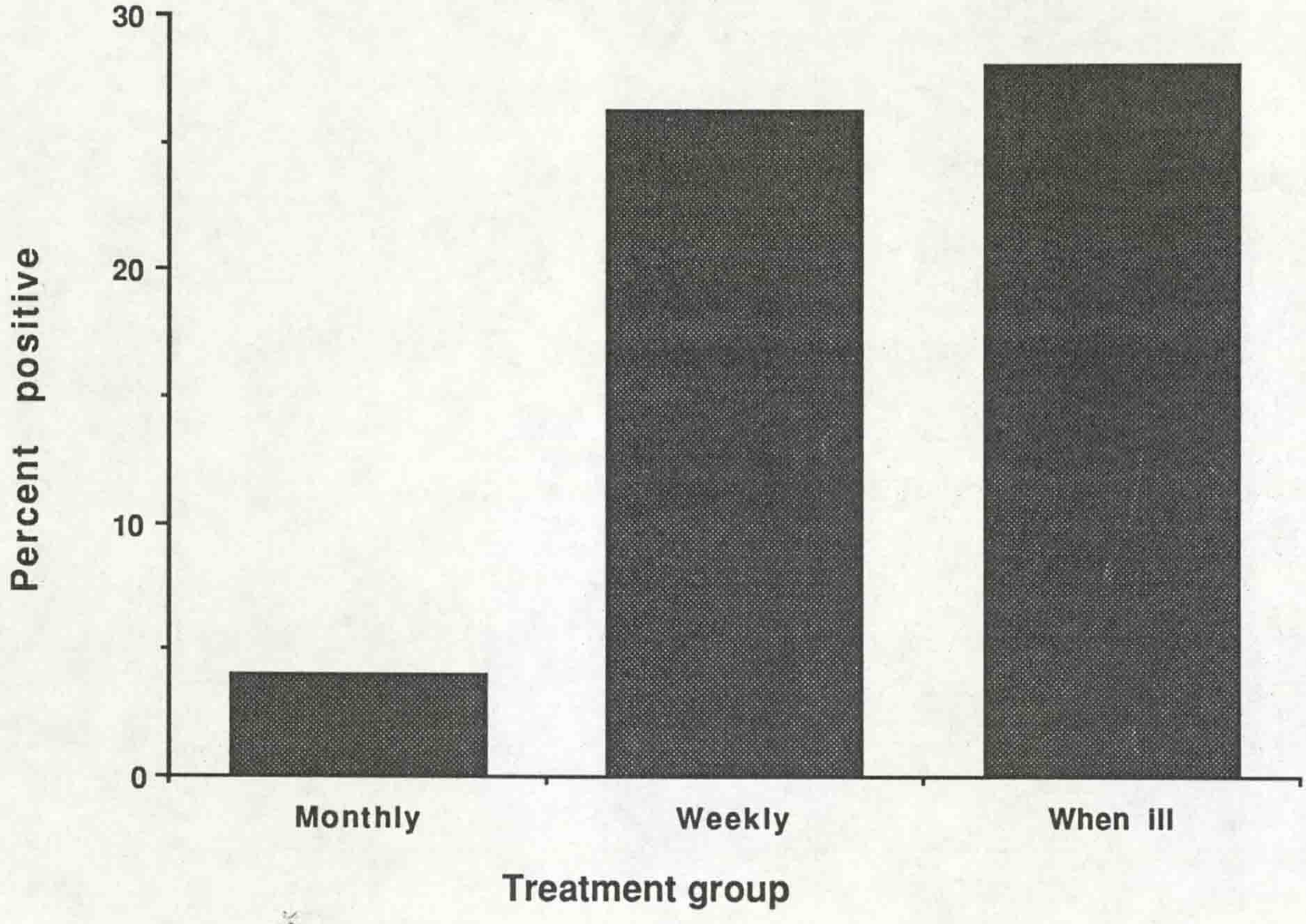


Fig 10.8 Percent parasite positive placental specimens by treament group



CHAPTER 11

CHILD SURVIVAL AND NUTRITIONAL STATUS IN THE FIRST YEAR OF LIFE

11.1 INTRODUCTION

This chapter is devoted to the infants born to the mothers who were in the study. The health of the mother during pregnancy and the outcome of pregnancy for the mother and newborn have been presented in chapters 8, 9 and 10.

Two main outcomes are presented in this chapter: (1) the survival of the infants in the first twelve months of life and the (2) nutritional status of infants during the same period. Each of these is presented in the light of explanatory variables that could affect them. These variables are: area of residence, season of recruitment and delivery months, age and parity of mother, antimalarial chemoprophylaxis during pregnancy, malaria infection in the mother at the time of delivery, in the placenta or in the cord and birthweight.

Newborns were examined at the time of birth for malaria parasites and nutritional status based on anthropometric measurements as described in chapter 8. The infants were then followed up at 6 months and at 12 months.

At each of these follow up visits it was noted whether the child was alive or not. If the child was dead then the date and the reported cause of death was noted. If the child was alive then the child was examined for malaria parasites and nutritional status by repeating the anthropometric measurements taken at birth.

11.2 MALARIA INFECTION IN THE INFANT

Of the 194 children examined at 6 months 107 (55.2%) were parasitaemic. The infection types were mainly P.falciparum (99.1%) and the rest were P.malariae (0.9%) of 222 infants examined at one year 153 (70.7%) had parasites in their blood.

The infection type was predominantly P.falciparum (95.8%) with P.malaria increased to 3.52 and P.ovale 0.64%.

The infant parasite density at 6 months and 12 months were not signficantly different and did not differ significantly by the treatment groups to which their mothers belonged. Thus, whether the mother was receiving monthly or weekly chemoprophylaxis or was being treated when ill did not affect the mean parasite densities of infants at 6 and 12 months of age. This should have been expected given the intensity of transmission (Chapter 3) and the fact that the infants seemed to loose their immunity rather rapidly after birth (Chapter 4). See table 11.1.

These parasite densities differed neither by mothers' age, parity nor by their area of residence. The parasite prevelance differed by the age of the infant but not by their mothers' treatment group, age, parity or area of residence.

11.3 NUTRITIONAL STATUS

The mean weight of the infants at 6 months was 7.2 $\pm$ 1.34kg and 8.75 $\pm$ 1.5kg at 12 months. The mean height of the infants at 6 and 12 months were 61.86 $\pm$ 5.31cms and 68.72 $\pm$ 4.95cms respectively.

There was no statistically significant difference between the mean weights of the infants either at 6 months or at 12 months by area of residence. The mean height at 6 months was significantly greater in area A 86.3 $\pm$ 4.6 cms) followed by area B (62.022 $\pm$ 5.814 cms) than area C. ($F = 4.58$, Df. 2/198, $P < 0.025$). By 12 months the difference in mean height by area was no longer statistically significant.

These anthropometric measurements were compared to international standards and Z scores were obtained and used for comparing nutritional status by various factors.

11.3.1 Area of residence

The area of residence did not make any statistically signficant difference on the mean weight for age, weight for height and for age Z scores at 6 and 12 months.

The data shows that by six months the infants' weight for age values compared favourably with the international standard but that by 12 months they fell considerably below the international standard of weight for age score. This may be due to the fact that in the first 6 months the infants are relatively protected from malaria and other infections notably measles, by maternal antibodies passively received in uterus. This may also reflect on the inadequacy of the weaning practices in terms of quality, quantity and frequency of feeding and the hygienic practices surrounding it. Judging from the causes of death, many of these infants would have suffered many bouts of gastroenteritis, malaria, acute respiratory infections and perhaps an encounter with measles.

Weight for height scores suggested that infants were doing quite well. Their weight was normal for their height both at 6 and 12 months. They, however, were short for their age compared to international standards.

11.3.2 Season of recruitment and delivery

Although the effect of seasonality was seen in mean birth weights, which were significantly lower for newborns whose mothers were recruited at the beginning of the wet season, there was no seasonal variation in the mean Z scores for weight for age, weight for height or height for age. Thus the months of recruitment and the month of delivery had no association to the anthropmetric values of the infant either at 6 months or at 12 months (Tables 11.1, 11.2, 11.3).

It was expected that the babies delivered at the beginning of the

wet season might have been so exposed to intense malaria transmission that their nutritional status, as judged by their mean weight at six months would have been poor. Infants born during wet months had slightly higher mean weight at six months than the infants born during drier months, but the difference was not statistically significant. The same was true of the weight of infants at 12 months.

The height for age of the infants was found to be similar to those of other children in the same area reported in Chapter 7. The infants were generally too short for their age as compared to the international standard of height for age, but their weight was proportional to their height.

11.3.3 Birth weight

The birth weight did not signifcantly affect the weight of the infants at six months, 7.151 $\pm$ 1.277kg was the mean weight of infants with normal birth weight as compared to the mean weight of infants with low birth weight which was 6.97 $\pm$ 1.913. This difference was not statistically significant.

The height for age at 6 months revealed the effect of low birth weight. The infants born with low birth weight had a mean height for age Z score of -3.388 $\pm$ 2.687 as compared to -0.662 $\pm$ 1.95 of infants who had normal birth weight (2.5kg and over). The differences were statistically significant ($F = 6.40$, df 1/176, $P<0.025$).

The mean weight for height Z score at 6 months was higher for infants who had normal birth weight than their low birth weight counterparts, but the difference was not statistically significant. The same was true of the mean weight for age Z scores (Table 11.5).

11.3.4 Chemoprophylaxis status of the mother (Treatment group)

The results of the study showed no statistically significant

difference between the treatment groups in terms of each of the anthropometric measurements at six months and at twelve months.

The treatment group of the mother did not significantly affect the mean weight for age Z score at 6 and 12 months (Table 11.5).

Although the infants whose mothers received monthly chemoprophylaxis did better in their weight for age at 6 months (-0.186 $\pm$ 1.280) as compared to those receiving weekly chemoprophylaxis (-0.651 $\pm$ 1.363) and those whose mothers were treated only when ill (-0.417 $\pm$ 1.729). The difference was not statistically significant.

11.3.5 Maternal age and parity

The age or parity of the mother did not appear to influence the weight of the infants either at six months or at 12 months. Neither did they significantly affect height and the other anthropometric mesurements at 6 or 12 months (Table 11.4 and 11.6).

The infants of the women aged 15 to 19 appeared to have lower mean Z scores than the rest of the infants but the difference was not signficant at six months ($F = 0.30$, df 21/158 $p > 0.05$) or 12 months ($F = 0.44$, df = 24/182, $p > 0.055$).

From these results it was clear that the age and parity of the mothers in this study had no statistically significant influence on the nutritional status of their infants at the ages of 6 and 12 months as measured by weight for age, weight for height and height for age Z score values.

11.3.6 Maternal, placental and cord parasites at the time of delivery

The maternal peripheral parasitaemia at delivery affected the weight of the child at six months slightly and the differences observed were not statistically significant. The maternal peripheral parasitaemia had no effect on the height of the infants at six months and at 12 months.

The mean weight at 12 months, of infants whose mothers had parasitaemia at the time of delivery was 7.88kg as compared to 8.63kg for infants whose mothers were parasitaemic. This difference was statistically signficant ($F = 4.59$, $df = 1/49$ $P<0.05$).

The infants whose mothers had placental infection when they were born had mean weights and mean height that did not differ significantly from those whose mothers' placentae were parasite free, this was so both at 6 and 12 months. The weight for height Z scores at 6 months however, were lower for the infants who had had parasitised placentae (-2.42 ± 2.52) than those who had had parasite free placentae (-1.60 ± 1.86) and the difference was statistically significant ($F = 4.67$, $df = 1/148$: $P<0.025$). The impact of placental parasitisation was thus detectable in these infants 6 months after birth. This effect was not observed at 12 months.

Cord parasitaemia had no effect on weight or height at 6 or 12 months. The infants who were parasitaemic at their first follow-up visit and examination appeared to have lower mean weight for age Z scores (-0480 ± 1.619) than those that were aparasitaemic ($-0.26 \pm 1,27$) but the difference was not statistically significant.

Plotting weight at six months against height and arm circumference at the same time, a relationship exists between the height weight and arm, circumferance, which is to be expected. The correlation coefficient of 0.311, 0.553, $p < 0.001$, respectively were observed. The correlation between height and head circumference weight and chest circumference at 6 months, all had correlation coefficient value of over 0.3 and hence all the values were different from 0, this was statistically significant in each case ($p<.001$).

Similar correlation coefficients were seen at one year where weight and height was .334, $p<0.001$; weight vs arm circumference was 0.392, $p<0.001$ weight vs head circumference 0.337, $p<0.001$ and weight and chest circumference was 0.523, $p<.001$.

11.4 INFANT SURVIVAL

There were 318 births recorded in the study. Of these, 8 (23 per thousand live births) died in the first month of life, and 43 (135.2 per 1000 live births) died in the first year of life. Thus neonatal and infant mortality rates in the study were 23 per 1000 and 135.2 per 1000 live births respectively. The causes of death were acute respiratory infection (ARI) (34.8%). Measles (26.1%) diarrhoea and vomiting (17.4%), malaria (15.2%) and others.

11.4.1 Area of residence

The one year survival rate was higher in area C where 41 out of 46 (891 per thousand live births) infants survived as compared to 97 out of 118 (822/1000) in area A and 86 our of 107 (811/1000) in area B (see Table 11.7). Thus the rate of survival was highest in area C and lowest in area B but the difference was not statistically significant.

11.4.2 Season of recruitment and of delivery of the mother

As was expected, the month of recruitment, which related closely to the month of conception, significantly affected the survival of their infants in the first year of life. The wettest season had the lowest survival rate of 757 live births for those whose mothers were recruited during the dry months, but the difference was not statistically significant (see Table 11.7).

Maternal parasitaemia at the time of delivery did not vary significantly by month of recruitment and the same applied to parasite counts, which seemed high in the month of December but still the difference was not statistically signficiant.

The survival of the infants was highest for infants born during the wet season (846.9 infants per 1000 lives births) followed by those born during the dry months (818.2 per 1000 live births) and those born during the moderately wet season (781 per thousand live births) (Table 11.7). This could be explained

by the fact that these infants entered the intense transmission season after losing most of their maternal immunity against malaria.

The difference observed was however not statistically significant ($X^2 = 1.03$, 2Df $p > 0.05$).

11.4.3 Age and parity of the mother

Age and parity of the mother did not significantly affect the survival rate of the infants. The infant survival appeared to be highest among women aged 35 years and over (916.7 infants per 1000 live births) and lowest in the age group 15 to 24 but the difference was not statistically significant ($X^2 = 0.86$ 2df, $p > 0.05$) see Table 11.7.

The table also appears to suggest that survival of the infants increases with increasing parity. Thus 841.7 infants per 1000 live births in the study population survived the first twelve months among women who were para 2 and over as compared to 8,24,6 among para ones and 818.2 among primigravidae who thus had the lowest infant survival rate. This difference was again not statistically significant.

11.4.4 By chemoprophylactic status

The effect of chemoprophylaxis during pregnancy on the survival and nutritional status of infants was difficult to predict. Regular prophylaxis had a significant impact on placental parasites (Chapter 10). This impact would be expected to show not only birth weight but also on-child survival and of health status. Regular prophylaxis could also have reduced the amount of maternal antibodies passed on to the baby *in utero* and hence the infant would as a result be more susceptible to malaria sooner after birth than those whose mothers were not on regular effective chemoprophylaxis. The results showed the highest survival rates among the infants of mothers who received weekly chemoprophylaxis and the rate was lowest among infants whose mothers were

only treated when ill. The difference was however not statistically significant. See Table 11.8.

11.4.5 Birth weight and nutritional status at 6 months

Nearly half of all the children (48%) born below the normal birthweight of 2.5kgs died before they were 1 year of age. The risk of dying increase threefold as compared to that of normal birth weight children. The birth weight was thus the single most important factor that influenced the survival of infants in their first year of life (x^2 = 25.29 idf, p <0.001), Table 11.8. The mean birth weight of survivors was 3.117 $\pm$ 0.517 as compared to 2.85 $\pm$ 0.463 for the infants who died. This difference was also statistically significant. (F = 9.99, df 1/251, p <0.005).

The infants who survived the first six months, but died before twelve months exhibited poor nutritional status at 6 months. Their weight for height was adequate based on the Z scores, but both their weight for age and height for age scores were below international standards. This was true of the infants who survived 12 months but was worse for the infants who died before the completion of 12 months. The mean height for age Z score for the infants who died was -2.56 $\pm$ 2.7 as compared to -1.56 $\pm$ 1.9 for those who survived. This difference was statistically significant (2.63, df = 1/175, p<0.025). Similar Z scores for weight for age and weight for height were lower for the non-survivors but the differences were not statistically significant.

11.4.6 Malaria parasites: maternal, placental and cord

The infants of mothers who were parasitaemic at the time of delivery had a slightly lower survival rate (806.5 compared to 834.1 per thousand live births) but the difference was not statistically significant (see Table 11.8).

The mean maternal peripheral parasites counted at delivery was

lower for the mother of surviving children than for the mothers of the children who died but again the difference was not statistically significant.

Placental infection appeared to affect the survival rate of the infants. 44 of 58 (758.6per 1000 live born children) infants who had parasitised placentae survived as compared to 135 of 157 (or 859.9 per 1000 live born children) with no placental infection but the difference was again not significant (X^2 = 3.11m, 1df. p>0.05).

Table 11.8 presents the data on cord parasitaemia and its influence on infant survival. Cord parasitaemia substantially increased the risk of death in the first year of life. The survival rate of cord blood positive infants was 666.7 as compared to 845.5 per thousand infants born alive in the study whose cord-blood samples were negative for malaria parasites. (X^2 = 3.80, 1 df, p< 0.050).

The cord-blood parasite counts were much higher for the non-surviving infants than for the surviving infants. The difference was statistically signficant (F = 4.14, df = 1/234, p<0.050).

The mean Z score for the infants who died in infancy was lower at six months (-1.02 $\pm$ 1.43) as compared to those who survived (-0.33 $\pm$ 1.43). The mean weight for height Z scores of the infants who survived (1.17 $\pm$ 2.27) did not differ much from those of the infants who died (1.26 $\pm$ 1.51).

In summary the key factors that affected infant survival in the first year of life were birthweight and cord parasitaemia. Both of these factors can be improved through effective antimalarial chemoprophylaxis. This study has also shown that the months of conception may also be an important factor, since the season of recruitment (not of birth) signficantly contributed to the survival of the infant. It would appear that children born just before the wet season are able to go through the high transmission season when they still have adequate maternal protection while those born in the dry season enter the wet high transmission with inadequate maternal protection. The month of delivery

would not be easily amenable to effective intervention. One option would be to target the children born in dry months for chemoprophylaxis during the wet months.

11.5 DISCUSSION

The main findings presented in this chapter are that parasite prevalence reached half the population of infants (55%) by the age of six months and that nearly three quarters (71%) are infected by the age of 12 months. Similar studies in Nigeria tend to show a slower increase in parasite prevalence under six months with a more rapid increase thereafter reaching higher peak prevalence rates of 80-90% by the age of one year (Bruce-Chwatt, 1952, Gilles et al., 1969 and Molineaux and Gramiccia, 1980). The peak observed in West Africa is certainly higher. The reasons for this are not clear but a universal access to antimalarials in Saradidi may be partly responsible for the lower peaks in prevalence rate of parasitaemia among the infants.

Pregnant women who were not from Saradidi and were examined for malaria parasites at the time of delivery had a rate that was twice as high as pregnant women in Saradidi. This is presented and discussed in Chapter 10. This may support the explanation given above for lower peak parasite prevalence rate in Saradidi.

The parasite density among the infants was high and remained so for up to 12 months and was not affected by maternal, placental or cord experience with malaria parasites or by whether or not the mother was on chemoprophylaxis during pregnancy. The age, parity and area of residence also had a significant effect on it.

The data presented show that the nutrition status of the infants, based on weight for height Z scores compared well with the international standard.

The weight for age measure gave a slightly different picture.

According to the weight for age Z values the nutritional status was good only up to six months but by 12 months the infants were below the international standard. The height for age Z scores were negative throughout infancy indicating that the infants were always shorter than the international standard.

It would be reasonable to conclude that the nutritional status varied in the first year of life but also to recognise that the health of the infants may be affected by the onset of weaning and the beginning of exposure of the infants to various infections. It is after six months that most of the passively acquired maternal antibodies against various diseases have waned and the infant is more susceptible to such diseases.

The weaning process itself may affect nutritional status due to the practice itself. The quality and frequency of feeding and the hygiene surrounding it may cause various problems leading to poor nutrition status.

Placental parasites at the time of birth and current parasitaemia in the infant tended to affect the nutritonal status in terms of lower weight for age. Other workers have described this relationship between parasitisation and malnutrition (McGregor and Smith, 1952; Walter and Waterloo, 1954). McGregor and his colleagues conclude that although there is a relationship between parasitaemia and malnutrition it is difficult to unravel. Workers in West Africa have also shown that children tend to loose weight during wet seasons when malaria transmission is also at its peak. (Gilles et al., 1969). The project demonstrated better anthropometric measurements among children who were protected (Molineaux and Gramiccia, 1980).

Another factor that was expected to affect the nutritional status in infancy was birth weight. This impact was not demonstrated in the study. This might have been so partly because nearly half of the children with birth weights below 2.5kg died and there might not have been enough of them at 6 and at 12 months for valid statistical comparisons.

Neonatal mortality rate in the study population was 25 per 1000 live births and infant mortality rate was 135.2 per thousand live births. These rates are the national rates of 18/1000 and 89/1000 respectively (Kibet, 1981).

The most important determinant of infant survival is birth weight. Nearly half of all the children born below the normal birth weight of 2.5kg died before their first birthday. The findings in this chapter are consistent with the findings of many studies (Wilcox, 1983) found a close relationship between birth weight and survival through the neonatal period. Victora and her colleagues studied 5,914 Brazillian children; they concluded that there was a strong association between birth weight and neonatal and post-neonatal mortality (Victora et al, 1987). The neonatal mortality rate in our study was even higher than 21.3 per 1000 in the Brazillian study (Victora et al., 1987).

The risk of death in our study for the underweight newborns was increased threefold. A similar risk increase was described by Victora and her colleagues, 1987. This phenomenon has been observed for many years. Blacklock and Gordon noted high perinatal and neonatal mortality in highly malarious areas (Blacklock and Gordon 1925). Bruce-Chwatt could not confirm this finding (Bruce-Chwatt, 1952). More recently, studies have shown low birth weight to be one of the most important predictors of neonatal mortality (McCormick, 1985). In the United States of America, birth weights below 2,500gm have been associated with 5 fold increase in post-neonatal death (McCormick, 1985).

The Brazillian study did not find other factors like socio-economic status, maternal age, parity and area of residence as important determinants of infant survival (Victoria et al., 1987). Our findings were similar. As in their study we found that many of the infants died of infections like API, measles and diarrhoea and vomiting. They considered that low birthweight and related factors could have contributed to 40% of the infant deaths.

In this study the women who were recruited into the study at

the beginning of the wet season (March-April) and those who delivered at the beginning of the dry season (November-December) tended to have infants of higher survival rates than the other women who were recruited into the study during the beginning of the dry season (November-December) and those who delivered at the beginning of the wet season (March-April) have infants of higher survival rates than the other women. In the case of pregnancy the foetus is most affected if it is exposed to intense malaria transmission in early pregnancy. The infants are affected more if they are exposed to intense malaria transmission when their maternal acquired antibodies have declined.

Our data demonstrated the effects of cord and placental parasitisation on infant survival. These effects were not significantly influenced by any of the three strategies of chemoprophylaxis that were being evaluated. This lack of effectiveness could be attributed either to ineffectiveness of the drug used as the rate of chloroquine resistance was already very high in this community (Chapter 4) or non-compliance as described by Kaseje et al., (1987).

Malaria certainly contributes considerably to infant deaths. An effective intervention in highly malarious areas must include effective and timely chemoprophylaxis in pregnancy and particularly for women pregnant for the first time (World Health Organisation, 1984).

The main object of this study was to evaluate the effectiveness of monthly chemoprophylaxis to pregnant women in terms of maternal, foetal and infant outcomes. We have found no advantage of weekly chemoprophylaxis over the monthly approach. It would appear that a more effective drug than chloroquine given early in pregnancy at monthly intervals for at least four months would improve the survival of infants by improving birth weight which appears to be the key predictor of infant survival.

Table 11.1 The mean Z scores of height for age at 6 and 12 months by month of recruitment.

Month of Recruitment	No.	Mean Z score (12 months)	No.	Mean Z score at (6 months)
January	21	-2.52 ± 1.84	21	-2.11 ± 1.56
February	10	-2.2 ± 1.76	14	-1.84 ± 2.00
March	16	-1.59 ± 1.46	18	-0.99 ± 1.41
April	13	-2.72 ± 2.22	13	-1.68 ± 1.23
May	5	-2.76 ± 2.38	5	-2.72 ± 0.09
June	3	-1.66 ± 1.64	4	-0.69 ± 1.09
July	12	-1.51 ± 1.29	13	-1.19 ± 1.63
August	21	-2.44 ± 1.55	21	-2.38 ± 2.59
September	34	-1.07 ± 2.05	23	-1.83 ± 2.10
October	32	-2.33 ± 1.31	22	-1.60 ± 2.28
November	18	-1.66 ± 1.29	12	-1.61 ± 2.64
December	23	-1.57 ± 1.30	17	-0.69 ± 1.14

Table 11.2 The mean scores of height for age at 6 and 12 months by month of birth.

Month of Birth	No.	Mean Z score (12 months)	No.	Mean Z score (6 months)
January	20	-2.79 ± 1.90	18	-1.89 ± 2.14
February	26	-1.91 ± 1.51	19	-2.03 ± 2.60
March	24	-2.13 ± 2.00	13	-1.90 ± 2.02
April	19	-1.78 ± 1.28	14	-1.63 ± 2.11
May	20	-1.92 ± 1.40	19	-1.51 ± 2.96
June	26	-2.39 ± 1.40	19	-2.20 ± 2.27
July	18	-2.44 ± 1.23	19	-1.78 ± 1.43
August	12	-2.61 ± 2.10	17	-1.71 ± 1.85
September	15	-1.98 ± 1.70	17	-1.40 ± 1.22
October	7	-2.41 ± 1.26	7	-1.89 ± 1.64
November	6	-2.32 ± 2.28	7	-2.28 ± 2.29
December	17	-1.83 ± 1.49	17	-0.92 ± 1.49

Table 11.3 The mean Z scores for weight for height and weight for age by month of recruitment of the mother.

Month of Recruitment	No.	Mean Z Scores (wt. for age)	No.	Mean Z Scores (wt. for ht.)
January	21	-0.13 ± 1.51	20	1.69 ± 1.62
February	14	-0.58 ± 2.20	14	2.79 ± 3.09
March	18	-0.43 ± 0.85	18	0.47 ± 1.62
April	13	-1.00 ± 0.85	13	0.45 ± 1.67
May	5	-0.85 ± 1.04	5	1.80 ± 1.14
June	13	-0.31 ± 0.82	4	0.21 ± 0.89
July	21	-0.19 ± 1.30	13	1.10 ± 3.01
August	21	-1.09 ± 1.38	19	0.70 ± 2.14
September	23	-0.45 ± 1.84	15	0.81 ± 2.26
October	22	-0.17 ± 1.43	22	1.82 ± 2.91
November	12	-0.76 ± 1.22	11	0.82 ± 2.91
December	17	-0.16 ± 1.02	17	1.48 ± 1.64

Table 11.4 Mean height for age Z scores at 6 months by parity, age of mother and birth weight.

Independent Variable	Mean Z score	Significance Test F	df	P
Parity				
0	-2.19 ± 2.20			
1	-1.40 ± 1.25	1.56	4,177	>0.05
2	-1.46 ± 2.89			
>2	-1.54 ± 1.70			
Age of mother				
<25 years	-1.90 ± 2.11			
25-34 years	-1.31 ± 1.55	1.37	2,177	>0.05
>34 years	-1.36 ± 1.89			
Birth Weight				
<2.5kg	-3.39 ± 2.69	6.4	1,176	<0.025
2.5kg+	-0.66 ± 1.95			

Table 11.5 The mean weight for age, weight for height and height for age Z scores by treatment group.

Z Score	Treatment group and mean Z score values			P
	Monthly	Weekly	When ill	
Weight for age:				
6 months	-0.18 ± 1.28	-0.65 ± 1.36	-0.42 ± 1.73	>.05
12 months	-0.89 ± 1.13	-0.89 ± 1.13	-0.85 ± 1.80	>.05
Weight for height:				
6 months	1.49 ± 2.22	1.04 ± 2.20	1.14 ± 2.42	>.05
12 months	0.75 ± 1.77	0.63 ± 1.77	1.09 ± 2.21	>.05
Height for age:				
6 months	-1.61 ± 2.13	-1.945 ± 1.70	-1.67 ± 1.23	>.05
12 months	-1.99 ± 1.57	-2.20 ± 1.88	-2.31 ± 1.55	>.05

Table 11.6 Mean weight for age, weight for height and height for age by placental parasite status.

Z Score	Placental Parasites +	Placental Parasites −	P
Weight for age: at 6 months	-0.72 ± 1.82	-0.43 ± 1.26	>0.05
Height for age:			
6 months	-2.42 ± 2.52	-1.595 ± 1.86	<0.05
12 months	-2.58 ± 1.80	-2.055 ± 1.53	>0.05
Weight for height:			
12 months	0.704 ± 1.75	1.04 ± 2.48	>0.05

Table 11.7 The number of infants surviving the first 12 months of life per thousand live births by various factors (area of residence, seasons of recruitment and birth age, and parity of mothers).

Factors affecting Survival	No.	Survival rate per 1000 live births		Stat. X^2	Significance 2	P
		No.	rate per 1000			
Area of Residence						
A	118	97	822.0			
B	107	86	803.7	1.75	2	>0.05
C	46	41	891.0			
Season of recruitment						
Wet	92	76	826.1			
Dry	47	36	757.0	0.92	2	>0.05
Moderate	126	104	825.4			
Season of delivery						
Dry	110	90	818.2			
Wet	98	83	846.9	1.03	2	>0.05
Moderate	55	43	781.8			
Age of mother						
15-24	192	156	812.5			
25-34	58	48	827.6	0.86	2	>0.05
25 & over	12	11	916.7			
Parity of mother						
0	88	72	818.2			
1	57	47	824.6	0.21	2	>0.05
2 & over	120	101	841.7			

Table 11.8 The number of infants surviving the first 12 months of life per thousand live births by various factors (treatment group, birthweight and parasite prevalence).

Factors affecting	No.	Survival Rate per 1000		Statistical Significance		
		No.	rate per 1000	X^2	df	P
Monthly prophylaxis	89	74	883.15			
Weekly prophylaxis	88	77	875.5	3.97	2	>0.05
Treatments when ill	88	67	761.4			
Birth weight >2.5	232	2982	853.4			
Birth weight <2.5	21	11	523.8	25.29	1	<0.0001
Mother parasite +ve	31	25	806.5			
Mother parasite -ve	153	129	843.1	0.25	1	>0.05
Placental Parasite +ve	58	44	758.6	3.11	1	>0.05
Placental Parasite -ve	157	135	859.9			
All infants	**215**	**179**	**832.6**			
Cord parasite +ve	18	12	666.7			
Cord parasite -ve	220	186	845.5	3.80	1	>0.05

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SARADIDI COMMUNITY HEALTH PROJECT - PREGNANCY FORM LSTM/SCHP 1.B

RECRUITEMENT FORM NO [1] (1)

AREA _____ AREA ID [ ] (2) VILL [ ] (3 4) HOUSE [ ] (5 6 7) INDIV [ ] (8 9 10)

VILLAGE _____

NAME _____

TREATMENT GROUP (11)
10 mg [1]
5 mg [2]
Village [3]

DATE OF ENTRY/ DAY [ ] (12 13) MTH [ ] (14 15) YR [ ] (16 17)
STARTED TREATMENT

AGE (Years) [ ] (18 19)

PARITY [ ] (20 21) [ ] (22)

HEIGHT (cms) [.] (23 24 25 26)

RISK STATUS (27)
High Risk [1]
Normal [2]

If High Risk indicate reason

Hb electrophoresis (28)
AA [1]
AS [2]
SS [3]
Other [4]
Not done [9]

If Other then specify -

SARADIDI COMMUNITY HEALTH PROGRAMME - ANTE-NATAL HISTORY

LSTM/SCHP 2.0

NAME _____

ID AREA (3) VILL (4 5) HOUSE (6 7 8) INDIV (9 10 11)

	2,1 (12 Weeks)	2,2 (20 Weeks)	2,3 (36 Weeks)	2,4 (Delivery)	2,5 (+6 Weeks)
FORM & VISIT	1 2	: 2	: 2	: 2	: 2
DATE	12 13 / 14 15 / 16 17	12 13 / 14 15 / 16 17	12 13 / 14 15 / 16 17	12 13 / 14 15 / 16 17	12 13 / 14 15 / 16 17
WEIGHT (Kgs)	18 19 20	18 19 20	18 19 20	18 19 20	18 19 20
HISTORY OF FEBRILE ILLNESS (21) Yes 1, No 2, nk 9					
MALARIA PARASITE (22) Yes 1, No 2, nk 9					
TYPE OF INFECTION	1st (23) 2nd (24) 3rd (25)				
P. falciparum 1, P. malariae 2, P. ovale 3, P. vivax 4, Not Known 9					
DENSITY OF INFECTION (999=NK)	26 27 28	26 27 28	26 27 28	26 27 28	26 27 28
HAEMOCRIT (99=NK)	29 30	29 30	29 30	29 30	29 30
	Blood (31) Urine (32)				
CHLOROQUINE Yes 1, No 2, nk 9					
MALARIA ANTIBODIES TITRE (999/999=NK)	33 34 35 / 36 37 38	33 34 35 / 36 37 38	33 34 35 / 36 37 38	33 34 35 / 36 37 38	33 34 35 / 36 37 38

Appendix 1a

SARADIDI COMMUNITY HEALTH PROJECT - PARTURITION

LSTM/SCHP 3.C

FORM NO [3] 1

NAME (of Mother) _____

AREA ID [ ] 2 | VILL [ ] 3 4 | HOUSE [ ] 5 6 7 | INDIV [ ] 8 9 10

DATE OF DELIVERY DAY [ ] 11 12 MTH [ ] 13 14 YR [ ] 15 16

GESTATION AT PARTURITION (In weeks) [ ] 17 18

OUTCOME OF PREGNANCY 19
- Abortion [1]
- Still Birth [2]
- Live Birth [3]

METHOD OF DELIVERY 20
- SVD [1]
- Forceps/Vacuum [2]
- Caesar. [3]

PLACE OF DELIVERY 21
- Home [1]
- Hospital [2]
- Other [3] If Other specify

PRIOR CHOICE OF BIRTHPLACE 22
- Home [1]
- Hospital [2]
- Other [3] If Other specify

RESULT (1=singleton 2=twin etc) [ ] 23

CONDITION OF MOTHER 24
- Alive [1]
- Dead - Medical [2]
- Dead - Obstetric [3]
- Dead - Other [4]
- Dead - Nk cause [5]
- Unknown condition [9]

PLACENTA

PLACENTA WEIGHT (Kgs) (After washing, membranes intact +6 cms cord) [ ] 25 26

MALARIAL PARASITES 27
- Yes [1]
- No [2]
- Not Done [9]

TYPE OF INFECTION 28 29 30
- P. falciparum [1] [1] [1]
- P. malariae [2] [2] [2]
- P. ovale [3] [3] [3]
- P. vivax [4] [4] [4]
- Not Known [9] [9] [9]

DENSITY OF INFECTION

Parasites per 300 WBCs 999 = Nk [ ] 31 32 33

MALARIA PIGMENT 34
- Yes [1]
- No [2]
- Not Done [9]

Appendix 1c

SARADIDI COMMUNITY HEALTH PROJECT - INFANT AT BIRTH LSTM/SCHP 4.C

FORM NO [4] (1)

NAME (of Mother) _____

NAME (of Infant) _____ DATE OF BIRTH

ID of MOTHER — AREA [ ] (2) VILL [ ] (3 4) HOUSE [ ] (5 6 7) INDIV [ ] (8 9 10) CHILD NO. IF TWIN [ ] (11) DAY [ ] (12 13) MTH [ ] (14 15) YR [ ] (16 17) SEX (18) Male [1] Female [2]

CORD BLOOD

MALARIAL PARASITES (19) Yes [1] No [2] Not Done [9]

TYPE OF INFECTION (20) (21) (22)

	20	21	22
P. falciparum	1	1	1
P. malariae	2	2	2
P. ovale	3	3	3
P. vivax	4	4	4
Not Known	9	9	9

DENSITY OF INFECTION Parasites per 300 WBCs (999 = Nk) [ ] (23 24 25)

MALARIAL ANTIBODIES 999 / 999 = Not Known [ ] (26 27 28) / [ ] (29 30 31)

HEAMOCRIT (99=NK) [ ] (32 33)

MEASUREMENTS

WEIGHT (Kgs) [ ] (34 35 36)

HEIGHT/LENGTH (Cms) [ ] (37 38 39)

ARM CIRCUMFERENCE (Cms) [ ] (40 41 42)

HEAD CIRCUMFERENCE (Cms) [ ] (43 44 45)

CHEST CIRCUMFERENCE (Cms) [ ] (46 47 48)

Appendix 1 d

SARADIDI COMMUNITY HEALTH PROJECT - INFANT EXAMINATION LSTM/SCHP 5.B

NAME (of Infant) _____

NAME (of Mother) _____

ID AREA [3] VILL [4 5] HOUSE [6 7 8] INDIV [9 10] CHILD NO. IF TWIN [11]

FORM & VISIT NO. [5,1] (6 Months) [5,2] (12 Months)
(1 2)

	DAY	MTH	YR			
DATE OF VISIT	[12 13]	[14 15]	[16 17]	[12 13]	[14 15]	[16 17]
WEIGHT (Kgs)	[18 19 20]			[18 19 20]		
HEIGHT/LENGTH (Cms)	[21 22 23]			[21 22 23]		
ARM CIRCUMFERENCE (Cms)	[24 25 26]			[24 25 26]		
HEAD CIRCUMFERENCE (Cms)	[27 28 29]			[27 28 29]		
CHEST CIRCUMFERENCE (Cms)	[30 31 32]			[30 31 32]		

MALARIAL PARASITES [33]

Yes	1	1
No	2	2
Not Done	9	9

TYPE OF INFECTION [34] [35] [36]

P. falciparum	1	1	1
P. malariae	2	2	2
P. ovale	3	3	3
P. vivax	4	4	4
Not Known	9	9	9

DENSITY OF INFECTION
Parasites per 300 WBCs
999 = Nk [37 38 39]

HAEMOCRIT 99 = Nk [40 41]

MALARIAL ANTIBODIES
999 / 999 = Not Known [42 43 44] / [45 46. 47]

SARADIDI COMMUNITY HEALTH PROJECT - FIRST INFECTION / SURVIVAL LSTM/SCHP 6.B

FORM NO [6] (1)

NAME (of Mother) _____

NAME (of Infant) _____

ID of MOTHER — AREA [ ] (2) VILL [ ] (3 4) HOUSE [ ] (5 6 7) INDIV [ ] (8 9 10) CHILD NO. IF TWIN [ ] (11)

FIRST MALARIA INFECTION

Did the Infant experiance a MALARIA infection in the first 12 months (12)
Yes [1]
No [2]

Date of first experiance of patent MALARIA — DAY [ ] (13 14) MTH [ ] (15 16) YR [ ] (17 18)

SURVIVAL

Did the Infant SURVIVE the first 12 months (19)
Yes [1]
No [2]

DATE OF DEATH — DAY [ ] (20 21) MTH [ ] (22 23) YR [ ] (24 25)

REPORTED CAUSE OF DEATH

	(26)	(27)	(28)
Malaria	1	1	1
Measles	2	2	2
Diarr. & Vomit	3	3	3
Malnutrition	4	4	4
A.R.I	5	5	5
Chronic Cough	6	6	6
Accident	7	7	7
Others	8	8	8
Not Known	9	9	9

Appendix 1f

COMMUNITY CROSS SECTION SURVEY

Name of household head_____ Area_____ Village_____

I.D. No. ☐☐☐/☐☐☐ Date ☐☐/☐☐/☐☐

1. Fill in names, I.D., age and sex for all members of the household in the table below:-

I.D.	NAME	SEX 1- Male 2- Female	AGE (YRS) 1-00	IF < 5 YEARS	
				Date of birth	ID of Mother
01					
02					
03					
04					
05					
06					
07					
08					
09					
10					

2. Was any baby born in this household in the last 12 months?

   1- Yes

   2- No ☐

   If yes, fill in the table below.

	Name of baby	Date of birth
1		

a)

	Name of deceased	Date of death	Age at death	Cause of death
1.				
2.				
3.				

b) Deaths < 1 year -

Deaths 1 year + -

Total deaths -

4. Fill in the following table for women 15 - 49 years:-

Name	Age (Yrs)	Yrs of Completed education. 0-None 1=1-4 yrs 2=5-8 yrs 3=7 8 yrs 8=still in schoo-ol. 9=DK	Marital Status 0=Never married 1=Mono married now 2=Poly-Married 3=Divorced or separated	Children born alive living in household.	Children born alive living elsewhere	Children born alive who have died	Pregs. not surviving since last

ill in the follwing table for children under 5 years.

Name	I.D. of Mother	Age in months	Splenomegaly 1-Yes 2-No	Blood slide (MPs) 1-Pdsitive 2-Negative	Haematocrit. 1-(20% 2-20%+	Height (Cms)	Weight (Kgs)	History of fever in past 2 weeks 1-Yes	N 3 w