THE EPIDEMIOLOGY

OF

MATERNAL AND NEONATAL HEALTH

IN

PAPUA NEW GUINEA

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ABSTRACT

In 1986 a Rapid Rural Appraisal (RRA) conducted in the Wosera subdistrict of Papua New Guinea identified questions related to neonatal and maternal health that required applied research. This thesis describes the epidemiological health project that followed, whose three objectives were: to determine the size, gestation and body proportions of Wosera newborns; to describe maternal factors that might be important determinants of low birth weight; and to review findings in relation to district and national planning.

A population based cross-sectional study of 1008 women was conducted. Women who were pregnant were followed up at antenatal clinics in a longitudinal study of pregnancy. Within a few days of delivery, these mothers and their newborns were visited at home.

The results show maternal nutrition was poor, and was made worse by childbearing. Maternal malaria, anaemia and iron deficiency were common. Perinatal mortality was high and mean birthweight was lower than seen in many developing countries; however, the proportion preterm or acutely growth retarded newborns was similar to than seen in other population based studies. Wosera infant size may be a consequence of maternal stature, malaria, smoking and nutrition. Maternal compliance to weekly chloroquine prophylaxis through antenatal clinics was irregular; on the other hand, umbilical cord care packs were better received by the mothers, and reduced neonatal morbidity.

In the Wosera, focused antenatal care will have a limited impact on neonatal mortality in the absence of socio-economic development. Women should be encouraged to stop smoking in pregnancy. There is potential for reducing perinatal mortality in village births by emphasising umbilical cord care and providing mothers with cord care packs. Nationally, there is a need to simplify the national antenatal standard drug protocol; and further research is needed on the standard management of anaemia in pregnant women.

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GLOSSARY

APH	antepartum haemorrage
BBA	born before arrival
BMI	body mass index
CI	confidence interval
DDT	Dichloro-diphenyl-tri-chloro-ethane
DOIC	district officer in charge
ELISA	enzyme linked immunosorbent assay
FEP	free erythrocyte protoporphyrin
HB	haemoglobin value
IM	intramuscular
IUGR	intra-uterine growth retardation
KM	kilometre
LBW	low birthweight
LMP	last menstrual period
LSCS	Low segment caesarian section
MCH	maternal child health
MDH	Maprik District Hospital
MUAC	mid upper arm circumference
PNG	Papua New Guinea
PNGIMR	Papua New Guinea Institute of Medical Research
PPH	postpartum haemorrage
PCV	packed cell volume
RRA	rapid rural appraisal
SE	standard error
SGA	small for gestational age
STD	standard deviation
TFR	total fertility rate
WHO	World Health Organization

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THE WOSERA, AND THE MATERNAL AND NEONATAL HEALTH PROJECT

1 PAPUA NEW GUINEA

Papua New Guinea lies in the tropics between the equator and 12 degrees south, with a land area of 463,840 square kilometres (km). For many years the indigenous Melanesian people lived in many thousands of small autonomous communities, separated by fast flowing rivers and mountainous terrain. The Melanesians first had contact with European traders, plantation labour recruiters and missionaries. The British, Dutch, and Germans controlled parts of the country at various times. Eventually colonial administration was passed to the Australians. Until the 1960's, there was virtually no economic or political development. There were democratic elections in 1972, and full political independence in 1975.

The country has large mineral resources, particularly gold and copper, and exports *copra,* cocoa and coffee. In 1985, the population was estimated at 3.3 million, mostly living in rural areas.

2 THE WOSERA [1]

2.1 History

The Wosera people are an ethno-linguistic group living south west of Maprik town in the East Sepik Province (figures 1-1 and 1-2). They occupy some 800 square kilometres in the North and South Wosera census divisions, and are thought to have migrated into the area from the Sepik river.

The Australian administration established a permanent base in the area in 1937, and the first mission station was a Catholic base at Kunjingini set up in 1938. In 1942, many Wosera were killed during the Japanese occupation; after heavy bombing and fighting in occupied villages, the Japanese were defeated in 1945.

After the war return of administrative control was slow and missionaries did not come back to 1948. There were clashes between mission and traditional culture. One priest burnt down the *haus tambarans* (traditional religious houses). Protestant missionaries arrived. Government development remained slow.

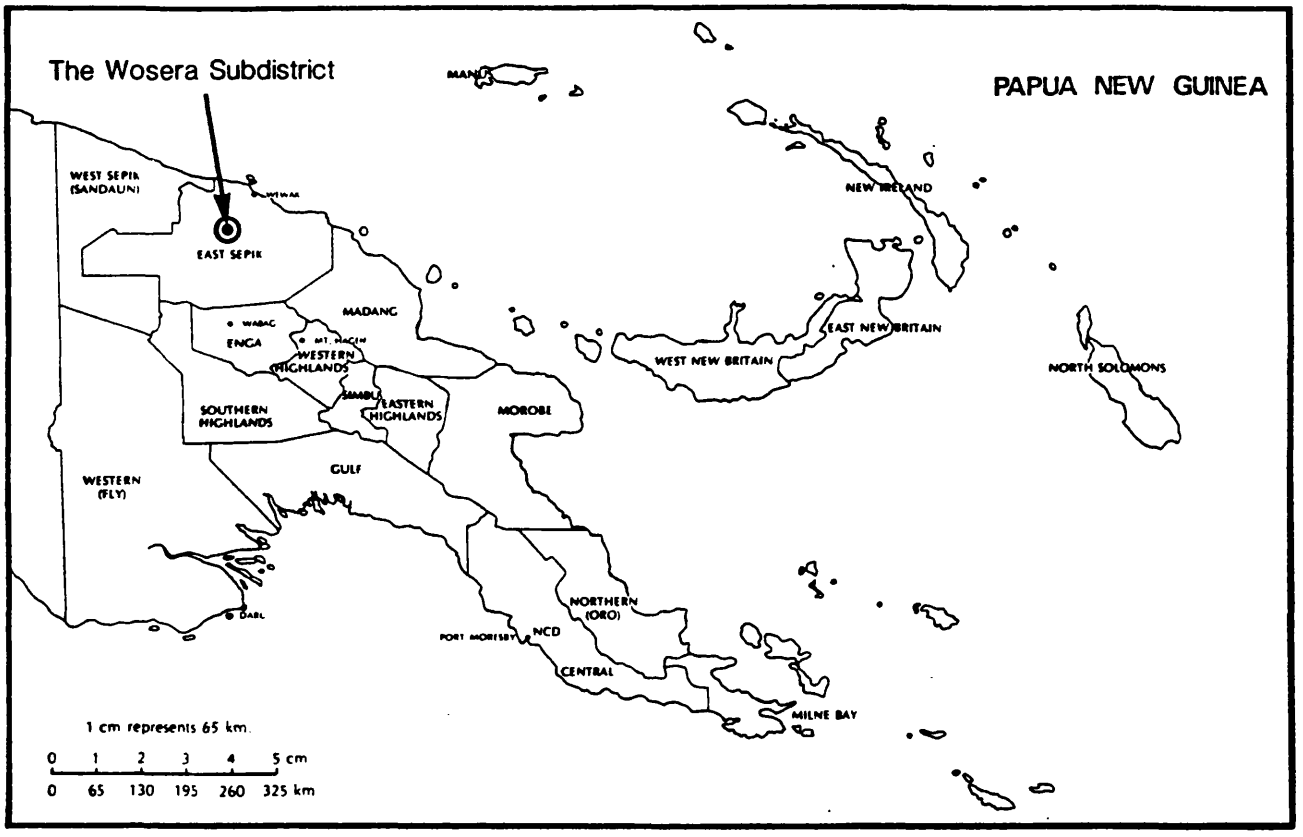
Rice and peanuts were introduced as cash crops and were adopted with enthusiasm, but failed. Government reports of land disputes and land scarcity added to the impression of chronic food shortage, malnutrition, high population growth rates and an ailing agricultural system. The solution as viewed by the administration was to resettle part of the Wosera population elsewhere in the country. This led to a few families moving to the Gwanga area to the west of the North Wosera, and a larger proportion moving to oil palm resettlement

[1] adapted from the report of a rapid rural appraisal of the Wosera, carried out in 1986. The author of this thesis was a member of the appraisal team (Heywood et al. 1986).

schemes in West New Britain Province.

In summary, contact with the outside world for the Wosera people has been traumatic and difficult.

Figure 1-1. Map of Papua New Guinea, showing the position of the Wosera



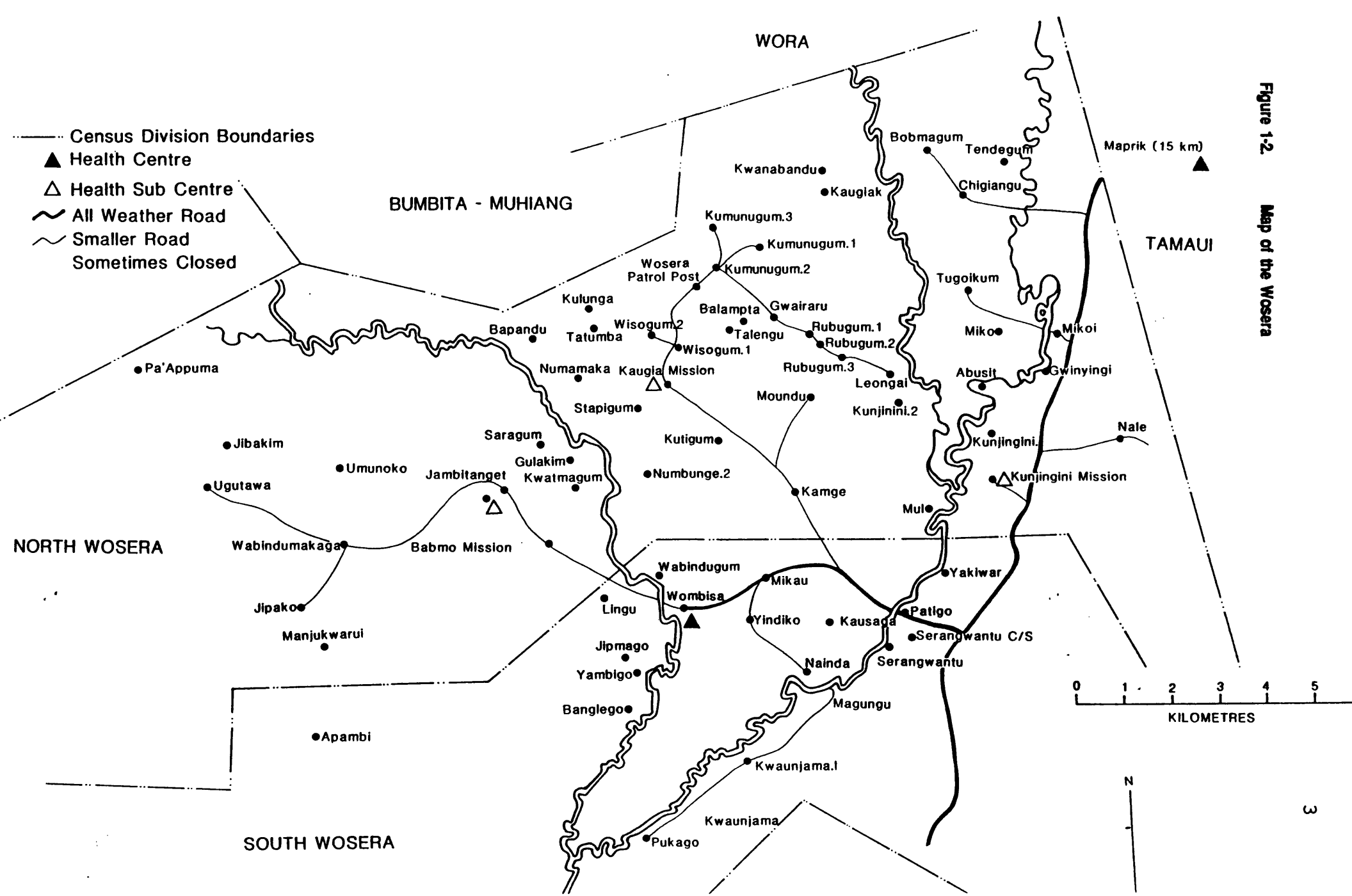


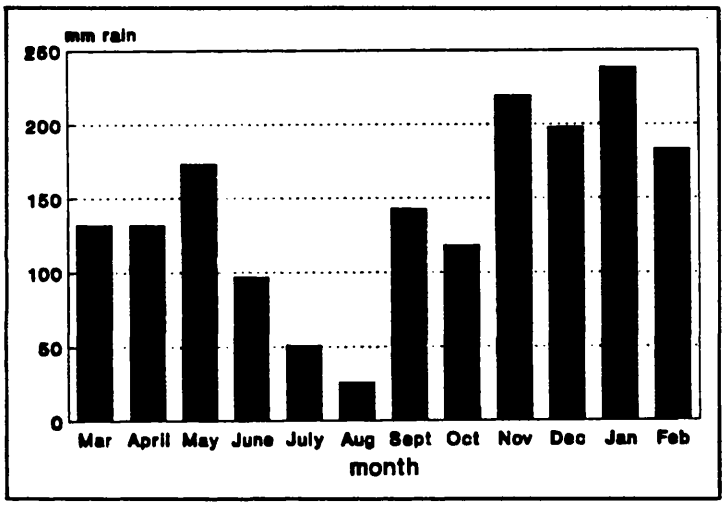
Figure 1-2. Map of the Wosera

2.2 Geography

Physical: The physical features fall into three main categories: low hills, relict alluvial plains and recent riverine plains. The relict alluvial plains lie east of the Amogu River (figure 1-2), and are sparsely populated. The more recent alluvial plains are west of the Amogu, and flood intermittently. Most of the Wosera is low lying hills.

Rainfall: Data is sparse, but annual rainfall is likely to be 1500-2000 mm per annum, with 40% falling in the six months April-September. Rainfall data for 12 months (collected while field work for this thesis was being carried out) is shown in figure 1-3. The total rainfall was 1710 mm, with 36% falling April to September.

Figure 1-3. **Monthly rainfall at Kunjingini: March 1987 - March 1988**



Soil: Soils of the **low lying hills** are well drained and moderately fertile, with considerable local variation in soil type. The soils of the **relict alluvial plains** are acidic clay or clay loams. They are poorly drained, and of low fertility and phosphorus content. The soils of the **alluvial plains** are usually well drained and moderately fertile.

Vegetation: The natural vegetation in the area is lowland hill forest, but in most areas this has been disturbed by human activity and gardens. The main vegetation is re-growth following cultivation. Sago palm forests vary from pure sago forests in permanently flooded areas to mixtures with other trees in seasonally flooded areas.

Population: The 1979 resident population was estimated in the 1979 census of North and South Wosera to be 21,588. This gives a mean population density of 27 people per square km. In the densely populated central area near Kaugia station, this rises to over 150 persons per square km. The resident population has been growing slowly between the 1962 and 1979 census at the rate of 1.3% per annum. This is in contrast to the predictions made in the mid-sixties and is lower than the 2.3% in the country as a whole.

Extensive migration is the likely cause of this low rate of increase. Ross (1984), compared

male:female ratios by age from census data by age. Although the overall ratio in the Wosera was 1, in people aged 20-29 years the ratio was 0.82 indicating an out-migration of young men. If the male/female ratio at birth is the same as the rest of the country (1.10), then some 25% of men aged 20-29 years have left the area. This assumes there is no accompanying female loss due to migration.

2.3 Economy

Poverty in the Wosera is marked. Total income was estimated in 1986 to be K 30 [2] per head per annum, of which cash crops contribute K 14.8. The average cash income for the East Sepik from cash crops is K 28 a head, which is still low in comparison to the national average of K 53 a head.

The major source of income is small-holder *robusta* coffee, accounting for an estimated 45% of sub-district income in 1985. World market coffee prices therefore have a marked affect on household income in the Wosera.

There has been widespread cocoa planting in the Wosera and coffee plots have been neglected as coffee prices fell. There is little supervision and advice from the Government Department of Primary Industry or the commercial cocoa buyers. Villagers expectations of the returns from cocoa may be overly optimistic.

2.4 Agriculture

The Wosera agricultural system is comprised of five main components: **horticultural production** of annual crops of yams ("mami", *D. esculenta*), bananas and *colocasia* taro; **sago production**; **perennial tree crops,** which include coconut, greens (" tu-lip", *Gnetum gnemon*), and breadfruit; **cash crops** of peanuts, tobacco, coffee and cocoa; and **pig husbandry.**

2.5 National political structure

In the mid 1960's criticism by a United Nations mission speeded up the preparation for independence. At this time, decentralisation emerged as an important political issue. In 1977, 3 years after Independence, political power was devolved to 20 provincial governments, including health service administration. Although concurrent power exists with the national government in health, agriculture, and secondary education, in practice this confers little authority of national over provincial departments.

Locally, elected village councillors meet at subdistrict level. North and South Wosera census divisions have separate councils, each with a small budget. Prior to 1986, a representative from each council sat on a district management committee in Maprik, with government

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1K (1 kina) is worth about £0.80

administrative officers. The potential for local action was curbed in 1988 by legislation which appeared to suspend local councils entirely.

Wosera elected representatives are national parliamentary members (1), elected provincial parliamentary members (2), and local government councillors.

2.6 Local administration

Based in the Wosera are public servants from the divisions of agriculture, district services, local government, health and education. Others are based in Maprik (officers for lands, commerce, forests, works and community development).

Up to 1986, public servants at the district level were responsible to their departmental heads in the Provincial Capital at Wewak through the district officer in charge (DOIC) at Maprik. The DOIC was responsible for co-ordinating departments at district level. In 1986, the Department of the East Sepik was restructured in an attempt to give greater control of spending within divisions. Now district public servants within divisions are directly responsible to the departmental heads in Wewak.

In the Wosera, government services to the community were generally poor. Supervision of staff at the district and subdistrict level was lacking. There was little communication between government officers, and between them and the villagers. Many public servants were acting on positions several levels above their substantive positions. Disciplinary procedures were slow, and were the responsibility of the departmental heads and the provincial secretary in Wewak.

2.7 Health

Components of the rural health care system in PNG are:

Aidposts: Staffed by health workers with a basic training who provide outpatient curative care;

Health subcentres: Staffed by nurses and nurse aides, they provide outpatient services, maternal and child health clinics, and sometimes obstetric and inpatient care;

Health centres: Staffed by a paramedical officer (health extension officer), these facilities provide services similar to a health subcentre, but generally have a greater patient capacity. They administer and supervise all health services in the area.

50% of rural health services are run by missions, but funded by the government (Department of Health 1987).

Wosera services: One government health centre at Wombisa was in operation during 1987 without nurses and usually with no Health Extension Officer. Obstetric and family planning services were absent. The quality of inpatient care was poor (Heywood et al. 1986).

Three of the 4 mission health subcentres in the Wosera were open: Kaugia and Kunjingini (Catholic Mission), and Nungiwia (South Seas Evangelical Mission). The Assembly of God centre at Jambitanget had no nurse and had been downgraded to an aidpost.

There were six government aidposts but only 4 staffed.

The local **referral centre** was Maprik District Hospital (MDH), a 110 bedded government unit covering 110,000 people living in surrounding districts. The doctor at MDH administered to Wosera government services, and also supervised mission health work.

Staffing ratios for outpatient units/population in the Wosera showed too few nurses conducted MCH work, but otherwise were consistent with levels elsewhere in the country (Heywood et al. 1986).

Some innovative health schemes designed to improve health and nutrition through village aides and women's groups had been introduced in the Wosera from 1982. Many of these schemes have not been sustained.

3 THE WOSERA RAPID RURAL APPRAISAL

Population density, poverty, land shortage and an agriculture system under stress have been repeatedly commented on by staff working in the Wosera since the 1960's (Forge 1963; Oxer 1965; Lea 1973; Ross 1984).

The National Nutrition Survey of 1983 showed malnutrition in under 5 year old children in the Maprik district (which includes the Wosera) to be amongst the highest in the country (Heywood et al. 1988).

In view of the persisting economic, agricultural, nutrition and health problems in the area, and the absence of any co-ordinated solution, the Papua New Guinea Institute of Medical Research (PNGIMR) collaborated with Provincial Government in 1986 to conduct a Rapid Rural Appraisal (RRA) in the Wosera. The aims of the multidisciplinary team were to work together to identify:

1. specific interventions which could be implemented without collection of further information;

2. further information which was needed for planning and implementation of projects, and which could be collected in a period of 12-18 months.

Recommendations for immediate intervention included road improvement, evaluation of the widespread cocoa planting, and rehabilitation of smallholder coffee. Health and nutrition recommendations related to urgently required staff and service improvements. Details are contained in the full report of the RRA (Heywood et al. 1986).

Health findings: In summary, infant mortality appeared high, and a large proportion of deaths seemed to be occurring in the neonatal period. The rapid appraisal team had access to information from a recently conducted study that indicated birthweight was low (mean 2.6 kg) and that as many as 73% of infants were preterm (Winkvist 1988). Maternal malnutrition, malaria and anaemia were also recognised problems in the Wosera.

The RRA team suggested further work to examine "the reasons for the high proportion of low birthweight infants in the Wosera with a view to designing specific interventions". Table 1-1 summarises some of the information known about the Wosera in relation to the high infant mortality rate (left side) and outlines some of the questions that still remained (right side).

The PNGIMR decided to set up a small field station in the Wosera for a year (1987), funded by the PNGIMR malaria and nutrition programmes, to carry out health research work arising from the RRA. This thesis describes the maternal and neonatal health study.

Table 1-1. Wosera maternal and newborn health: the level of understanding by the end of the RRA 1986

KNOWN	UNKNOWN
high infant mortality	whether neonatal deaths were more common than post-neonatal
birthweight low	aetiology of low birthweight: preterm, or growth retarded
gestation possibly short (>30% preterm)	accuracy of this preterm rate
maternal malnutrition present	extent of maternal malnutrition and its effect on birthweight
endemic malaria, including chloroquine resistant *Plasmodium falciparum*	effect of malaria on birthweight, and in inducing preterm labour
widespread poverty	whether improving health services would have a beneficial effect if the main constraint on health was poverty
home deliveries usual	influence of hygiene and traditional birth practices on perinatal mortality
	role of health services in improving perinatal mortality
	level of maternal mortality

4 THE WOSERA MATERNAL AND NEONATAL HEALTH PROJECT

4.1 Objectives

The primary objectives of the maternal and neonatal health project were to:

i **Determine the size, gestation and body proportions of Wosera newborn infants.**

ii **Describe and quantify the maternal factors that might be important determinants of low birth weight or high perinatal mortality: such as maternal fertility, education, nutritional status and level of malarial infection; work and illness at the time of delivery; delivery events; and health service obstetric utilisation patterns.**

iii **Review the findings of the study in relation to planning and policy at district and national levels.**

iv **Review the findings of the study in relation to knowledge that was available from previous studies in more developed and less developed countries.**

4.2 Project justification

i **The high preterm rate of village deliveries needed confirming.**

Up to 73% of the 50 consecutive village births seen by Winkvist (1988) in the Wosera were classified as preterm by gestational age assessment. This high rate, if confirmed, could account for the apparent high perinatal mortality.

ii **The extent of maternal malnutrition, malaria and other factors related to birthweight required investigation to determine priorities for maternal health programmes.**

Some international maternal child health authorities were recommending focused interventions directed toward reducing the risk factors associated with low birthweight to reduce perinatal mortality (Puffer and Serrano 1987; Lechtig 1988). In the Wosera, the level of various factors (such as maternal malnutrition, malaria, anaemia and smoking) likely to influence infant birthweight were unknown. Whether and how health staff should divert their attention to focused antenatal interventions required research.

iii **The feasibility of implementing the nation-wide standard antenatal drug protocol was unknown.**

Standard management of pregnant women living in malarious areas included a standard weekly prophylaxis schedule (PNG Society of Obstetrics and Gynaecology

1986). Anecdotal evidence suggested compliance with the regimen in many areas of the country was poor. Before embarking on strategies to improve drug compliance, the practical problems implementing the regimen needed examining in a group of people most likely to benefit from it.

iv **The apparently high neonatal death rate needed further examination before specific health interventions could be recommended.**

If a large proportion of infant deaths were occurring in the neonatal period, this might implicate low maternal tetanus immunization coverage, inappropriate village childbirth practices, poor hygiene in the perinatal period, or high newborn preterm rates. Potential interventions included: increasing tetanus toxoid vaccination coverage of pregnant women; encouraging supervised deliveries at the health centres (which had implications for nursing time); training and encouraging traditional birth attendants; health education for mothers about hygiene at delivery; and improving hygiene at birth through water supply projects.

v **While poverty remained the major underlying factor in determining the poor health in the population, service improvement, particularly for mothers and their newborn babies, might have some value.**

The RRA included specific recommendations relating to cash income generation. However, some improvements could be obtained through preventive health services in relation to maternal and newborn health.

4.3 Rationale

i **To work through health services where possible, and to include a health service support component.**

The Wosera health services that were operational were working under trying circumstances with minimal supervision. Kaugia subcentre showed signs of impending service collapse. The research team worked through health services where possible, and time was spent in direct health worker supervision and support, without becoming primary providers of health care.

ii **To feedback findings from the study into the local, provincial and national health care systems.**

The study was relevant to health care delivery for Wosera people. Part of the responsibility of the research staff was to ensure that findings were communicated in an appropriate way to health staff at all levels.

4.4 Methods

There were 2 phases to the main project. In the first, a population based cross-sectional study of women of childbearing age was conducted in a sample of Wosera villages. Women classed as pregnant by the cross-sectional study were recruited into antenatal clinics, which were run jointly between health subcentre and PNGIMR staff: this constituted the longitudinal study of pregnancy. When the pregnant women from the longitudinal study delivered, PNGIMR staff visited them in their village to collect details of the delivery, and examine both mother and baby: this constituted the postnatal study. Any woman that had been seen at the population based cross-sectional survey and subsequently became pregnant was also included in the study.

In the second phase, nursing staff at Kunjingini health subcentre re-organised antenatal clinics in villages that had not been covered by the cross-sectional survey. The PNGIMR supervised and sometimes helped with the initial recruitment and follow-up antenatal clinics. These data contributed to the longitudinal study of pregnancy.

Outcome of pregnancy in the women recruited in the second phase of the study was determined through MCH clinic reports combined with village visits. The same follow-up procedure was employed for pregnant women from the villages seen at the cross-sectional study who had not delivered when the village postnatal study finished in December 1987. In summary, the following studies were carried out:

A cross-sectional study of women of childbearing age to estimate perinatal mortality retrospectively; to determine maternal fertility; to measure maternal malarial and nutritional status; and to provide a population based cohort of pregnant women.

A longitudinal study of pregnant women recruited through antenatal clinics to examine the use and compliance of a prophylactic drug regimen and to measure weight gain in pregnancy.

A postnatal study of pregnant women derived from the population based cohort to determine the characteristics of newborn infants.

An intervention study to improve umbilical cord care in village deliveries.

A retrospective study of the use made by women of obstetric services.

Data analysis of local hospital births.

Table 1-2 summarises the thesis structure.

Table 1-2. Thesis chapter structure

Chapter 1	Introduction to the Wosera and the maternal and neonatal health study.
Chapter 2	Literature review of birthweight and perinatal mortality: the causes of low birthweight, maternal determinants and associations, with particular reference to birthweight in developing countries.
Chapter 3	The Wosera, and literature review of maternal and neonatal health in PNG.
Chapter 4	Population based cross-sectional survey.
Chapter 5	Longitudinal study of pregnancy.
Chapter 6	Postnatal study.
Chapter 7	Umbilical cord care study.
Chapter 8	Obstetric health service user study.
Chapter 9	Maprik hospital birth data analysis.
chapter 10	Study results summary; discussion; recommendations for local health managers; implications for national health policy; suggestions for further research.

4.5 Ethical considerations

4.5.1 Expectations of the community and country

Health research in developing countries can be criticised on ethical grounds if they make no contribution to health care of the people studied. Frequently studies do not fulfil villagers expectations of improved health care (sometimes mooted by researchers to build up study compliance) and do not train local health staff in research (Poon 1985). These issues were considered from the outset of the Wosera project:

The study should make some long term contribution to improved health care in the area or in the country: The basis of the study was how the health and survival of newborn babies could be improved through health service intervention.

Frequently well intentioned research studies fail to contribute anything to the country because researchers do not communicate or attempt to influence health policy. In this study, relevant initial findings were used as a basis for in-service for nurses and voluntary village health workers in the Maprik area; preliminary results were presented to doctors and health staff throughout the country (Garner and Heywood 1987); and findings from the umbilical cord study have already been used to change the standard management protocol of the newborn in PNG (PNG Society of Paediatrics 1988: 8-10). Provincial and national staff have already received a preliminary report of the work.

The study should not generate expectations in the local people for improved health care that are not fulfilled: The RRA and the presence of the research team did improve health care in the Wosera area, but by indirectly influencing existing health services. The

Wosera people understood that the PNGIMR team could not take the place of health services.

Nevertheless, the doctor on the team frequently participated in health care of particularly sick patients attending the subcentres. The study vehicles were used as ambulances for genuine emergency referrals to MDH, and this took precedence over research work.

The study should assist the host country in developing its research capability: Two health extension officers undertook their community health training by participating in the project; and a masters student from the University of Papua New Guinea was a member of the survey team before starting on his own project in the Wosera.

4.5.2 Consent

Informed consent from participants in a scientific study is different in developing countries. Decision making in a village is often made as a group, and men are frequently dominant. Individual consent may not relevant in this context.

Before starting work in any of the villages, there was a preliminary meeting with the village leaders to explain the study, why it was being done it and how it would affect them. All wished the work to go ahead. Leaders then called a second meeting with the rest of the village to explain the work. It was indicated at this time that participation in the work was optional.

Non-participation in the study would not affect provision of emergency care by the research team through the existing health services.

4.5.3 Diseases discovered during field work

Clinical examination and basic haematological tests of women or babies sometimes revealed conditions that required medical treatment. In these circumstances, mothers were advised carefully, given referral letters and encouraged to attend the appropriate facility for treatment. Transport was provided for anyone needing inpatient care.

Anaemia detected during surveys was managed using the Standard Treatment Manual for Adults (Physicians Society of PNG 1984). Asymptomatic malarial parasitaemia was not acted on when found as malaria was endemic.

4.5.4 Control and treatment groups

Malaria drug prophylaxis is generally recommended for pregnant women living in malarious areas, and is part of the standard antenatal management in PNG. It was therefore judged unethical not to offer chloroquine prophylaxis to women attending antenatal clinics.

4.5.5 Anonymity

Sensitive information was collected at interview when women gave opinions of treatment received in Maprik District Hospital. Interviewees understood that this information may be helpful in improving the service, and that a summary of the information (without their names or village) may be used in a report to the Provincial Government.

4.5.6 Access to Information

Study data may be relevant should an individual subsequently fall ill, but is often inaccessible to health service providers. In addition, subjects frequently complain they are not told of the results of blood tests taken during research. To overcome these 2 problems, women seen during the cross-sectional study were given a card which recorded the date, their name, age, ID number and their haemoglobin value. This they kept so that the team could readily access information should it be needed by local health services. Cross-sectional, antenatal and postnatal data were treated like hospital notes, and used in writing letters should the women need referral. Antenatal summary history cards were supplied to all the clinics.

When blood was taken, subjects watched the initial field processing and were shown the digital read-out on the field haemoglobin instrument, and reassured where appropriate. The results of urine tests for 4-aminoquinoline metabolites were discussed with mothers at subsequent clinics.

Newborn babies were supplied with a baby book where details of the newborn examination and the mothers health at the postnatal visit were recorded.

chapter 2

BIRTHWEIGHT AND PERINATAL MORTALITY IN DEVELOPING COUNTRIES

- a literature review -

1 INTRODUCTION

Why is birthweight is routinely recorded in health facilities throughout the world? Birthweight is predictive of mortality risk: perinatal mortality, for example, is more common in babies falling into lower birthweight groups (Karn and Penrose 1951). However, it is often forgotten that perinatal mortality is also high in very large babies (Bromwich 1986), but this risk affects a smaller number of infants born.

Comparisons between countries have shown an association between the proportion of babies born that are low birthweight (LBW: defined as a birth of less than 2500 g) and infant mortality: a high incidence of LBW within a population is associated with a high infant mortality (Puffer and Serrano 1987). The connection between birthweight and mortality has led health planners to recommend antenatal interventions designed specifically to alter maternal risk factors for LBW in order to reduce perinatal mortality (WHO 1978b; Ashworth and Feachem 1985; WHO 1984). Some highly focused interventions include food supplementation for pregnant women, which may be on a large scale. This is happening in an aid programme in the slums of Calcutta, for example (Calcutta Metropolitan Development Authority 1988). However, it is unknown whether improving birthweight alone will affect mortality at all (Rush 1987).

In communities where perinatal mortality is high, mothers are naturally fearful that their babies may die. Few mothers are concerned about the birthweight of their infants - in fact, many mothers fear large babies because of the risk of obstruction in labour (Townsend 1986). There appears to be no appeal, therefore, for mothers to participate in a health intervention that aims to increase birthweight. It therefore becomes particularly important to ensure that interventions that focus on increasing birthweight do actually improve survival of newborns before they are recommended and implemented. This requires an understanding of what birthweight actually measures, and its relationship to perinatal mortality. This chapter examines these questions.

2 BIRTHWEIGHT DISTRIBUTION IN POPULATIONS

The distribution of birthweights within a population is a mixture of primary, normal distribution, and a secondary distribution located in the lower tail (Goldstein 1981). This distribution causes difficulties in summarising population birthweight data statistically. Thresholds are commonly used (the proportion less than 2.5 kg, for example); however, thresholds are inefficient in their use of birthweight data, and do not represent the shape of the distribution.

Wilcox and Russell (1983) suggest the birthweight distribution of a population can be described by the mean of the main distribution, its standard deviation, and the proportion of residual births in the tail. The difficulty with this model is how to truncate the predominant

distribution in order to determine the residual births. The authors use a maximum likelihood estimate [1], and apply the procedure to three large British data sets. The residual proportions were found to be between two and five percent.

The residual proportion includes the smallest infants, who have the highest mortality risk. Wilcox and Russell illustrate the importance of the residual proportion in determining mortality rates (using data from Karn and Penrose 1951). They show that although females are more likely to weigh less than 2.5 kg because the mean of the main Gaussian distribution is lower, they have a smaller residual proportion when compared with male infants, and consequently lower fetal and neonatal mortality rates. Interestingly, the preterm rate in males is slightly higher than in the females. If only term infants are included in the analysis, then the distribution follows the primary distribution closely, confirming that the residual proportion contains most of the small, preterm infants.

This subset of infants outside the Gaussian distribution are therefore a very important group in determining mortality rates, and they are not described adequately by mean birthweight values of the population as a whole.

This description of birthweight provides a framework when looking at birthweight determinants. Many biological variables may affect birthweight, and the level or extent of these variables in a population of women will affect the primary Gaussian distribution. However, there may be other factors that primarily influence the size of the residual proportion: these factors are likely to have a powerful influence on perinatal mortality.

3 NEWBORN CHARACTERISTICS AFFECTING BIRTHWEIGHT

Two neonatal problems may influence both birthweight and neonatal mortality risk: preterm delivery and acute growth retardation. Neonates falling into these two groups seem to account for the residual proportion of the birthweight distribution described in 2.

Birthweight of **abnormal babies** is often lower than the population mean. The mortality risk of this group is higher than normal babies. Abnormalities may be chromosomal, congenital or the result of a congenital infection, and are usually obvious at birth.

3.1 Preterm delivery

Infants that are delivered before 37 completed weeks (259 days) of pregnancy [2] are classified as preterm births. They tend to be lighter than term births, although a baby can be preterm and still fall anywhere within the population birthweight distribution.

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[1] The authors truncate successively below the birthweight mean. Each lower point yields a better fit to the predominant distribution until the truncation point falls within the residual portion, when the goodness of fit begins to deteriorate.

[2] By convention, the normal length of pregnancy is 280 days (40 weeks) from the first day of the last menstrual period (LLewellyn-Jones 1986).

Diagnosis: Gestation is usually calculated from the date the last menstrual period (LMP) in the mother began. Ultrasonic examination of the fetus at 12-16 weeks gestation allows estimation of the bi-parietal head diameter, which can be used as to accurately assess gestational age.

The Dubowitz neonatal examination at birth is also accurate in determining newborn gestation. Infants are examined by a trained observer who scores 21 clinical criteria. The score is then read against a regression line to determine gestation. In the original studies of 167 white infants with known gestation, Dubowitz and Dubowitz (1977) showed a high correlation between gestation assessed by LMP and by Dubowitz assessment (r=0.93; 95% CI ± 2.0 weeks).

Mortality risk: Mortality risk is greater than that of normal term infants. Puffer and Serrano (1987: tables 36 and 37, using data collated from a WHO study) show that the early neonatal death rates in LBW infants from various developed countries were 5-10 times higher in the preterm group compared with term LBW babies. For babies weighing 2.5 kg - <3.0 kg, the rate was increased by 2.1 to 3.6 times in preterm babies, depending on the country.

3.2 Growth retardation

The definition and significance of growth retardation is confused in much clinical and epidemiological literature. This is partly because of the assumption that small infants must be growth retarded.

There are various definitions of growth retardation currently in use: for example, birthweight less than the fifth centile of standard weight for gestational age; birthweight less than 2.5 kg in a term infant; or birthweight less than 2 standard deviations below the population mean (Kramer 1987).

If LBW infants are categorised as preterm or term, then some will be small and term (Wilcox 1983). These babies are often described as **small for gestational age** (SGA). In view of the increased mortality seen in low birthweight groups, it is a small conceptual leap to regard SGA babies as being the result of a disease process that has caused them to be growth retarded. This thinking equates SGA with **intrauterine growth retardation** (IUGR). These terms are often used interchangeably in the literature. For example, Seeds (1984) states that "up to 40% IUGR babies may...(be) constitutionally small". However, if a fetal genome determines that the infant will be SGA, then it is incorrect to classify the infant as growth retarded.

Further confusion ensues when birthweight characteristics are compared across populations. The proportion of newborns that are LBW may be 30% in India or 3.8% in Norway (Kramer 1987). The reason often given for the high prevalence of LBW in developing countries such as India is growth retardation (Villar and Belizan 1982a). This implies unwanted pathological processes impeding fetal growth in Indian infants, and that a medical intervention is required

to relieve the retardation; and it presumes that if the forces of "growth retardation" could be removed in Indian mothers they would deliver infants of a similar size to their Norwegian counterparts. However, if this were indeed possible, and the mean birthweight of Indian infants increased from 2.5 kg to 3.5 kg, there is little evidence that perinatal mortality would be improved; indeed, it seems likely that pregnancy outcome could be adversely affected, with increased perinatal mortality as a result of cephalopelvic disproportion.

Problems with current definitions of growth retardation can be summarized as follows:

1. They ignores biological variability in birthweight (Wilcox 1986). This means biologically small infants are classified as growth retarded.

2. They are not a true measure of retarded growth, as they are based on one measurement only (Altman and Hytten 1989).

3. Normal weight infants who have suffered weight loss in late pregnancy as a result of placental insufficiency may not be classified as growth retarded.

4. Application of standards of birthweight for gestational age from one population to another with a different mean for the primary birthweight distribution may be invalid.

"Growth retardation" can be categorized into **asymmetric growth retardation**, where fetal growth slows near term; and **symmetric growth retardation**, where sub-optimal growth probably is the result of processes occurring throughout pregnancy (Altman and Hytten 1989). Some authors consider symmetric retardation to have occurred in the first trimester (Villar and Belizan 1982b). Terminology used to refer to growth retardation is profuse and summarized in table 2-1. This classification is important as it identifies asymmetrically growth retarded infants, a group that can be determined using morphological criteria, and whose mortality risk may be higher than other term infants.

Table 2-1. Terms used to describe babies whose low birthweight cannot be entirely explained by preterm delivery

TERM USED IN THIS REVIEW	symmetric growth retardn.	asymmetric growth retardn.
TERMS USED ELSEWHERE	chronic growth retardation; proportional growth retardn.; stunted; non-wasted; type 1 IUGR; miniatures	acute growth retardation; disproportional growth retardn.; malnourished; wasted; type 2 IUGR (Villar et al. 1982b);
TERMS COMMONLY USED TO COVER BOTH		small for gestational age; small for dates; light for dates; dysmature.

3.2.1 Asymmetric growth retardation

There seems little doubt that infants growing at a particular velocity which then falters because of some pathological process will be growth retarded. Altman and Hytten (1989) suggest that growth retardation be restricted to fetuses where there is evidence of intrauterine weight loss or faltering growth.

Detection of faltering in late pregnancy is possible through repeated symphysis-fundus height measurement or serial ultrasonography. Acutely growth retarded infants may show clinical evidence of the faltering at birth: they are characteristically thin, with low body fat, and at risk of hypoglycaemia.

The underlying pathology is thought to be due to reduced maternal utero-placental blood flow (Chiswick 1985). In many cases there is no apparent cause for this. Pre-eclampsia, smoking and excessive alcohol consumption are strongly associated with acute growth retardation. Famine conditions in the third trimester can cause a reduced mean birthweight of 200-400 g (Rush 1989). Very little is known about the incidence of acute growth retardation in newborns in populations where maternal malnutrition is widespread and long standing.

Diagnosis: Infant weight for length is reduced. The ponderal index is often used as a measure of "thinness" of a baby ([weight in g*100]/[length in cm]^3). A low ponderal index value indicates a thin infant. Ponderal index standards for newborns of varying gestation is given by Lubchenco et al. (1966). Miller (1971) suggests that term infants with a ponderal index of less than 2.2 could be regarded as "malnourished".

Growth of the head in acute growth retarded infants tends to be preserved. Neonatal fat, laid down late in gestation, tends to be diminished. Thus skinfolds thickness in asymmetrically growth retarded babies is reduced.

Mortality risk: This is dependent on the severity of the retardation, and the complications associated with this such as asphyxia or hypoglycaemia.

3.2.2 Symmetric growth retardation

The rate of fetal growth appears to be dependent on fetal genome; maternal health and nutrition; and a maternal growth modulating effect.

Length velocity is at a maximum 14-16 weeks of pregnancy (Hill and Milner 1988). Infants exposed to maternal factors that impair growth in the first half of pregnancy are likely to be "stunted"; subsequent growth may be at a normal rate, producing infants whose body proportions are appropriate for their size. Such infants may be regarded as growth retarded, and the body proportions will indicate a symmetric type.

Fetal genome will influence birthweight (Snow 1989), but an infant small as a result of genome should not be described as growth retarded.

The maternal modulating influence on growth is thought to be mediated at least in part by uterine constraint on fetal growth, occurring particularly in late pregnancy (Hill and Milner 1988). The latter effect is suggested by animal experiments (Snow 1989), and is probably an important factor in the small size for gestation seen in human twins (Campbell and Samphier 1988). This constraint seems to result in symmetrically small infants. Whether they should be described as "growth retarded" is debatable.

Diagnosis of symmetric growth retardation: By definition, these infants cannot have normal body proportions and their skinfolds values (reflecting body fat) are not reduced.

Given that birthweight follows a normal distribution, on theoretical grounds it is difficult to classify a low birthweight infant as symmetrically growth retarded as the distribution expects some small babies to occur by chance. Is it possible to determine whether symmetric growth retardation has occurred in an otherwise normal baby? This is a difficult question. Two criteria are given below in an attempt to answer this.

Symmetric growth retardation may have occurred in an infant if a maternal factor is detected which:

1. has been shown to be causally related to reduced weight for gestational age in other studies;

2 should fetal exposure to it have been altered, then the expected effect on birthweight would be to increase it.

For example, smoking has a clear direct effect tending to reduce birthweight (Kramer 1987). It can be surmised that if the mother had not smoked in pregnancy, the baby may have been larger. Therefore it is reasonable to deduce that growth retardation has occurred in infants born to mothers that smoke. On the other hand, fetal sex influences birthweight (males tend to be heavier than females); however, it is not possible to modify the exposure of a female fetus, so the lower weight cannot be attributed to growth retardation.

Mortality risk of symmetric growth retardation: Symmetrically small infants do not have the same clinical conditions seen in the asymmetrically growth retarded baby; it could be anticipated that their risk of mortality is less.

Symmetric growth retardation and mortality risk are confounded by factors such as socio-economic status (see 5.6). The risk of death as a result of the biological effect of birthweight *alone* is difficult to determine. Some clues are given by the work of Bakketeig et al. (1984). They show that perinatal mortality in infants that are long for their birthweight is higher than infants of the same weight category but of medium length for weight. This suggests that mortality risk is higher in asymmetrically growth retarded infants compared with the symmetrically retarded.

4 NEWBORN CHARACTERISTICS IN DEVELOPING COUNTRIES

Birthweight: There are several reviews of birthweight data from various countries (for example, WHO 1978a; Villar and Belizan 1982; WHO 1984; Puffer and Serrano 1987; Kramer 1987). The percentage of LBW infants varies from 5% (Japan) to 30% (India). Mean birthweight of populations within countries varies from 2.5 kg (India) to 3.5 kg (Norway).

Preterm babies: Using date of last menstrual period, a WHO study (1978a) found that the percentage of preterm births varied from 20% (Hungary) to less than 3% (Japan). Using national data from over 3 million people in the United States, Puffer and Serrano (1987: table 32) quote preterm rates of 17.3% in blacks and 8% in whites. In a South East Asia study (Perera and Lwin 1984), the percentage of preterm babies, using date of last menstrual period in community based surveys, was 2.5% (rural Thailand); 7.7% (rural India); and 12.5% (rural Burma). Table 2-2 summarises preterm rates from various studies (preterm rates from smaller studies in malarious areas are detailed in section 5.2).

Table 2-2. Percent preterm births (singleton) reported in various countries

COUNTRY	SOURCE	YEAR OF PUBLICATION	% PRETERM	GESTATION ESTIMATION	MALARIOUS AREA?
Hungary	WHO	1973	19.8	LMP	NO
Cuba	WHO	1973	11.2	LMP	NO
Austria	WHO	1973	10.8	LMP	NO
USA	WHO	1973	7.3	LMP	NO
Sweden	WHO	1973	5.0	LMP	NO
New Zealand	WHO	1973	4.5	LMP	NO
India, Delhi cohort	PAHO [a]	1987	8.4	LMP	NO
India, Delhi Hosp.	PAHO	1987	9.1	LMP	NO
Ivory Coast	Reinhardta	1978	23.0	Dubowitz	YES
Papua New Guinea	Winkvist	1988	30.0	Dubowitz	YES

a original studies by Ghosh et al., used by Puffer and Serrano 1987

Accurately determining gestation is a major methodological problem in many of these studies. Using the date of the last menstrual period is particularly problematic in developing countries as accurate recall of date of last menstrual period may be limited in societies where calendar dates are not in frequent use. Further, high fertility and prolonged breast feeding may mean that the resumption of regular menstruation may not occur between pregnancies.

Determination of gestation by Dubowitz examination requires sophisticated paediatric clinical skills to ensure an accurate and reliable estimation.

Incidence of growth retardation: The literature on growth retardation in developing countries has been confused because of problems defining what it is. Table 2-3 summarises 6 studies that have measured several anthropometric variables in newborns. In all cases, the anthropometry values are remarkably similar.

Table 2-3. Studies measuring neonatal anthropometry including birth length

COUNTRY	source	year	N	BIRTH WEIGHT	LENGTH	HEAD CIRCUM	PONDERAL INDEX
USA	Lubchenco	1963/66	4700	3.1	48.6	33.8	2.6
USA	Miller	1971	1692	---	50	34	2.5
India	Ghosh	1971	5031	2.9	49	33.5	2.5
Lima	Frisancho	1977	4787	3.3	49.6	34.2	2.7
Woods	S. Africa	1984	1957	3.0	48.5	34.1	2.6
Adair	Philippines	1988	1971	3.0	49.2	----	2.5

* the average value of the fiftieth centile in infants 37-40 weeks gestation
* primigravida only
(Birthweight in kg; length and head circumference in cm; ponderal index in g*100/cm³)

5 MATERNAL DETERMINANTS OF BIRTHWEIGHT

The main problem with identifying birthweight determinants is separating their affect from confounding variables. For example, height, socio-economic status and a woman's age at birth of her first child may all appear to correlate with birthweight: but is it this factor that is having an affect on birthweight, or are they just measures of something else that is actually directly influencing birthweight, for example, nutritional status? Kramer (1987), in an exhaustive review of 921 papers, examines the effect of 43 potential determinants of birthweight, estimating the magnitude of the effect where appropriate using meta-analysis.

Generally, studies examining birthweight determinants use regression analysis explore the effect of factors on mean cell values for birthweight. It is important to remember that while these models may explain much of the variation in mean birthweight between cells, they are of little help in predicting individual birthweight (Chapman and Fryer 1980).

In this chapter, factors that could possibly be important in the study area are reviewed briefly, particularly in relation to potential interventions.

5.1 Nutritional factors

Maternal height: A woman's height is determined partly by her genome and partly by her nutrition during childhood. In the Wosera, poor childhood nutrition is widespread (chapter 3), and likely to have an important influence on final adult height. In addition, women in PNG may continue growing in their 20's, so in younger women age will affect maternal height.

Maternal height and birthweight may be confounded by socio-economic factors. Short stature from poor childhood nutrition may be related to poverty. During pregnancy, these same socio-economic factors could affect maternal diet, and thus affect the birthweight of her infant. In an analysis of the British Perinatal Survey Goldstein (1981) showed that taking maternal height into account virtually eliminated the apparent effects of maternal age and social class on birthweight.

Kramer reviews 79 relevant publications, and concludes that maternal height does affect birthweight after socio-economic status had been controlled for; and that in populations with a high prevalence of short women, stature is important in determining the proportion of low birthweight infants.

In this thesis maternal height is important because it may:

1. affect birthweight;
2. reflect childhood nutrition;
3. act as surrogate measure for socio-economic status;
4. be a factor to control for when reporting maternal weight. This can be done by using weight for height standards, or calculating body mass index (weight/height$^{2)}$.

Pre-pregnancy weight: The influence of pre-pregnancy weight on birthweight is potentially confounded by maternal height (taller women are likely to be heavier); smoking (cigarettes may act as an appetite suppressant); genetic factors (affecting height); and socio-economic status.

Kramer's analysis of 74 studies which controlled for confounding factors suggests that preterm births are more common in underweight women, based on studies from Western countries. This has important implications as a higher preterm rate may have a marked effect on mortality (section 3.1). Pre-pregnancy weight also seems to affect birthweight after gestation has been controlled for.

Weight gain in pregnancy reflects maternal fluid retention, fat deposition, breast enlargement, in addition to placental, uterine and fetal growth. Weight gain and birthweight may be confounded by gestation: weight gain will be less when infants are born preterm. Weight gain also represents fetal growth, and may be expected to correlate with birthweight. Kramer's meta-analysis suggests that, controlling for gestation, weight gain does appear to have a positive relationship with birthweight. The effect is greater if the initial maternal weight is low.

However, in developing countries fetal growth alone may account for almost 50% of the weight gain observed (for example, if the gain is 6 kg, and birthweight 3 kg, the baby accounts for 50% of the gain). This suggests weight gain, especially in populations where overall gain is low, may be more of a reflection rather than a determinant of birthweight.

Maternal work in pregnancy: The potential effect of work on birthweight may be important because of the heavy workload of many women in rural areas of developing countries. Work

may reduce birthweight by shortening gestation; by reducing calories available for fetal growth; or by reducing blood flow to the uterus, through exertion or posture. Studies from developed countries give conflicting results (Kramer 1987). Developing country studies on the effect of seasonal variation in work on birthweight are confounded by the seasonal variation in food availability. In the Wosera, maternal nutrition seems poor (Ross 1984) and maternal work appears high: separating the effect of work, socio-economic status and food intake on birthweight is likely to be difficult.

Anaemia: Severe or moderate anaemia could theoretically impair oxygen supplied to the fetus during pregnancy, thus reducing growth, or causing fetal stress especially in late pregnancy.

Haemoglobin and iron status changes in pregnancy previously regarded as abnormal may well be physiological (Mahomed and Hytten 1989). Fluid retention will lower haemoglobin levels by dilution: a fall in haemoglobin is therefore expected. In fact, a high haemoglobin may be associated with poor weight gain or pre-eclampsia and an adverse pregnancy outcome. On the other hand, a large baby or twin pregnancy is associated with a bigger increase in plasma volume and a greater decline in haemoglobin values.

Studies looking at haemoglobin in relation to birthweight need to measure haemoglobin early in pregnancy to minimise confounding by fluid retention. Birthweight and iron status may also be confounded by maternal nutritional status (Kramer 1987).

The influence of haemoglobin and iron status on birthweight becomes more complicated in endemic malarious areas. Malarial infection may cause anaemia, and iron deficiency is frequently associated with the anaemia of chronic malaria (McGregor 1982). Control of malaria in endemic areas may cause a rise in haemoglobin (Schofield et al. 1964), and chloroquine prophylaxis has been shown to be more effective than iron in preventing anaemia in primigravida (Fleming et al. 1986). Malaria control also increases mean birthweight in primigravida (McGregor et al. 1983). It may be that malaria influences birthweight through its effect on haemoglobin.

Zinc: It has been suggested that fetal growth and gestational length could be affected by low maternal zinc status. Well designed randomized double blind trials of zinc supplementation have failed to show an effect of supplementation on birthweight (Hunt et al. 1984; Mahomed et al. 1989); Swanson and King (1987) suggest that the relationship between zinc status and pregnancy outcome remains an open question.

The importance of considering zinc in this study is that it is known that hair levels of zinc are low in the Wosera (Ross et al. 1986). However, zinc is related to protein intake, and low zinc levels may simply reflect poor diet, rather than an isolated micronutrient deficiency.

Low zinc levels may also have an independent effect on birthweight specific mortality. Some authors have suggested that low zinc levels in the mother may reduce the bacteriostatic effect of amniotic fluid and thus increase the risk of congenital bacterial infections (Tafari et al. 1977).

5.2 Malaria

Malaria could act on birthweight by impairing fetal growth or by reducing gestation.

The epidemiology of malaria in pregnancy is reviewed by Brabin (1983) and McGregor (1984). In summary, women appear to be more susceptible to malarial parasitaemia during pregnancy, particularly if nulliparous; the peak prevalence of infection occurs between 10-20 weeks; and antenatal malaria can result in anaemia (Gilles et al. 1969).

Malaria and preterm delivery: Asymptomatic parasitaemia is common in semi-immune individuals, and appears to relate to partial immunity to the parasite. Presumably a clinical episode of malaria is likely in a person with a parasitaemia, but the significance of the increased parasitaemia in pregnant women in relation to systemic symptoms is not clear. It is generally accepted that any systemic illness, especially those causing prolonged fever, can precipitate a woman into preterm labour. Menon (1972), for example, reports on three preterm deliveries and five intrauterine deaths in 17 women with symptomatic malaria in late pregnancy. However, the study inclusion criteria are not clearly defined, nor is the immune status of the women: malaria infection in a non-immune woman may cause severe illness that can threaten both mother and fetus, whereas malarial illness in a woman resident in a highly endemic malarious area may be attenuated by maternal malarial immunity.

McGregor et al. (1983) report on the effect of malaria on 6427 singleton birth weights in the Gambia. Mean birthweight values were higher in babies born with malarious placentae, even after stratifying for place of residence, parity of the mother and sex of the infant. The trend was consistent in almost all parity groupings but was only significant in primigravidae. There appeared to be no relationship between placental size and presence of placental malaria at parturition. The authors note the data cannot exclude the possibility that malaria causes a reduction in placental efficiency, or that it causes early delivery. The placental weight/birthweight ratio, which tends to decrease as gestation progresses, was higher in primigravidae with placental infection, consistent with shorter gestation in this group.

Reinhardt et al. (1978a) studied 204 consecutive hospital deliveries in an endemic malarious area of the Ivory Coast and clinically assessed gestational age by Dubowitz examination. Twenty-three percent of newborns were preterm, but this seems to be calculated using a preterm cut-off of less than 38 weeks, whereas the conventional definition is taken as less than completed 37 weeks (Reinhardt et al. 1978a: table 4). The preterm rate in first born babies was 38%, and the mean gestation of this group was 38 weeks. In babies of parous women the mean gestation rose to between 38.7 and 39.1 weeks: the difference in mean gestation between first born babies and subsequent births was significant. Reinhardt et al. (1978b) also examined the influence of maternal malaria (defined as either a positive placental blood smear or a venous blood sample at delivery positive for parasites) on mean gestation. They showed no significant relationship; however, *Plasmodium falciparum* indirect immunofluorescence antibody titres were elevated in the preterm group.

The Ivory Coast study is particularly important as it documents clinically assessed gestational

ages in babies born in a malarious area. The study finds the preterm rate to be much higher than reported in many other studies, and the effect is particularly pronounced in the babies of primigravidae, whose susceptibility to malaria is already known to be increased. However, the study does not clearly state who conducted the Dubowitz examination and whether the assessments were checked; and the study group were obstetric hospital admissions, who could be a biased group with obstetric conditions making preterm delivery more likely.

Malaria intervention studies and birthweight: Morley et al. (1964) conducted a controlled trial of monthly pyrimethamine chemoprophylaxis in Nigeria. Weight gain in the second half of pregnancy was greater in the intervention group (4.0 compared with 3.2 kg), and the mean birthweight in the intervention group was significantly greater than in the control group by 157 g. Stillbirths and twins were excluded, and gestation was not controlled for. Thus the improvement in birthweight could be due to a reduction in proportion of preterm births.

MacGregor J. and Avery (1974) show an increase in mean birthweight in the Solomon Islands corresponding with the introduction of effective malaria control by residual DDT (dichloro-diphenyl-trichloroethane) spraying. Mean birthweight in first born babies increased by 252 g; the overall mean rise was 165 g. The birthweight rise is unlikely to be the result of overall improvements in health and nutrition of the people as they occurred rapidly and were closely associated with the spraying programme. Gestation was not measured or controlled for in the assessment of the increase in birthweight gain; however, it would require a high preterm rate in the control period with a subsequent dramatic reduction in this as a result of the intervention to explain these weight differences entirely in terms of a reduction in the number of preterm babies. [3]

In a controlled trial of *Maloprim* (dapsone plus pyrimethamine) birthweight of babies born to primigravidae were significantly greater in women from the prophylaxis group compared with the women from the control group, and LBW infants were less common in the prophylaxis group (Greenwood et al. 1989). However, the study did not measure gestation so it is not obvious whether the effect on birthweight is related to a reduction in the number of small, preterm infants or by an effect on birthweight in all infants.

In summary, the extent of preterm delivery caused by symptomatic malaria in endemic malarious areas remains elusive. This is partly because accurate assessment of gestation is often difficult in such populations (section 4). A parallel study to the one reported in this thesis conducted in 1987 by the PNGIMR in Madang, an area where malaria is highly endemic, suggests that most first born infants were term (Bernard Brabin, personal communication). Kramer suggests further work is required to determine the effect of malaria on birthweight.

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[3] For example, if the mean weight of preterm and term babies were 2.0 kg and 3.0 kg respectively, and the pre-intervention preterm rate were 20% which was reduced to 3% with intervention, then the mean weights of babies born pre-intervention and after the introduction of spraying would be 2.80 and 2.97 kg.

5.3 Smoking

Smoking may affect fetal growth, either directly, through carbon monoxide displacing oxygen from haemoglobin and moving the oxygen dissociation curve to the left, so that less oxygen is released at a given partial pressure of oxygen; or, by the effect of nicotine reducing appetite; or by a direct effect on fetal metabolism by cyanide in smoke (Kramer 1987).

Literature on the effect of smoking on birthweight in developed countries is prolific. Kramer (1987) found 121 articles and in his summary, smoking appears to have a small effect on gestation (-0.05 weeks per cigarette smoked per day), but this effect remains questionable. However, the effect of smoking on birthweight seems indisputable. Smoking seems to have a dose response effect on birthweight, and is particularly marked in mothers who smoked in the last trimester. A recent study of 1860 women suggested that the birthweight reduction due to smoking was approximately 5%, corrected for gestation (Brooke et al. 1989), which is roughly equivalent to the estimate reached by Kramer of -11 g per cigarette per day.

Smoking and perinatal mortality: A recent population based prospective study (Cnattingius et al. 1988) showed a clear relationship between smoking and late fetal death (odds ratio 1.4), controlling for age group, parity group and whether a single or multiple delivery. This study is important because it directly examines the effect of smoking with perinatal mortality, rather than indirectly through its effect on birthweight.

5.4 Arecia nut chewing

Many lowland Papua New Guineans regularly chew the arecia nut, taken with pepper (*piper betle*) and slaked lime (calcium carbonate and calcium hydroxide). This combination, referred to as betel nut chewing, causes a reduction in blood pressure by its effect on the parasympathomimetic nervous system (Taylor 1980). This could reduce uterine blood flow and potentially influence birthweight. The influence of betel chewing on blood pressure beyond the time it is being chewed in lowland Papua New Guineans is not known, and there are almost no epidemiological studies examining betel nut consumption, maternal blood pressure and fetal growth.

5.5 Morbidity

Illness may act on birthweight by reducing maternal appetite and food intake, or by increasing her metabolism and reducing nutrients available to the baby. Acute febrile illness may reduce uterine blood flow or induce preterm labour.

Birthweight and maternal morbidity is confounded by poverty. Poor people are more likely to be ill, to be malnourished and to deliver LBW babies.

Symptomatic urinary tract infection appears to have a small effect on birthweight, but information on the prevalence of antenatal urinary tract infection in developing countries is sparse (Kramer 1987). Genital tract infection, especially by *Ureaplasma urealyticum*, seems

to be associated with preterm delivery, but again the prevalence of this and other genital infections has been little studied in developing countries. However, gynaecological infections may be more common than previously imagined (Bang et al. 1989).

5.6 Socio-economic status

Kramer examines a number of studies for an effect of socio-economic status that is independent of nutritional status. Many factors are linked to socio-economic status (for example, nutritional status of the mother, workload, and education). Therefore to demonstrate an "independent" effect of socio-economic status is of little relevance. However, it is likely that improvement of socio-economic status will be very important in reducing perinatal mortality in the long term, possibly by its effect on other intermediary birthweight or mortality determinants.

5.7 Parity

Neonatal outcome seems to follow a 'U' shaped relationship with parity, with mortality lowest in the mid-parity range and rising in high parity women. Several factors confound the relationship. Age, for example: young women may be less well nourished and more likely to be nulliparous.

Mean birthweight values tend to increase with parity up to parity 5 in the UK (Pethybridge et al. 1974), but with the most marked difference between first born babies and subsequent births. Kramer calculates an effect of 43.3 g /birth from seven studies. A parity effect was only shown in a study conducted in a developing country between first born babies, whose mean birthweight was 100 g less than that of babies born to parous women (DaVanzo et al. 1984). The effect of parity on preterm rates in developing countries is not well described.

5.8 Preceding birth interval

A short birth interval may reduce birthweight through the preceding nutritional burden of pregnancy and lactation.

Infant death frequently shortens the subsequent birth interval. Death is more common in low birthweight infants, and mothers who have had one low birthweight infant are more likely to have another. This may confound a relationship between birth interval and birthweight. Kramer's analysis of 26 studies suggest that there is no effect of interval on gestation or birthweight. However, fertility patterns will effect the nutritional demands on the mother, and may effect subsequent offspring indirectly through maternal nutritional depletion.

5.9 Antenatal attendances

Kramer reviews the evidence that antenatal clinics effect birthweight, looking at time of first visit, number of visits made, and the quality of the clinics. Women that attend antenatal clinic early and attend regularly may be relatively privileged in some way, and therefore more likely to have larger babies irrespective of the effect of the antenatal clinic. In summary, no firm

conclusions can be drawn about the impact of antenatal care on birthweight.

5.10 Conclusions

A. Identifying birthweight determinants is obscured by confounding between causative and associated factors.

B. Maternal nutrition, malaria, socio-economic status and smoking habits stand out as important probable determinants. Malaria and poor nutrition are particularly important because part of their effect may lie in shortening gestation, thereby increasing the residual proportion and perinatal mortality.

C. Parity, anaemia, malaria and iron status are likely to be related in endemic malarious areas.

D. High preterm rates have been reported from some studies conducted in malarious areas.

6 PERINATAL MORTALITY AND BIRTHWEIGHT

6.1 Low birthweight and mortality

Comparison of mortality between infants whose birthweight was less than 2.5 kg or more than 2.5 kg was the start of understanding the relationship between mortality and birthweight. The grouping creates a threshold value below which mortality starts increasing rapidly but gives the impression that there is something intrinsic in birthweight determining mortality. The evidence for this is thin. In fact, low birthweight may be simply representing an increased chance that the baby is preterm, acutely growth retarded, or abnormal is some way; it may be the result of maternal growth modulation related her size; or it may be a consequence of some other factor which itself has an independent effect on perinatal mortality, such as socio-economic status.

This section reviews birthweight specific mortality, and examines some of its determinants within populations. A model using birthweight distribution and weight specific perinatal mortality curves adopted from Wilcox and Russell (1986) is used to describe overall perinatal mortality rates in populations. Finally, factors confounding the relationship between birthweight and mortality are discussed.

6.2 Birthweight specific mortality

Developed country data: Variation in mortality rates between babies falling into different birthweight categories has been recognised for many years. Karn and Penrose (1951) studied 13,730 infants born at University College Hospital, London, and showed a marked increase in neonatal death in babies whose birthweight was below 4.5 pounds (2.05 kg). In

the USA, Chase (1969) looked at infant mortality from registration of 109,861 deaths from 1960. Overall the infant mortality rate in those weighing 2.5 kg or less at birth was 192/1000 live births, and for those above, the rate was 10/1000. Infant mortality rates in non-whites were 59/1000 in 2.0 kg -, 25/1000 in 2.5 kg - and 17/1000 in the 3.0 kg - group.

Developing country data: Information on birthweight and mortality from developing countries took longer to emerge. Few countries have national birth registration, and data on births, birthweight measurement and infant outcome often require special studies.

Trends were similar to those found in the West. Morley (1973) shows the neonatal death rate increases with declining birthweight in Nigeria (neonatal mortality rate for babies weighing 2.0 - < 2.5 kg was 49/1000 compared with 15.2/1000 in the 2.5 kg - category).

A large study by the Pan American Health Organisation (Puffer and Serrano 1973) showed that a higher proportion of low birth weight babies (2.5 kg or less) died in the neonatal period compared to babies in the higher weight group in data from hospitals in Argentina, Bolivia, Brazil, Chile, Colombia, El Salvador, Jamaica, Canada and the USA. These data are only indirect evidence of a higher mortality in low birthweight infants as no denominator is available to calculate death rates by weight category.

Data from 1973 from Austria, Cuba, Hungary, Japan, New Zealand and Sweden on singleton live births as part of a large collaborative study by the WHO go further by defining mortality rates by birthweight group (WHO 1978a). The authors point out the problem of comparing statistics across countries when interpretation of definitions vary. For example, a live birth, by the WHO definition is when any sign of life ("beating of the heart, pulsation of the umbilical cord, definite movement of voluntary muscles...") which can occur in obviously non-viable early fetuses; but in Austria, for example, the primary criterion for a live birth was the presence of natural breathing. Using the perinatal mortality rate helps minimise between country bias caused by misclassification errors between fetal and newborn death. The study shows a high perinatal mortality in the low birth weight groups. In Cuba, for example, the perinatal mortality in the <2.5 kg and 2.5 kg+ group was 170/1000 and 11.5/1000 respectively. Mortality rises a little in birthweight groups above 4.0 and 4.5 kg. The authors rank countries by perinatal mortality and percentage of total births which are less than 2.5 kg, which shows a clear association.

Another WHO study looked at perinatal mortality in relation to birthweight in rural areas of Burma, India, Indonesia and Thailand (Perera and Lwin 1984). Birthweight and mortality were determined from a population base: the study in each country commenced with a house to house enumeration and registration of pregnant women. This contrasts with most birthweight data from developing countries which is usually from hospital confinements (birthweight and infant mortality data based on hospital deliveries in areas where only some of the population deliver in hospital is subject to selection bias, as the births may not be representative of the population as a whole). Birthweight were divided into 0.5 kg groups, and, in the 1- <3 kg range, perinatal mortality rates decreased with increasing birthweight categories, shown in table 2-4.

Table 2-4. Perinatal mortality rate per 1000 births by birthweight from rural studies in 4 countries (data from Perera and Lwin 1984)

birthweight	Burma	India	Indonesia	Thailand
1.0 -	733	545	850	1000
1.5 -	250	400	237	466
2.0 -	50	45	106	56
2.5 -	22	22	25	23
3.0 -	18	12	26	10
3.5 -	24	16	23	..
4.0 +	14	..	135	154

.. indicates the rate above is derived from a combination of the 2 weight groups

Puffer and Serrano (1987) review data available on mortality in relation to birthweight; they note that Burma, India and Indonesia have high rates of LBW infants and high infant mortality; South American countries are somewhere in the middle; and European countries have low rates for mortality and have fewer LBW infants.

6.3 Weight specific mortality curves

Karn and Penrose (1951) show a hump shaped relationship between neonatal survival and birthweight. They plotted the logarithm of the ratio of the number of deaths divided by the number of survivors at a given birthweight against birthweight, producing a parabola of survival odds against birthweight. They point out there is an optimum birthweight, at which the mortality curve reaches a minimum.

Wilcox and Russell (1983b) used a similar logistic scale (log[deaths/survivors]) and plot mortality against survival using data from Karn and Penrose. Mortality plots above 4 kg and below 3 kg were roughly straight, with slopes equal in magnitude but opposite signs. They found that the best fit of birthweight specific mortality in the 3-4 kg range came from using a formula incorporating the risk from low birthweight, the risk from high birthweight, and a constant.

The fact the slopes for the mortality at high and low birthweight are of equal magnitude does not imply that as many babies die of high as low birthweight. To begin with, the birthweight distribution is not symmetrical but skewed towards lower birthweight (section 2). Secondly, the "optimal" birthweight is heavier than the population mean, so that more deaths occur in low birthweight than high birthweight categories.

Mortality determinants in lower birthweight groups: Mortality rates in LBW groups will be particularly influenced by the proportion of preterm and asymmetrically growth retarded babies in them (section 3).

Table 2-5 shows projected perinatal mortality rates at various preterm rates, assuming the relative risk for perinatal mortality between term and preterm is 7. For example, a perinatal mortality rate of 40/1000 in term infants and 280/1000 in preterm infants will result in an overall perinatal mortality of 47/1000 with 3% preterm rate, or 83/1000 with an 18% preterm rate.

Table 2-5 also shows how different twin pregnancy rates can alter overall mortality perinatal rates. Twin deliveries are more frequently preterm, and mortality of twins is higher overall than singletons: perinatal mortality rate for all births will be influenced by the twinning rate of the population. Mortality rates are calculated for 2 twinning rates: 1% (approximately what the Western rate of 1 in 90) and a second of 2%. It can be seen that the overall mortality is increased by a factor of 1.23 (61.5/50) in the higher twinning rate group.

Table 2-5. Projected perinatal mortality rates at different levels of preterm rate, and different twinning rates

PROJECTED PERINATAL MORTALITY RATES AT DIFFERENT LEVELS OF PRETERM BIRTHS

	PERINATAL MORTALITY RATE (/1000)		
perinatal rate in term	30	40	50
perinatal rate in preterm	210	280	350
% preterm	calculated PNMR		
3	35	47	59
6	40	54	68
12	51	68	86
18	62	83	104

the relative risk of death preterm / term infants is set at 7 (section 3.1)

PROJECTED PERINATAL MORTALITY AT DIFFERENT TWINNING RATES

perinatal rate in singleton births: 50
perinatal rate in multiple births : 250 (Llewellyn-Jones 1986: relative risk=5)

1 % twinning rate (1000 births contains 20 twin babies): perinatal rate=50/1000
2 % twinning rate (1000 births contains 40 twin babies): perinatal rate=61.5/1000

Mortality determinants in the higher birthweight groups: Morbidity and mortality in high birthweight groups is common. Complications at delivery relate mainly to the difficult passage of the infant through the maternal pelvis, and include death or permanent mental retardation from asphyxia, meconium aspiration and brachial plexus injury (Bromwich 1986).

In a series of 1019 perinatal deaths in urban Addis Ababa, Naeye et al. (1977) showed a 9.5/1000 perinatal mortality from obstructed labour, accounting for 15% of the perinatal deaths.

Offringa and Boersma (1987) therefore question the value of food supplementation given during pregnancy to increase birthweight and reduce neonatal mortality. Increasing birthweight may simply shift birthweight distribution to the right, exposing infants to the high mortality rates in the upper part of the weight specific mortality curve for that population. In areas where obstetric services are poor, this may have a detrimental effect on the outcome of pregnancy.

Overall determinants of perinatal mortality: Some factors are likely to influence mortality whatever the size of the infant, such as the socio-economic status of the population, and the

quality and accessibility of health services serving them.

Socio-economic factors: Perinatal mortality will in part depend on the level of hygiene at delivery. It may also depend on the understanding of the mother and her attendants of the process of labour, and the particular way that delivery is managed. Cash income, quality of housing, and accessibility to clean water are likely to be important in perinatal risk. Overall level of education of the mother, and specific understanding of the processes of childbearing (which is not necessarily learnt through formal Western schooling) may be factors influencing care of the child in the critical perinatal period.

Health services: The use made of obstetric services and their quality may be an important influence on perinatal mortality. Where services are widely used for normal deliveries, the presence of health staff at delivery is likely to reduce perinatal mortality. If services are used only when a woman develops complications in labour, the impact on overall perinatal mortality is likely to be much less.

These overall determinants of perinatal mortality may well be predominate in areas where perinatal mortality is very high. In effect, the position of the weight specific mortality curve is simply move upward against the log scale of mortality rate. This means that the high perinatal mortality is occurring across all weight groups. The mid range weight groups tend to contribute most of the deaths as they contain the bulk of the population. In such areas, medical input through at risk policies focusing on LBW infants is unlikely to have any impact on overall perinatal mortality.

6.4 Factors confounding birthweight and mortality

During the 80's, the validity of the causal relationship between birthweight and mortality was questioned (for example, Goldstein 1981). Tompkins et al. (1985) point out that in descriptive studies each birthweight grouping is analyzed independently, with the assumption there are no other important between the statistical cell groupings other than those studied. However, birthweight is a surrogate measure for many other factors that may have an independent effect on risk, and thus confound the apparent relationship. It is possible to classify some of these factors under 3 broad headings, outlined below, with examples of potentially confounding factors:

Maternal factors: A mother who is poorly nourished may be at higher risk of giving birth to a low birthweight infant. Her malnutrition may influence lactation and poor newborn nutrition, thus tending to increase infant mortality.

Socio-economic: Socio-economic status of the family may effect both birthweight through its impact on nutrition, and may effect mortality in a variety of ways: for example, by its effect on the level of hygiene, child care management, water supplies, and health service use.

Baby: LBW is associated with abnormal clinical conditions such as congenital disorders, preterm delivery or acute growth retardation (section 3). These conditions may in themselves

cause an increase in mortality risk in comparison to normal infants.

In summary:

A. The proportion of infants that are preterm or asymmetrically growth retarded may have an important bearing on perinatal mortality rates because of their higher risk of death.

B. Low birthweight infants are not necessarily growth retarded.

C. In populations were perinatal mortality is high, it is likely that the poverty is acting through various channels resulting in infants of low birthweight; and that poverty may also be independently responsible for the high mortality risk in the population.

7 INTERVENING IN PERINATAL MORTALITY

The Wosera maternal and neonatal health project examined interventions that could be introduced through the existing health care system and which could possibly have some impact on perinatal mortality (chapter 1).

If the high preterm rate of Wosera newborns reported in 1986 by Winkvist (1988) was confirmed and was due to a specific aetiological factor, then a highly focused intervention to reduce the preterm rate would be possible. For example, if maternal malarial illness was resulting in many mothers labouring before term, malaria control measures could reduce the problem. On the other hand, if low birthweight infants were asymmetrically retarded, then possible causes of placental insufficiency in late pregnancy would need to be considered, such as malaria, workload or pre-eclampsia.

If the growth retardation appeared symmetrical, then other causes, such as smoking or poor nutrition may be contributing to the small infant size. Identifying a "quick and cheap" effective health intervention would less likely if nutrition seemed the main influence on birthweight: relief of poverty through gradual social and economic development would be a more sustainable solution.

Finally, if the high perinatal mortality were not confined to an excess of preterm or asymmetrically growth retarded infants, then limited health intervention by improving perinatal care by the mothers and her delivery attendants remained a possibility.

chapter 3

MATERNAL AND NEONATAL HEALTH IN THE WOSERA

1 WOMEN

1.1 Role in Sepik society

Women in the Wosera play a key role in subsistence food production. They maintain the gardens, plant harvest crops other than yams, and carry out a large part of the sago harvesting. At home, they are responsible for child rearing and child care, firewood and water collection, care of the pigs and food preparation. Their social status is lower than that of men, who dominate decision making and political life at all levels.

1.2 Education

Formal education: Primary school education throughout the country is provided by community schools, run by government or missions. In some provinces it is free, and in others parents are expected to pay a small registration fee per child at the beginning of each school year. Coverage of schools shows a lot of regional variation. Overall it is not high, and girls are less likely to attend schools than boys.

At Kaugia (figure 1-2), 320 children were enroled in 1987, but only 37% were girls. At Kunjingini and Jambitanget schools there was 48-50% female enrolment. Overall, 46% of grade six enrolment in Wosera primary schools were girls (Peter Konia, school inspector, 1987 figures: personal communication).

In the East Sepik Province (figure 1-1) 76% of women aged 15-49 years had not attended school (data from 1980 census). Forty six percent of men and 69% of women aged 12-25 years at the time of census had received no education. Only 10% of men and 5% of women had attended any secondary education. These figures are lower than for the country overall (Department of the Prime Minister 1984; Townsend 1985).

Absenteeism is higher in girls and thus enrolment figures probably underestimate the sex differential in schooling. The female role in household chores and child care means that young girls will often be kept at home to help mother; performance and achievement in boys tends to be more highly valued by the parents.

Non-formal education: Over the last five years there has been activity in the area through women's groups linked to the East Sepik Women's network. Workshops in Kaugia in subsistence agriculture, nutrition and health have been carried out. A *merisin meri* (women village health aides) scheme had been set up through Maprik health centre. However, non-formal education is sporadic, and reaches only a limited number of women.

1.3 Fertility

1.3.1 levels of fertility

In the Sepik: Data collected by Ross in 1984 showed that 34% of women attending Kaugia MCH clinics were of parity five or more, and 7% were parity nine or above (Heywood et al. 1986).

Sturt and Sturt (1974) collected longitudinal data 1963-1969 in the Anguganak area of the West Sepik. The total fertility rate (TFR) was 5.4 births per woman. [1] Fertility was not as high as in some developing countries, and could be due to the late age of menarche, estimated at 18.4 years (Wark and Malcom 1970); late marriage and birth of first child (average age 23.2 years); and maintenance of a long average birth interval (over 3 years).

Roscoe (1989) analyzed birth spacing retrospectively from MCH clinic data in the Yangoru subdistrict (East Sepik Province) and showed no obvious birth spacing changes from 1970 to 1987.

The National census provides an estimate of lifetime and current fertility from a stratified cluster sample survey conducted in 1981 (Baaker 1986b). The total fertility rate in the country as a whole was 5.4/woman of childbearing age. In the "rural village sector" it was the same, and in this group mean age at childbearing was 29.8 years. The crude birth rate was 33.9/1000 population. The rates for the Northern Coast, which includes the East Sepik, were very similar to these national averages.

Baaker also shows that fertility had not changed much overall between 1966-1980. Fertility in women with some primary education was higher than women with no education, probably because women living traditionally in villages were less likely to attend school and more likely to adhere to traditional family spacing practices.

Religious denomination seems to have some bearing on fertility. A comparison of fertility data from 1966-70 and 1976-80 separating Catholics from Major Protestant adherents shows the TFR had increased in Catholics and declined in Protestants, although the difference was not large.

1.3.2 Fertility control

National government population policy: In response to a national growth rate estimated at 2.3% per annum, the country has moved toward setting up a National Government population policy. An early document points out that growth may increase to 3% with a population doubling time of less than 25 years (Department of Finance and Planning 1988).

1

Total fertility rate represents the total number of children that would be born to a woman should she experience the current age specific fertility rates (Pollard et al. 1974)

Health department policy on contraception: Access to methods of fertility control remains part of PNG national health policy. However, fewer than 7% of adult women aged 15-44 years are using clinic supplied methods of family planning. A national programme started in 1978 lost its impetus with decentralisation in 1981 (Department of Health 1986).

Delivery of family planning through MCH clinics is hampered by the fact that 27% of health centres and 64% of health subcentres are run by Catholic agencies, who do not participate in provision of contraceptives (Groos and Garner 1988). This is rarely discussed in relation to the poor progress in providing family planning through the health services. The target for availability of contraception through health subcentres of 100% by 1989 (Department of Health 1986) seems to ignore this problem.

East Sepik and the Wosera: In the East Sepik Province there were 721 new contraceptive acceptors in 1984, accounting for less than 1.5% of the total number of women aged 15-44 years. In Maprik, a community based contraceptive pill distribution scheme was set up through an existing women's village health aide scheme. The project was quickly thwarted in the Wosera by expatriate Catholic priests, who announced in church that women using the contraceptives would give birth to "headless chickens or deformed children". There were fewer than 20 new acceptors over the two years, and none from the Catholic area (Bill Lapai, health educator: personal communication).

1.4 Pregnancy

1.4.1 Traditional beliefs and practices

Townsend (1986) has reviewed this subject.

Women recognise the signs of early pregnancy even if amenorrhoeic due to lactation. The delay in clinic attendance frequently attributed to women not knowing they are pregnant is usually related to a reluctance to attend. Pregnancy occurring prior to weaning of the previous child indicates the woman has broken the traditional postpartum taboo on sexual intercourse, and they may be too ashamed to attend antenatal clinic.

There are no social mechanisms for reducing maternal work during pregnancy, and no indication that women do less at this time. Some Sepik women believe that if the mother is "lazy" during pregnancy the inactivity may cause a heavy birth and difficult labour. Winkvist (1988) reported that women threw themselves from trees in late pregnancy to induce labour.

Food taboos during pregnancy do exist, but are usually specific on peripheral foods, such as game, which the women are unlikely to eat very often anyway. The taboos alone are unlikely to affect women's nutrition during pregnancy.

Almost all deliveries in the Wosera occur in the village in the traditional *haus graun* (thatched buildings with earth floors). Schofield's description (1963a) has changed little. Attendants are frequently other women relatives or in-laws, or other women from the village.

The husband may help. Delivery is onto a *limbum* (a flat piece of bark). After delivery of the baby, there is no active management by mother or attendants of placental delivery. Once the third stage is complete, the mother washes with a cup of water; the infant is then attended to. The cord is cut short, using a razor blade or specially sharpened fresh bamboo. The houses are dark inside, and contamination of the baby or placenta with earth is not uncommon during delivery. The placenta is buried, and the infant put to the breast if crying. Sometimes the infant may be rinsed to remove vernix.

Parturition difficulties such as delay in delivery of the baby are first managed on a psychosocial level within the village. Traditional beliefs and interpretations are discussed in the extended family group. If this fails the family will often use health services.

1.4.2 Maternal mortality

The national maternal mortality rate is estimated to be 8/1000 births, and 20/1000 in some rural areas (Mola 1985). The main causes are postpartum haemorrhage, puerperal sepsis, and prolonged labour. Maternal mortality in the Sepik is likely to be high. In Anguganak, maternal mortality was 16/1000 (based on 877 births). Women delivering a stillbirth had a higher mortality (7/55 died: Sturt 1974).

1.4.3 Health services

Health department policy for antenatal care: the National Health Plan indicates more emphasis will be place on maternal health, particularly "identification of, and antenatal screening for, high risk pregnancies; provision of iron, folic acid and antimalarial drugs to pregnant women..." (Department of Health 1986: paragraph 21.40).

Specifics of the national programme are found in the standard management manual (Papua New Guinea Society of Obstetrics and Gynaecology 1986). This booklet contains standard regimens to be used "unless their are specific professional reasons to do otherwise". Pregnant women should be seen monthly until 28 weeks, then fortnightly until 36 weeks, and then weekly. At her first antenatal visit she should be supplied with treatment course of chloroquine for malaria, and mebendazole for intestinal parasites; and supplied with a prophylactic regimen of folic acid daily (5 mg) for a month and then weekly; oral ferrous sulphate (200 mg) twice daily; and chloroquine (2 tablets) weekly. Women are also given tetanus toxoid if they have not previously been vaccinated. Tetanus toxoid booster injections are given to nulliparous women who have not received a booster in the last five years, and women having their fourth child.

Health department policy for care at parturition: Institutional deliveries have more than doubled during the decade 1972-83 and account for approximately 40% of all births. The Health Department are aiming for an increase up to 65% of all deliveries by 1990 (Department of Health 1986). Mola (1985) suggests that over the past 10 years the increase in institutional deliveries has been a urban phenomenon, and that increase in rural sector has been only 14%.

National health department policy encourages training in maternal care for voluntary community health volunteers where suitable primary health care schemes are running; and innovative ways of improving care, such as developments in village birth attendants and maternity villages in districts where there is commitment from the community or health workers.

Antenatal care in the Wosera: Antenatal coverage in the Wosera was variable (table 3-1). At Jambitanget there were no maternal child health clinics at all. At Kaugia the clinics were held but in 1985 only 34% of the target population of pregnant women were registered. At Kunjingini clinics coverage was high, but 52% of total visits were new attendances, suggesting many women were only attending antenatal clinic once.

Table 3-1. Antenatal clinic attendance in the Wosera 1986

clinic	new attend ances	re-attend-ances	target popul-ation	coverage (%)	new/total attendances (%)
Kaugia	85	95	248	34	47
Kunjingini	406	374	350	12	52
Jambitanget	0	0	305	0	0

Sources: target populations: Heywood et al. 1986; antenatal figures from clinic registers

1.5 Nutrition

Nutritional status and age: Nutritional status of both women and men in PNG appears to peak in the 20-29 year age group, and then decline. In women, the decline is usually greater and the final difference larger than in men (Groos and Garner 1988). The decline is seen in weight, skinfolds thickness, and in weight for height. Sinnett (1973) shows lower nutritional status in older women in a highland community. The problem with cross-sectional data is that an apparent decline in nutritional status with age may be due to a secular change in nutritional status. A longitudinal study from the Solomon Islands shows that at least some of the nutritional differences between age groups seen in cross-sectional studies is the result of a true decline in the nutritional status of the cohort (Friedlaender and Rhoads 1982).

Changes during pregnancy: Information on nutritional changes during pregnancy in rural PNG women is sparse. Estimates derived from cross-sectional data on pregnant women of known gestation suggest women gain about 6 kg in body weight during pregnancy (Groos and Garner 1988). There is some evidence from the Highlands that maternal physical activity is reduced during pregnancy and lactation (Greenfield and Clark 1975).

Changes during lactation: Harrison et al. (1975) demonstrate declining weights in the course of lactation. An intensive study by Orr-Ewing in the Madang area shows an average loss of 5.6 kg of fat for 12 months of lactation, with a sharp increase to 1.0 kg a month from 12 to 15 months (Groos and Garner 1988). Data from the Maprik District (Schofield 1962) show weight differences within age groups between lactating and non-lactating mothers. This is particularly marked in women aged 21-25 years, with a 3.11 kg difference in weight

between nulliparous women and those who had been lactating for up to a year.

Relationship with maternal parity: Analysis of maternal nutritional status in relation to parity could reveal a lasting affect of pregnancy and lactation on maternal nutritional status. A small study in the Highlands demonstrates a decline in weight and skinfolds measurements between nulliparous and primiparous women. (Greenfield and Clark 1973). Bailey (1963) reports lower weight for height and skinfolds thickness in women with one live child compared to nulliparous women. Harrison (1975) describes a significant decline in skinfolds measurements with parity.

Nutritional status of Wosera women: Table 3-2 summarises studies of nutritional status of Wosera women. Where data has been given by age group, a weighted average has been taken.

Table 3-2. Summary of anthropometric means of Wosera women from different studies

source, year	villages	n	mean height	mean weight	wt/ht	remarks
Bailey, 1961	Stapigum	54	146.6	42.8	87	<40 years, not pregnant
Schofield, 1963	Kaugia + others	520	145.2[1]	38.6	81	tinea villages
Ross, 1984	Kaugia clinic area	356	151.1	43.7	85	MCH clinic not pregnant

[1] mean height and weight calculated from weighted averages

Bailey examined women from one village. Schofield conducted surveys in villages chosen because of a high prevalence of *Taenia imbricata*, and demonstrates an association between the disease and poor nutritional status, suggesting a possible bias in the villages selected, and estimates of nutritional status are likely to be lower than the overall level in the Wosera. Data from Ross (1984) are more recent but does not include nulliparous women.

Interestingly, mean height in Ross's study is greater than in the other 2 studies conducted 21-23 years before. This could reflect a true secular change in nutritional status with time.

1.6 Smoking

Materials: In the Wosera, the traditional smoking material is a leaf tobacco grown in the garden and air dried inside the house (*brus).* This is smoked by both men and women either rolled in a tobacco leaf or newspaper, or in a pipe.

Processed tobacco (in the form of sticks ("twist"), loose tobacco ("spear"), or cigarettes) has been introduced recently. Abuse is known to be on the increase in urban and rural areas because of extensive advertising even in the most isolated areas. In the Wosera cigarettes

and tobacco account for 4.1% of trade store expenditure (Heywood et al. 1986).

Constituents: Data on the smoke constituents of *brus* are limited. Levels of tar and nicotine (Brott 1981) showed a mean value of 48.5 mg/*brus* cigarette for tar (compared with 17 mg/manufactured cigarette) and 5.4 mg/*brus* cigarette for nicotine (compared with 1.1 mg/manufactured cigarette). Carbon monoxide levels were not measured.

Women's smoking habits: In a study in a rural community in the Highlands of PNG, Vallance et al. (1987) showed an overall reduction in the proportion of smokers over the last 15 years. In women aged 15-24 regular smokers declined from 47 to 6%; however, in those aged 25-44 years the decline was less marked, from 64 to 56%, with an increase in occasional smokers from 1 to 11%. There was a large increase in the use of commercial tobacco up to almost 100% in smoking women of childbearing age.

Wosera women are also exposed daily to carbon monoxide and smoke from household fires.

1.7 Malaria

1.7.1 Malaria in PNG

Malaria in PNG varies from highly endemic stable malaria in the lowlands, to epidemic unstable malaria in parts of the highlands (Moir and Garner 1986). Characteristics of malaria in the stable endemic areas of the country include:

Persistence of splenomegaly in adults: Traditionally, moderate malaria endemicity (hyperendemic) is defined by populations with high spleen rates in children (>50%) and adults (>25%) whereas the highly endemic areas (holoendemic) are defined by populations with high spleen rates in children and low rates in adults (Bruce-Chwatt 1985). The classification is based on supposition that low spleen rates in adults in areas of stable transmission is because the adults are highly immune as a result of heavy exposure during childhood. This classification does not fit well into the pattern observed in New Guinea. Metselaar (1956) described a population in West New Guinea where parasite rates declined after the age of 5 years, and yet spleen rates remained high in adults (78%). Peters and Standfast (1957) observed this in rural Maprik (46% spleen rate in adult males). In Madang, rates of between 31.3 and 37.0 were observed in some 5500 adults (Cattani et al. 1986). It has been suggested that splenomegaly of groups of people living in the same area may be dependent on their region of origin (Brabin et al. 1988).

Drug resistance: Resistance of *Plasmodium falciparum* to chloroquine is now widespread. Seventy three percent of strains show *in vitro* resistance, and 54% *in vivo* resistance (Dulay et al. 1987). *In vivo* resistance is lower probably because host immunity contributes to parasite clearance, and is usually at the R1 level. This means that although parasitaemia with or without symptoms recurs within 28 days of a full treatment course of 4-aminoquinoline drugs, the treatment does initially clear the parasitaemia and eliminate symptoms (Sapak and Garner: submitted manuscript).

Shift in parasite formula: Cattani et al. (1986) noted a shift from a predominance of P. vivax in the Madang area in 1977 to P. falciparum in 1981-3. This may reflect differential drug sensitivity.

Vectors: The vectors in PNG are from the *Anopheles punctulatus Donitz* complex. There are three species that transmit malaria: A. punctulatus, A. koliensis, and A. farauti No.1. (Charlwood et al. 1986).

1.7.2 Malaria in the Wosera

The first malaria survey in Maprik was in 1956 and later a field station was established when the area became a pilot project for the country's malaria eradication program (Peters 1960). In Wosera males, parasite rates were 62% (Peters and Standfast 1957).

In 1984 malaria blood slides were collected in the Wosera as part of an evaluation of a primary health car programme. Parasite rates in adults were 38-51% depending on age group (n=1352: PNGIMR, unpublished data). *Plasmodium malariae* infection rate was 10%. This is high in comparison with the 1-3% infection rate for this parasite found in rural Madang (Cattani et al. 1986).

Schofield et al. (1964) studied parasitaemia and haemoglobin values in 2 census divisions close to the Wosera. The parasite rate in women aged 16-20 years was 53%, and in older women ranged from 18-27%. Spleen rates varied from 55 to 67% in adult women. The parasite ratio was 1:3:1 falciparum:vivax:malariae (includes mixed infections). Mean haemoglobin values in non-pregnant women were between 12-13 g/dl, and 9.9-10.2 g/dl in pregnant women. With the introduction of surface spraying with DDT plus mass administration of a single dose of pyrimethamine plus chloroquine, mean haemoglobin of the population rose. In pregnant women, the rise was 1.4 g/dl.

2 INFANTS

2.1 Mortality

Infant mortality rates: Before health services had been introduced in Maprik, the infant mortality was estimated as higher than 500/1000 (Peters 1960). This seems very high and barely compatible with demographic survival of the population. Retrospective birth history data collected by Ross in 1983 from mothers attending Wosera MCH clinics gave a combined infant mortality rate plus stillbirth rate of 130/1000, with the deaths concentrated in the neonatal period (Heywood et al. 1986). Accuracy of mortality rates derived from retrospective birth histories are limited by problems determining age at death. In addition, the estimate includes both recent deaths and others occurring up to 20 years previously, when the risk of death may have been higher. Nevertheless, the data suggests a reduction since the 1960's.

Census data, using indirect measures of infant mortality, suggests infant mortality fell from 183/1000 to 94/1000 between 1971 and 1980 in the East Sepik. These provincial rates are among the highest in PNG (Baaker 1986a).

In the country infant mortality has declined from around 240/1000 in 1946 to 72/1000 in 1980 (Riley and Lehmann 1989). In Anguganak, Sturt (1972) documented the fall with the introduction of basic curative services from 108/1000 in 1963 to 61/100 in 1971. The decline in infant mortality from staggering high levels down to 60-80 /1000 has occurred in many areas of the country like Angugunak with very little social or economic development. Further mortality reduction probably requires not only health services but broader development.

In summary, the East Sepik is one of the 3 most disadvantaged provinces in PNG as measured by infant mortality. The level in the Wosera may be higher than the provincial average, and higher than that found in other deprived areas after the introduction of health services.

Neonatal mortality rates: In the Wosera, Ross's survey in 1983 indicated that up to 79% of infant deaths were occurring in the first month. This seems high, and may reflect problems with defining age at death. Schofield et al. (1961) estimated neonatal mortality at 200/1000 excluding those due to neonatal tetanus in the Maprik district.

There are some direct estimates of neonatal mortality from village based studies. In Tari (Highlands), Lehmann (1984) determined levels by direct enumeration of up to 27,000 people: the neonatal mortality rate was 19.4/1000 and the postnatal mortality rate was 54.8/1000, suggesting neonatal mortality accounted for less than a third of infant deaths. However, continuous demographic surveillance may miss neonatal deaths. A study in a rural area of Madang in 1982-4 following a cohort of 1002 births showed a neonatal mortality rate of 22/1000 and a postnatal mortality rate of 24/1000 (Moir et al. 1989).

The contribution of neonatal deaths to infant mortality is viewed as important to health service planning and priorities (Aitken 1987).

Stillbirth rates: Stillbirth rates are even more difficult to determine. In the Tari study, the rate in 1980-83 was 16.3/1000 births. In Anguganak (Sturt 1972) the rate was 62/1000 births.

2.2 Preterm Infants

Incidence data: Previously, "prematurity" described low birthweight (for example, Jansen 1962). There is little information on preterm incidence. A recent study of 738 women delivering in Port Moresby General Hospital and Goroka Base Hospital revealed a mean gestation of 38.7 weeks by Dubowitz ascertainment (R Primhak, personal communication).

Winkvist (1988) determined a preterm rate of over 30% by Dubowitz ascertainment. This rate seems remarkably high. The need to verify her findings were important in the study planning.

Mortality and preterm newborns: "prematurity" appears to be an important cause of death in hospitals and health centres. Aitken (1987) shows that 44.5% of registered neonatal deaths were due to "prematurity", with 36.9% due to infection (1982 figures). However,

prematurity was defined at that time by birthweight (less than 2.2 kg). The 44.5% of neonatal deaths thus represent deaths in neonates with low birth weight, and, although some of these deaths may be indeed be preterm, how many is unknown.

2.3 Birthweight

Birthweight in the Sepik: The mean birthweight of infants born in the East and West Sepik Provinces are low compared to values seen in Western populations. Schofield (1962) reports a mean birthweight of 2.72 kg from Maprik district. In an adjacent province, Wark and Malcom (1969) report on a mean birthweight of 2.4 kg (SD=0.5, n=63).

Ross (1984) estimated birthweight of 2.7 kg in females and 2.9 kg in males using regression from weights taken at MCH clinics in the month after birth, using z scores for weight, weight for height and weight for age compared against international standards. Interestingly, intercepts for weight for height were close to zero but for height for age were negative, suggesting that Wosera babies surviving to 1 month are stunted rather than wasted in comparison to their American counterparts.

Winkvist (1988) recruited village deliveries at birth in the Wosera. Her families were self-selected in that they reported the births to her, and as such the sample could be a biased. The mean weight of 2.6 kg obtained from the 60 or so mothers is the best estimate of mean birthweight in the Wosera area to date.

Variation between populations: there is substantial geographic variation in birthweight in PNG. Babies born to mothers from the Highlands tend to be heavier than their coastal counterparts. Ferro-Luzzi et al. (1978) showed a 400 g difference between highland and lowland newborns; Greenfield (1983) found a difference of about 150 g; and Aitken (1987) describes mean birthweight ranging from 2.8 to 2.9 kg in coastal institutions and 3.0 to 3.2 kg in highland centres. Babies of Highlands mothers delivering in Port Moresby General Hospital are heavier than the babies of other mothers delivering there (R Primhak, personal communication). The reason for the coastal/highlands difference is not known.

Secular trends: Evidence for secular trends is limited. Both Lourie (1986: data from Milne Bay Province 1936-78) and Greenfield (1983: data from Goroka 1964-73) show no evidence of a significant trend in mean birthweight. Improving economic, nutritional and health status within populations would still require a long observation period to demonstrate an effect on birthweight. The mean birthweight determined by Schofield of 2.72 kg in the Maprik area compared with that determined by Winkvist of 2.6 kg 25 years later does not suggest that there has been any improvement in the nutrition or health of women in the Wosera.

Sex differences: Several studies have shown the mean weight of male infants at birth to be greater than that of females, consistent with findings from other countries (Scragg 1955; Ferro-Luzzi et al. 1978; Greenfield 1983).

Variation with maternal parity: In the Highlands mean birthweight is lower by 205 g in babies from nulliparous compared with parous women (Greenfield 1983: table 4, weighted

averages calculated).

Birthweight and mortality: Scragg (1955) looked at neonatal mortality by birthweight. Neonatal period starts increasing rapidly below 2.3 kg (5 lbs), although the numbers of infants are small.

Heywood and Lehmann (1984) followed up a cohort of 1635 babies in the Tari basin born in health facilities to one year. The mean birthweight 3.01 kg. By demographic surveillance they were able to ascertain that this was 47% of the births in that area. Infant mortality in the 24 infants weighing less than 2.0 kg at birth was 500/1000; this levelled out to between 46/1000 and 78/1000 in weight groups up to 4.0 kg. In the 68 children above 4.0 kg, there were no deaths. This gives a good indication of mortality by birthweight in mothers attending facilities for delivery.

2.4 Causes of neonatal mortality

Neonatal tetanus was once common in the Wosera, causing an estimated 61/1000 neonatal deaths (Schofield et al. 1961). This has been reduced to almost zero probably due to tetanus toxoid vaccination of females, either as children or during pregnancy. In the absence of MCH services in the Jambitanget area, new cases of neonatal tetanus may arise.

The incidence of neonatal sepsis is not known. The diagnosis is rarely made: a review of two months of inpatient records from the Wombisa Health Centre uncovered two cases with this diagnosis (Heywood et al. 1986). Neither had been treated with the correct antibiotics. Health workers reported that neonatal sepsis was uncommon: the incidence may in fact be low. However it is not clear whether health workers accurately diagnose the condition, and many mothers with sick neonates may fail to attend health centres.

3 SUMMARY

A. Fertility in the Sepik appears constrained by traditional marriage and birth spacing practices.

B. Maternal nutritional status declines with age and childbearing.

C. Malaria is widespread in the Wosera, with *Plasmodium falciparum* predominating.

D. Infant and neonatal mortality in the Wosera appears high, and is probably higher than the national average. Causes of neonatal mortality are not clear.

E. Birthweight in lowlands PNG tends to be low, and this is the case in the Wosera.

chapter 4

WOMEN OF CHILDBEARING AGE: A CROSS-SECTIONAL STUDY

1 AIMS

The aims of this study were to:

i describe fertility, education, nutrition, and malaria in women of childbearing age in the Wosera subdistrict;

ii estimate the perinatal mortality rate of Wosera babies from maternal birth histories;

iii identify a cohort of pregnant women to commence a longitudinal study of pregnancy, with non-pregnant women providing control data for comparison.

2 METHODS

2.1 Sampling

The densely populated North Wosera with an estimated total population of 18 152 [1] was selected as the area to carry out the cross-sectional survey. Maternal child health (MCH) clinics were not used to identify the population of women because this would cause a selection bias: non-pregnant nulliparous women did not attend MCH clinics, parous women attended only if they had a child under 5 years old, and other clinic non-attenders might be particularly disadvantaged.

Wosera villages are found in groups, for example, Kumunigum 1, 2 and 3. Sampling units were these village groups. Women living in mission stations formed an additional group. The sample was chosen to include villages in all 3 clinic zones (Kaugia, Kunjingini, Jambitanget); villages situated on the fertile river flats (Kunjingini zone) and the low lying hills (Jambitanget and Kaugia zones); and villages that were distant from the main Wosera roads and mission stations.

The Jambitanget zone lacked maternal health services, had poor roads and was a long way from the PNGIMR base, and therefore fewer villages from that zone were surveyed. Altogether, twenty one of the 47 villages in the North Wosera were surveyed (table 4A-1).

The aim of the work was discussed with village leaders. There followed a meeting with villagers to explain the study. Surveys in the Kaugia and Jambitanget zone were conducted in March and April over 4 weeks, and Kunjingini in June 1987. Village coverage was measured against the 1987 electoral roll (Electoral Commission 1986).

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[1] Estimates of the size of the resident population were made by using the 1979 census data and the growth rate of 1.03% overall (calculated from 1962 and 1979 census information in the rapid rural appraisal). estimated population in the North Wosera population in 1987 is 18 152, and the South Wosera 7 185, giving a total estimated population of 25 337.

2.2 Data collected

Name: Each mother's Christian "Catholic" name, traditional family name were recorded with the traditional name of her father and mother. Marital status was omitted after pilot testing the questionnaire: polygamy, remarriage and prolonged absence of the male partner meant that a meaningful response would require 3-5 questions. Instead, women were asked their husband's name. They were regarded as 'not married' if the gave no name, and 'ever married' if they gave a name.

Village of origin: Women were asked the village they had been born in. Care was taken to ensure the response recorded was not the village the women had married into.

Age: Most women did not know their year of birth. Some families possessed census cards. These often contained an estimate of the woman's age when she was much younger (and the error in the estimate likely to be less). When age was not known, one experienced team member estimated the age of all women surveyed, using an algorithm (table 4A-2) before taking a birth history. The algorithm ensured the estimate was not based on maternal parity. In some instances age was estimated by back tracking from an estimated age of the woman's first born child. Consistency of census cards against the algorithm method was checked at each village [2]. Estimated age was checked by a second observer (PG) as each woman completed the survey.

Ever born children: Mothers were asked about past births to enumerate by sex living children, stillbirths, first week deaths (one week corresponds to the time a woman is traditionally confined indoors postnatally), and later deaths. Women were asked the number of times they had delivered twins, and whether their first born child was still alive.

Fertility: Date of birth of the last born child was taken from infant clinic books and recorded. Absence of MCH clinics meant these data were not available in Jambitanget area, so villages in this zone were excluded from age specific fertility rate estimates.

School: Completed grade at school was noted, equivalent to number of years attended. Years of attendance at catechetical classes (where religious doctrine and basic hygiene had been taught) was also recorded.

Maternal mortality: Women were asked if they had sisters, and if those sisters were still alive. If women reported a death they were asked if the sister had been of childbearing age, if she had been pregnant or if the death was connected with childbirth. No attempt was made to enumerate the age or number of sisters, and this method is separate and arose independently to the more sophisticated sisterhood method (Graham et al. 1988). A short history of the circumstances of death was recorded in all adult woman that had died. Names of relatives that the team could be interviewed later was recorded.

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[2] Census cards at Kunjingini 2 appeared inaccurate, and were not used to estimate age

Anthropometry: Standard procedures for anthropometric measures were used by staff already experienced in the techniques. Equivalence between observers was maintained by weekly training sessions set up by Professor R. Gibson, University of Guelph.

Height was measured twice barefoot using a *Holtain* anthropometer (supported by a wooden stand) to the nearest mm. Weight was measured to the nearest 100 g using a heavy beam scale or an electronic scale. Calibration of scales was tested weekly against a known volume of water contained in a plastic bottle of known weight. Mid upper arm circumference (MUAC) was measured twice using an *inser-tape* (Ross, Canada) to the nearest mm. Triceps (TSF) and subscapular (SSF) skinfolds values were established from the mean of three measurements taken sequentially from the right arm, using new Holtain skinfolds callipers. Ninety one percent of all women seen had their skinfolds estimated by one observer.

Spleen assessment: The spleen was measured by Hackett's grading in non-pregnant women (Bruce-Chwatt 1985), and measured in cm in pregnant women. Assessments were done by 1 of 2 doctors without inter-observer checking.

Haemoglobin values: Blood samples were taken by venepuncture. The haemoglobin estimation was done at the survey using a 'Hemacue' (Akiebolaget, Sweden) device. Five percent of field haemoglobin estimations were cross-checked in the laboratory.

Free erythrocyte protoporphyrin and packed cell volume values: From blood transported in EDTA, packed cell volume (using a microhaematocrit centrifuge) and free erythrocyte protoporphyrin estimations (using a hematofluorometer model 205, Aviv Diagnostics, USA) were carried out. 50 µl of blood was pipetted (Gilson, France) into PBS-tween and frozen for chloroquine ELISA estimation in Madang.

Chloroquine ELISA estimation: This was performed by PNGIMR laboratory staff using reagents supplied by Dr Teunis Eggelte, from Koninklijk Instituut voor de Tropen (KIT). This ELISA is thought to be more specific than the Dill Glazko urine test when for measuring urinary 4-aminoquinoline metabolites (Shenton et al. 1988).

Malarial parasitaemia: The thin blood films were fixed with methanol, stained with Giemsa dye, and parasitaemia determined by a standard protocol. One hundred fields were examined in each slide. If no parasites were seen the slide was recorded as negative. If positive, a density count was carried out. Parasites were counted against 200 white blood cells and recorded by species. If the parasitaemia was scanty (10 or less parasites per 100 fields) then the count was carried out against 100 fields, and at least one parasite ringed by the examiner. A 10% sample was re-examined by a second examiner blind to the findings of the first. Density differences and species inconsistencies between the two examinations were reviewed by the laboratory supervisor.

2.3 Pregnant women

Women who reported being pregnant were seen by a nurse, who recorded details of past pregnancies; their smoking and betel nut consumption habits; whether seen already at antenatal clinic, and, if so, their understanding of prophylactic drugs that may have been issued to them. The nurse explained the possible benefits of chloroquine and folic acid prophylaxis to the mother, detailed the weekly regimen, and supplied an initial treatment course of chloroquine to clear malarial parasitaemia. Abdominal obstetric examination was conducted, and their blood pressure and axillary temperature measured. Women were encouraged to attend the next antenatal clinic.

2.4 Referrals

Women sick at the survey were treated appropriately. Subjects who were anaemic were treated as outlined in the Standard Treatment Manual for Adults (Physicians Society of Papua New Guinea 1984), or, if pregnant, as outlined in the Standard Treatment Manual for Obstetrics (Society of Obstetricians and Gynaecologists of Papua New Guinea 1987). [3]

2.5 Data management and analysis

Data were checked on the day of the survey. IF inconsistent blood results were identified the samples were reanalysed. Anthropometric or demographic data which were missing or inconsistent were followed up by a village visit. Data were computerized during and after the study in Kunjingini. An interactive data checking programme was used to enter the data twice. Analysis was done using Statistical Analysis Systems (SASPC), release 6.03 (SAS Institute Inc. 1988).

Maternal weight for height standards were taken from Jelliffe (1966). [4] Body mass index was calculated using the standard formula:
body mass index=weight in kg/height in m^2.

Infant mortality was estimated by a modification of the Brass technique, incorporating multipliers originally developed by Trussell (chapter 3, Manual X: Department of International and Economic Affairs 1983). West life tables were used as they have appear to be appropriate for PNG (Deborah Lehmann, personal communication).

Skinfolds measurements, free erythrocyte protoporphyrin (FEP) and blood chloroquine estimations were skewed to the right. Logarithms of these variables were used in analysis.

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[3] One woman recruited as pregnant died a month later in Wewak General Hospital. It appears she died shortly after a laparotomy at the hospital, and may not have been pregnant. A second woman with a fixed abdominal mass who was referred to the Wewak surgeon. Both these women were excluded from the analysis.

[4] The height in 13 women was less than the lowest point of 139.5 cm. A plot of Jelliffe's standards was extrapolated back to obtain standard weights for these women.

3 RESULTS

3.1 Sample

A total of 1008 women aged 15-49 years from 21 villages were seen, plus women from the 3 mission stations (who formed 3.5% of the sample). The villages surveyed are listed in table 4A-7. Table 4-1 shows the female population of each clinic zone derived from the electoral roll, and the number seen at survey. The sampling fraction was highest in the Kaugia zone and lowest in the Jambitanget zone.

Table 4-1. Total population and surveyed population of childbearing women by clinic zone (pop.=population)

clinic zone	adult female population	SURVEY SAMPLE: women seen	% seen	total village-ages	distant villages	pop. from distant villages	pop. from mission stations
KAUGIA	941	502	53	10	2	64	14
JAMBITANGET	941	177	19	3	1	46	17
KUNJINGINI	1318	329	24	7	0.5 *	25	25
Total	3200	1008	31	20	3.5	135	35

adult female population derived from 1987 electoral roll, corrected to exclude post-menopausal women (reduced by 8.7%, correction derived from National Statistical Office 1983)

* 0.5 as only one hamlet of Abusit was relatively isolated

Absentees: Total absentees from the villages surveyed was estimated at 155 (13.8%). This excludes women classified as post-menopausal ("lapune pinis"). The numbers absent varied between villages, and usually there was some practical explanation in villages where absentee rates were high. [5] Given between-village mobility and the heavy female commitment to agricultural activities attendance can be viewed as good.

3.2 Age structure

Age was known in 61% of women (known date of birth, reliable census registration, or women knew the school grade completed and the year they left school). In the remaining 39% age was estimated using the algorithm (table 4A-2). A frequency distribution of individual ages shows no heaping on particular numbers. Whipple's index (the number of women with a recorded age ending in 0 and 5 divided by a fifth of the total women seen) was 97%, indicating no preference for these digits (Newell 1986).

[5] For example, Wisogum 1, which was the first village surveyed (14 absent); Kumunigum 3 (24 absent), a very spread out village-the message about the survey may simply not have reached some households; Jambitanget (22), a village with high mobility and movement to and from Maprik and Wewak; and Nale 1 (12 absent), where a village group had gone to a school function for several days at the Sepik River.

Figure 4-1. Age distribution of women seen at the cross-sectional survey (n=1008)

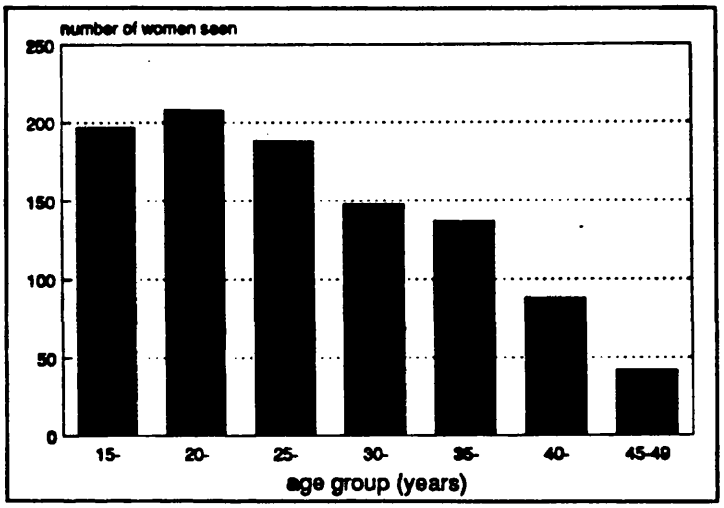


Table 4-2 shows maternal age by parity. Parity is defined as the number of pregnancies resulting in live born infant(s).

Table 4-2. Age and parity* of women seen

age group (years)	number seen	parity group				
		0	1	2-3	4-6	7+
15-	197	145	45	7	0	0
20-	208	70	74	56	8	0
25-	188	23	27	89	49	0
30-	148	9	5	36	77	21
35-	137	5	4	17	61	50
40-49	120	4	5	11	35	75
TOTAL	1008	256	160	216	230	146

* parity is number of birth events with a live outcome

Data sheets were checked in young women of high parity. In the 7 women aged 15-19 years with 2 birth events, the age was certain in 2; and in the 8 women aged 20-24 years with 4 birth events, the age was certain in 3. Infant deaths, which may shorten the subsequent birth interval, were more common in these young women: 6 had experienced at least one infant death.

3.3 Province of origin

Most women were from the Wosera (960). Of the remaining 48, 33 came from elsewhere in the East Sepik Province, 9 came from mainland New Guinea or New Guinea Islands, and 6 from the New Guinea Highlands.

3.4 Education

Table 4-3 shows that 60% of the women had not completed even one year at school. Older women were less likely to have attended school but the percentage of young women who had not received schooling was still high. The level of primary schooling of women in the Wosera seems to be slightly better than the provincial average as determined by the 1980 census.

Table 4-3. Frequency distribution of schooling by age group

	----completed years at school-----			
	none	1-6(%)	7+(%)	women (n)
VILLAGE WOMEN				
age group (years)				
15-	53.4	42.6	3.9	380
25-	64.9	29.8	5.2	328
35-	66.8	32.7	0.5	223
45-49	92.9	7.1	0.0	42
STATION WOMEN	2.9	42.9	54.2	35
TOTAL	60.0	34.8	5.2	1008

Fifty two women had received secondary schooling; 19 of these women were mission station residents, and only 33 out of 973 village residents (3.3%) had attended secondary schools.

Catechetical school training showed that attendance regularly for more than a year was more common in older women: 11.5% (68/593) of 15-29 year old women and 38% (157/415) of the 30-49 year old women had attended.

3.5 Fertility

Fertility indices are summarised in table 4-4. Overall 10.7% of the women reported being pregnant or this was found on abdominal examination. The 30-34 year age group contained the highest percentage of women categorised as pregnant (13.5%).

There were 17/1008 aged 25 onwards who reported that they not married. In the 40-49 year age group all women gave the name of a husband.

The crude birth rate was lower than the national census figure of 33.9/1000 population for the rural village sector.

The twinning rate was calculated per maternity. The rate was marginally higher than rates seen in the West. Scragg and Walsh (1971) reported higher rates in other areas of PNG (3.15% in New Britain, for example).

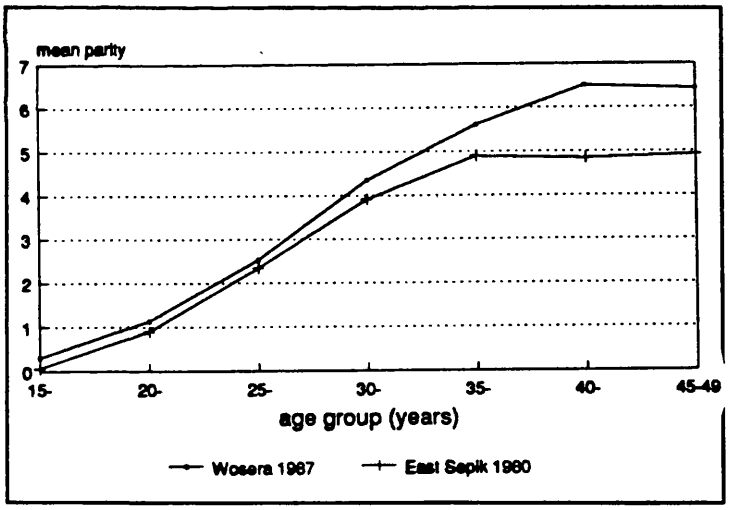
Table 4-4. Fertility indices derived from the cross-sectional survey

pregnant at survey	107/1000	women
general fertility rate (aged 15 49) [a]	184/1000	women
crude birth rate (estimated denominator)[b]	17/1000	population
twinning rate	22/1000	birth events
overall fertility (vs. Hutterites)[c]	0.451	
average completed family size	6.5	
total fertility rate	5.7 /woman	

[a] 149 births in 811 women (excludes Jambitanget zone)
[b] assuming women of childbearing age represent 9.1% of the population (national statistical office 1984)
[c] Newell 1986

Mean parity [6] by age group is compared with National Census data for village women of the East Sepik Province in figure 4-2. The mean parity of Wosera women aged 40-44 years and 45-49 years was 6.5 and 6.4 respectively, which does not suggest a recall bias in the older women, who might be more likely to forget births. Mean parity was higher in the Wosera compared with the provincial data by 1.8 births.

Figure 4-2. Mean parity of women from the cross-sectional survey compared with the East Sepik rural village sector from the National census 1980 (Baaker 1986b)

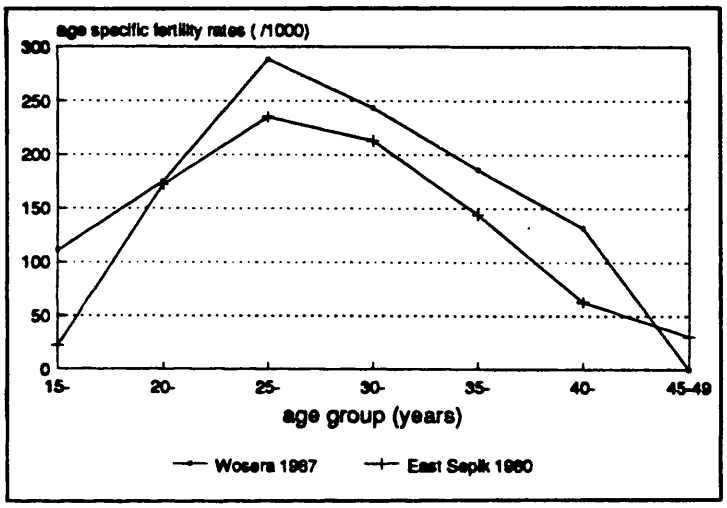


[6] In these fertility calculations parity is defined as live births per woman, and twins therefore count as 2. Elsewhere in the thesis, parity refers to live birth events per woman.

The Wosera total fertility rate (TFR) was 5.7 per woman. This is higher than the unadjusted TFR from the census of 4.4 for rural East Sepik.

Age specific fertility rates from this study are shown in figure 4-3 compared with the census data for East Sepik village women. In both, the rates peak in 25-29 age group. Wosera women 20-44 years old show slightly higher fertility overall. The provincial level for women under 20 years is higher than in the Wosera; this may reflect the traditional practice of not women not marrying until they are in their 20's in the Wosera.

Figure 4-3. **Age specific fertility rates from the Wosera cross-sectional survey compared with the East Sepik derived from 1980 census (Baaker 1986b)**



3.6 Mortality

Male:female ratio: Child survival from maternal birth histories is shown in table 4A-3. The male/female ratio for live births was 1.08, consistent with the ratio found in other parts of PNG (Lea and Lewis 1975).

Stillbirth rate: The stillbirth rate was 17 per 1000 births. This is likely to be an minimum estimate: some may not report a stillbirth, and older women may forget.

Perinatal mortality rate: Perinatal mortality rate calculated by age group of the mother shows a lower rate in older women (table 4A-3). This may reflect older women forgetting or not reporting early deaths or stillbirths from the distant past. Table 4-5 shows perinatal mortality rates reported by mothers aged 29 years or less, who are unlikely to omit deaths through memory loss. The proportion of children born alive and now dead shows that over 13% of live births born to women aged 15-29 years in the Wosera have died.

Table 4-5. Survivorship of ever born children from mothers aged 15-29 years

maternal age group (years)	women seen	births	perinatal mortality per 1000	live births now dead (%)
15-19	197	59	84.7	13.6
20-24	208	243	90.5	13.2
25-29	188	488	84.0	15.4

First born survivorship: Survival data concerning first born children (Kaugia and Kunjingini clinic zones only) showed that 91 first born children died out of 508: there were 12 stillbirths, 32 first week deaths, and 47 after the first week. The proportion dead (17.9%) was higher than the overall proportion for all women (14.8%), and the perinatal mortality of 87/1000 in first born children was higher than the value for all birth orders combined of 70/1000 (table 4A-3). Bias could account for these apparent differences. Asking a special question may improve recall of a deceased infant; and the higher percentage dead may simply be the result of first born children being older and therefore exposed to the risk of mortality for longer. The data does not demonstrate a greatly increased perinatal mortality risk for first born children.

Perinatal mortality and maternal education: The percentage of children dead classified by maternal age and schooling is shown in table 4-6. Schooling was associated with a slightly lower percentage of dead children.

Table 4-6. Percentage of children alive by age and schooling of mother

maternal age (years)	NO SCHOOLING: live births	NO SCHOOLING: child deaths	NO SCHOOLING: percent dead	RECEIVED SCHOOLING: live births	RECEIVED SCHOOLING: child deaths	RECEIVED SCHOOLING: percent dead
15-29	401	72	17.9	259	40	15.4
30-49	1256	247	19.6	662	88	13.3

Infant mortality: The estimate of infant mortality (using a modification of the Brass technique) gave a rate of 104/1000 live births (appendix 4A-4). However, the numbers of women in the 15-19 year age group on which this estimate is based is small.

Maternal mortality: Sixteen women stated that they had a sister that had died during pregnancy or shortly after, but only 3 of these reports had occurred in the previous 5 years (1982-). A further 30 reported a sister of childbearing age dying, but not connected with pregnancy; terminal symptoms reported by the relatives were later reviewed and did not reveal any that suggested a missed maternal death.

The 3 reported maternal deaths were followed up later by village interviews with the dead woman's husband or in-laws. In one case, delivery of the placenta was delayed and the

mother died shortly after the sister at Jambitanget health subcentre had removed it. In the second case, the woman died in labour. The village interview in the third case revealed it was not a maternal death. These results are combined with other data and reported later (chapter 5, section 4.11).

3.7 Nutrition

3.7.1 Descriptive statistics

Mean nutritional measures of non-pregnant women are presented in table 4A-5, and summarised in table 4-7.

Table 4-7. Mean anthropometric measurements in non-pregnant women

age (years)	N	height (cm)	weight (kg)	muac (cm)	wt. for height	BMI	tsf (geom. mean)	ssf
15-	361	152.1	49.6	23.9	95.9	21.39	11.42	14.4
25-	416	151.7	46.7	23.5	90.7	20.24	9.07	12.5
40-49	122	149.2	43.4	23.2	86.6	19.43	8.15	11.4
ALL	899	151.6	47.4	23.6	92.2	20.59	9.81	13.1

MUAC= mid upper arm circumference BMI= body mass index
tsf= triceps skinfold thickness ssf= subscapular skinfold thickness

The data show:

1. The women were short.
2. Low skinfolds values, reflecting low fat reserves and poor nutritional status.
3. Some women aged 15-19 years were still growing (the mean height and weight is lower in 15-19 year old women compared with 20-24 year old women). The body mass index (BMI) between these 2 groups was virtually the same, suggesting weight gain accompanies the increment in height.
4. Older women were shorter (the difference in mean height between the 20-24 year old women and the 45-49 year old women was 5.3 mm).
5. Nutritional status deteriorates with increasing age, commencing in the 20-24 year age group. Weight for height, body mass index and triceps skinfolds were lower in the older age groups.

Secular change in nutritional status: The height difference between younger and older women may be in part due to physical shortening with age, and partly a cohort effect: that is, socio-economic and nutritional improvement with time may have reduced childhood stunting in the population, so that younger women tend to be taller. This is difficult to answer without longitudinal information, but it is possible to compare this study with earlier data.

The mean height in this study is similar to that found by Ross (1984), but greater by 5 mm or more compared with earlier studies from the 1960's (summarised in table 3-2). Schofield (1963) found the mean height of women aged 21-30 years was 147.8 cm. These women

represent a cohort in 1987 aged 46-55 years. The mean height of 45-49 year old women in this study was 147.7 cm, very similar to their approximate cohort equivalent 25 years previously. Although this data must be interpreted with caution, it does suggest that the large difference in height by age demonstrated at the cross-sectional study is due, at least in part, to a cohort effect.

3.7.2 nutrition in relation to parity

Weight and height: Table 4-8 shows the mean weight of non-pregnant women by age and parity (number of birth events with a live outcome). Mean weight, as has already been shown, increased initially with age and then fell progressively in the older age groups. The table shows an affect of parity within each age group: weight declined as parity increased. The effect is obvious through all age and parity groups (apart from older women of parity 1 or 2, where the numbers are small).

If there were a relationship between short stature and high fertility (that is, if women from poorer, more malnourished families were more likely to marry younger and have more children) then height could confound this apparent relationship between parity and nutritional status. To test this, mean body mass index was tabulated against parity and age. This shows the effect of parity on nutritional status is maintained when controlling for height.

Table 4-8. **Mean weight and body mass index by age and parity for women not pregnant at the time of the cross-sectional survey. (weight in kg; number ())**

MEAN WEIGHT

age (yrs)	para 0	para 1	para 2-3	para 4-6	para 7+	ALL
15-	50.36 (195)	49.72 (100)	47.30 (58)	46.73 (8)	- -	49.61 (361)
25-	50.56 (36)	46.68 (33)	47.62 (118)	45.83 (165)	45.00 (64)	46.69 (416)
40-49	40.15 (4)	48.62 (5)	44.95 (10)	42.55 (31)	43.40 (71)	43.42 (121)
ALL	50.22 (235)	48.96 (138)	47.37 (86)	45.36 (204)	44.16 (135)	47.42 (898)

MEAN BODY MASS INDEX

age (yrs)	para 0	para 1	para 2-3	para 4-6	para 7+	ALL
15-	21.79 (195)	21.19 (100)	20.56 (58)	20.26 (8)	- -	21.39 (361)
25-	21.74 (36)	19.91 (33)	20.45 (118)	20.00 (165)	19.77 (64)	20.24 (416)
40-49	18.33 (4)	20.72 (5)	19.76 (10)	19.08 (31)	19.50 (71)	19.43 (121)
ALL	21.72 (235)	20.87 (38)	20.45 (186)	19.87 (204)	19.63 (135)	20.59 (898)

Analysis of variance indicates that there were highly significant effects of age on maternal weight, both linear and quadratic (table 4A-6). The effect of age was not adequately fitted by a straight line (both the initial increase and the subsequent decrease in weight are non-random effects). Neither is the effect of parity (decreasing weight with increasing parity) due to chance. However, the two effects were independent of one another (the interaction

term was not significant). Thus the age effect is the same, irrespective of parity.

Analysis of variance using body mass index overcomes the problem of the quadratic relationship between weight and age. The effect of parity group remained significant after age has been accounted for ($f=7.27$, $df=(4,892)$, $p<0.0001$), and there was no interaction. Mid upper arm circumference and subscapular skinfolds measurements showed a similar independent significant effect of parity group after age had been taken into account. Triceps skinfolds thickness showed a significant interaction between age and parity. The patterns for the different measures were remarkably similar.

3.7.3 Variation between villages

Analysis of covariance taking age, age^2 and parity group into account shows that village of residence has significant effect on both weight ($f=7.27$, $df=(23, 869)$, $p<0.0001$) and height ($f=4.10$, $p<0.0001$). Standardisation of weight and height for age and parity of the non-pregnant women by village of residence (using the least squares means of the GLM procedure in SAS) shows geographical variation in both these standardised nutritional indices (appendix 4A-7). The mission stations show high mean standardised weight and height, although the numbers of women seen was small. This is in keeping with the fact they are more privileged communities.

The villages where women had a higher mean standardised height (greater than 152.5 cm) were Abusit, Guiningi, Jambitanget, Kunjingini 1, Kunjingini 2, Nale 1 and Palgere. None of these villages are in the Kaugia zone. The villages where women had a low standardised height (less than 150.0 cm) were Kumunigum 2, Kutigum 1, Lingu, Talengu, and Wisogum 1. All of these villages except Lingu (near Jambitanget) are in the Kaugia clinic zone.

These data indicate poorer long standing nutrition in the Kaugia zone compared with the Kunjingini zone. Village sampled in the Jambitanget zone were few and showed a mixed picture.

3.8 Haematology

Mean measurements for haemoglobin, packed cell volume and free erythrocyte protoporphyrin (FEP) are shown in table 4-9. FEP measurements were only done in the latter part of the study which limited the sample size.

The mean haemoglobin measurements were low by Western standards. In women not pregnant at survey, the population mean was 10.1 g/dl; 13.2% (119) had haemoglobin values below 9.0 g/dl, and 2.7% (24) had levels below 7.5 g/dl. The mean FEP values were raised, [7] indicating the presence of iron deficiency.

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[7] Piomelli et al. 1976 report a mean value of 1.69 ± 0.67 µg of FEP per g Hb. for 48 normal adults. The upper limit of normal is taken as above 95% C. I. ie. 3.0 µg/g Hb.

The mean haemoglobin of the pregnant women was 8.7 g/dl (n=108), 1.4 g less than the non-pregnant group; but mean FEP were similar (pregnant women FEP mean: 3.9 µg/g Hb; n=29).

Table 4-9. Mean haematologic parameters of non-pregnant women at the cross-sectional survey

age (years)	n*	PCV	HB	MCHC	N	FEP
15-	361	35.0	10.1	28.9	104	4.0
25-	415	34.9	10.1	28.8	150	4.0
40-49	122	35.2	10.1	28.7	30	3.7
ALL	898	35.0	10.1	28.8	284	4.0

* missing data: age (n=1); haemoglobin (n=1)
UNITS: packed cell volume (%), haemoglobin (g/dl), mean cell haemoglobin concentration (g/dl) and free erythrocyte protoporphyrin (g/g hb); WESTERN NORMAL VALUES (women): PCV = 36-48; HB = 11.5-15.5; MCHC =30-35

3.9 Malaria

3.9.1 Basic epidemiology

Spleen rates: The spleen rate in the Kaugia and Jambitanget zones determined by one observer (GE) was 51%. The spleen rate in the Kunjingini clinic zone, determined by a second observer (PG), was 63%. Although the Kunjingini zone was surveyed some 2 months later, it is likely the difference in spleen rates is caused by an observer effect. Therefore spleen measurements were not examined by village or month.

Spleen rates by age group in women not pregnant at the survey varied between 51-61% : the overall rate was 55% (table 4A-8). There was little trend with age. The rate is similar to the spleen rates found by Peters et al. (1957) in adult males in the zone, and a little lower than the range found by Schofield (1962) of in the Maprik district.

Spleen rates by parity grouping (table 4A-10) showed slightly lower rates in nulliparous women compared with those in parity groups 1 and 2-3, but the differences were not dramatic. This is consistent with the findings of Schofield (1962).

***Plasmodium falciparum* species:** Species infection rate was 54% for all women: the rate showed a gradual decline with age from 65% in 15-19 year groups, to 38% in 40-49 year groups (figure 4-4). There was a similar gradient across parity, from 60.1% in nulliparous women to 40% in women of parity 7+ (figure 4-5). The data also shows that 32% of all infections were mixed (210/655).

Figure 4-4. Species parasite rates by age group in women not pregnant at the cross-sectional survey (all infections)

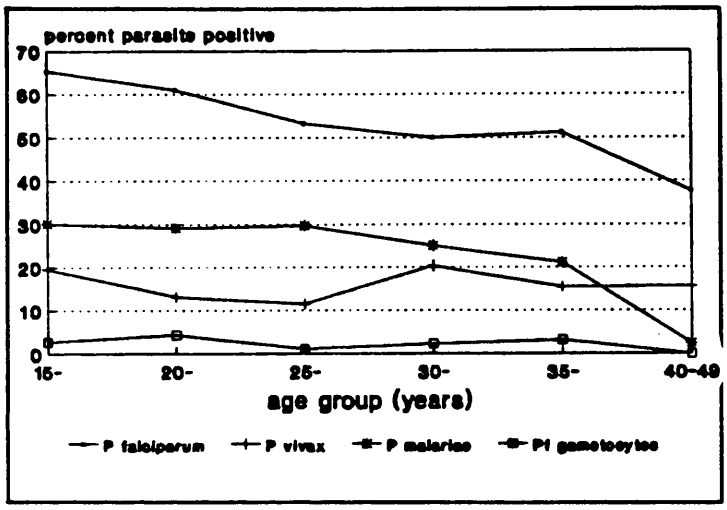
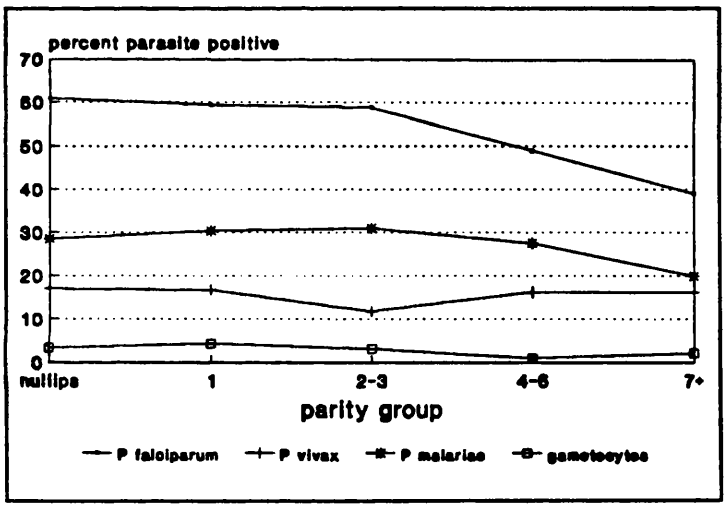


Figure 4-5. Species parasite rates by parity group in women not pregnant at the cross-sectional survey (includes all infections)



The lower prevalence of *Plasmodium falciparum* in older age groups was also reflected in a fall in mean species density with age, from 31 to 5.6 parasites/µl blood (figure 4-6).

Plasmodium falciparum contributed proportionally less to the parasite formula for single infections with increasing age (figure 4-7), moving from 74% of single infections in the youngest women to 48% in the oldest group.

The gametocyte rate was 2.8% overall which is consistent with findings in adults in other stable endemic areas of the country.

Figure 4-6. **Geometric mean species parasite density, all non- pregnant women, mixed infections included**

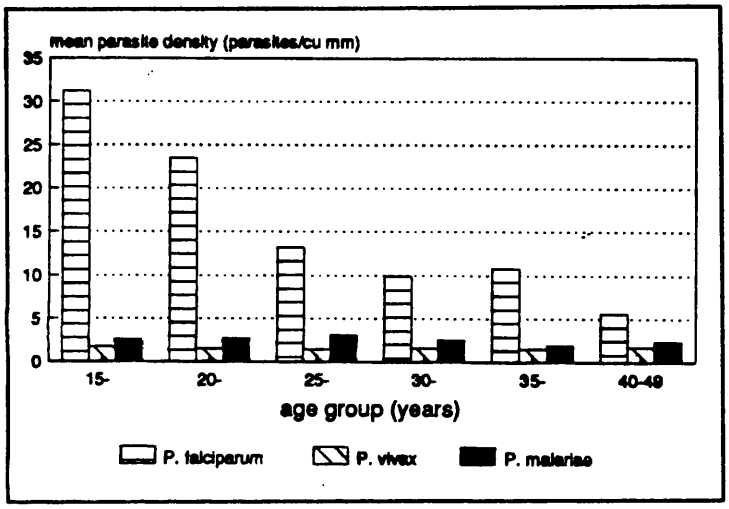
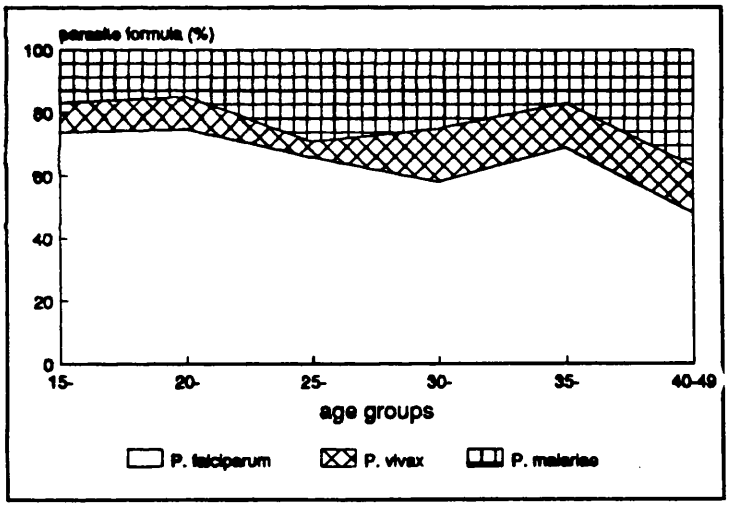


Figure 4-7. **Single species parasite formula by age group in non-pregnant women** (single species infections only used in the calculation)



***Plasmodium malariae* species:** *Plasmodium malariae* infections occurred in 28% of women that were found to be not pregnant, and was the second most common infection. This species infection rate is high in comparison to elsewhere in PNG. There was a downward trend with age (table 4A-9 and 4A-10).

***Plasmodium vivax* species:** *Plasmodium vivax* infections were seen in 16% of women that were not pregnant. There was no trend either with age or parity; and in all parity age groups, (except the 40-49 year old women), *Plasmodium vivax* was less common than the other 2 species (table 4A-9 and 4A-10).

Village variation: There is dramatic village variation in species parasitaemia (table 4A-11). *Plasmodium falciparum* rates varied from 39% (n=43) to 76% (n=50); *Plasmodium vivax* rates

varied from 8% (n=62) to 33.3% (n=15); and *Plasmodium malariae* rates varied from 14% (n=80) to 67% (n=24).

3.9.2 Blood chloroquine levels

Blood levels of chloroquine determined by ELISA were skewed to the right. In the 1006 women seen at the cross-sectional survey (including the women found to be pregnant) the median was 17.6 nanogram/l (ng/l); the range 0-1897 ng/l; and the 25th centile 9.5 ng/l. Table 4-10 shows blood chloroquine levels and malaria species parasite rates.

Table 4-10. Species parasite rates blood chloroquine levels for all women surveyed (n=1006)

		CHLOROQUINE LEVEL (ng/l)	N	-------SPECIES PARASITE RATES-------- falciparum	vivax	malariae
AGE GROUP	15-29	0-	156	60	15	23
		10-	354	62	14	32
		100-	77	54	10	21
		1000-	5	00	00	20
	30+	0-	108	55	17	23
		10-	248	46	19	25
		100-	56	29	07	25
		1000-	2	50	50	00

The results show that the blood chloroquine values for the population are low in comparison to other studies conducted in malarious areas where people have access to chloroquine (Teunis Eggelte, personal communication). Species parasite rates tended to be lower at levels of chloroquine above 100 ng/l.

3.10 Women pregnant at the cross-sectional study

Pregnant women already seen at antenatal clinic: One hundred and nine of the 1008 women seen during the cross-sectional survey were classed as pregnant. Of the 109, 33 (30%) had already been seen at MCH antenatal clinics. Table 4-11 shows that the main determinant of whether they had been seen was the clinic zone. More pregnant women had been seen at clinic already in the Kunjingini zone. In Jambitanget, where there no MCH services, only 2 had been to an antenatal clinic.

Table 4-11. Pregnant women already seen antenatally by clinic zone

Clinic zone	women already seen	total women	percent seen
Kunjingini	19	33	58
Kaugia	12	53	23
Jambitanget	2	23	9
ALL	33	109	30

Table 4-12 shows then number of time women had been seen antenatally already: most had only been once, and attendance was not related to parity.

Table 4-12. Number of previous attendances at antenatal clinic during the current pregnancy by maternal parity

PARITY	ATTENDANCES		
	nil	once	two plus
0	14	6	1
1	17	5	0
2-3	18	8	4
4+	27	4	5
all	76	23	10

Table 4-13 shows the stage of pregnancy when the pregnant women were seen at the cross-sectional study (calculated retrospectively from the date of delivery), and whether they had been seen already, grouped by education of the mother. This shows that only 7 women out of a possible 65 (11%) had been seen at existing clinics prior to 31 weeks gestation; and that educational status was not related to whether women had been seen at clinic.

Table 4-13. Education of women by their stage of gestation at survey and whether they had been seen already at antenatal clinic

SCHOOLING	STAGE OF PREGNANCY (WEEKS)			
	0-20 seen/women	21-30 seen/women	31-40 seen/women	all seen/women
no school	0 / 17	3 / 18	13 / 24	16 / 59
attended school	2 / 14	2 / 16	13 / 20	17 / 50
ALL	2 / 31	5 / 34	26 / 44	33 /109

Effect of previous antenatal attendance on malaria parasite rates: Women that had already been seen at antenatal clinic may have received chloroquine treatment or supplied with chloroquine prophylaxis. Of the 33 in this group, 19 reported having received a single dose of chloroquine at the preceding antenatal clinic, and 13 had been supplied with

prophylaxis (data missing in 1 woman).

Of the 13 supplied with prophylaxis, 5 appeared to have good understanding of the drug regimen and reported good compliance; in 4, comprehension and compliance appeared poor; in 3 the compliance appeared good but the regimen was erroneous; in one understanding was good but compliance unknown. Thus only 5% (6/109) of all the pregnant women seen at the cross-sectional survey could potentially be adhering to weekly chloroquine prophylaxis.

Intermittent chloroquine ingestion by pregnant women clinic might affect malaria parasite rates. Table 4-14 examines this by tabulating malaria species infection and chloroquine levels by age, pregnancy status and whether the woman had already been seen at antenatal clinic. In younger women the percentage infected with *Plasmodium falciparum* was highest in pregnant women who had not been seen at clinic, and lowest in pregnant women already seen at antenatal clinic. In the older women the falciparum species rate was lower overall and lowest in previously seen pregnant women. Mean chloroquine levels show little difference between the groups, except in older pregnant women already seen antenatally, where the level was higher.

These results show the parasite prevalence was lower in younger women already seen antenatally, but this was not associated with higher blood chloroquine levels. In older women, the parasitaemia reduction was associated with higher mean chloroquine levels. However, confounding factors such as parity and stage of gestation were not controlled for. It is possible that women in early pregnancy, when malaria parasitaemia is more common, were less likely to have been seen at antenatal clinic. Analysis stratifying by parity, whether a woman had been seen antenatally, and stage of gestation is described in chapter 5 (section 4.9).

Table 4-14. Species infection rates and mean blood chloroquine by age group, pregnancy status and whether previously seen antenatally

age (years)		N	fal	viv	mal	mean blood chloroquine (ng/l)
15-29	not pregnant	526	60	15	30	27.3
	pregnant, no ANC	47	66	06	17	25.2
	pregnant, seen at ANC	20	45	05	10	29.9
	ALL	593	60	14	28	27.2
30+	not pregnant	373	46	17	25	26.4
	pregnant, no ANC	29	45	10	24	27.2
	pregnant, seen at ANC	13	23	23	0	47.9
	ALL	415	46	17	24	26.9

fal=Plasmodium falciparum; viv=Plasmodium vivax; mal=Plasmodium malariae
ANC=antenatal clinic

4 DISCUSSION

4.1 Findings

Maternal **education** was poor, even in young women, despite the Wosera community school network. Whether females were particularly disadvantaged compared with males can not be deduced as the survey was of women only. However, 1987 enrolment figures suggest females in the Kaugia zone are less likely to attend school than males (chapter 3, section 1.2).

Fertility was high, greater than the provincial level but similar to other small studies in the Sepik (for example, Sturt and Sturt 1974).

Mortality of infants was higher than the national level in rural areas (Baaker 1986). Many deaths appeared to be occurring in the perinatal period. Perinatal mortality may be slightly higher in first born children. This phenomenon is common in other populations. Maternal education was weakly associated with reduced mortality. Education is often a marker of socio-economic factors. As education levels were so low and poverty so uniform then differences in mortality related to these variables is unlikely to be striking.

The study confirms previous findings of poor **nutritional status** of women in the Wosera. Differences in height and weight between women in different age groups was demonstrated: although this may be in part a cohort effect due to shorter, older women, the lower body mass index in older women indicates a true decline nutritional status with age.

Lower maternal nutritional status was associated with **increasing parity** within age groups. This was demonstrated convincingly with all nutritional parameters measured. Thus the more children a woman has delivered at a particular age the worse her nutritional status is likely to be. It can be argued that the age/parity relationship is the result of confounding by another factor (Graham and Danso-Manu 1988) such as socio-economic status. For example, poorly nourished women from marginalised families may exhibit higher fertility and likely to begin childbearing early. However, it is unlikely to be the explanation for the findings from this study for the following reasons:

1. Women from poorer families are more likely to be stunted; however, the relationship between nutritional status and parity is preserved in analyses using weight for height and body mass index.

2. The effect is consistent across parity groups, and the distribution within statistical cells suggests this is an effect spread through the population. For example, the effect is evident between parity 0 and parity 1 women in the 15 to 29 year age groups, which constitute 64% (341/526) of women in these age groups.

3. The affect of increasing parity causing falling nutritional status is consistent with data on the nutritional demands of pregnancy and lactation, and the knowledge that the

Wosera diet is marginal and lacking protein and energy dense foods (Heywood et al. 1986).

4. Additional longitudinal data of weight gain in pregnancy and some information on maternal weight 6 months postnatally suggests the process of childbearing and lactation does deplete maternal nutritional reserves (chapter 5, section 4.8).

The **variation between villages** and zones of maternal height may reflect differences in socio-economic characteristics. The Kunjingini area is closer to the Pagwi-Wewak highway, and where Wosera women sell food to travellers; and access to sell coffee or cocoa is less hampered by poor roads and lack of public transport. Groos and Hide (1989) report height differences in women from South Simbu, and relate this to cash income and female education.

The haematological findings confirm the **high prevalence of anaemia** in the population, as was **iron deficiency.** This is probably related to the high prevalence of malaria as it is now becoming recognised that iron deficiency is a common consequence of recurrent malaria in a stable endemic area (McGregor 1982).

The malariometric findings confirm the area is highly endemic. Blood chloroquine levels were low, although experience with this measure in cross-sectional malariometric surveys is limited. Parasite rates were similar to levels in the 1950's except that *Plasmodium falciparum* rather than *Plasmodium malariae* has become the dominant species. Even so, *the Plasmodium malariae* parasite rate was high compared with other areas of PNG surveyed in the last 10 years. This may be related to high levels of malarial transmission, or low use of chloroquine in the study population.

The existence and regularity of antenatal clinic appear to be important determinants of whether pregnant women attend, rather than maternal factors such parity or education. Women that do attend do so in late pregnancy. Only 5% of the pregnant women in the survey could have been taking chloroquine prophylaxis.

4.2 Critique of methodology

Sample: Sampling by village groups meant that larger village units contributed more to the final number of women in the sample than smaller village groups. The Kumunigum village group, for example, was responsible for 12% (123/1008) of the total sample of women seen and yet were responsible for only 1/21 or 5% of the village sample. This multiplicative affect may cause a bias in that the large village groups may be relatively over represented in the sample. This could have been avoided if Kumunigum 1, 2, and 3 were considered as separate units each with an equal chance of being sampled.

Having stratified by village group, it can be argued that village selection should have been randomised. In view of the small number of villages, it was decided to choose villages representing different types of village on quite an arbitrary basis, but ensuring that some smaller, more distant villages were included. It is possible that some bias determined our

choice of villages, although how this potential bias might be operating is difficult to know.

5 SUMMARY

In women of childbearing age in the Wosera:

A. the majority had received no formal education despite a network of community schools;

B. their fertility was high, and higher than the level for the Province;

C. their nutritional status was poor and appeared to decline with age. Frequent childbearing may have accelerated this decline;

D. anaemia and iron deficiency was common;

E. malaria was highly endemic and evidence of malarial infection was found in the majority of women;

F. few women had the opportunity to attend antenatal clinic, and those that did attended late in pregnancy;

G. regular chloroquine prophylaxis was not generally taken;

H. Infant mortality was high, especially in the perinatal period.

chapter 5

LONGITUDINAL STUDY OF PREGNANT WOMEN

1 AIMS

The aims of this study were to:

i. quantify factors that might affect birthweight and survival of infants born to cohort of pregnant women. The factors include maternal fertility, smoking,pregnancy weight gain, and the presence of pre-eclampsia, malaria and anaemia;

ii. institute a programme of chloroquine prophylaxis for pregnant women through existing MCH clinics, and to monitor prophylaxis compliance;

iii. determine the outcome of pregnancy of women in the cohort, and to follow up mortality in their infants to one year of age.

2 INTRODUCTION

Some maternal risk factors for low birthweight and perinatal mortality were identified through the cross-sectional study (chapter 4), but a more detailed study of pregnant women was required to assess antenatal factors that could be important, such as maternal smoking, and the presence of malaria or pre-eclampsia.

Chloroquine prophylaxis in pregnancy was likely to be beneficial as malaria was highly endemic in the Wosera. The national antenatal drug regimen of 4 drug types (chapter 3, section 1.4.3) was obviously too complicated for many women. Health staff frequently modified regimens, possibly in an attempt to improve compliance.

The standard drug regimen was therefore simplified to a more realistic schedule. Women were supplied with 3 day course of chloroquine (3 tablets) and folic acid (5 mg), followed by weekly chloroquine (2 tablets) and folic acid (5 mg). [1]

3 METHODS

3.1 Sample

Women seen at the cross-sectional survey who were classed as pregnant, or who subsequently became pregnant, were followed up through antenatal clinics. Other women came from the reorganization of antenatal clinics in the Kunjingini clinic zone in villages not

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[1] RATIONALE FOR THE MODIFIED REGIMEN: Intestinal helminths are not considered to be major aetiological factor in anaemia in PNG (Kariks and Woodfield 1972; Shield et al. 1987). **Mebendazole** was therefore omitted. **Oral iron** was omitted: compliance was likely to be poor as the drug should be taken daily; symptoms of gastric irritation are common (Finch 1980); and stool colour alteration caused maternal anxiety.

Chloroquine has been shown to be more effective than iron given alone in preventing anaemia in nulliparous women (Fleming et al. 1986); **folic acid** requirements increase in malaria induced haemolysis (Fleming 1989). These drugs were therefore prescribed.

covered in the cross-sectional study (chapter 1, section 4.4).

A control group of postnatal mothers not seen at antenatal clinic had been planned for the postnatal study (chapter 6, section 2.1), but was abandoned after 7 women had been seen. Data from this group were used in the analysis of fertility and personal habits analysis only.

3.2 The first antenatal visit

Forms used are reproduced in appendix 4. The following data were collected:

Demographic: Demographic data were identical to the data collected at the cross-sectional survey. Maternal age was assessed using the same method (chapter 4, section 2.2). Age estimations made by the nurse seconded to the project (MP) were discussed after each clinic with the demographic field officer (MEB) to ensure standardization.

Birth history: Past pregnancies were listed individually in all parous women. The mothers were asked whether the baby was born alive, the baby's sex, place of delivery (home or health centre), complications at birth, and if the child was still alive. Birth date accuracy was categorized as known to the day, to the month, to within 6 months, or a date that was a complete guess. The age at death of deceased children was determined from clinic records where possible; otherwise developmental milestones or school grade attended at the time of death were used.

Personal habits: Women were asked if they smoked every day, some days or not at all; how frequently they smoked the 4 main tobacco types (*brus*=home grown air cured tobacco; *stik*=stick tobacco sold in trade stores; *spia*=tobacco sold in tins; *sigaret*=cigarettes); how they smoked the tobacco (in a pipe, rolled up like a cigar, or rolled in newspaper), and whether they had smoked during the 4 periods (morning, afternoon, evening, during sleeping hours) of the previous day. Women were asked how frequently they chewed betel nut, and whether they had chewed in the 4 periods of the previous day.

Previous medical and obstetric history: A history of serious illness, operations, tuberculosis, caesarean section or receiving a blood transfusion were asked.

Prior antenatal attendance and current health: The number of times women had attended antenatal clinics already during the pregnancy was noted, and drugs dispensed were inquired after. If women had been given drugs to take home, they were asked what the medicines were, what they were for, how often they were supposed to take them and whether they took them. Women were asked if they had experienced a fever in the last 7 days, and the source of medicine if the episode had been treated.

Nutritional assessment: Height, weight, mid upper arm circumference, triceps and subscapular skinfolds were measured using standard techniques and equipment (chapter 4, section 2.2). Weight was measured with a heavy beam balance or an electronic weight scale (accurate to 100 g).

Laboratory tests: Maternal blood was taken for haemoglobin, packed cell volume, malaria parasite and chloroquine ELISA determinations. *Autolet* (Owen Munford, England) spring loaded capillary samplers were used to minimise maternal discomfort. Urine samples were tested by the Dill-Glazko test (which detects 4-aminoquinoline metabolites for up to 48 hours after a single dose of chloroquine: Lelijveld et al. 1970). Urine samples were also tested for glucose, nitrites, protein, and ketones (*Ames Multistix*, Miles Laboratories, Australia). For logistic reasons, urine samples were not collected from women classed as pregnant at the cross-sectional study.

Physical examination: This was done in the privacy of a specially constructed portable examination shelter. A brief general physical examination was conducted, including axillary temperature, sitting blood pressure using a mercury sphygmomanometer, chest examination for evidence of tuberculosis, and splenic size recorded in centimetres. The obstetric examination included measurement of the symphysis-fundus height in centimetres and fetal palpation.

Women who were ill or anaemic were advised appropriately. Those requiring admission and who were willing to go were transported to Maprik District Hospital.

3.3 Antenatal follow up visits

Follow up clinics were conducted every 6-8 weeks. Women were asked about recent illness. They were examined, and anthropometric measurements (except height) were repeated.

Prophylaxis compliance was measured in 4 ways:

1. Women were asked when they last took chloroquine. This was asked in a non-judgemental way to obtain an accurate answer (for example, "how are you getting on with the tablets?" or, "are you able to remember to take the tablets?").
2. The number of returned tablets were counted and bottles replenished to a known number of tablets.
3. Urine samples were tested using the Dill-Glazko reagent.
4. Blood chloroquine ELISA determination was done at alternate clinic visits.

Informal village visits were carried out shortly after the antenatal clinics had been reorganized to ascertain attitudes and compliance to prophylaxis.

Data management packages were used to generate current lists of pregnant women by village and stage of gestation estimated from the symphysis-fundus height. Women in late pregnancy were weighed at home between scheduled clinics in order to estimate weight gain in pregnancy more accurately. Women that were due to deliver were weighed every week.

3.4 Pregnancy outcome

Pregnancy outcome was determined through the postnatal study (chapter 6), through MCH clinics and by regular village visits. All live born infants were followed up for 12 months. In the event of a maternal or infant death, circumstances and details were recorded from relatives or health staff, employing forms and methods developed in an earlier mortality study in Madang (Moir et al. 1989).

The first 45 women who delivered were followed up postnatally at six months when their nutritional status was assessed.

3.5 Maternal mortality

A small qualitative study was conducted throughout 1987, examining retrospectively causes of past maternal deaths. Deaths were identified by villagers during informal discussions, and the husband or relatives interviewed where possible to determine the circumstances of the death.

3.6 Data analysis

The stage of gestation of mothers seen at antenatal clinic was estimated using the date of birth of infants determined at follow up. This assumes that all infants were term.

4 RESULTS

4.1 Demographic description of the cohort

In all, 287 women pregnant women were recruited: 109 at the cross-sectional study, 65 seen at the survey who subsequently became pregnant, and 113 through the reorganized antenatal clinics in the Kunjingini zone.

This sample of pregnant women, as a percentage of the total number of women of childbearing age living in each clinic zone, represented 12% in the Kaugia zone (116/941), and 11% in the Kunjingini zone (148/1318). The percentage was less (2%, from 23/941) in the Jambitanget clinic zone (denominator data derived from table 4-1). There were slightly more nulliparous women from the Kaugia clinic zone, but this was not significant (chi-squared test). The number of pregnant women from each village is shown in table 5A-1.

Table 5-1 shows the age group of the mother tabulated against parity group, and compares the parity distribution of this group with that of the women seen at the cross-sectional study. The pregnant cohort shows a lower proportion of nulliparae and grand multiparae (7+), and a higher proportion of women of parity 2-3. This probably reflects that young unmarried women, and older, high parity mothers have lower fertility levels than the married women in their 20's.

Table 5-1. **Age and parity description of the cohort of pregnant women (excluding women who miscarried); parity distribution compared with women surveyed in the cross-sectional study**

AGE GROUP (years)	PARITY GROUP					
	0	1	2-3	4-6	7+	ALL
15-	35	8	1	0	0	44
20-	22	36	14	1	0	73
25-	4	9	36	22	0	71
30-	1	4	20	24	7	56
35+	0	1	2	19	18	40
ALL	62	58	73	66	25	284
column percent	21.8	20.4	25.7	23.2	8.8	99
X-sectional study (%)	25.4	15.9	21.4	22.8	14.5	100

4.2 Extent of antenatal follow up visits

Table 5-2 shows antenatal visits made by women during the study by parity group. Only 62/284 (22%) of women were seen on 3 or more occasions at clinic. This was partly because initial recruitment included women in late pregnancy who were likely to deliver after the first or second clinic visit.

Table 5-2. **Number of antenatal visits of pregnant women during the study by parity group (excludes women who miscarried)**

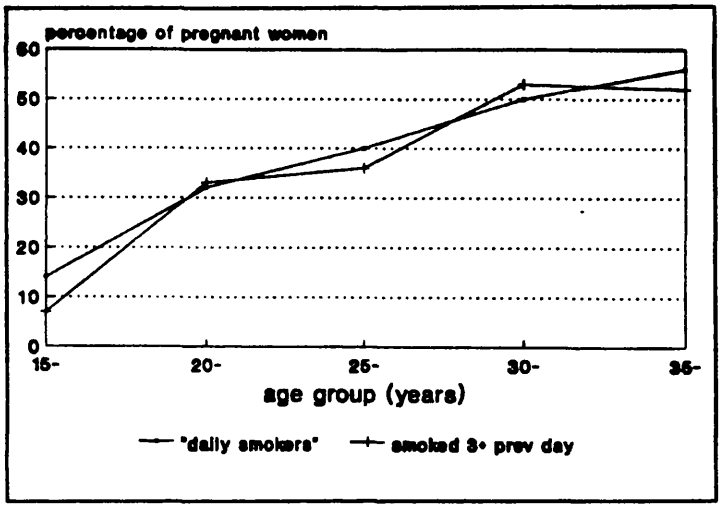
parity group	women	number of antenatal visits 1	2	3	4	5
0	62	30	17	9	4	2
1	58	25	19	8	5	1
2+	164	74	57	17	13	3
all	284	129	93	34	22	6
% all	100	45	33	12	8	2

4.3 Personal habits [2]

Smoking frequency during pregnancy: Sixty nine percent (192/278) of pregnant women reported smoking in pregnancy. Overall 38% of women classified themselves as daily smokers, and 37% of women had smoked in 3 or more periods of the previous day. Figure 5-1 shows this data by age group: smokers show a definite upward trend with age, from 14% in the youngest group to 56% in the eldest classifying themselves as daily smokers. The pattern with age is very similar either way the smoking habit is defined.

[2] Data were missing from antenatal histories for smoking in 9 women, and betel nut consumption in 10. Miscarriages and women recruited postnatally only were excluded from the analysis, leaving 278 smoking and 277 betel nut histories.

Figure 5-1. **Pregnant women: self classified daily smokers, and those who smoked on 3 or more occasions the previous day, by age group**



The percentage of women reporting occasional smoking is similar in all age groups except in the older category, where fewer women report themselves as occasional smokers (table 5A-2). The age effect is so marked that it seems unlikely to be the result of different habits in different cohorts of women.

Table 5-3 shows smoking frequency the previous day classified by reported smoking regularity. Notably 27% of women classifying themselves as occasional smokers had smoked in 2 periods in the previous day, and 24% in 3 or more periods. Of the daily smokers, 77% had smoked during 3 or more periods the previous day. In fact, 10 women reported getting up at night to smoke. The data shows that consumption can be high in women that report themselves as occasional smokers, and women reporting themselves as regular smokers usually smoke in every period of the waking day (morning, afternoon and evening).

Table 5-3. **Times smoked the previous day and reported smoking regularity in pregnant women**

	yesterday smoking frequency (row percent)				
	nil	once	twice	3 plus	total
occasional smoker (n=86)	16	33	27	24	100
daily smoker (n=105)	4	7	12	77	100

In smokers, *brus* was smoked by all. A few women smoked other materials in addition: twist tobacco (5 women), rolling tobacco (5 women), and cigarettes (2 women, both less than 30 years old).

The main smoking method was tobacco rolled in newspaper: 65% (122/188) used newspaper, while 27% (55/188) smoked the tobacco rolled as a cigar ("brus to brus"), and

2% (4/188) used a pipe. Six percent (11/188) reported using several methods as their main method.

Betel nut consumption: 68% (189/277) of pregnant women reported chewing betel nut during pregnancy. Table 5A-3 shows that 14% (38/277) of pregnant women reported chewing daily, and 12% (34/277) had chewed during 3 or more periods the previous day. Women reporting themselves as daily chewers increased with age. This trend was reflected in women chewing 3 or more times in the previous day, except in the 35+ age group, where the percentage chewing dropped to 15% (6/39). It is possible that the gum disease, widespread in older women due to lifetime betel nut chewing, restricts their daily intake.

Table 5A-4 shows consumption history from the previous day tabulated against the regularity of chewing reported. Notably in women classifying themselves as occasional chewers, 49% (74/151) had chewed the previous day; and 63% (24/38) of daily chewers had chewed in 3 or more periods the previous day. Three daily chewers reported getting up at night to chew. These figures demonstrate the high level of betel nut consumption in the Wosera.

Self reported regularity of smoking classified against the same for betel nut is shown in table 5-4. Eighty one percent (86/106) of daily smokers chew betel nut, and 84% (32/38) of daily chewers smoke.

Table 5-4. Reported betel nut consumption by reported smoking in pregnant women

BETEL CONSUMPTION	SMOKING FREQUENCY			
	non-smoker	occasional smoker	daily smoker	ALL
non-chewer	42	26	20	88
occasional chewer	37	55	59	151
daily chewer	6	5	27	38
ALL	85	86	106	277

4.4 Birth history analysis

Data: There were 777 births from the retrospective birth histories, including stillbirths and twins, from 230 parous mothers. These data were used in the estimation of mortality and the analysis of health service use. In the analysis of birth intervals, births of pregnant women from the longitudinal study were added to the data, giving 761 completed intervals.

4.4.1 Birth intervals

When the preceding child survived infancy, the mean birth interval was 35.7 months. If the preceding child survived 2 years the interval was very similar (35.5 months). Health services do not provide any form of family spacing in the Wosera: the relatively long birth interval is probably the results of traditional taboos on sexual intercourse while a child is still breast

feeding. In the event of infant death, these taboos do not apply: hence the correlation between survival of the preceding child and subsequent birth interval as shown in table 5-5.

Table 5-5. Mean birth interval by outcome of preceding pregnancy from birth history analysis

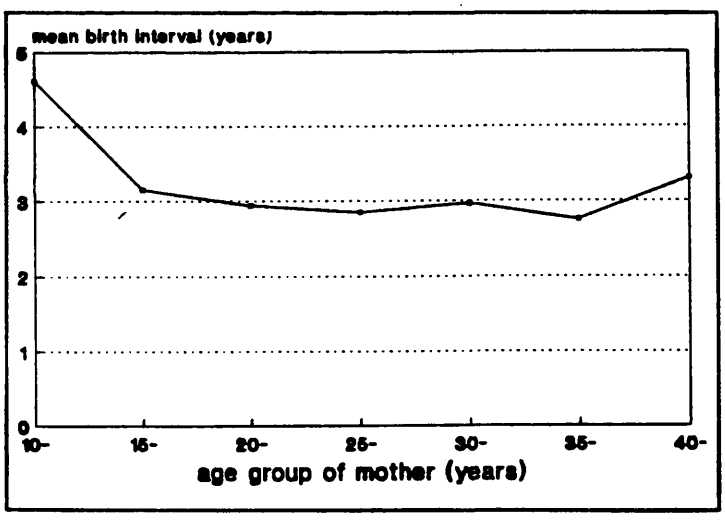
PRECEDING PREGNANCY OUTCOME	BIRTH INTERVAL (YEARS) mean	95% CI	N
stillbirth	1.67	1.22 - 2.11	19
neonatal death	1.91	1.58 - 2.25	77
post-neonatal death	2.22	1.68 - 2.76	41
survived first year	2.98	1.87 - 3.09	624
ALL	2.80	2.69 - 2.90	761

To ensure observer bias in estimation of dates of birth of children had not affected the estimates given in table 5-5, mean intervals were calculated using only intervals where both dates of birth were known to the nearest month. The mean birth intervals were similar, and the trend with outcome was the same (n=430).

The mean birth interval did not vary with interval number (excluding intervals where the preceding child died before completing infancy).

The mean interval by age of mother (at the time of delivery of the first child in the interval) is shown in figure 5-2. The interval before a subsequent pregnancy was relatively long in the 3 women that delivered the first child before the age of 15. Mean interval values remained constant in relation to age of mother in age groups from 15 years.

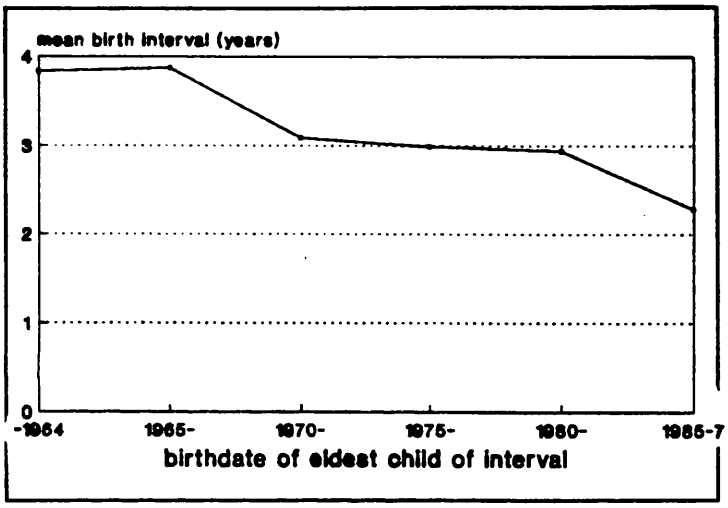
Figure 5-2. Mean birth interval by age of mother at the start of the interval



Mean interval by year of birth of the first child of the interval are shown in figure 5-3 (data in table 5A-5). A preceding infant death is likely to shorten the subsequent interval; because the sample is taken from pregnant women, those with a recent antecedent death are more likely to be included in the sample. To avoid this bias, the data are examined excluding

intervals where the child died before the age of 2. Thus the decline in birth interval seen from 1985 onwards suggests a real decrease in birth interval.

Figure 5-3. **Mean birth interval by year of birth of first child of the interval**



Multivariate analysis: The factors affecting the length of birth intervals were examined by analysis of covariance, using log-transformed interval lengths as the response variable. There was a highly significant effect of outcome on the length of the succeeding birth interval ($F = 8.0$, $df = (4, 734)$, $p = 0.0001$), with the main difference being between birth intervals where the child survived and cases of infant mortality. Quality of the estimate of interval length had a significant effect, ($f = 2.64$, $df = (3, 734)$ $p = 0.048$), which differed depending on the outcome. Age of mother at the beginning of the interval had no significant effect on the length of interval. The trend in length of interval with year of birth of child was not accounted for by confounding with these other variables: birth intervals appeared to be longer in the past, even when effects of quality of data were taken into account ($F_{1,734} = 8.24$ $p = 0.004$). There were no significant effects associated with birth order or the ages of the mothers, once quality and outcome had been allowed for.

4.4.2 Mortality of children

Direct estimates of perinatal, neonatal and infant mortality were calculated from the maternal birth histories. Births in the two years prior to the survey were excluded: because the sample was of pregnant women, those who had experienced the death of a child born within the last two years were more likely to be pregnant again (and therefore included in the sample) than those women with surviving children. This effect would bias the population mortality rate estimates of children born within the last two years.

Infant mortality: The direct estimate of infant mortality was 133/1000. This corresponds to the estimate made at the RRA of 130/1000 (although this data included stillbirths), but is much higher than the country rate of 72/1000 (chapter 3, section 2.1). Table 5-6 shows the rates by date of birth of the child; the lower rate in births 1960-74 may reflect omission in the histories of infants dying many years previously. Mortality from 1975 onwards does not

suggest a dramatic improvement in recent years.

Table 5-6. Mortality rates from retrospective birth histories of pregnant women, excluding births in 1985-86

date of birth of child	live births (N)	MORTALITY RATES (/1000) perinatal[a]	neonatal	post-neonatal	infant[b]
1960-74	125	79	88	32	120
1975-79	185	63	76	59	135
1980-84	350	89	94	43	137
ALL	660	80	88	45	133

[a] stillbirths and deaths in the first week of life per 1000 births
[b] neonatal, post-neonatal and infant rates per 1000 live births

Perinatal mortality: The perinatal mortality rate was 80/1000; the neonatal rate was 88/1000; the post-neonatal rate was 45/1000. Perinatal mortality rates were similar to those found at the cross-sectional survey, where the direct estimate in women aged 15-29 years was 84/1000, and this is to be expected as the pregnant cohort contained 174 women surveyed in the cross-sectional study. Of the neonatal deaths, 57/1000 occurred in the first week, and 31/1000 in the subsequent period. Bias caused by systematic misclassification of late neonatal deaths as early neonatal (or from post-neonatal into neonatal) is possible, but is unlikely to account entirely for these findings. In conclusion, the data show the infant mortality rate was high, and confirm previous evidence that many deaths were concentrated in the neonatal period.

4.4.3 Use of obstetric services

Health centre or hospital deliveries accounted for 6.7% (52/772) of births occurring prior to 1987; 93.3% (720/772) delivered in the village (place of birth data was missing in 5 of the 777 births). Women pregnant while away on plantations in the New Guinea Islands tended to deliver at health facilities while away, but not if they returned home.

Of the 230 parous women, 9.6% (22) delivered once in hospital, 2% (5) delivered twice, 2% delivered 3 times and one woman delivered 5 times in hospital. Thus only 14.5% (33/230) parous Wosera women have ever delivered in a health centre or hospital. Of the 52 deliveries occurring in hospital, 25 were in women that finally had more than one delivery in a health facility.

Obstetric complications reported by the mother were probably incomplete. The placenta was retained at 13 deliveries, and a further 3 there was a history of postpartum haemorrhage. Incidence of life threatening complications can thus be estimated from this data at 20 per 1000 births (16/772).

4.5 Prior antenatal clinic attendance

Table 5A-6 shows the stage of gestation when first seen as part of this study, and whether those women had already been seen at MCH antenatal clinics. Only 9% (7/77) of women seen already were less than 20 weeks and 28% (25/77) were less than 30 weeks, suggesting that those that do attend antenatal clinic in the absence of a special study do so late in pregnancy.

Information on preceding MCH drug dispensing was available from 74/77 women already seen at antenatal clinic. Seventy three percent (54/74) had received at least one single dose of chloroquine at MCH antenatal clinic and 49 of these women were from the Kunjingini zone. Twenty seven percent (20/74) had been supplied with prophylaxis (8 from Kunjingini clinic and 10 from Kaugia clinic).

Of the 20 women receiving prophylaxis, 4 reported poor compliance, and 6 reported taking it but were taking an erroneous regimen. Only 9 women seemed to understand the weekly regimen and reported good compliance. Thus out of 74 women seen at standard antenatal clinic, a maximum of 12% (9/74) could possibly be taking malaria prophylaxis as outlined in the standard treatment schedule. Even in these 9, compliance was unknown.

4.6 Morbidity and past medical history

Eight women reported fever in the preceding week. When asked specifically if they had been febrile in the previous 7 days, a further 4 reported a recent febrile episodes, possibly reflecting that mild symptoms from malaria are often not regarded as an illness in adults living in a highly endemic area. The incidence of possible malaria episodes was 43 per 1000 women per week (12/278), or 2.2 episodes per woman per year. A further 3 women reported a fever in association with some illness, such as a cough or an abscess.

From the total of 15 with a history of fever, information on whether they received treatment was recorded in 13. Nine went without any treatment. One attended an aid post and 2 a health subcentre, each receiving a single dose of 3 tablets of chloroquine; one woman treated herself with 2 tablets of chloroquine. These data illustrate that clinical malaria episodes were often not treated.

Only 2 women gave a history of tuberculosis. Neither women showed evidence of active disease. Although the disease is common in particular areas of the East and West Sepik (Garner and Giddings 1986), this suggests that the Wosera is relatively spared of the disease.

Ten women had previously received a blood transfusion or intravenous iron dextran. No woman had a history of caesarean section and this was confirmed by abdominal examination.

4.7 Blood pressure and pre-eclampsia

Table 5-7 shows mean blood pressure of pregnant women at first visit by stage of gestation at that time. From this cross-sectional data, gestation does not correlate with mean blood pressure.

The mean blood pressure of the 281 pregnant women at first visit was 108.2 / 67.4 mm Hg. Hypertension was rare: the number of women with diastolic pressures above 90 mm Hg was 3, and with systolic pressures above 135 mm Hg was 2. One woman showed persistent proteinuria, oedema and hypertension; she was also anaemic, and this did not respond to standard management, and she may have had nephrotic syndrome. In summary, pre-eclampsia was not a common condition is this cohort of pregnant women, and it is therefore unlikely to be a major contributor to intra-uterine growth retardation in this population.

Table 5-7. Mean blood pressure of pregnant women * at first visit by gestation

gestation (weeks)	n	----blood pressure (mm Hg)------- SYSTOLIC mean	SE	DIASTOLIC mean	SE
0-	69	110	1.3	67	1.1
20-	117	107	0.9	66	0.8
30-term	93	108	1.0	69	0.9

* 2 women excluded (migrated out:delivery date unknown)
SE= standard error

Table 5-8 shows mean blood pressure at first visit of all women who had a live birth. Mean systolic blood pressure tended to decline with age, while the diastolic pressure remained static. These results are consistent with findings from other, non-pregnant PNG populations (Vines 1970).

Table 5-8. Mean blood pressure of pregnant women at first visit by age group

AGE GROUP (years)	n	SYSTOLIC mean	std	DIASTOLIC mean	std
15-19	42	111	9.5	66	11.8
20-24	73	109	9.6	68	8.4
25-29	70	108	9.6	67	8.9
30+	96	106	10.2	68	8.8
ALL	281	108	9.9	67	9.2

4.8 Nutrition

4.8.1 status at first visit

Table 5-9 shows the mean values of maternal nutritional measures at first visit by their stage of gestation. Weight and body mass index was greater in women whose pregnancy was more advanced. Triceps skinfolds were greater with advancing pregnancy but lower in the 30 weeks plus group, a phenomenon seen in other pregnant groups (Taggart et al. 1967). Subscapular measurements were higher in women at later pregnancy. The low overall values reflect the low reserves of fat in these women, also observed in non-pregnant women at the cross-sectional study.

Table 5-9. Nutritional status mean values of pregnant women at first visit compared with non-pregnant women

PREGNANCY STATUS (weeks)	N	mean weight	body mass index	skinfolds: triceps	skinfolds: subscapular
non-pregnant	834	47.4	20.6	9.8	13.1
0-	69	49.3	21.3	9.7	13.8
20-	117	50.7	21.8	9.6	14.9
30-term	93	52.9	22.8	9.0	15.2

non-pregnant excludes women later who became pregnant (65)

4.8.2 Presence of ketonuria

Maternal ketonuria has been associated with starvation, poor antenatal weight gain and perinatal mortality (Naeye 1983). Urine samples collected at antenatal clinics showed the prevalence of ketonuria was 2.6% (9/341). In all but 2 of these cases the amount was 'trace'. Thus ketonuria reflecting moderate acidosis associated with starvation was uncommon in this cohort.

4.8.3 Changes in nutritional status

Weight gain during pregnancy was estimated in two ways. First, by fitting a straight line by least squares through all weight measurements made during the last 6 months of pregnancy, providing the period between the first and the last such measurement exceeded 4 weeks. A separate line was fitted for each woman, and the gradient of the line used as an estimate of rate of weight gain in the last 6 months of pregnancy. [3] In the 99 women on whom there was data to fit a regression, mean weight gain was 0.202 kg/week (95% CI 0.169 - 0.235), giving an estimate of the overall weight gain of 4.8 kg (24 weeks*0.202), or 4.8 kg per pregnancy. If it is assumed that weight gain prior to 16 weeks is maximum of 10% of total weight gain, then the estimated maximum overall weight gain is 5.3 kg.

[3] Other studies on nutritional status during pregnancy in developing countries have made assumed that weight gain during the second and third trimesters to be approximately linear with time, and the gain in the first trimester to be less than 1 kilogram (Gueri et al. 1982)

A second estimate of weight gain was made by selecting only those women seen prior to the 14th week of pregnancy, and who were reweighed within 4 weeks prior to delivery (28 women). The minimum gain was -1.1 kg, and the maximum was 14.8 kg, the latter to a woman with twins. Figure 5-4 shows the distribution of weight gain in women with singleton pregnancies. Their mean gain was 4.83 kg (95% CI 0.3.92 - 5.73: n=27), and the median 5.0 kg.

Figure 5-4. **Weight gain in women with singleton pregnancies seen prior to 14 weeks gestation and reweighed within 4 weeks of delivery**

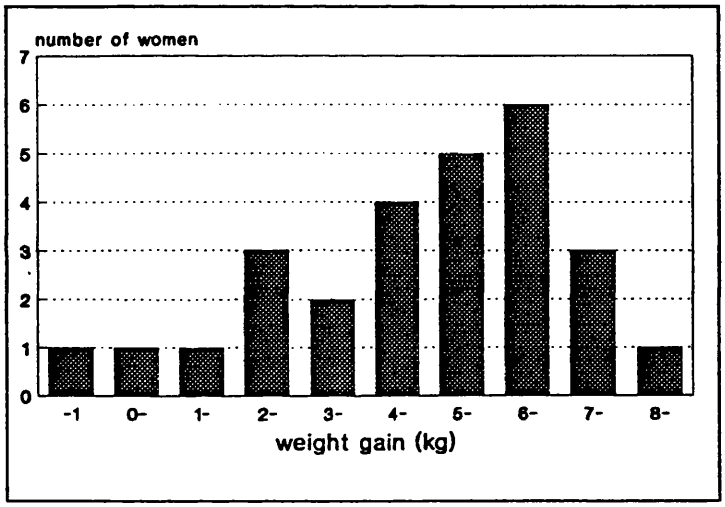
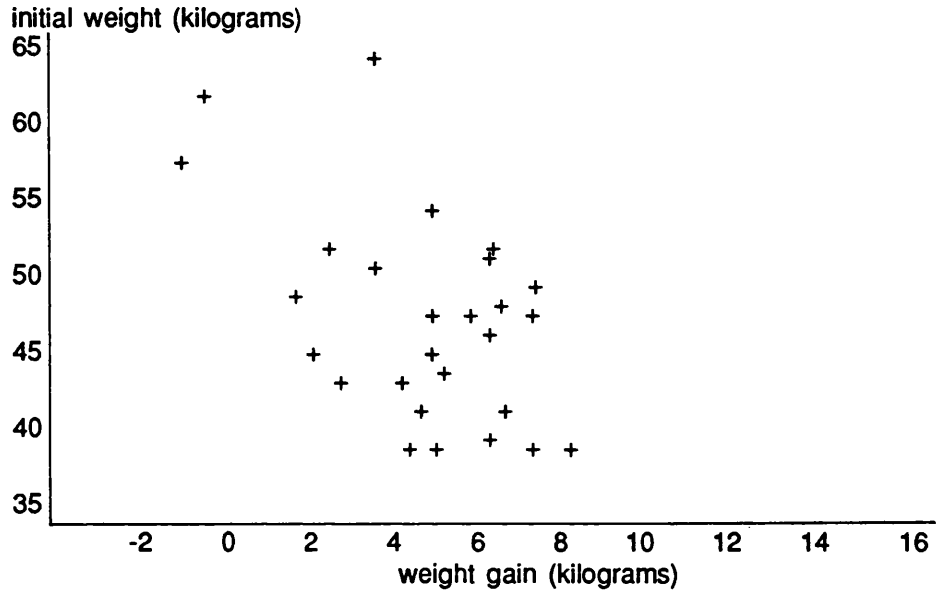


Figure 5-5 shows weight gain plotted against initial weight in these 27 women. It shows that the lighter women tend to gain more weight. Linear regression showed a significant negative correlation between initial weight and weight gain per week. (initial weight= 54.5 -1.48*weight gain: f=9.236, df (1,25), t=0.0055).

Figure 5-5. **Initial weight and weight gain in pregnant women seen in the first 14 weeks of pregnancy and reweighed within 4 weeks of delivery n=27)**



4.8.4 Postnatal weight change

Postnatal weight at 6 months was collected from the first 45 women who delivered. Weight change between the immediate postnatal period and six months postpartum varied from -8.2 kg to +7.5 kg. The first quartile (25%) was 2.5 kg and the third quartile (75%) was -0.2 kg. The mean weight change was -1.39 kg (std 2.58). This decline in weight from the postnatal value was significant (t=3.61, p=0.0008).

4.9 Malaria

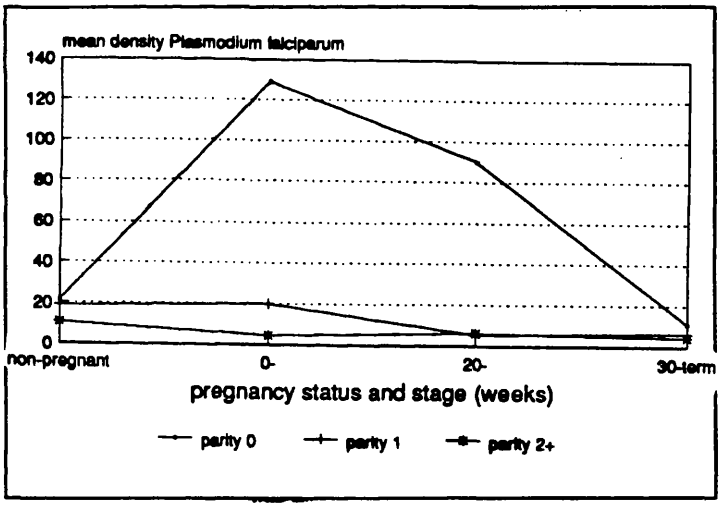
4.9.1 status at first visit

Table 5-10 shows mean parasite density, splenic enlargement, blood chloroquine, haemoglobin and FEP values of women by parity group, pregnancy status and, if pregnant, gestation. The relationship between *Plasmodium falciparum* density and stage of gestation in nulliparous pregnant women is striking: it peaks in the early weeks (0-19 weeks), and then declines to levels similar to non-pregnant women (figure 5-6). This pattern in pregnant women living in endemic malarious areas is seen elsewhere (Brabin 1983). Pregnancy and stage of gestation showed little affect on *plasmodium falciparum* density in parous women.

Table 5-10. Mean malaria parasite densities, size of spleen, chloroquine concentration, haemoglobin and FEP by parity of mother, pregnancy status and stage of pregnancy

PARITY GROUP	pregnancy status + weeks gestation	n	mean values pf	pv	pm	spleen (cm)	chloro-quine	hb	fep	fep (n)
0	non-preg	220	21.3	1.7	2.5	1.90	25.3	10.3	4.1	81
	0-	18	128.9	1.2	1.2	3.56	26.9	8.6	3.8	11
	20-	24	90.1	1.2	2.0	4.13	70.0	8.3	4.5	18
	30-term	17	11.1	1.2	1.5	1.71	54.5	8.6	5.5	9
1	non-preg	128	18.6	1.6	2.9	2.32	30.0	10.0	3.9	38
	0-	14	20.0	1.2	1.4	3.86	46.1	9.3	3.0	7
	20-	25	5.4	1.0	1.4	3.56	51.8	8.3	4.0	18
	30-term	18	6.4	1.1	1.3	1.22	75.0	8.6	4.8	14
2+	non-preg	486	10.6	1.5	2.5	2.10	26.2	10.0	3.9	150
	0-	37	4.6	1.2	1.6	1.88	54.3	9.2	2.9	19
	20-	68	6.2	1.1	1.9	2.09	64.9	8.8	3.5	55
	30-term	58	4.4	1.6	1.1	1.55	47.3	9.1	3.9	41
all	non-preg	834	13.9	1.6	2.6	2.08	26.5	10.10	4.0	269
	0-	69	14.7	1.2	1.5	2.73	43.9	9.07	3.2	37
	20-	117	10.4	1.1	1.8	2.82	62.9	8.64	3.8	91
	30-term	93	5.63	1.42	1.2	1.52	53.0	8.96	4.3	64

Figure 5-6. Mean density of *Plasmodium falciparum* species by pregnancy status and parity group



Mean density of *Plasmodium vivax* and *Plasmodium malariae* were marginally higher in non-pregnant compared with pregnant women whatever the parity, and similarly the mean levels of chloroquine were higher in pregnant women than they were in non-pregnant women. One explanation for this finding is that pregnant women received chloroquine from MCH clinics, which was sufficient to eliminate the parasitaemia due to these species. However, table 5A-7 suggests this was not so: mean chloroquine levels varied little between those previously seen and those not previously seen at clinic, (except the parity 1 group); and mean species parasite densities of Plasmodium vivax and *Plasmodium malariae* did not show an obvious difference between women previously seen and those not previously seen at antenatal clinic.

Plasmodium vivax and *Plasmodium malariae* mean parasite density did not show any relationship to parity or gestational age (table 5-10). Mean spleen size tended to peak in the 20-29 week group in all parities. Chloroquine levels were higher at all stages of gestation in all parity groups compared with non-pregnant women, except nulliparous women in early

pregnancy.

4.9.2 Anaemia and iron deficiency

Haemoglobin levels were lower in pregnant women compared with non-pregnant women. Overall the difference between women in late pregnancy and non-pregnant women was 1.46 g/dl. The difference was most marked in nulliparous women in late pregnancy (haemoglobin 1.93 g/dl less than in non-pregnant nulliparous women: table 5-10).

Table 5-11 shows the number of women by parity group by different haemoglobin categories. The percentage of women with haemoglobin values of less than 8 g/dl at first visit was higher in nulliparous women (43%) compared to women of parity 1 (23%), and women of parity 2 and above (14%). This is mirrored in the women with haemoglobin values greater than 10 g/dl: only 6% of nulliparous women had haemoglobin values in this category. The increased risk of low haemoglobin in nulliparous women probably reflects the higher susceptibility of this group to malaria.

Table 5-11. Haemoglobin groups at first visit of pregnant women by parity group, with column percent ()

first visit haemoglobin	parity group 0	1	2+	ALL
<8	23 (43)	13 (23)	23 (14)	59
8-	31 (51)	33 (59)	105 (64)	169
10-	7 (6)	10 (18)	35 (21)	52
ALL	61 (100)	56 (100)	163 (99)	280

Although the numbers of free erythrocyte protoporphyrin determinations were less, there was a slight but consistent increase in mean levels with stage of gestation within parity groups, suggesting that iron deficiency may increase as pregnancy progresses (table 5-10).

4.9.3 Mosquito net use

At later village visits (for postnatal infant follow up), women were asked if they had slept under a mosquito net the previous night. Twenty five percent (27/107) of women reported sleeping under a mosquito net. This is a low rate of use, but slightly higher than the rate of 10% determined previously in households (survey conducted in 1984: Dr Timothy Brown, personal communication).

4.9.4 Evaluation of the measures of prophylaxis compliance

Pilot study: Dill-Glazko urine positivity after a single dose of 2 chloroquine tablets was tested in a small pilot study of 1 staff member (pregnant), 1 staff member (non-pregnant) and a mission station resident (pregnant). Each was given a prophylactic dose of chloroquine and urine collected daily for 4 days. In all 3, the Dill-Glazko test remained positive (+++) for the 2 days following the dose. On the third day (72 hours after the dose) the positivity dropped to ++ in the pregnant women and remained at +++ in the non-pregnant

woman. By the fourth and fifth day, the results were +, which was not regarded as a positive test. Although the numbers are small, this indicates a higher degree of positivity than the original report of the test, where positivity persisted after a single dose of 5 mg/kg chloroquine base for 24 hours in 100% of patients, and 63% for up to 48 hours (Lelijveld and Kortmann 1970).

Maternal drug history: Women were asked the day they last took a prophylactic dose of chloroquine. Although the question was asked in a neutral form to obtain an accurate answer, the correlation between the mother's answer and the urine test for 4-aminoquinoline metabolites was poor (table 5-12). All women that reported having taken the drug within the last day should have a positive Dill-Glazko test, but in 24% the test was negative.
This suggests that drug history reporting is not reliable, and confirms the impression of field staff that the answer to the question was strongly influenced by what the women gauged the questioners wanted to hear.

The table also shows a high urine positivity rate in women that appeared not to be taking prophylaxis. In 2 of the 13 women, the urine test was strongly positive (+++) for 4-aminoquinoline metabolites, suggesting that either the women's history was unreliable, or the test was showing false positive results.

Table 5-12. Comparison of reported time since prophylactic chloroquine dose taken and Dill-Glazko test postivity

	|---days since chloroquine taken--|				|-- not --|
	1	2	3-4	5-21	taking
number of samples	30	13	48	35	13
percent positive	76	69	71	65	38

Chloroquine ELISA results: The mean chloroquine ELISA values for various levels of positivity of the Dill-Glazko test are shown in table 5-13, indicating that mean levels of chloroquine were associated with strongly positive urine Dil-Glazko tests. Dill-Glazko positive (++) urine tests were not associated with mean blood chloroquine ELISA greater than the mean level for women with urine samples negative for Dill-Glazko. This may reflect that blood chloroquine peaks rapidly before distributing into body tissues, and active urine excretion means chloroquine metabolites are found in the urine for up to 6 weeks after a single dose.

Table 5-13. Mean blood chloroquine levels as measured by ELISA measured against urine test for 4-aminoquinoline positivity

----------DILL-GLAZKO---------- COLOUR	POSITIVITY	CHLOROQUINE ELISA (geometric mean) ng/l	N
no change	NEG	86.2	30
+	NEG	74.1	37
++	POS	88.6	40
+++	POS	206.8	47
++++	POS	336.9	19

+ : slight pinkish coloration, but the change is faint or inconstant
++: definite coloration

The mean blood chloroquine concentration by ELISA in the presence of a negative urine test was 79.4 ng/l; in the presence of a positive test it was 164 ng/l. If 100 ng/l is used as a cut-off of positivity in the ELISA test, then of the 98 women with negative urine tests, 28% were positive on ELISA; and of the 135 with positive urine tests, 57% were positive on ELISA. Thus ELISA chloroquine estimations seem to correlate poorly with the urine Dill-Glazko test. Further work validating the blood chloroquine ELISA against blood levels determined by conventional methods in adults living in malarious areas is required.

Tablet counting: Tablets dispensed at clinic were recorded, and the number taken checked at subsequent clinic visits. The data correlated poorly with urine tests, chloroquine ELISA values and drug history taking. Some women returned completely empty bottles but did not appear to be taking their prophylaxis, and actually declared that this was so. Analysis of tablet counting as a proxy for consumption was therefore not undertaken.

In summary, tools for measuring compliance with weekly chloroquine prophylaxis were limited.

4.9.5 Evaluation of compliance

Despite the limitations of the various compliance measures, effectiveness of the drug prophylaxis programme was examined in several ways: first, urine positivity for 4-aminoquinoline metabolites at clinic visits; second, the relationship between positivity and blood parasitaemia; and finally, by interviews with pregnant women examining attitudes to the programme.

Urine positivity: Table 5-14 shows the number of times a woman had a positive Dill-Glazko urine test by the number of times she was seen at follow up antenatal clinic. Of the women only seen once at follow-up during the study, 54% (47/86) had positive urine tests. Of those seen twice, 41% (14/34) had positive urine tests on both occasions. In women seen 3 or more times, only 20% (5/25) had positive tests each time seen. Compliance in nulliparous

women was slightly better, but the numbers are small. Of the 12 nulliparous women seen on 2 or more occasions, 66% (8/12) were positive each time. These results may be encouraging, as the urine tests were taken at clinics occurring 1-4 days after the day women were instructed to take their prophylactic dose (Sunday), so some tests could be negative even if the women had taken the weekly dose.

Table 5-14. Number of times Dill-Glazko test positive by number of times seen at follow up clinic (excluding initial visit)

	TIMES POSITIVE				
TIMES SEEN	0	1	2	3	4
1	39	47			
2	7	13	14		
3	1	7	9	4	
4	0	1	1	1	1

Effect on parasite rates: Analysis of urine test positivity on parasite rates did not show a strong relationship. In the 98 with a negative urine test, the *Plasmodium falciparum* species parasite rate was 47%; in those with a positive urine test, it was 39%.

However, if the geometric mean parasite density is examined by urine test positivity and parity, it appears that positive urine tests were associated with a lower density of parasitaemia but only in nulliparous women with respect to *Plasmodium falciparum* species (table 5-15). To test whether gestation is confounding the apparent association between parasite density and urine positivity (women in late pregnancy may be more likely to have been seen at clinic and taken chloroquine when malarial parasitaemia is lower anyway), tests done after 20 weeks were examined by 10 week bands and parity. The association between positive urine tests and lower *Plasmodium falciparum* density is maintained (table 5A-8).

Table 5-15. Mean parasite density by urine positivity by Dill-Glazko test in pregnant women

	DILL-GLAZKO TEST					
MATERNAL PARITY	negative			positive		
	fal	viv	mal	fal	viv	mal
parity 0	19.4	1.7	1.2	4.4	1.2	0
parity 1+	3.0	1.1	1.1	2.7	1.1	1.1

fal=P. falciparum; viv= P. vivax; mal= P. malariae

Attitudes to the prophylaxis programme: After the first set of follow up clinics in the Kaugia zone, staff gained the impression that chloroquine prophylaxis compliance was low. Informal village interviews were conducted over the following week. One field officer (MEB) saw 31 women and assessed that consumption was irregular in at least 11, and 5 not taking them at all. Table 5-16 summarises some of comments made by the mothers in relation to the chloroquine prophylaxis.

Table 5-16. Some of the problems encountered while taking prophylactic chloroquine remarked on by mothers

PROBLEMS	MOTHER'S COMMENTS
side effects	dizziness itchy weakness of whole body
problems taking them	nasty taste chokes on the tablets
other fates	simply dislikes them prefers the yellow ones (folate) saving them until sick takes them when her husband tells her to throws them away her child emptied the bottle

4.10 Outcome of pregnancy

All pregnant women were followed up. One woman was untraceable: the village could simply not identify her. Table 5-17 summarizes the outcome of pregnancy, and mortality rates. The small denominator means the rates are likely to be affected by random variation, but are included to give an approximate indication of their magnitude.

Table 5-17. Outcome of pregnant cohort (N=287)

OUTCOME	N	ESTIMATE OF RATE	
miscarriages	3		
maternal deaths	3	maternal mortality rate	(N= 3): 10/1000
stillbirths	6	perinatal mortality rate	(n=13): 45/1000
neonatal deaths in singleton births	10	neonatal mortality rate	(n=13): 45/1000
neonatal deaths in twin births	3	post-neonatal mortality rate	(n= 8): 28/1000
post-neonatal deaths	8		
twins	5 sets	twinning rate:	1 in 57 deliveries

* 2 of these stillbirths were also maternal deaths

4.11 Maternal mortality

The follow up of the 287 women revealed 3 maternal deaths, reflecting the high maternal mortality seen in many parts of rural PNG (National Health Plan 1986). Wosera maternal deaths were also identified by the sister question of the cross-sectional survey (chapter 4, section 2.2), and by informal village discussions (see methods in this chapter). Details of deaths identified as maternal obtained from the husband or relatives is outlined in table 5-18. It is not known what proportion these deaths represent of all the maternal deaths in the Wosera, or whether there was some selection bias in deaths discovered by informal village discussions. Nevertheless, causes of maternal mortality can be examined in this aggregated data. Six out of the 14 deaths were related to retained placentae or postpartum haemorrhage.

Table 5-18. Circumstances of maternal death in Wosera women

SOURCE	YEAR	PARITY	TIME	CIRCUMSTANCES	POSSIBLE DIAGNOSIS
cross-sectional study	'79	2	in labour	2 days in labour	obstructed labour
	'83	0	postnatally	retained placenta (4 hrs) died just after removal	PPH
informal village visits	'80	4	postnatally	normal delivery; ill and died 1 week later	puerp. sepsis
	'78	2	in labour	difficult labour (15 hrs); died 20 mins. after placenta delivered	PPH
	'84	7	antenatally	oedema and weak	anaemia
	'85	0	postnatally	retained placenta; died after 4 hrs	PPH
	'87	6	antenatally	died 10-30 weeks; sudden illness, severe headache	?meningitis
	'82	9	in labour	not known	?
	'81	6	postnatally	retained placenta; died 1 hour after delivery	PPH
	'85	4	postnatally	antenatal anaemia; died just after placenta delivered	?PPH
	'85	6	postnatally	had operation ?LSCS for delivery died 2 weeks later	?puerpural sepsis
pregnancy study	'87	0	postnatally	gross malnutrition and anaemia died 4 days after stillbirth	?infection ?anaemia
	'88	2	postnatally	APH; retained placenta; admitted; died 18 hours later	APH/PPH
	'87	4	postnatally	stillbirth; admitted with puerpural sepsis; died after antibiotics for 9 days	?retained products

5 DISCUSSION

Mortality: Retrospective infant mortality analysis confirmed high mortality, particularly in the perinatal period, with little evidence of secular improvement. The prospective study of the pregnancy cohort showed a lower perinatal and infant mortality, which can be anticipated from the non-specific effect of the research team working in the area: nevertheless, it is salutary to note that despite health services, the PNGIMR team and a special emphasis on maternal and neonatal care, there were still as many maternal and neonatal deaths.

The follow-up study does confirm that deaths in the neonatal period predominate over those in the post-neonatal period.

Pregnancy: The higher risk of nulliparous women in relation to malaria and anaemia in pregnancy was confirmed, and it was in this group only that an impact of chloroquine prophylaxis had a demonstrable effect on parasitaemia due to *Plasmodium falciparum*.

Weight gain during pregnancy was low, at around 5 kg, or about 11% of the average Wosera woman's weight. This study was not large or long enough to take into account the possible effects of seasonal alterations in food supply. However, these effects are not as marked in the Wosera as in other countries, and the diversity of the agricultural system means there is a variety of foods available at different times of the year (Ross 1984). Seasonal variation in food supply may reflect in different mean birthweight values by month, but this was not found to be the case when data from Maprik hospital were analyzed (chapter 9). Given the weight gain estimate is valid, it means that nutritional fat reserves at

the end of pregnancy are likely to be minimal, and lactation is likely to further deplete maternal nutrition. [4] Indeed, the 6 month postnatal follow up showed declining maternal weight. In view of the known lactational demands on maternal nutritional reserves in the second half of the post-neonatal period, and the prolonged breast feeding and late introduction of solids, it is likely that this weight loss would continue. This data suggest that the relationship between parity and nutritional status shown in the cross-sectional survey may be causal: childbearing impairs maternal nutritional status.

Smoking in pregnancy was widespread. The effect of traditional tobacco on the fetus is unknown, but it is likely to be associated with impaired fetal growth (chapter 2). Health awareness programmes would be useful, particularly aimed at young women.

Antenatal clinics: The study was too short to evaluate the sustainability and effect of antenatal clinics delivered through the health services. However, it was evident the process of properly conducted antenatal care required well trained staff and time, with each consultation requiring 5-15 minutes.

Prophylaxis compliance: The study was hampered by poorly validated methods of evaluating compliance. Nevertheless, it seems that compliance with weekly chloroquine prophylaxis was poor, despite the effort put into explaining it. The overall picture suggests that prophylaxis compliance nationally is likely to be very difficult to implement through health services in many rural areas. Adult Wosera people dislike taking medicine. Preventing illness with drugs associated with curing illness is conceptually difficult to understand, and the potential influence of the tablets on the pregnancy is viewed with concern. Some women are concerned that the tablets could cause twins. Other women, frightened of dying from obstructed labour, are unlikely to want to take drugs that increase the size of their baby.

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[4] Using the components of weight gain given by Llewellyn-Jones 1986 scaled down by 0.75 (average Wosera birthweight of 2.5 kg /average Western birthweight of 3.3 kg) estimates of component weight gains can be made. If the fetus accounts for 2500 g, the placenta and liquor 900 g, the uterus enlarges by 650 g and the breasts by 300 g this total is 4350 g. This leaves the weight gain from fat deposition, increase in blood volume and extra-cellular volume expansion at 650 g in Wosera women.

6 SUMMARY

In pregnant women living in the Wosera:

A. regular smoking in pregnancy was widespread, especially in older women;

B. the average birth interval was about 3 years when the previous child survives, although this may be becoming shorter;

C. self reported febrile illness was common, and usually went untreated;

D. hypertension and pre-eclampsia were rare;

E. weight gain during pregnancy was low;

F. mean *Plasmodium falciparum* density was high and mean haemoglobin levels reduced in pregnant nulliparous women in early pregnancy. Chloroquine prophylaxis reduced *Plasmodium falciparum* parasitaemia density. An effect of pregnancy on malaria infection was not demonstrated in pregnant multigravida;

G. compliance with a simplified prophylaxis regimen of the national standard management schedule was erratic even when administered through this special project.

chapter 6

CHARACTERISTICS OF WOSERA NEWBORNS

1 AIMS

The aims of this study were to:

i. determine the birthweight, gestational age and body proportions of Wosera newborns;

ii. examine birthweight and gestation in relation to maternal factors associated with low birthweight in other populations.

2 METHODS

2.1 Study population

The target population was all live infants born from April - December 1988 born to women seen at the cross-sectional study.Two villages seen at the cross-sectional survey had to be excluded from antenatal follow up and the postnatal study. [1] To compensate for this loss, births from Tuwaikum village (not covered in the cross-sectional study) were included in addition.

Initially it was hoped to evaluate the antenatal programme by including infants born in villages not covered by the antenatal clinics forming a control group. However, this was abandoned after 7 women had been recruited into this group for three reasons: bias in birth reporting were apparent (the team were more likely to be informed if a baby was ill); reporting of births in villages where the team did not have a high profile was unlikely; and the detailed postnatal visit was not easily conducted with people that the team did not know personally. The seven births were excluded from the analysis of infant characteristics (3.1 and 3.3), but included in the analysis of village delivery details (3.2) and the umbilical cord study (chapter 7).

2.2 Delivery reporting

Mothers were asked to inform the PNGIMR team as soon as they delivered. The husband or male relative usually did this. Lists of pregnant women were maintained on microcomputer. An estimated delivery date was calculated using symphysis-fundus height measured at their first visit. Updated lists were used by staff to check with villagers whether deliveries had occurred.

1

Jipako, because the road became impassable; and Jambitanget, because of its distance, lack of health services and local village unrest

2.3 Postnatal visit

Two or more PNGIMR staff visited the mother at home to examine both baby and mother. Measurements taken are listed in Table 6-1.

Table 6-1. Newborn and maternal measurements at the postnatal visit

NEWBORN	MOTHER
general examination	general examination
Dubowitz assessment	palpation of uterine fundus
umbilical cord length	spleen size
axillary temperature	axillary temperature
head circumference	blood pressure
weight	weight
crown-heel length	mid upper arm circumference
crown-rump length	triceps and subscapular skinfolds
haemoglobin	haemoglobin
FEP	FEP, PCV
malaria parasitaemia	malarial parasitaemia
	blood for chloroquine ELISA

Baby examination: Infants were weighed using a single standard spring scale (CMS weighing equipment, England). Scale calibration was checked monthly. Crown heel and crown rump lengths were measured using a standard length board. An *inser-tape* (Ross, Canada) was used to measure head circumference. Gestational age assessment using the Dubowitz method was carried out outside the hut in good light. The length and condition of the umbilical cord was documented. Rectal temperature, using a digital thermometer (Toshiba or Terumo, Japan) were taken initially. Despite careful explanation the procedure caused some anxiety and latterly the axillary site was adopted.

Haemoglobin values and malaria parasites were measured from a heel capillary blood sample (*Autolet*, Owen Munford, UK). Haemoglobin was measured in the field ("Haemocue" haemoglobin photometer: Aktiebolaget, Sweden). If possible, blood was also taken in EDTA (microtainer: Becton Dickinson, USA) for free erythrocyte protoporphyrin measurement (Hematofluorometer model 250: Aviv Diagnostics, USA).

One of two staff performed the baby examination (PG or MEB). Every month the 2 researchers conducted several neonatal examinations together to maintain consistency in measurement. The second team member (DL) was an experienced interviewer, and also trained in neonatal anthropometry and gestational age scoring.

Maternal examination: parameters measured are listed in table 6-1. Instruments used were the same as used in the cross-sectional and longitudinal pregnancy studies.

In view of the possibility that viruses or malaria were implicated in the preterm or low birthweight infants (chapter 1), paired maternal sera for viral and malarial antibody titre

estimation were taken: one sample antenatally, and a second at the postnatal visit. Blood was also used to measure malaria parasitaemia, haemoglobin, PCV, FEP and blood chloroquine (by ELISA).

Maternal Interview: The questionnaire is reproduced in appendix 4. The interview was conducted in privacy, usually inside the house. Mothers were asked:

1. the number of times she had attended antenatal clinic;
2. reported regularity of compliance with prophylaxis, by asking about the regimen, and taking a drug ingestion history over the previous weeks;
3. illness episodes in pregnancy (systematic inquiry using 9 symptoms);
4. illness in the week and day prior to onset of labour;
5. work in the week and the day preceding labour, which was then classified as **resting** (no work); **light work** (carrying water, collecting greens from the garden, climbing "tulip" (*Gnetum gnemon*) trees or weeding); **heavy work** (cutting firewood, clearing the jungle, pulping sago, washing sago, or carrying yams);
6. the most senior attendant at delivery;
7. the time labour commenced. If mothers were unsure, the time of onset of painful contractions was used;
8. the time the membranes broke;
9. the date and time of delivery: the time was determined during the day by using the position of the sun; and at night, by using the traditional method (the number of times the cockerels had crowed -roughly equivalent to the time in hours);
10. labour difficulties;
11. the length of the third stage. The placenta usually delivered soon after the infant, or after a short delay ("istap liklik") which meant 15-30 minutes after delivery. A prolonged third stage was defined as longer than 30 minutes. Delay reported by the mother was corroborated against events occurring at delivery. For example, if the infant had delivered, and an attendant had then left the house and walked to a river 1 km away and back again before the placenta had delivered, then the length of the third stage was estimated at 45-55 minutes.

The same routine was used for women in the cohort that delivered in Maprik hospital.

2.4 Definitions

Birthweight was measured to the nearest 100 g. LBW is defined as those weighing less than 2500 g, in other words, their recorded weight was 2.4 kg or less.

Gestation at birth was calculated from the Dubowitz score, using the standard regression equation (gestation=(0.2642*score) + 24.595; Dubowitz and Dubowitz 1977). Infants less than 37 weeks were classified as preterm, and those 37 weeks onwards were regarded as term, corresponding to the World Health 1975 classification (Puffer and Serrano 1987). Histories from the mother or relatives were used to identify stillbirths.

2.5 Validation

Birthweight: Some infants were seen after 48 hours of birth. To determine whether weights recorded after 48 hours and before 120 hours were valid as a measure of birthweight, an intensive follow up study was conducted. Twenty three consecutively born study infants were seen within a day of delivery, and then reweighed at 2, 4, and 7 days. Although analysis of variance revealed that differences between weights in the same infants were significantly different depending on the day, the means taken in the first 5 days were more or less identical (appendix 3, section 1). In view of these findings, babies seen after 5 days were excluded from the birthweight, gestation and body proportion analysis. Multiple regression of infants seen within 5 days in the main study showed no correlation between the day the infant was weighed and the weight, after gestation had been adjusted for.

Gestation: Pilot newborn examinations at Madang General Hospital revealed minor morphological differences between Melanesian and Caucasian infants in relation to ear cartilage formation and pinna shape. Correspondence with Dr Dubowitz helped clarify how these criteria should be scored.

During the study it was found that most newborns were term, in contrast to the study by Winkvist (1988). Dr and Professor Dubowitz visited the project because of this discrepancy. Dr Dubowitz (LD) formally tested observer PG and checked observer MEB when she arrived in the East Sepik. Correlation of scores between study observers and LD, both by individual criteria and the overall score, was very close. The maximum error was estimated at 0.5 weeks (appendix 3, section 2). LD examined 10 babies born in villages in the Wosera, some of whom were part of the study.

3 RESULTS

3.1 Infant characteristics

3.1.1 Sample

One hundred and forty infants were born to the cohort of women during the study. One died shortly after birth (a twin); 5 were live infants of twin deliveries; 13 were seen after 120 hours. This leaves 121 live singleton births. [2]

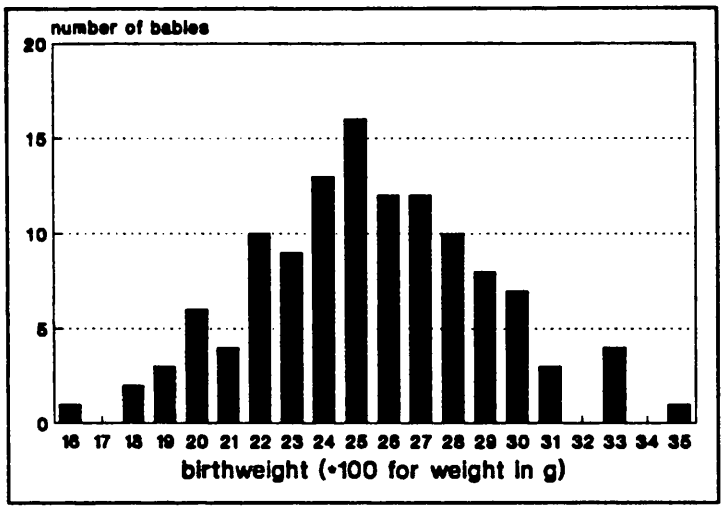
Babies seen after 5 days: Table 6A-1 lists the 13 infants that were not seen before they were 120 hours (5 days) old. Seven were visited late as they were born while the birth reporting system was being set up. Of the remaining 6, 1 birth occurred while the mother was visiting relatives in the provincial capital; 1 baby was born in a village where a newborn in the study had recently died (chapter 7); and the father of another baby had recently died. In 3 the cause of delay was unknown. The mean weight of the 13 exclusions was 2.68 kg (range 2.1-3.6 kg; n=12, one infant not weighed), and all survived the neonatal period. This analysis suggests that these exclusions are unlikely to bias the findings from the 121 newborns.

3.1.2 Birthweight

Birthweight distribution: The birthweight distribution of the 121 infants is shown in figure 6-1, and is approximately normal: the median and the mode was 2.5 kg. The figure suggests slight digit preference for 2.5 kg, but this is not apparent in a plot that includes the 7 control infants; nor is it evident if the observer imagines one less infant in the 2.5 kg category.

[2] There were 7 births in the control study, which was abandoned early (see methods). Of these 7, 1 had been brought to the team because the child was ill due to preterm delivery; the infant died. The mean birthweight of the 7 was 2.50 kg (compared with 2.54 kg in the main study) and the mean gestation was 37.9 weeks (compared with 39.3 in the main study); and 4/7 of the infants were less than 2.5 kg.

Figure 6-1. Distribution of birthweight for singleton births seen before 120 hours (n=121)



Mean birthweight: The mean birthweight was **2.54 kg** (table 6-2). This is based on 108 women from the population based cross-sectional survey, plus 13 women recruited through antenatal clinics at Tuwaikum village (population 423). Strictly speaking, these 13 were not sampled from a population based frame and depended on attendance of those women at antenatal clinic. This could cause bias if antenatal attendance was poor. However, coverage in Tuwaikum appeared complete. The mean birthweight of the Tuwaikum women was 2.62 (n=13, std=0.41), which is not significantly different from the main study less Tuwaikum (2.53, n=108, std=0.35; t test, p=0.4). [3]

Table 6-2. Mean and centile values for gestation, birthweight, length and head circumference of Wosera newborns

	number measured	mean	standard deviation	CENTILES 5	10	50	90	95	95% confidence limits
birthweight	121	2.54	0.358	2.0	2.1	2.5	3.0	3.1	2.47 - 2.60
gestation	121	39.3	1.02	37.2	38.2	39.4	40.4	40.7	39.1 - 39.48
length	121	46.7	1.94	43.6	44.1	46.6	49.1	49.7	46.4 - 47.0
crown-rump	120	30.4	1.72	27.7	28.1	30.4	32.2	33.1	30.1 - 30.7
head circum.	121	33.0	1.14	31.1	31.7	33.1	34.6	34.8	32.8 - 33.2
ponderal index	121	2.48	0.227	2.16	2.22	2.46	2.77	2.89	2.44 - 2.52

weight in kg; gestation in weeks; ponderal index in g*100/cm ^{3} ;other measurements in cm

3

Tuwaikum is close to Abusit and Gwingini villages, where standardised heights of women showed they were taller than other Wosera villages surveyed (chapter 4, section 3.7.3. A higher mean birthweight is consistent with this.

Table 6-3 compares birthweight from this study with other studies in lowland PNG: the mean is similar to Winkvist (1988), but lower than Ross's estimates from regression analysis using first clinic weights. Birthweight determined in Lumi, West Sepik in 1969 was lower, but not significantly so (Lumi mean birthweight 2.40, n=63, SD=0.6; normal test z=1.7).

The mean birthweight from this study is significantly lower than the mean birthweight of the 122 Wosera women delivering in Maprik Hospital 1984-1987. (chapter 7; n=122, sd=0.485, normal test z value=2.74; p<0.01).

Table 6-3. Mean birthweight values from various studies in lowland PNG

Area	Source, year published		pop*	Birth-weight	STD	n	comment
Wosera	POSTNATAL STUDY		V	2.54	.358	121	population based
Wosera	HOSPITAL STUDY (chapter 8)		H	2.69	.485	122	Woseras delivering in Maprik
Wosera	Winkvist,	1986	V	2.6	.399	54	opportunistic village recruits
Wosera	Ross,	1984	V	2.8	-	-	estimate from 1st clinic visit
Maprik	Schofield,	1964	?	2.72	-	-	Wingei and Wam census div.
West Sepik	Wark + Malcom,	1969	H	2.4	.5	63	Lumi, West Sepik
Coastal	Aitken,	1987	H	2.8-2.9	-	-	means from coastal centres

* pop: the source of data: V = village; H = hospital

Infant sex and birthweight: The mean birthweight of female infants was 2.52 kg (std 0.372) and of males, 2.56 kg (std 0.342). This slight difference is consistent with the higher mean weight of male infants found in PNG (chapter 3) and elsewhere (chapter 2). The sex difference in developing countries has been estimated as 93 g (Kramer 1987): this study is too small to anticipate demonstrating a significant sex difference.

The Wosera compared with other countries: Wosera mean birthweight were low compared with national data other developing countries except India, where some studies have shown mean birthweight to vary from 2.49 to 2.87 kg (Kramer 1987).

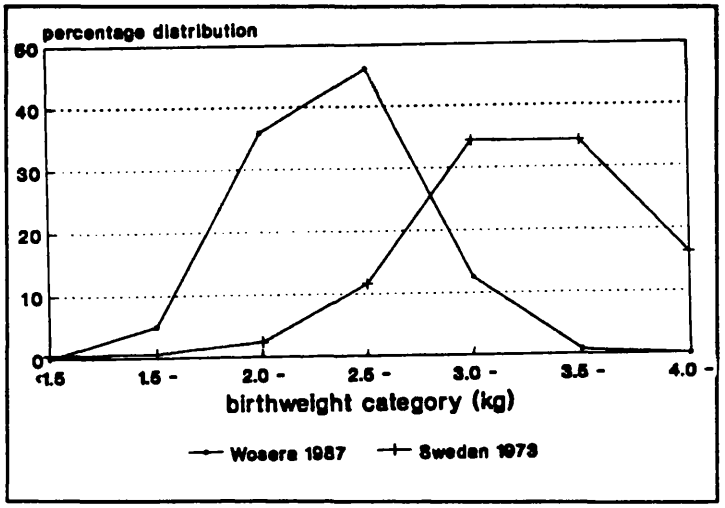
Low birthweight prevalence: Low birthweight prevalence was high (table 6-4). Slightly fewer males were low birthweight, consistent with their higher mean birthweight.

Table 6-4. Number and percent () of low birthweight newborns by sex

SEX	WEIGHT CATEGORY <2.5 kg	2.5+ kg
male	20 (37)	34 (63)
female	28 (42)	39 (58)
ALL	48 (40)	73 (60)

The distribution of babies by birthweight category is compared with the distribution from Sweden (figure 6-2). The Wosera distribution shows a marked shift to the left.

Figure 6-2. **Birthweight distribution of Wosera newborns (500 g groups) compared with Sweden (WHO 1978)**



3.1.3 Gestation

Eighty one Dubowitz measurements were done by one observer (PG), and 37 by the second observer (MEB). Two were performed by a third observer (DL), who had been trained previously to undertake them, and 1 by a visiting doctor (GE). Mean scores between the 2 main examiners showed a slight difference (0.5 weeks, MEB>PG). This was probably due to selection bias: the team leader was more likely to examine babies that were ill or preterm (Appendix 3, section 3).

Three males and 2 females were preterm, giving a preterm percentage of 4.1% (95% C.I. 1.3 - 9.6%). [4]

There was no sex difference in mean gestation. (Females:39.3 weeks, std 1.02); males: 39.3 weeks, std 1.03).

Tuwaikum village had a mean gestation of 39.5 weeks (n=13, std=1.02). This was very similar to the mean gestation of the main study with these births excluded (39.2 weeks, n=108, std=1.00).

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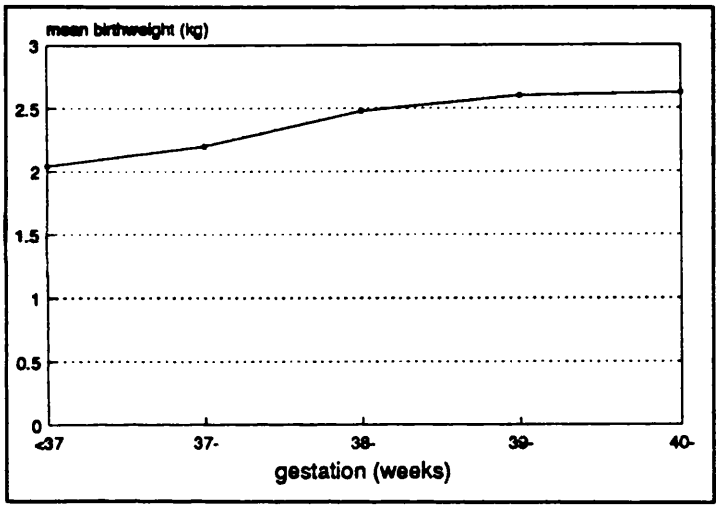
[4] Confidence limits were estimated using the Poisson distribution

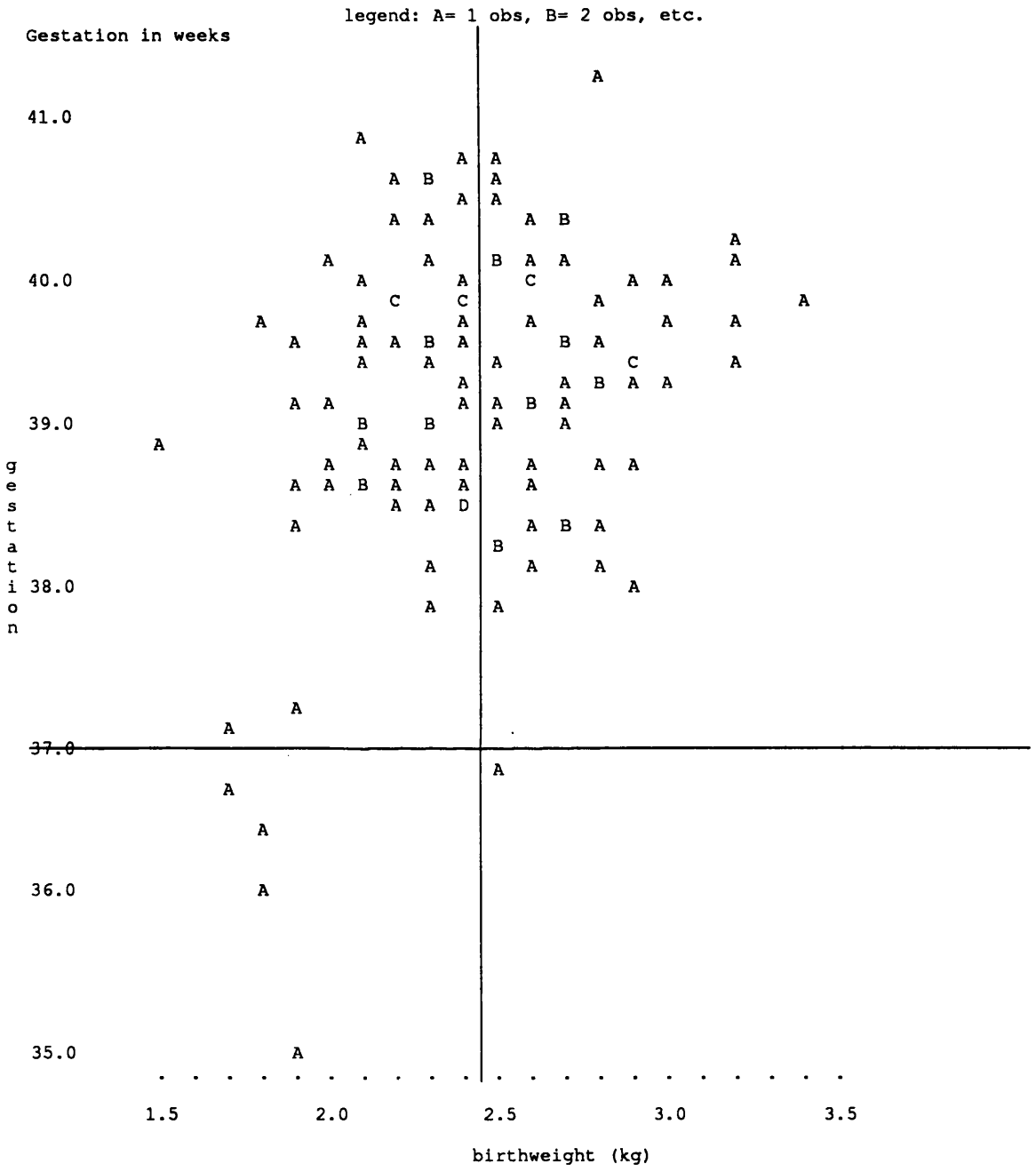
Of the 5 singleton preterm infants, 4 were low birthweight. Mean birthweight by gestation is shown in figure 6-3. The mean birthweight was higher in term deliveries compared with preterm. The correlation coefficient between birthweight and gestation was 0.322 (p=0.0003).

Figure 6-3. Mean birthweight by gestation (data presented in table 6A-2)



A plot of gestation by birthweight shows the association between birthweight and gestation was due to a few preterm, low birthweight infants (figure 6-4). There was no relationship between gestation (by the Dubowitz method) and birthweight above 37 weeks.

Figure 6-4. Gestation by birthweight



3.1.4 Neonatal anthropometry

Birthweight and other anthropometric parameters were analyzed to determine whether infants were asymmetrically or symmetrically growth retarded (chapter 2, section 3.2).

Ponderal Index: The mean ponderal index (table 6-2) was similar or marginally lower than data from other developing countries (reviewed in table 2-3), and practically the same as reported by Ghosh (1971) in India.

Using standard ponderal index centiles at 40 weeks gestation (Lubchenco et al. 1966), thinness was divided into 4 categories between the 10, 50 and 90 centiles. The frequency distribution of Wosera infants by centiles derived from Lubchenco and birthweight group is shown in table 6-5. Ten percent of term infants (12/116) were under the 10th centile. This suggests that acute growth retardation, determined by a low ponderal index, is no more common in this population compared with Lubchenco's.

Table 6-5. Frequency distribution of infants by birthweight group and standard ponderal index centiles

BIRTHWEIGHT	---PONDERAL INDEX CENTILE GROUP [a]				
	0-	10-	50-	90-	all
<2.5 kg	10	32	6	0	48
2.5+	3	42	27	1	73
ALL	13	74	33	1	121

[a] centiles from Lubchenco et al. 1966: 40 wks gestation
10c=2.25 50c=2.62 90c=3.11

There was a positive correlation between ponderal index and birthweight. Linear regression confirms this (f=40.4, df=(1,119),p=0.0001). This was noted by Woods in South African newborns (1984). It may reflect that ponderal index is a method of examining relative thinness of babies within birthweight categories, but does not fully correct for length at various birthweight values. This could explain why the mean ponderal index from this study is slightly lower than found in the studies reviewed in table 2-3 (except Ghosh 1971) as the mean weight in this study was lower than the mean birthweight in the other reviewed studies.

Length and head circumference: Mean length and head circumference values of Wosera newborns were lower than values from other developing country studies (tables 6-2 and 2-3).

3.1.5 Newborn malaria, haematologic and iron status

Infants recruited in the control (n=7) group are included in this analysis. Blood slides for malaria parasites and haemoglobin estimations were available on 125 infants taken within 5 days of birth. None were positive for malaria parasites. [5] This contrasts with 4/52 in a study in Madang (Lehner and Andrews 1988) and 4/104 in another study in Madang (Sehgal 1988). However, both these studies peripheral infant blood was usually taken within two hours of birth, when transient parasitaemia may occur.

The mean haemoglobin was 17.6 g/dl (std 2.16). The values were normally distributed. Free erythrocyte protoporphyrin determinations were available on 94 infants. The geometric mean

[5] Slides were read using a PNGIMR standard technique (Cattani et al. 1988)

value was 5.01 µg/g Hb. The highest value was 10.3 µg/g Hb, and the lowest 2.6 µg/g Hb. Mean levels of both haemoglobin and FEP show no difference when divided between infants below and those equal to and above 2.5 kg.

Hospital deliveries: Infants delivering in hospital had lower haemoglobin values. Using singleton births of the 121 babies of mothers recruited antenatally (data missing n=2), the mean haemoglobin of village births was 17.7 g/dl (std 2.06, n=107) and, for those delivering in hospital it was 15.4 g/dl (std 2.25, n=12). The difference was significant (variances equal, exact t test: t=3.6, df=117, p=0.0005). Traditionally, the cord is cut in the village after the placenta has been delivered (chapter 3) and blood transfer from the placenta is likely. In hospital deliveries, the cord is cut as the anterior shoulder is delivered before completion of the third stage.

3.2 Delivery details

3.2.1 Antenatal attendance

This analysis includes women seen postnatally after 120 hours, and 7 women seen in the control study. Four women (all from the control group) had not attended antenatal clinics. Of the attenders, 12% (18/144) had attended once, 27% twice, 29% 3 times and 10% 4-5 times.

Compliance with malaria and folic acid prophylaxis was assessed in antenatal clinic attenders. Compliance was judged to be poor in 31% (45/144) of women. 53% (76/144) of women understood the regimen and reported taking it regularly. 3 women reported taking the drug with an erroneous regimen (for example, one tablet every day). In 4 cases data were missing.

3.2.2 Health prior to delivery

Three women reported a fever which resolved in the week prior to delivery; 8 reported a fever that persisted to the day prior to delivery; and 5 reported some other illness such as a cough in the week and day prior to delivery. In only 2 instances was there a fresh illness just prior to delivery. There was a maximum of reported illness at the parity extremes (table 6-6).

Table 6-6. Maternal illness in the week prior to delivery by parity

PARITY GROUP	N	---ILLNESS PREVIOUS WEEK-------- N	%	N	%
		---ANY---		-MALARIA LIKE-	
0	27	4	(15)	3	(11)
1	27	2	(7)	1 }	
2-	36	3	(8)	2 }	(7)
4+	50	7	(14)	5 }	
ALL	143	16	(11)	11	

There was no obvious excess of febrile malaria like illness in nulliparous pregnant women on the week or day prior to delivery. These data therefore do not support they hypothesis that higher susceptibility of primigravida to malaria causes frequent clinical episodes in late pregnancy, precipitating labour.

3.2.3 Work prior to delivery

Few women rested in the week before labour started, and the majority were working on the day before labour commenced (table 6-7).

Table 6-7. Reported work in the week and day prior to labour

Work prior[a] to labour	week before N	%	day before N	%
resting	23	(16)	67	(47)
light work	70	(49)	64	(45)
heavy work	49	(34)	11	(8)

[a] data missing n=2

Table 6-8 shows women by parity group and whether they were engaged in heavy work in the week before, or working at all in the day before delivery. There was little variation in the level of work reported by parity, except in higher parity groups, where fewer women reported heavy work in the preceding week.

Table 6-8. Work practices in preceding day and week by parity

parity group	N (%) heavy work in preceding week	N (%) working on preceding day
0	10 (38)	11 (42)
1	15 (41)	18 (49)
2-	15 (38)	22 (56)
4+	9 (18)	24 (48)
ALL	49 (34)	75 (53)

Of those women reporting heavy work in the previous week, 39% (19/49) reported resting, 40% reported light work and 20% reported heavy work in the day preceding labour.

3.2.4 Place of delivery

Ninety percent of women delivered in the village (129/144); 13 women delivered in a health centre or hospital; one woman delivered on the way to the hospital; and another in a town house in Wewak while visiting relatives.

Seven women delivering in health facilities were nulliparous. Five of the 13 chose to deliver in a health centre, 3 went because they were advised to at antenatal clinic, and 5 went because of problems during labour. In 4 this was due to delay with delivery (final time from labour onset to delivery was 55, 54, 52, and 51 hours). One woman delivered at Kaugia health subcentre but was sent to Maprik Hospital with a retained placenta. Four women that delivered in the village were later admitted: 2 with retained placental parts; 1 with a perineal tear; and 1 because of a preterm infant.

3.2.5 Length of labour

This ranged from under one hour (n=1) to 138 hours, with a median of 13.5 hours. The distribution was positively skewed. The geometric mean for village births was 11.3 hours. Labour was longer in nulliparous women (geometric mean 16.3 hours). This difference was significant (approximate t test: t=2.06, df=68, p=0.04). Table 6-9 shows the distribution of labour length. As most Wosera villages are within 3 hours walk of a health subcentre, 95% could have reached such a unit if they commenced walking when labour pains commenced.

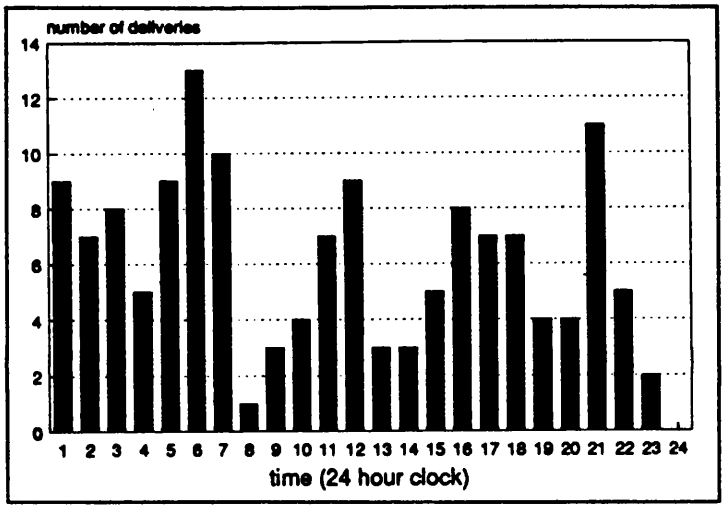
Table 6-9. Length of labour in village births

Time * (hours)	N	%
0-	7	5
3-	13	10
6-	35	27
12-	43	33
24-	28	21
48-	5	4

*data missing n=3

The distribution of births by time of delivery (figure 6-5) suggests digit preference at 6.00, 12.00, and 21.00 hours. Thirty five percent (51/144) of deliveries occur between 1-6 hours, 24% (34) between 7-12 hours, 23% (33) between 13-18 hours and 18% (26) between 19-24 hours. The proportion of births born in the light (6.00-18.00 hours) is similar to that born during the dark (18.00-6.00 hours). The apparent predominance of births in the early hours may be the result of a misclassification effect: the estimated birth time during the night may have been estimated as later than it actually occurred.

Figure 6-5. Reported time of delivery



The length of time from reported membrane rupture to delivery varied from 0-26 hours, with a mean of 2 hours and a median of 1 hour. In 85% (111/131) of women the time was from 0 up to 3 hours, in 11.5% the time was from 3 up to 12 hours, and in 4% (5/131) the time was in excess of 12 hours (13, 16, 17, 18, and 26 hours). There was no difference in this time period between parity groups.

3.2.6 Attendants

The woman delivering on the way to hospital was attended to by the mission nurse and excluded from this analysis; the woman delivering in Wewak was attended by relatives and included. Overall, 27% (35/130) births at home were unattended, the mother herself delivering the child. All nulliparous women (22 of them) were attended. Of the 7 women delivering within 2 hours of onset of labour, 3 were unattended.

Sixty one percent (80/130) of deliveries were attended by female relatives or relatives, while in 12% (15/130) the husband was the attendant.

One nullipara and 1 parous mother reported breech deliveries. It is possible that the incidence was higher than this. Traditionally, a breech is regarded as unlucky and indicates the mother has broken a taboo during the pregnancy.

3.2.7 Third stage of labour

Five percent (7/130) reported excessive blood loss during the third stage of labour. Two women reported retained placental pieces; in the remaining 128, 79% (101/128) delivered the placenta without delay. Thirteen percent (17/128) reported a delay, but this was estimated to be less than one hour.

However, 8% (10/128) reported significant delays with placental delivery in excess of 1 hour. In 5 this was 3 hours or more. Interestingly, the delivery delay beyond 1 hour was more common in primigravida (3/22) or women of parity 4 + (5/45). In 2 of the women with delayed placental delivery, PNGIMR field staff (DL) assisted placental delivery at home.

3.3 Birthweight, gestation and associated maternal factors

3.3.1 Birthweight

Birthweight and possible maternal factors influencing it was examined, using singleton infants seen within the first 120 hours. Table 6-10 summarizes the factors investigated.

Table 6-10. Maternal factors examined in relation to birthweight

parity
education
nutritional status (height, head circumference, postnatal weight, weight gain)
blood pressure at first visit
smoking habits
betel nut consumption
antenatal clinic visits
prophylaxis compliance
recent illness
level of physical work in week prior to delivery;
Malaria at first visit (size of spleen, Plasmodium falciparum density, and haemoglobin)

Table 6-11 shows means of some variables with birthweight category. Mean maternal height, weight gain in pregnancy and booking in haemoglobin all have lower means in the low birth weight category. As expected, mean gestation of low birthweight infants is lower due to the 6 preterm babies in this group.

Table 6-11. Means of maternal height, infant gestation, maternal haemoglobin at first antenatal visit and pregnancy weight gain by, infant birthweight category

weight group	N	maternal height[a] (cm)	infant gestation (wks)	booking haemag (g/dl)[b]	pregnancy weight gain rate[c] N	(kg/wk)
<2.5	48	151.0	39.1	8.4	38	0.128
2.5+	73	152.2	39.5	8.8	59	0.232
ALL	121	151.7	39.3	8.6	97	0.191

T-TEST: [a] p=0.2; [b] p=0.085 (missing data n=2); [c] p=0.0004

Parity: Mean birthweight varies little in relation to parity (table 6A-3); although it is slightly lower in primigravida, the difference is not significant (mean birthweight of infants born to primiparous women = 2.43 kg; to multiparous women = 2.56 kg [t test t=1.665, df=119, p=0.0986]).

Clinic attendance: Mean birthweight was higher in women that had attended antenatal clinics more frequently (table 6A-4). In women that seemed to understand the drug regimen, mean birthweight did not differ between good and poor compliers (table 6A-5). Women that had a poor understanding of the drug regimen had a low mean birthweight.

Maternal morbidity: Mean birthweight in women reporting an illness in the week prior to delivery was lower (table 6A-6); if women with any illness, be it malaria like or some other illness, are grouped together, then the difference in the mean birthweight between the two groups is significant (t test, $p<0.04$). This is probably because women reporting illness having a lower mean gestation (well group: gestation=39.4 weeks; ill group: gestation=38.4 weeks; approximate t test: $p=0.06$). This is examined in more detail in section 3.3.2.

Spleen and parasite density at first antenatal visit: There was no trend in mean birthweight with spleen size. Nor was there a relationship between birthweight and *Plasmodium falciparum* parasite density at first visit.

Haemoglobin at first visit: There was a relationship between haemoglobin at first visit and birthweight: mean birthweight was lower in anaemic women (table 6A-6). Although the regression between haemoglobin and birthweight was significant, stage of gestation at first visit could confound the relationship. Haemodilution as pregnancy progresses tends to reduce haemoglobin, and occurred with this study (table 6A-8); higher birthweight may be associated with women that book early.

When gestational age at first visit was categorised into 0-, 20-, and 30- weeks, a generalised linear model examining birthweight in relation to haemoglobin value at first visit, taking into account stage of gestation, the relationship between birthweight and haemoglobin value became non-significant. When ponderal index was substituted for birthweight the relationship to booking haemoglobin approached significance ($f=3.09$, $df=91,115$) $p=0.0814$: table 6A-9).

Maternal education, smoking and betel nut chewing: Mean birthweight did not vary with maternal education, or with regularity of smoking or betel nut chewing. There was no relationship between mean birthweight and the level of reported work in the week prior to delivery (table 6A-6).

Blood pressure at first visit: Low birthweight infants were not associated with higher mean blood pressure at first visit ($n=117$).

Height: A plot of maternal height suggested a weak association with birthweight, confirmed by regression ($f=5.14$, $df=(1,119)$, $p=0.0252$). There was no relationship between ponderal index and maternal height.

Maternal head circumference: Mean maternal head circumference was very slightly greater in women delivering babies weighing 2.5 kg or more compared to those delivering LBW infants (table 6A-7).

Weight gain: Pregnancy weight gain (chapter 5) correlated with infant birthweight. This correlation can be anticipated as the fetus alone represents approximately half of the total maternal weight gain.

3.3.2 Gestation and low birthweight

Table 6-12 lists singleton infants that were either preterm or weighed less than 2 standard deviations below the study birthweight mean (infants weighing 1.8 kg or less).

In 3 of the 5 preterm infants, their mothers had experienced an illness in the week prior to delivery: 2 histories suggested lower respiratory tract infection, one suggested urinary tract infection, and a fourth woman with evidence of nephrosis in pregnancy. The fifth woman was well, but had a dense *Plasmodium falciparum* infection. This had been present at her initial visit and had subsequently cleared. None of the 5 women were nulliparae.

The remaining 2 of the 7 were small but term infants; the mother of one reported an illness in the last few weeks (accompanied by a fall in haemoglobin value 1.9 g/dl between first visit and postnatal period); and the second exhibited severe antenatal anaemia, but refused regular treatment or a blood transfusion.

Two infants show obvious evidence of acute growth retardation with low ponderal indexes: one preterm infant, and the other the infant of the mother with severe anaemia.

In conclusion, the majority (5/7) of singleton infants 2 standard deviations below the mean birthweight were preterm; and in all 7, there was evidence of recent maternal illness, chronic illness or persistent dense malaria parasitaemia.

Table 6-12. Preterm or small (<1.9 kg) infants: maternal booking haemoglobin and malaria parasitaemia, reported illness in late pregnancy and postnatal haemoglobin and malaria parasitaemia

				booking data					postnatal information			
	gest-ation	birth weight	ponderal index	par-ity	HB	spleen	falci-parum	history of illness	temper-ature	fal dens	hb
	weeks	kg	g*100/cm³		g/dl	cm	per cc		°C	per cc	g /dl
	35.0	2.0	2.67	7	5.9	30	12	nephrosis; a/n hbs 4-7	36.2	0	5.7
	36.1	1.9	2.53	1	8.8	0	24	dysuria and backache:recent	36.6	0	8.6
	36.5	1.9	2.14	4	8.0	2	0	fever, chest pain and cough:recent	38.2	0	9.4
	36.7	1.8	2.36	5	8.3	0	0	fever and cough:recent	36.2	0	10.2
	36.9	2.6	2.67	1	9.1	10	1310	nil	36.2	1880	10.2
	37.1	1.8	2.31	6	8.4	0	0	fever, headache, diarrhoea prev month	37.1	0	6.5
	38.9	1.6	1.97	1	6.4	2	0	antenatal anaemia: 4.5-6.9g/dl	37.6	0	4.4
mean	36.7	1.94	2.38	3.6	7.8	6.2		(n=7)			
REST	39.4	2.57	2.49	2.9	8.7	2.05		(n=114)			

4 DISCUSSION

The study shows the birthweight of infants born to a cohort of Wosera women derived from a population based study were low. Mean birthweight was consistent with recent local data (Winkvist 1988). The gestational age assessment indicates there were not an usually high proportion of preterm deliveries compared with Western populations (table 2-2). The analysis of newborn proportions, using ponderal index, suggests that acute growth retardation is no more common in this population than in other populations studied in the USA. In other words, the low birthweight in the Wosera is associated in general with proportionally small, term infants.

Whether Wosera newborns can be termed "chronically growth retarded" is a matter for debate. This study was not large and therefore differentiating the effect of multiple factors influencing birthweight was not possible. However, what emerged was that low birthweight preterm infants were associated with recent illness in the mother.

Interestingly, clinic attendance was positively associated with birthweight. However, this may simply reflect that women that attend clinic early and frequently may also deliver larger babies, rather than the effect being causal.

Haemoglobin values did not correlate with birthweight after stage of pregnancy when the haemoglobin was determined were taken into account.

The high prevalence of malaria in the population suggests that adequate control of this factor could increase birthweight, particularly in nulliparous women. However, this was not shown by this study, probably because the intervention was not effective in eradicating in malaria in mothers. This was in part due to poor compliance generally with the prophylaxis regimen (chapter 5); the role of *Plasmodium falciparum* chloroquine resistance is a further factor that could impair the efficacy of chloroquine prophylaxis.

It could be hypothesised that maternal malaria and smoking were both factors present which have been shown in other studies to be causally related to reduced weight for gestational age from other studies; and that, should both exposures have been removed during pregnancy, the expected affect on birthweight would be to increase it. This fulfils the 2 criteria proposed in chapter 2 (section 3.2.2) as evidence for growth retardation. Thus the low birthweight in Wosera newborns is in part due to symmetric growth retardation. However, the extent of this growth retardation, over and above small infant size related to maternal uterine constraint, is not clear. Certainly the 800 g difference between a typical Western birthweight of 3.3 kg and the Wosera birthweight of 2.5 kg is unlikely to be entirely the result of growth retarding factors in the mother.

It could be argued that low birthweight in Wosera newborns is in part an adaptive phenomenon; the size may be appropriate for maternal pelvic capacity so that obstructed labour is not common; and the lactational demands of a proportionally small infant on the mother are likely to be less, resulting in less stress on an already marginal maternal nutritional status.

5 SUMMARY

In neonates born to a cohort of women from a population based survey:

A. mean birthweight of singleton infants was low (2.54 kg, 95% C.I. 2.47 - 2.60), and 40% of infants were low birthweight (<2.5 kg);

B. the proportion preterm was similar to that found in Western countries (4.1%, 95% C.I. 1.3 - 9.6);

C. acute growth retardation, determined using ponderal index, was similar to that found in Western populations;

D. mean birthweight of the cohort was significantly lower than that of Wosera women delivering in the local hospital over the previous 4 years;

E. neonatal haemoglobin was lower in study babies born in hospital compared with those born in the village;

F. birthweight was positively associated with maternal height, and number of times women attended antenatal clinic;

G. those weighing less than 2 standard deviations below the population mean were usually preterm, and were often associated with recent maternal illness.

chapter 7

UMBILICAL CORD CARE

1 AIMS

The aim of this study was to evaluate methods of reducing neonatal sepsis by providing pregnant mothers attending antenatal clinic with simple umbilical cord care packs.

2 INTRODUCTION

At the beginning of the postnatal study the causes of neonatal death in the Wosera were unknown. At the postnatal visit, any findings that may be of relevance to risk of perinatal death were observed (table 6-1).

In the postnatal study neonatal sepsis was diagnosed in some newborns. The definition used was that contained in the standard treatment manual for children in PNG (PNG Paediatric Society 1988), sometimes termed sepsis neonatorum (for example, Siegel and McCraken 1981). Another group of newborns were pyrexial, but otherwise apparently well. Yet others had evidence of septic umbilical cords (the cord smelt offensive or cellulitis of the peri-umbilical skin was noted).

In the presence of mild fever in the newborn with no evidence of systemic disease, the mother was advised that the baby had a mild fever; and, in the event of any signs of illness (such as poor feeding) they were to immediately take the child to the local health facility. Ideally, infants with a fever or evidence of cord sepsis should have been admitted to a health subcentre; as mothers did not perceive the child as ill, this was simply not possible. This management was discussed with paediatric colleagues in Port Moresby General Hospital, and felt to be appropriate and ethical.

Infected umbilical cords were painted with acraflavine 10% in spirit at the postnatal visit. Mothers were given applicators and acraflavine in spirit to apply to the cord themselves over the following 3 days. In some circumstances, the mother was advised to attend the health centre the following day so the umbilical cord and the baby could be reviewed by health staff.

One infant who was well postnatally but had a mild fever died 6 days later. A second infant, febrile postnatally but otherwise well, was seen aged 21 days with neonatal sepsis. At this point field data was reviewed (table 7A-1), and in light of the emerging evidence, new procedures for umbilical cord care were introduced in two phases. Maternal compliance with these procedures was evaluated, and their outcomes were compared with those preceding the interventions.

3 METHODS

Initial intervention: Women usually severed umbilical cords with sharpened bamboo or a razor blade. Both could be contaminated: the razor blade was rarely new, and the bamboo was often prepared using a general purpose agricultural knife. It was therefore decided to supply mothers with a new razor blade to cut the cord, and acraflavine 10% in spirit and swab sticks to paint the cord stump. A tie was not supplied as it was judged that the process of tying could result in cord contamination from handling. Cord clamps were not distributed as it was felt mothers and attendants were likely to be anxious about something so foreign to their current cultural practices.

One disadvantage of supplying a new razor blade without a tie was there could be more than usual blood loss from the cord stump. This is because the usual blunt instrument induces vessel spasm in the cutting process (Professor Biddulph, personal communication), and a sharp blade would not provide this stimulus. However, this was weighed against the problems that providing a tie could cause.

Use of the blade was demonstrated at antenatal clinics, and it was suggested that the cord should be cut leaving 2-3 cm rather than flush with the skin to avoid excessive blood loss. Application of the spirit was also demonstrated, and women were instructed to use the spirit on the day of delivery and the following day.

Umbilical cord care and neonatal sepsis were subjects at in-service teaching conducted at Kunjingini and Kaugia health subcentres.

Modification of intervention: Excessive blood loss from the cord was noted by 4 mothers using the razor packs in the subsequent 46 babies born. This was associated with low neonatal haemoglobin values (14.0, 13.8, 13.5, 13.1 g/l). Prior to the introduction of razor blades, no newborn had a haemoglobin below 14.0 g (n=53, haemoglobin value missing=7).

After deliberation, plastic umbilical cord clamps were then added to the packs and women instructed in how to use them. Mothers and their relatives practised using clamps at antenatal clinics using a plastic hose. Postnatally the cord was examined for correct application of the clamp.

Morbidity follow up: All newborn infants in the postnatal study were supplied with infant clinic books, which were used by health staff to record morbid episodes. Wherever possible, any neonate reported as ill by subcentre staff was reviewed by PG. All illness episodes in the neonatal period were transcribed from infant clinic books by PNGIMR staff. Admissions were followed up. The PNGIMR was active in the area into January 1988: morbidity surveillance activities were maintained even though the postnatal study was completed.

Temperature recording: Rectal body temperatures were measured in the first 24 infants. Despite careful explanation to the mothers, it became evident that the procedure caused some mothers unnecessary anxiety. Therefore rectal recording was abandoned in favour of the axillary method. To correct for the difference in measurement technique, 1 degree

Centigrade was subtracted from all rectal measurements to make them equivalent to the axillary measures. This was based on some newborns where both methods of temperature recording were employed, showing the axillary temperature to be approximately 0.5 - 1.0 °C less.

Newborns examined prior to the start of the intervention study formed the "traditional" umbilical cord care group; those supplied with a razor and acraflavine were classed as the "razor" group; and those born to mothers supplied with an umbilical cord clamp in addition to the razor formed the "clamp" group. Some women delivered in hospital and they classed as a separate group.

4 RESULTS

This analysis includes twin infants; infants in the control group (chapter 6, section 2.1); and babies seen in the first week of life. In all, there were 138 infants in the study. Fourteen of these had delivered under health service supervision, leaving 124 village births.

4.1 Compliance

Fifty seven village born infants were seen prior to intervention. Of those mothers seen before the postnatal study finished, 32 mothers had been issued with razor blade packs in the first intervention, and 33 mothers had been issued with packs that contained cord clamps. In 2 babies where mothers had been issued with packs, it was not clear whether the packs had been used.

The majority of women had used all the components of the pack: 69% (22/32) of those issued with the razor pack used the razor and the acraflavine, and 51% (17/33) of those issued with the addition of a clamp used all 3 components. Seventy three percent (24/33) of mothers issued with a clamp had used it (table 7A-2).

4.2 Outcome

Body temperature: Data could be biased by variation in ambient temperature with season or through some undetected change in measurement method. To examine for overall secular change, body temperature was plotted in order of infant recruitment (figure 7A-1). The plot does not suggest any secular change, although there are a few higher temperatures seen in the earlier babies related to neonatal sepsis.

Table 7-1 shows the number of infants with raised body temperature by intervention group. A raised body temperature appears more common in the traditional cord care group. There were 3 babies with raised axillary temperatures in the razor group. Although 22/32 of mothers in this group had used acraflavine, it had not been used in any of these 3.

Table 7-1. Infant's temperature at the postnatal visit by intervention group

CORD CARE	postnatal baby temperature °C		
	35-	37.5+	
traditional	49	9	58
razor	34	3	37
clamp	24	0	24
hospital delivery	14	0	14
ALL	121	12	133

rectal temperatures corrected
temperature missing in 3 infants

Table 7A-3 outlines clinical details of the infants who developed definite or possible neonatal infections, collating data from clinic books and health centre and subcentre records. Four of the 5 cases of definite neonatal sepsis had been seen by a doctor; the fifth case, in the intervention group, had been seen on day 28 by health subcentre staff with signs indicative of pneumonia (fast breathing and intercostal recession). The 3 cases of probable neonatal sepsis were babies whose history and clinical findings were highly suggestive of neonatal sepsis, but who had not been reviewed by a doctor: 1 was febrile with skin pustules; a second was febrile, with diarrhoea and died; and a third was ill with an axillary temperature recorded as 38.9 °C.

Table 7-2 shows morbidity follow up in the neonatal period for all infants, extracted from the baby's clinic record book. It shows a higher incidence of neonatal infection in the control group compared with the intervention groups.

Table 7-2. Follow-up morbidity in the neonatal period by intervention group

	N	NEONATAL SEPSIS	?NEONATAL SEPSIS	FEVER	URTI/LRTI	OTHER
controls	61	5	3	0	3	2
razor	37	1	0	0	1	5
clamp	24	0	0	2	0	4
hospital delivery	14	0	0	0	0	2
ALL	136	6	3	2	4	13

This study was not blind. If the researchers were biased toward the intervention working, bias could be introduced by classifying illness in the intervention group as less severe than in the non-intervention group. Examination of the illness episodes described as "fever" suggests that this was not the case. In 1 case, the infant was brought to the clinic with a cough at day 8, was afebrile with no signs of sepsis; the infant was brought back for review on day 12, with a reported history of fever: the axillary temperature was recorded as 36.5 and the infant given amodiaquine. The second case of "fever" was on day 27 with no other complaints. The temperature was recorded as 37.1 and the child given amodiaquine.

In the illnesses classified as "other", there were none suggestive of neonatal infection. Of the 9 cases in this category that were part of the intervention group, 3 were cases of simple conjunctivitis, 3 were brought because the parents were worried about constipation, 1 was mild diarrhoea, 1 was brought with physiological breast swelling and 1 was an incidental finding at a MCH clinic that an infant had a "damp" but uninfected cord. It is likely that PNGIMR encouragement of parents to take their child to the centres should they be worried increased the number of consultations made.

Table 7-3 is a 2x2 table with cases of probable and definite cases of neonatal sepsis grouped together. There are significantly fewer cases of neonatal sepsis in the intervention group compared with the control group (Fishers exact's test: p=0.032).

Table 7-3. Umbilical cord intervention study: neonatal sepsis (definite and probable) by intervention or non-intervention group (hospital deliveries excluded)

	no sepsis	sepsis	total
control	53	8	61
intervention	60	1	61
total	113	9	122

(2-tailed Fishers exact test:p=0.032)
excludes 2 infants where it was not known
whether they had used the intervention

Table 7-4 describes the cord at the postnatal visit by intervention group. It can be seen that both umbilical cord flare and a smelly cord are common in both intervention and pre-intervention groups. One problem with interpreting this data is that initially the examiners were less aware of umbilical cord sepsis. Once it was perceived as a problem, the examiners looked harder for it and were more likely to detect mild evidence of flare. The table also shows that 2 cords were grossly contaminated with earth. This reflects the poor living conditions of the people.

Table 7-4. Cord care group and description of the umbilical cord at the postnatal visit

		------CORD CARE GROUP ---------		
STATE AT POSTNATAL	N	traditional	razor	clamp
dry or clean	93	45	27	21
umbilical flare	19	11	6	2
pus on base only	3	2	1	0
smell only	6	2	3	1
contaminated	2	2	0	0
ALL	123	62	37	24

Some of the women in the clamp group were part of the intensive study examining weight change in the first week of life (appendix 3, section 1). No treatment or care of the cord was provided during this study: however, the state of the cord was noted at each visit and the axillary temperature of the baby measured. The 25 newborns were seen a total of 99 times and an axillary temperature above 37.5 oC was recorded on only one occasion.

5 DISCUSSION

This study demonstrates that umbilical cord sepsis was common in village deliveries. There were fewer serious neonatal bacterial infections in the second part of the study when umbilical cord care had been introduced, and this was associated with a reduced prevalence of fever detected incidently at the postnatal visit.

The umbilicus has been known as an important entry point for micro-organisms in newborns. The incidence of neonatal tetanus is affected by traditional methods of cutting or dressing the cord (Woodruff et al. 1981). Neonatal tetanus was very common in the Maprik district prior to the introduction of widespread maternal vaccination: Schofield (1961) estimated that neonatal death rate from the disease was 61/1000. Widespread maternal immunisation has reduced the incidence of neonatal tetanus in the area, but the birth practices that in the past led to *Clostridium tetani* infection have not changed (chapter 3, section 1.4).

Bacteria may invade through the stump and seal within the umbilical artery (Forshall 1957). The focus of infection may then discharge externally from the umbilicus, or cause septicaemia from infection of the iliac vessels or by metastatic spread. In this study, infection of the umbilicus was manifest by abdominal skin flare, indicative of spreading cellulitis from the umbilicus, and by pus discharge from the umbilicus stump. Neonatal sepsis in 2 infants neonatal sepsis included septic arthritis: this is presumptive evidence of blood born bacterial spread from a septic focus, and was reported by Forshall (1957).

The clinical picture seen in the cases of neonatal sepsis diagnosed by the author were very similar to the findings in other studies of umbilical cord sepsis, reviewed by Cullen (1916).

The study did not have concurrent controls because the staged analysis during the field work provided sufficient evidence to introduce cord care without trying to nest a controlled trial within the study. It is possible that external factors that changed with time could have biased the results. For example, seasonal changes could alter the ambient temperature. However, confounding factors are unlikely to explain all the various strands of evidence in relation to postnatal fever, and the reduction in neonatal morbidity. Interestingly, fever occurred in the razor only group, and 2 of these women delivered at the time the clamps were being issued and used. The intensive baby weight follow up study showed that newborn fevers were extremely rare in the group using clamps, even when the measurements were repeated over the first week of life.

The study suggests razor blades should be used in combination with a method of clamping the cord to avoid excessive blood loss. Mothers were able to use umbilical cord clamps, and were quite enthusiastic about them: the loss of blood after cutting the cord, whether by

traditional methods or with a bamboo, was a source of concern. However, the clamps were introduced in women who had, by the end of the study, confidence and trust in the PNGIMR team, and this is likely to have influenced use. Nevertheless, women are unlikely to participate in activities that they do not see as potentially beneficial, which was one of the problems of the programme of chloroquine prophylaxis during pregnancy. Their attitude and interest to umbilical cord suggests that women are likely to use them, provided they are shown how to.

6 SUMMARY

A. Neonatal sepsis was relatively common in Wosera neonates;

B. Many women delivering in the village used the umbilical cord care packs supplied to them through antenatal clinics;

C. A reduction in neonatal morbidity was associated with the introduction of a health care package which included emphasis on umbilical cord care, and provision of cord care packs to mothers.

OBSTETRIC HEALTH SERVICES USE

1 INTRODUCTION

The aims of this retrospective user survey were to:

i. describe Wosera women using obstetric health facilities, and why they went there;

ii. elicit their impressions of the care they received.

2 METHODS

Demographic data of Wosera women delivering in Maprik District Hospital (MDH) or Kaugia health subcentre 1984-86 were taken from labour ward birth registers. Women were located and interviewed. If a woman could not be found, the birth date was compared with children registered at child health clinics. This helped identify births in cases where there was little information to identify the mother. A standardised interview recorded demographic and social information, obstetric history, and details of the pregnancy, labour and circumstances surrounding the admission. Time of labour onset, how the women reached the facility, why they went, what happened when they got there and their impressions of the care provided were inquired after. Interviews were conducted by highly skilled field staff (DL, MEB or PG).

3 RESULTS

Study population: For the period 1984-6, there were 29 entries in the Kaugia health subcentre birth register, and 85 at MDH (Wosera residents only). Of these 114, 77 were found and interviewed; 16 women had migrated out of the area; and 17 women were not found. Four entries were either duplicates or were from women who had delivered twice in the 2 year period. In this latter group, the most recent delivery was enquired after at interview. [1]

Kaugia health subcentre administration had been erratic during 1986 and some deliveries there had not been recorded in the birth book. During village visits, 5 women were identified and interviewed who had delivered in an institution but who had not been listed in the birth registers. A further 7 women who delivered in a health facility early in 1987 and were not in the PNGIMR study area were also interviewed, giving a respondent total of 89. Fourteen births occurred in 1984, 37 in 1985, 31 in 1986 and 7 in 1987.

Characteristics of the women: Twenty eight percent (25) of women were privileged: either they or their husband were or had been clerks, teachers or army staff; or their husband was away in employment (and was therefore earning cash); or their husband owned a trade store

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1 The interview coverage of 82% (77/94) was a commendable achievement considering the incomplete name and residency data of many hospital birth register records, and was thanks to the local knowledge and field skills of DL

or a public motor vehicle business.

Mortality in previous births: Neonatal deaths in infants previously born to women delivering in hospital appeared little different the mortality rates derived from women in the longitudinal study of pregnancy (chapter 5). [2]

Place of delivery:

Sixty five women **delivered in a health facility**: 38 (59%, data missing=1) decided antenatally to do so. Reasons given where:
-they wanted to deliver in hospital (23 women);
-they had been ill antenatally (10 women);
-previous obstetric or neonatal problems (3 women);
-they thought they were overdue (2 women).
The remaining 26 (40%) went because of problems arising during labour. These included excessive pain or delay with delivery.

Twenty three women (26%) **delivered in the village** (1 delivered on the way to the health facility). Of these group, 18 (78%) went because the placenta was retained.

Transportation: Forty four (49%) reached the facility by ambulance or mission vehicle. Twenty five (28%) went in the village car, their families vehicle or a public motor vehicle. Twenty (23%) walked or were carried.

Reported care at health facilities: None of the women complained about treatment at Kaugia health subcentre: however, the PNGIMR team were associated with the service provision from this centre, and women dissatisfied with service there may well have been inhibited to reveal this at interview. Of those admitted to Maprik Hospital, 11% (7/61) complained about the care. Table 8-1 outlines the reported clinical details of these 7 women, and details their complaints. Most complaints concerned deliveries in the year preceding the study, and seem to reflect poor communication on the part of the nurses.

2

Stillbirths=5, neonatal deaths=19, later deaths=23, out of 250 births: neonatal death rate approximately 76/1000; compared with 88/1000, derived from birth histories collected in the same way from pregnant women in the Wosera (table 5-6).

Table 8-1. Criticisms of care received at MDH obstetric unit by Wosera women

BIRTH NUMBER	DATE	REASON FOR ACCESSING	MOTHERS COMMENTS
6	11/84	sick antenatally; long postnatal convalescence	told if she did not do as instructed, she would be sent to jail
10	03/86	?post-mature	delivered alone in the labour ward; nurses angry: "why bother come to hospital if you are going to deliver like a villager?"
1	10/86	delay	told her off for not delivering quickly
2	08/86	retained placenta	"too many Wosera, don't like helping them"
3	08/86	wanted to deliver supervised	became angry with her, does not know why
1	05/86	wanted to deliver supervised	disliked vaginal examination
4	05/86	previous retained placenta	got angry with her, does not know why

4 DISCUSSION

Although there is no population data to compare with the socio-economic information from women delivering in hospital, they are clearly a privileged group. This is consistent with the finding that mean birthweight of Wosera women delivering in Maprik was higher than that determined by a village based study (chapter 9).

Retained placentae in this study were the main reason for postnatal admission, confirming this condition as an important complication of labour in Wosera women.

Some of the women were obviously angry or distressed by the way they were treated at MDH. It is highly likely that their experience would be discussed by women in the village and is unlikely to encourage other women to attend the hospital even when they are in need.

5 SUMMARY

A. Wosera women that deliver in a health facility are often from a more privileged background.

B. A retained placenta was a common reason for maternal postnatal admission.

C. There was some consumer dissatisfaction with the attitude of health staff at the local hospital.

MAPRIK HOSPITAL BIRTHWEIGHT DATA 1984-87

1 INTRODUCTION

Institutional birth data from the district hospital at Maprik were analyzed in order to:

i. examine how women were using obstetric hospital services;
ii. examine secular, seasonal and geographic trends in birthweight;
iii. provide data to compare birthweight for Wosera hospital deliveries with results from village births (determined by the postnatal study described in chapter 6).

2 METHOD

The Maprik Hospital ward delivery book recorded information from 1984, with no missing periods evident. Data extracted were: age, parity, and village of origin of mothers; if the birth had occurred before arrival in hospital; delivery complications; infant sex; and birthweight. Birthweight had been measured in kg using a standard beam baby balance, which was in calibration when examined by PNGIMR staff in 1987.

3 RESULTS

Birthweight measurements of less than 1 kg were excluded (n=7). Birthweight had not been recorded in 52 babies, leaving 1499 birth events. Maternal parity was missing from a further 43 records.

3.1 Obstetric data

Coverage: The annual number of births in the Maprik district can be estimated at 3729 deliveries (using the national crude birth rate of 33.9/1000 and Maprik Hospital coverage of 110,000 people). Over the 4 years, Maprik hospital admitted 14% of this estimate (1551/3729*4). Over a third of deliveries (526/1499) were from the Maprik subdistrict (Maprik town and adjacent villages).

Women admitted postnatally: Born before arrival (BBA) accounted for 15% (222/1499) of deliveries. Almost a third of mothers with BBA infants had obstetric complications (32%, 70/222), compared with 8% (105/1277) for women delivering in hospital.

Obstetric complications: The commonest complications were twin deliveries and retained placentae, accounting for 3.5% each of all admissions (table 9A-1).

Obstetric complications and Wosera births: postnatal admissions were more common in women from the Wosera: 25% of deliveries were BBAs (26/131). This was higher than the rest of rural Maprik (16%, 116/740), but not significantly so (χ^2=1.3). This was associated with more obstetric complications recorded in Wosera women: 17% (22/131) had complications, compared with 13% (97/740) in the rest of rural Maprik.

Parity: Twenty six percent (383/1456) were primigravidae, and this group had significantly fewer complications (8%, 32/383) compared with parous women (13%, 138/1073: χ^2=5.98, p<0.05). Retained placentae, twins, breech and postpartum haemorrhage were all less common in primigravidae.

However, 41% (10/24) of the stillbirths occurred in primigravidae, and 42% (3/10) of delay in delivery was reported in this group. The stillbirth rate was 16 per 1000, similar to that found in the Wosera retrospective birth history study (chapter 4, section 3).

3.2 Birthweight distribution

The maximum weight recorded was 4.23 kg. Figure 9-1 shows the distribution of birthweight measurements (rounded to the nearest one decimal place) by sex of the infant. The distribution is biphasic, with peaks at 2.6 and 2.9 kg. This may reflect digit preference.

Figure 9-1. **Distribution of birthweight for all live births at Maprik hospital, 1984-87** (excludes sex unknown: n=14)

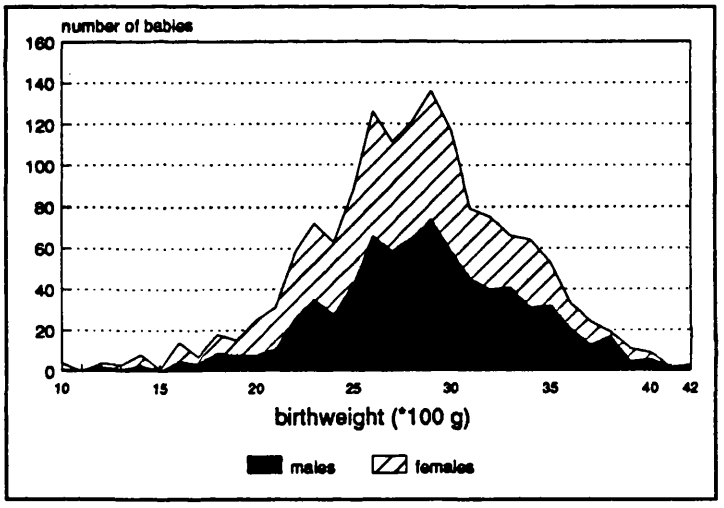


Table 9A-1 shows that mean birthweight was lower for stillbirths and twins. Women experiencing a delay in the second stage were few: mean birthweight in this group (3.1 kg) was higher than the overall level, but even in this group the mean was still less than Western average birthweight values (often greater than 3.3 kg: for example, WHO 1984).

3.3 Birthweight of live singleton infants

Mean birthweight: The mean birthweight by sex is shown in table 9-1. The sex difference was significant and consistent with findings from other countries (Kramer 1987). The male:female ratio was 1.09, similar to the ratio seen elsewhere in the country (Lea and Lewis 1975).

Table 9-1. Mean birthweight of babies from Maprik birth register 1984-7 by sex.

sex	mean birthweight	95% confidence limits
female	2.76	2.72 - 2.80
male	2.87	2.84 - 2.91
all	2.82	2.79 - 2.85
	t-test:p<0.0005	

Seasonality: A frequency plot of deliveries by month aggregated over the 4 years shows no seasonal pattern in numbers of babies born in hospital. Mean birthweight showed no trend with month of birth. The minimum value was in November (2.77 kg), and the maximum in September (2.93 kg).

Secular trends: Mean birthweight by year shows no detectable secular trend (table 9A-2).

Parity: The mean birthweight of infants born to primigravidae was 0.328 kg less than those born to parous women, a result that is clearly significant (mean birthweight in primigravidae=2.58 kg, 95% CI 2.55-2.62; mean birthweight in parous women= 2.91 kg, 95% CI 2.79-2.85).

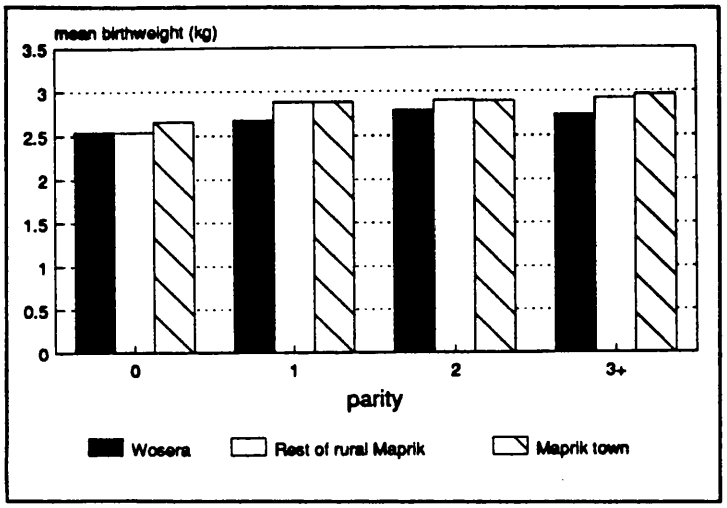
District analysis: The mean birthweight of Wosera infants is lower than any of the other areas (table 9-2).

Table 9-2. Mean birthweight of singleton live births by area of residence from Maprik hospital records

AREA	n	mean birth-weight	standard error
Wosera	122	2.69	0.044
Maprik town	507	2.87	0.022
Rural Maprik	699	2.79	0.019

The mean birthweight in Wosera primigravidae was very similar to the rest of rural Maprik, Maprik town and women from outside the district (figure 9-1, and table 9A-3). The difference in mean birthweight between first born and subsequent deliveries was 186 g for Wosera women compared with 376 g for all the rest. The difference in mean birthweight in table 9-2 is therefore largely accounted for by differences in birthweight of infants born to parous women.

Figure 9-2. **Mean birthweight of singleton infants born in MDH by area of maternal parity and area of residence**



The mean birthweight of Wosera women delivering in Maprik hospital was significantly higher than the mean birthweight determined at the postnatal study of pregnant women derived from a population based cohort (chapter 6, section 3.1.2). If mean birthweight within parity groups is compared, this trend remains consistent (tables 6A-3 and 9A-2).

4 DISCUSSION

Omission of complications by nursing staff completing the ward record book means the proportion of obstetric complications determined by this study represent a minimum estimate.

Twin deliveries and women with retained placentae were far more common than delay in the second stage. Third stage delay was common in Wosera village births (chapter 6, section 3.2.7). This hospital data suggests that in some women third stage delay results in pathological retention of the placenta requiring hospital admission.

Although primigravidae are at a higher risk of stillbirths than multigravidae, the cause of the very high frequency found in this study is not known. It may be the higher risk is magnified if access to obstetric services is poor.

The bimodal birthweight distribution suggests digit preferences by nursing staff. If the two peaks reflected a change in scales with a 300 g difference between them, then the mean birthweight by calendar year should have reflected this. It does emphasise the need for supervision of staff and checking of birthweight scales by obstetric ward supervisors.

The mean value is what could be expected from other data from lowland PNG (Aitken 1987). The absence of any seasonal pattern in birthweight suggests the birthweight data collected in

the Wosera postnatal study from March to December is not biased by season.

Wosera data: The higher proportion of BBAs in Wosera admissions suggests that Wosera women used the services when obstetric problems arose, rather than choosing to deliver there. Poor transport, cost and fear may be factors limiting the use made by Wosera people of Maprik obstetric services.

The lower mean birthweight in births to parous Wosera women compared with the other areas may reflect the declining nutritional status seen in Wosera women with advancing parity (chapter 4, section 3.7.2).

The higher mean birthweight of Wosera women admitted to hospital compared with the study of village births (chapter 6) suggests that Wosera women delivering in hospital are different in some way. Chapter 8 suggests that a higher proportion come from privileged families. This finding has important implications for the use of hospital birthweight data as a proxy for mean population birthweight: hospital data must be interpreted with caution on areas where most deliveries occur in the village.

5 SUMMARY

A. Obstructed labour was an uncommon complication of labour;

B. There were no seasonal trends in mean birthweight evident;

C. The mean birthweight of babies born to Wosera women admitted to hospital 1984-6 was greater than the mean birthweight of Wosera village births determined in 1987.

SUMMARY DISCUSSION

This chapter is divided into four sections. The first reviews the results from the various components of the maternal and neonatal health study in relation to study objectives (i) and (ii) (chapter 1, section 4.1); the second section reviews the results in relation to district management (objective iii, first part); the third section reviews the results in relation to national health policies (objective iii, second part); and the final section suggests areas requiring further research.

1 REVIEW OF RESULTS

1.1 Wosera newborns

The perinatal mortality rate was high. Low birthweight babies were common, and the mean population birthweight was low. Mortality and birthweight could not be explained by a large number of preterm infants or infants with acute growth retardation, as most were appropriately proportioned and term (chapter 6, section 3.1). However, twin births were more common than in Western populations (table 4-4), which is likely to increase perinatal mortality (table 2-5). Neonatal infection related to umbilical cord care probably resulted in serious morbidity and mortality risk (chapter 7).

1.2 Wosera women and childbearing

Maternal factors that might be important determinants of low birth weight and perinatal mortality were:

Fertility and nutrition: Fertility was higher than the rest of the province and country, but the levels were not as high as had been reported in some areas of PNG in the past undergoing demographic transition (Ring and Scragg 1973). Traditional cultural practices seem to limit fertility: relatively long birth intervals were demonstrated. However, there was evidence that birth intervals were becoming shorter (figure 5-3). This is consistent with the declining adherence to traditional practices.

Nutritional status of women was poor, and this is likely to directly influence fetal growth. Childbearing appeared to directly affect maternal nutrition (chapter 4, section 3.8.2). Longitudinal information on weight gain in pregnancy and postnatal weight loss (chapter 5, section 4.8.3) provided further evidence of the detrimental effect of childbearing on maternal nutrition.

Education: Maternal education was weakly associated with fewer child deaths (table 4-6). Female education reflects economic status, and differentials in the Wosera were not marked; a strong association was therefore unlikely.

Smoking: Smoking of traditional materials was widespread in pregnant women. Smoking reduces birthweight, and has been shown to increase perinatal mortality (Cnattingius et al.

1988). Although data on constituents of traditional tobacco is limited, it seems likely that smoking in pregnancy inhibits fetal growth in the Wosera.

Work: The relationship between strenuous physical work and birthweight is uncertain (Kramer 1988). However, in women whose dietary energy intake is marginal, energy expenditure is likely to influence their nutritional status before and during pregnancy. This study showed that strenuous physical work was common in pregnant women, reflecting traditional daily demands on women in the subsistence agricultural system (chapter 6, section 3.2.3).

Illness: Morbidity recording was limited in this study. At first antenatal visit, a recent history of illness was common, and usually had not been treated (chapter 5, section 4.6); postnatally, recent illness was associated with preterm births (chapter 6, section 3.3.2), emphasising the need for prompt and appropriate treatment of any illness episode in pregnant women.

Malaria: Malaria illness immediately preceding labour was not commonly reported, and did not seem to be associated strongly with preterm deliveries. This suggests that in stable malarious areas malaria may effect birthweight through an effect on fetal growth rather than length of gestation.

Plasmodium falciparum malaria and anaemia were more common in primigravidae at their first antenatal clinic visit, but there was no significant relationship between birthweight and haemoglobin when stage of gestation was controlled for. The high prevalence of malaria and anaemia in women of childbearing age (chapter 4, sections 3.8 and 3.9) suggests that effective malaria control could potentially reduce the amount of anaemia (Schofield et al. 1964).

Antenatal attendance: Mean birthweight increased with number of maternal antenatal clinic attendances. This may reflect that slightly more advantaged women are more likely to book early, and marginalised women may book late; or that chloroquine prophylaxis, even if taken intermittently, increases birthweight.

Delivery events: Labour appeared prolonged in some women delivering in the village, although prolonged rupture of the membranes was less marked (chapter 6, section 3.2.5). This did not seem to reflect obstructed labour with its associated perinatal mortality risk: no mothers required obstetric assistance with delivery; no woman had delivered previously by caesarian section; and no residual evidence of asphyxia was detected in newborns during the postnatal study.

Utilisation of obstetric services: Increasing institutional deliveries is one strategy for reducing perinatal mortality. The main obstetric service in the area was outside the subdistrict (Maprik District Hospital). Wosera women using MDH were frequently privileged, or had run into complications during labour (chapter 8).

1.3 Discussion

1. The high perinatal mortality, like a high infant mortality rate, indicates absolute poverty.

Both low birthweight and high perinatal mortality risk reflect poverty of the area, which had been shown in the Wosera by a rapid economic assessment (Heywood et al. 1986).

2. Poor hygiene as a result of poverty is likely to strongly influence perinatal mortality.

This is particularly true in the Wosera where most Wosera births take place in the village (chapter 5, section 4.4.3). While this study suggests that umbilical cord care may reduce the risk of serious neonatal morbidity (chapter 7), it is an example of a specific technique which does nothing to relieve the underlying deprivation (Newell and Nabarro 1989).

3. Health services have a role in reducing the severity of illness episodes.

Whilst socio-economic development is central to improving the health of mothers and their infants in deprived areas like the Wosera, prevention and treatment of illness in both groups of people may reduce the severity of morbid episodes and mortality levels.

4. The focus on birthweight internationally as a predictor of infant death has led to possibly misguided interventions that strive to reduce mortality by altering birthweight.

Control of maternal malaria or food supplementation in pregnant mothers may increase birthweight and reduce perinatal mortality by shifting the birthweight curve to the right (Ashworth and Feachem 1985). The effect of these interventions is now being questioned (Rush 1987). A large increase in birthweight could push the curve too far to the right, so the higher mortality seen in the up-going section of the weight specific mortality curve for the population becomes dominant (Offringa et al. 1986). Smaller increases could potentially reduce mortality, as the "optimal" birthweight of minimum mortality is usually slightly greater than the mean population birthweight (chapter 2, section 6). However, these suppositions are based on observational studies, and convincing evidence from developing countries does not exist. In the Wosera the overall risk of perinatal death was high, suggesting the birthweight specific mortality curve showed high mortality levels whatever the birthweight: in such circumstances, a focused intervention causing a small increase in population birthweight is unlikely to have a demonstrable effect mortality.

5. Maternal and neonatal health interventions should be concerned with maternal and neonatal health, rather than focus on birthweight.

Chloroquine prophylaxis is recommended to mothers living in endemic malarious areas, as malarial intervention tends to increase birthweight-whether this in itself reduces mortality significantly has not been demonstrated. An alternative way to judge the usefulness of

chloroquine prophylaxis is on its potential effect on the mother: primigravidae are particularly affected by malaria, with increased parasite levels, anaemia and probably, more morbid episodes: in these circumstances, it seems logical to recommend chloroquine prophylaxis to improve **maternal** health in pregnancy.

Similarly, a chronically poorly nourished pregnant woman is unlikely to gain much respite from her condition and the morbidity arising from it by a few supplementary meals during pregnancy, even though this was given at a time which had a maximum influence on birthweight. On the other hand, economic development with her community tending to improve the nutrition and health of the whole group is more likely to have a sustained impact on her health and that of her children.

2 IMPLICATIONS OF THE STUDY FOR LOCAL MANAGERS

Some study findings are specific to the Wosera subdistrict, and may be relevant to provincial administrators, combined with comments made in section 3 concerning implications for national health policy. [1]

2.1 Social and economic development are central to solving the poor health of the Wosera

The low income of households in the Wosera (chapter 1, section 2.3), the visible poverty, and the high infant mortality rate denote absolute poverty (Newell and Nabarro, 1989). Improvements in health require social and economic development. Health services will have little impact on the well-being of the population, their nutrition or morbidity burden. The Wosera appears particularly disadvantaged in comparison to the rest of the Province.

2.2 Female education needs improving

Understanding of health information and appropriate use of health services would be facilitated by primary education. Despite a network of operational primary schools in the Wosera, the majority of young women had not attended school at all (table 4-3). In Kaugia school, female enrolment was well below that of males (chapter 3, section 1.2) and higher absenteeism in girls was reported. Enrolment and attendance patterns at schools in the Wosera and elsewhere in the province needs examining. Focused campaigns encouraging female primary education could improve their basic education.

2.3 Health services in the Wosera could be improved

Health services in the Wosera were patchy (Heywood et al. 1986), and deteriorating, in common with many other rural areas of PNG (Bouten 1987). Experience elsewhere in the country suggests services alone could reduce the infant mortality to about 70/1000 (chapter

[1]Section 2 and 3 will provide the basis for summary reports which will be sent to East Sepik Provincial Government and the PNG National Health Department.

3, section 2.1.1). This study showed main constraint to maternal antenatal attendance in the Wosera were health service deficits (chapter 4, section 3.1).

To prevent health services deteriorating further, money for rural health services should be maintained, and management input should be boosted. Health workers could be part of development projects in the area (Garner 1988), as is proposed in the smallholder market access and food supply project that began in 1989 (Department of Agriculture and Livestock 1987).

Obstetric care at Kaugia health subcentre was intermittent; Maprik hospital was outside the subdistrict, and the quality of obstetric care described by the consumers was lacking (chapter 8). Data from various sources suggested maternal mortality in the Wosera was high (chapter 5, section 4.11); improving obstetric care could help reduce maternal deaths.

2.4 Pregnant women should be encouraged to stop smoking

In view of the known deleterious effects of smoking during pregnancy, a health education programme could be organized encouraging women to stop smoking in pregnancy. This could be organized through health workers, and included in community school teaching programmes.

2.5 Wosera women require access to family planning methods

The high fertility and the relationship between parity and nutrition indicate family spacing methods including contraceptives should be available to Wosera women. Despite a specially funded family planning project in Maprik, no contraceptive methods are available through health services in the Wosera (chapter 3, section 1.3.2) as mission services are Catholic, Wombisa Government Health Centre has no nurse, and local priests halted a community based pill distribution scheme.

Tubal ligation sterilisation could be performed in Maprik. Family planning clinics could be run from Wombisa health centre if a nurse was placed there. Discussion between senior health staff and the local Bishop are necessary in order to try and salvage the community based distributor scheme. Although the project began in 1985, no liaison has yet occurred (Bill Lapai, personal communication).

3 IMPLICATIONS OF THE STUDY FOR NATIONAL HEALTH POLICY

The implications in section 2 are of a general nature. They may also apply to national health policy makers. There are no easy solutions, and factors such as socioeconomic development and improved maternal education are likely to be very important. The following section has limited scope, and concerns some specific aspects of health care delivery that national health managers[2] may wish to consider.

3.1 Improve organisation of antenatal care

Antenatal care in PNG may be rushed and of poor quality (Garner and Giddings 1985), if it is provided at all. Women often present late in pregnancy, and are only seen once (table 3-1). Some of the problems with antenatal care provided through MCH clinics is outlined in table 10-1. Clinics could be re-organized, and outpatient departments at health subcentres could play a role in care.

MCH clinics: Antenatal consultations take time, and health subcentre clinic staff need to allocate staff or time slots to antenatal care. This may mean reducing the frequency with which children under five are seen or weighed.

A divide between MCH nurses and villagers is common. One nurse at Kaugia told me that Wosera women were " stupid, and most could not speak *tok pisin*" (the lingua franca for PNG). Neither statements were true. Respect for the mothers is important: examinations done in public may be embarrassing, and privacy for abdominal palpation should be ensured. In some instances, use of cloth screens hung from poles is all that is required. Health workers should encourage women to come to clinic even if they are unsure that they are pregnant: even if the health worker cannot confirm the diagnosis, the woman could be screened for anaemia. If pregnancy seems likely in a nulliparous woman, then chloroquine prophylaxis could be commenced, and the woman reviewed at the following clinic.

Outpatient clinics: Mothers attend outpatient clinics with sick children. This contact with health workers could be used more. If she is visibly pregnant, her antenatal booking status could be checked. In addition, health workers confronted with a sick woman may omit to consider whether she is pregnant. Use of outpatients has been useful in improving immunisation coverage in PNG (Keith Edwards, personal communication), and could help improve antenatal coverage and care.

Improving the care of illness in late pregnancy could potentially reduce preterm deliveries that result from treatable febrile illnesses (chapter 6, section 3.2).

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[2] The national health department can influence policy in provinces in a variety of ways. They provide guidelines for health workers, work on standard management schedules, influence health worker training, train supervisors, and organize health systems research.

Table 10-1. Objectives and problems with antenatal clinics in PNG

OBJECTIVES	PROBLEMS
preserve maternal dignity	often forgotten in a busy clinic
detect women at risk and advise health centre delivery	delivery care often poor quality; women too frightened to go
detect and treat abnormal conditions early	women often book late
provide routine drug prophylaxis and supplementation complex;	women present late; drug regimes too poor compliance with preventative regimes
prevent neonatal tetanus with toxoid immunisation	maintenance of vaccine supply
advice and discussion about childbearing ("health education") preliminary discussion about family planning	too busy; sometimes health worker has negative attitudes to patients no family planning service available

3.2 Review activities of antenatal care

3.2.1 Drug regimen

The national regimen for pregnant women (Papua New Guinea Society of Obstetrics and Gynaecology 1986) clearly needs simplifying. Secondly, the thresholds for the definition of anaemia in the manual need adjusting: if they had been followed, 21% (59/280) of women in this study would have been admitted to hospital for intravenous iron dextran infusion (table 5-11).

Chloroquine: *Plasmodium falciparum* malaria was not shown to be increased in parous pregnant women in this study. Studies examining the effect of malaria control on birthweight have only ever shown a convincing, significant affect in primigravidae. If low compliance with prophylaxis is confirmed from other areas of the country (see section 4) then standard management should be changed to with a better chance of maternal compliance, for example:

> Primigravidae, or women that are clinically anaemic (or with a haemoglobin of less than 8 g/dl): chloroquine treatment course, then weekly chloroquine prophylaxis.
>
> Multigravidae, not anaemic: supply with chloroquine course for presumptive treatment of malaria should they become ill at home.

Folic acid: Routine supplementation is probably unnecessary, although it may be of some value in poorly nourished women (Mahomed and Hytten 1989). In this population, a 5 mg of folic acid could be taken with chloroquine for women on prophylaxis; and for women with moderate anaemia, folic acid could be taken daily with ferrous sulphate.

Iron: oral ferrous sulphate, 200 mg daily as a daily dose could be taken with folic acid in women with haemoglobin concentrations of less than 8 g/dl. Women with a haemoglobin less than 7 g/dl in early pregnancy, anaemic women in late pregnancy, or in those whom

compliance is likely to be poor, could be encouraged to attend outpatients for parenteral iron dextran (30 mls). Women with a haemoglobin of less than 7 g/dl in late pregnancy should have a blood transfusion where possible.

Mebendazole: The potential teratogenic effects of mebendazole mean that it should not be used until after 16 weeks gestation. However, this recommendation is likely to confuse health workers and mothers, and the drug likely to be forgotten or given at the wrong time. This drug could be excluded as intestinal worm density in PNG adults tend to be low, and the evidence that helminths are contributing to antenatal anaemia is slim.

3.2.2 Health education

Communication with mothers at MCH clinic is often poor (Reid, 1984), and could be improved with supervision and in-service training. Discussion of the delivery and the problems that can occur is sometimes helpful. This can be done by a verbal rehearsal of the birth with the mother. Hygiene at delivery can be discussed. Packs as used in this study could be issued, possibly charging a nominal fee to cover pack cost.

3.2.3 At risk mothers

Antenatal clinics are used to detect "at risk" women. Health staff may recommend that women with a history of stillbirth or retained placenta should deliver in a health facility. Detecting women at risk of delivering a low birthweight infant by measuring their mid upper arm circumference, weight gain or increments in uterine fundus height is often recommended to detect growth retardation. However, it is difficult to know what action can be taken should any of these indicators show slow rates of fetal growth. Emphasis on fetal growth monitoring runs the risk of implicit criticism of the mother should the health worker detect growth slowing, and likely to cause the mother unnecessary worry and guilt.

3.3 Improve care at delivery

Improving maternal health care requires providing basic delivery facilities. While PNG is striving towards this, and institutional deliveries are rapidly increasing (chapter 3, section 1.4.3), the quality of care in rural areas may be poor, and staff may be unsympathetic to the needs of mothers, as was seen from consumer study reported in chapter 8 (table 8-1). Health workers should be encouraged, supervised, and receive regular in-service training by senior health staff.

3.4 Review national family planning programme

The National Health Plan (1986) aims for 100% availability of contraceptives through health subcentres by 1989: 64% of these units are run by Catholic agencies. This fundamental contradiction has been noted already (Reid 1984; Groos and Garner 1989) and needs facing at a national level.

4 FURTHER RESEARCH

This study raises further questions about maternal health care delivery in PNG. Further valuable work could be done as part of health programmes aimed at strengthening health care at national and provincial levels (Thomason 1988). National research institutions, such as the PNGIMR, should be collaborating with the health department, the University of PNG and other interested agencies. Some areas of useful future research may include:

4.1 Evaluation of antenatal care and prophylaxis compliance

In reviewing national antenatal drug schedules, rapid assessment of the coverage and range of care at antenatal clinics could be done. Interviews with nursing staff should reveal drug prescribing habits, and the nurses impression of the level of compliance. Clinic visits, interviewing mothers, counting tablets and performing urine tests for 4-aminoquinoline metabolites or analysis of capillary blood samples for chloroquine by ELISA, should give a more impartial measure of adherence to national protocols, and assist in designing a more realistic schedule, such as the one suggested in 3.2.1.

Some small scale preliminary studies are needed to evaluate Dill-Glazko urine test and blood chloroquine ELISA sensitivity and specificity to recent chloroquine prophylaxis ingestion.

4.2 Evaluation of standard management of anaemia in women

Anaemia as defined by Western criteria was widespread in both pregnant and non-pregnant women in this population. Definition of anaemia, and treatment protocols using anti-malarial, iron and folic acid preparations need evaluating in lowlands populations living in malarious areas. Part of this may included *in vivo Plasmodium falciparum* drug resistance testing, as drug resistance may cause chronic malarial illness despite treatment with 4-aminoquinoline metabolites; and thus lead to iron deficiency (McGregor 1982; Philip Harvey, personal communication).

4.3 Interventions to reduce maternal mortality

Delay with delivery of the placenta was common in Wosera village births (chapter 6, section 3.2.7). Maternal mortality in the Wosera seemed high, and appeared to be related particularly to retained placentae and postpartum haemorrhage (chapter 5, section 4.11). Postpartum haemorrhage is the commonest cause of maternal death in coastal PNG (Department of Health 1986), whereas puerperal sepsis is common in the Highlands. Assessment of the various possibly helpful interventions is required. Detecting and treating antenatal anaemia may be important (section 4.2); home remedies (such as early breast feeding or gentle uterine fundus massage) to speed placental delivery could be useful if accepted by the women.

4.4 Evaluation of a provincial programme improving umbilical cord care through antenatal clinics

The high compliance with the umbilical cord care packs in this study (chapter 6) may have been due in part to the relationship of the team with the village women. The intervention needs testing through routine services on a larger scale to examine if the packs are used. This could be combined with monitoring of antenatal clinic attendance and pregnancy outcome by MCH supervisors at district or provincial level.

4.5 Determination of national levels of smoking in pregnancy

Cross-sectional studies through antenatal clinics in different provinces, using the type of questions used in this survey (appendix 4), could be useful baseline information before launching campaigns encouraging women to stop smoking in pregnancy.

4.6 Consumer surveys of the quality of obstetric care

The retrospective consumer survey described in chapter 8 was logistically complex, but similar information could easily be obtained by sensitive interviews as patients leave hospitals ("exit" interviews: Lucy Gilson, personal communication). This could be used in assessing the quality of care provided, as part of schemes to improve this through management and training.

4.7 Impact of family planning programmes on maternal nutrition

The relationship between nutrition and fertility shown in this study warrants the setting up of comprehensive family planning in the Wosera subdistrict. Evaluation of the impact of a family planning programme on maternal nutrition could be a useful research study within such a programme.

4.8 Difference in birthweight between Highlands and lowlands regions

This study has not answered why Highland women have larger babies than lowlands women (chapter 3). Further comparative research on this could be useful, but it is difficult to see how such research may change health care practices.

4.9 New malaria interventions: the malaria vaccine

The Wosera has been chosen as a site to test future malaria sporozoite vaccines (Alpers et al. 1987). New methods of malaria control remain relevant to pregnant women, particularly primigravidae; and to young children, in whom malarial morbidity is high. On the other hand, it seems unlikely that a vaccine is likely to have a measurable effect on mortality in under five year old children while such a high proportion die in the perinatal period.

5 WOSERA WOMEN AND PROJECT PARTICIPATION: FINAL REMARKS

Drug prophylaxis was unpopular and compliance poor. The prophylaxis regimen had been emphasised from the outset and followed up through antenatal clinics (chapter 5). Women felt that taking medicine to prevent illness was illogical, and may "weaken" the effect of the medicine should the illness occur. Secondly, an intervention designed to increase the baby's birthweight was regarded with suspicion, as this was equated with an increased risk of obstructed labour and death of the mother or baby.

Although introduced late in the project, use made of the umbilical cord care packs was very high. Care of the cord were a concern to the mothers, and the intervention understood.

As health workers, many of us try to impose on our patients what we feel is right for them. Perhaps we should learn to listen a little more.

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appendix 1

TABLES

Table 4A-1. Villages selected for survey by clinic zone

Kunjingini zone	Kaugia zone	Jambitanget zone
Abusit	Balampta	Jambitanget
Gwiningi	Kumunigum	Jipako
Kunjingini	Kutigum	Lingu
Nale	Numbunge	
Palgere	Talengu	
Patigo	Wisogum	

Table 4A-2. Algorhythm for age estimation in women used throughout the Wosera study (developed by Monasseh Baea PNGIMR)

How old are you?	YES: age known
?	
Do you have an army census card?	YES: age established
NO	
Have you been to school?	YES: find out and year school completed. Most children enter school at 7-8 years of age, so add 8 to the completed grade at school and then subtract from the year she finished at school. eg completed grade 6 in 1974: 6 + 8=14; so estimated dob: 1974-14= 1960
NO	
How old is your first born child?	estimate age of first born from mother or relatives (adolescent, unmarried or woman; married, no children; married, with children; if the latter, how old was the son or daughter when they had children and estimate age of the grandchildren)
PLUS	
Did you get married when you were young?	YES: estimate age at marriage of 19
NO	
Did you wait a little/long time before years getting married?	YES: estimate age at marriage around 22-25
	USE age at marriage + 1 + age of first born and take away from 1987 for year of birth

Table 4A-3. Survivorship information on everborn children for all women seen at the cross-sectional survey

MATERNAL AGE (yrs)	RETROSPECTIVE BIRTH HISTORY: alive now		still-births		deaths first week		deaths after first week		twin deliv	women seen	perinatal mortality	live births now dead
	m	f	m	f	m	f	m	f	n	n	per 1000	(%)
15-	19	32	0	0	1	4	2	1	0	197	84.7	13.6
20-	107	97	4	4	6	8	9	8	4	208	90.5	13.2
25-	214	188	9	4	15	13	23	22	13	188	84.0	15.4
30-	300	241	4	7	18	21	38	25	17	148	76.5	15.9
35-	337	321	3	4	12	21	43	34	15	137	51.6	14.3
40-	244	256	6	3	14	14	23	21	13	88	63.7	12.6
45-49	110	109	1	3	7	9	24	11	3	42	73.0	18.9
TOTAL	1331	1244	27	25	73	90	162	122	65	1008	69.9	14.8

Table 4A-4. Infant mortality estimation from birth histories and parity ratios using a modification of the Brass technique (Department of Social and Economic Affairs 1984)

	WOMEN	MALES BORN	MALES DEAD	FEMALES BORN	FEMALES DEAD	RATIO M:F	M Pm(i)	F Pf(i)	TOTAL Pt (i)	M D m(i)	F D f(i)	TOTAL Dt(i)
AGE 15-	197	22	3	37	5	.59	.1117	.1878	.2995	.1364	.1351	.1356
AGE 20-	208	122	15	113	16	1.08	.5865	.5433	1.1298	.1230	.1416	.1319
AGE 25-	188	252	38	223	35	1.13	1.3404	1.1862	2.5266	.1508	.1570	.1537
AGE 30-	148	356	56	287	46	1.24	2.4054	1.9392	4.3446	.1573	.1603	.1586
AGE 35-	137	392	55	376	55	1.04	2.8613	2.7445	5.6058	.1403	.1463	.1432
AGE 40-	88	281	37	291	35	.97	3.1932	3.3068	6.5000	.1317	.1203	.1259
AGE 45-49	42	141	31	129	20	1.09	3.3571	3.0714	6.4286	.2199	.1550	.1889
TOTAL	1008	1566	235	1456	212	1.08	1.5536	1.4444	2.9980	.1501	.1456	.1479
PARITY RATIOS						P1/P2	.1904	.3457	.2651			
						P2/P3	.4376	.4580	.4472			

AGE (x)	M k (i)	F k (i)	TOTAL k (i)	M q (x)	F q (x)	TOTAL q (x)	M l (x)	F l (x)	TOTAL l (x)	M t(x)a	F t(x)a	TOTAL t(x)a
1	.9614	.5566	.7666	.1311	.0752	.1039	.8689	.9248	.8961	1.2829	2.1062	1.6792
2	1.0385	.9495	.9957	.1277	.1344	.1314	.8723	.8656	.8686	2.4959	3.3667	2.9146
3	1.0144	1.0157	1.0151	.1530	.1594	.1560	.8470	.8406	.8440	4.1590	4.6555	4.3966
5	1.0296	1.0573	1.0430	.1620	.1695	.1655	.8380	.8305	.8345	6.1003	5.9365	6.0191
10	1.0503	1.0891	1.0691	.1474	.1593	.1531	.8526	.8407	.8469	8.2420	7.2668	7.7693
15	1.0392	1.0814	1.0596	.1368	.1301	.1334	.8632	.8699	.8666	10.6639	9.0195	9.8685
20	1.0306	1.0711	1.0501	.2266	.1661	.1984	.7734	.8339	.8016	13.5789	11.7820	12.7102

Table 4A-5. Mean anthropometric values and standard errors (SE) of women at the cross-sectional survey

PREGNANCY	AGE GROUP		HEIGHT		WEIGHT		WT FOR HT	BODY MASS INDEX	
	(yrs)	N*	mean	SE	mean	SE	mean	mean	SE
not preg	15-19	179	151.2	0.39	48.6	0.45	95.0	21.2	0.15
	20-24	182	153.0	0.39	50.6	0.49	96.9	21.6	0.15
	25-29	165	152.8	0.39	48.4	0.45	93.0	20.7	0.16
	30-34	128	151.9	0.43	46.6	0.51	90.3	20.1	0.17
	35-39	123	150.2	0.51	44.5	0.50	87.8	19.7	0.18
	40-44	82	150.0	0.51	44.4	0.63	87.9	19.7	0.23
	45-49	40	147.7	0.77	41.4	0.86	84.0	18.9	0.31
	ALL	899	151.6	0.18	47.4	0.21	92.2	20.6	0.07
pregnant	ALL	109	152.0	0.51	50.7	0.57	98.4	21.9	0.18

		MUAC		TRICEPS SKINFOLDS		SUBSCAPULAR SKINFOLDS	
		mean	SE	mean	SE	mean	SE
not preg	15-19	23.6	0.14	11.6	1.02	13.9	1.02
	20-24	24.2	0.13	11.3	1.02	14.9	1.02
	25-29	23.7	0.14	9.9	1.02	13.2	1.20
	30-34	23.5	0.18	8.7	1.03	12.3	1.02
	35-39	23.3	0.18	8.4	1.03	11.9	1.03
	40-44	23.3	0.23	8.3	1.04	11.8	1.04
	45-49	23.0	0.32	7.9	1.06	10.7	1.05
	ALL	23.6	0.064	9.8	1.01	13.1	1.01
pregnant	ALL	23.3	0.150	9.5	1.02	13.6	1.02

* missing data: 1 woman age missing (excluded from all analysis)
1 woman missing weight
1 woman missing sub-scapular skinfolds

Table 4A-6. Analysis of Variance of weight and body mass index and age and parity on weight of non-pregnant women in the Wosera. (2 women were excluded because of missing data)

MODEL EXAMINING WEIGHT, AGE AND AGE*AGE AND PARITY GROUP, AND TESTING FOR AN INTERACTION
Dependent variable: weight

Source	DF	Type I SS	Mean Square	F Value	Pr > F
AGE	1	4545.8996	4545.8996	131.25	0.0001
AGE*AGE	1	452.1726	452.1726	13.06	0.0003
PARITGP	4	1437.4092	359.3523	10.38	0.0001
AGE*PARITGP	4	181.1840	45.2960	1.31	0.2653
Error	888	30755.1185768	34.6341425		
Corrected Total	898	37371.7839155			

MODEL EXAMINING BODY MASS INDEX, AGE AND PARITY GROUP
Dependent Variable: BMI

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	5	609.32342852	121.86468570	30.47	0.0001
Error	892	3567.51940859	3.99946122		
Corrected Total	897	4176.84283711			

R-Square	C.V.	Root MSE	BMI Mean
0.145881	9.7110921	1.99986530	20.59361897

Source	DF	Type I SS	Mean Square	F Value	Pr > F
AGE	1	492.981293	492.981293	123.26	0.0001
PARITGP	4	116.342136	29.085534	7.27	0.0001
Source	DF	Type III SS	Mean Square	F Value	Pr > F
AGE	1	64.64	64.64	16.16	0.0001
PARITGP	4	116.3421358	29.0855340	7.27	0.0001

Table 4A-7. Mean maternal height and weight by village, standardised for age and parity

village	mean weight (kg)	mean height (cm)	village	mean weight (kg)	mean height (cm)
Abusit	50.49	152.9	Kunjingini Stat.	54.99	155.9
Balampta	45.52	151.3	Kutigum 1	44.35	149.6
Guiningi	49.61	152.8	Kutigum 2	45.34	150.0
Jambi School	53.41	152.9	Lingu	44.47	148.5
Jambitanget	50.01	153.0	Nale 1	48.46	152.9
Jipako	45.11	151.1	Numbunge 1	46.24	150.2
Kaugia School	52.76	153.0	Numbunge 2	45.96	150.9
Kumunigum 1	47.54	152.4	Palgere	46.99	152.8
Kumunigum 2	44.07	149.7	Patigo	46.51	152.4
Kumunigum 3	45.93	150.9	Talengu	46.11	149.9
Kunjingini 1	48.95	154.0	Wisogum 1	44.86	149.1
Kunjingini 2	48.24	153.3	Wisogum 2	47.06	150.8

* Standardised to age 28.2 (the population mean), and parity uniformly distributed over the five classes (method of Searle, Speed, and Milliken 1980, referenced in Sas Institute Inc. SAS/STAT users guide 1988).

Table 4A-8. Spleen rates of non-pregnant women by age group

AGE GROUP	spleen rate (%)	N	hacketts grade 0	1	2	3	4	AES (Hack)	AES (cm)
15-19	59	178	73	34	46	25	0	1.91	5.35
20-24	49	182	93	24	38	27	0	2.03	5.20
25-29	61	165	64	26	42	31	2	2.09	5.53
30-34	53	127	59	20	32	16	0	1.94	4.69
35-39	54	123	56	22	15	27	3	2.16	6.29
40-49	51	121	59	20	28	11	3	1.92	5.22
ALL	55	896	404	146	201	137	8	2.01	5.37

(AES= average enlarged spleen)

Table 4A-9. Species infection by age group of women not pregnant at survey

AGE GROUP	N	NUMBER OF INFECTIONS BY PARASITE fal	viv	mal	f+v	v+m	f+m	f+v+m	SPECIES PARASITE RATES (%) fal	viv	mal	gametocytes
15-19yr	179	69	8	16	17	7	28	3	65	19	30	2.8
20-24yr	182	64	9	13	9	2	34	4	61	13	29	4.4
25-29 yr	165	54	4	24	10	1	20	4	53	11	30	1.2
30-34 yr	128	42	12	18	11	3	11	0	50	20	25	2.3
35-39 yr	123	40	8	10	8	1	13	2	51	15	21	3.2
40-49 yr	122	26	8	20	7	2	11	2	38	16	02	0.0
ALL	899	295	49	101	62	16	117	15	54	16	28	2.8

Table 4A-10. Spleen rates and species parasite rates by parity of woman

PARITY	N	SPLEEN RATE	NUMBER OF INFECTIONS							SPECIES PARASITE RATES			
			fal	viv	mal	f+v	v+m	f+m	f+v+m	falcip	vivax	malar	gametes
nullips	235	52	89	13	21	16	8	33	5	61	18	28	3.4
parity 1	138	60	43	6	12	11	2	24	4	59	17	30	4.4
parity 2-3	187	61	63	7	22	12	1	33	2	59	12	31	3.2
parity 4-6	204	52	65	13	31	14	4	19	2	49	16	27	1.0
parity 7+	136	50	35	10	16	9	1	8	2	40	16	20	2.2
ALL	900	55	295	49	102	62	16	117	15	54	16	28	2.8

Table 4A-11. Species parasite rates and blood chloroquine levels by village

VILLAGE	N	SPECIES PARASITE RATES			CHLOROQUINE LEVELS
		fal	viv	mal	(ng/l)
Abusit	80	49	16	14	1.4
Balampta	24	42	08	67	7.1
Guiningi	39	49	03	46	261.2
Wisogum 1	62	52	08	19	16.6
Wisogum 2	46	54	15	26	29.5
Jambitanget	80	62	09	32	55.6
Jipako	43	53	12	39	25.3
Kumunigum 1	41	61	24	24	13.2
Kumunigum 2	26	58	23	19	9.3
Kumunigum 3	36	56	11	22	15.6
Kunjingini 1	43	39	16	28	42.7
Kunjingini 2	28	61	32	25	7.8
Kutigum 1	37	49	24	30	10.0
Nale 1	15	07	33	40	162.5
Numbunge 1	43	61	14	30	13.3
Numbunge 2	50	76	14	34	3.4
Talengu	26	58	23	35	9.9
Lingu	28	50	14	29	66.9
Palgere	26	46	15	31	8.8
Kutigum 2	41	73	15	17	9.7
Jambi School	4	50	00	00	63.8
Kaugia School	14	43	14	00	3.2
Kunjingini Station	16	44	25	19	12.1

(fal=Plasmodium falciparum; viv=Plasmodium vivax; mal= Plasmodium malariae

Table 5A-1. Antenatal women recruited by village

village	women recruited	village	women recruited	village	women recruited
ABUSIT	13	KUMUNUGUM-2	11	NALE-1	5
BALAMPTA	4	KUMUNUGUM-3	15	NALE-2	1
BAPANDU	1	KUNJINGINI-1	7	NANGDA	3
BOBMAGUM	5	KUNJINGINI-2	10	NUMBUNGE-1	11
CHIGINANGU	11	KUNJINGINI-ST	3	NUMBUNGE-2	16
GWINYINGI	2	KUTIGUM-1	12	PALGERI	2
ISOGUM-1	11	KUTIGUM-2	9	PATIGO	8
ISOGUM-2	13	KWANJOMA-1	6	SERANGWANDU	12
JAMBITANGET	18	LINGU	1	TALANGU	3
JIPAKO	4	MANGUL	11	TUWAIKUM	12
KAMGE	6	MIKO-1	9	WAIGAMAGU	7
KAUGIA-SCH	1	MIKO-2	2	WEIKOR	2
KUMUNUGUM-1	13	MUL	5	YINDIGO	2

controls: BOBMAGUM (1), KUNJINGINI-2 (1), MOUNDU ( ), RUBUGUM-2 (1),TUWAIKUM (1)

Table 5A-2. Reported smoking habit by age group in pregnant women

age group (years)	smokers reported frequency: occasional (%)	daily (%)	% smoking in 3+ periods previous day	N
15-	35	14	7	43
20-	32	32	33	72
25-	32	40	36	68
30-	34	50	53	56
35+	18	56	52	39
ALL	31	38	37	278

Table 5A-3. Pregnant women who reported chewing betel nut daily, and who chewed betel nut during three or more periods in the previous day

AGE GROUP	daily chewer (%)	chewed in 3+ periods previous day (%)	N
15-	5	2	43
20-	6	11	72
25-	18	12	68
30-	20	20	55
35+	23	15	39
ALL	14	12	277

Table 5A-4. Pregnant betel nut chewing women: betel nut consumption the previous day compared with self classification of chewing regularity

	yesterday buai frequency (row percent) not	once	twice	3 plus	total
occasional chewer (n=151)	51	30	13	7	101
daily chewer (n= 38)	11	8	18	63	100

Table 5A-5. Mean birth interval by birth date of each interval's first child, where the first child survived 2 years or more (illustrated in figure 5-3)

year of birth of first child	birth interval (years) mean	st error	n
- 1964	3.83	0.703	6
1965 - 1969	3.87	0.373	28
1970 - 1974	3.09	0.165	75
1975 - 1979	2.99	0.126	156
1980 - 1984	2.94	0.065	293
1985 - 1987	2.28	0.057	59
ALL	2.96	0.054	617

Table 5A-6. Stage of gestation at first PNGIMR visit and whether already seen at MCH antenatal clinic

AGE GROUP (years)	0-20 weeks seen/women	21-30 weeks seen/women	31-40 weeks seen/women
15-	1 / 17	4 / 16	6 / 11
20-	4 / 17	8 / 28	13 / 28
25-	1 / 15	2 / 32	10 / 24
30-	1 / 14	2 / 21	14 / 21
35+	0 / 11	2 / 17	9 / 12
ALL	7 / 74	18 /114	52 / 96

Table 5A-7. Table of mean malaria parasite densities, size of spleen, chloroquine concentration, haemoglobin and free erythrocyte protophyrin by parity of mother, pregnancy status a and whether booked at clinic

PARITY GROUP	PREGNANCY STATUS + WHETHER BOOKED	N	mean values pf	pv	pm	spleen (cm)	chloro-quine conc	hb	fep	fep (n)
0	not preg	220	21.28	1.66	2.48	1.900	25.30	10.28	4.09	81
	unbooked	45	110.72	1.30	1.47	3.870	41.85	8.70	4.23	27
	booked	19	11.97	1.08	1.73	2.330	66.95	8.18	4.70	14
1	not preg	128	18.62	1.57	2.92	2.320	29.99	9.99	3.92	38
	unbooked	43	8.04	1.13	1.50	3.420	44.21	8.59	4.04	27
	booked	16	5.70	1	1	1.370	107.23	9.07	4.16	14
2+	not preg	486	10.58	1.55	2.54	2.100	26.18	10.05	3.94	150
	unbooked	123	6.25	1.23	1.74	2.080	57.92	9.07	3.45	84
	booked	41	3.23	1.57	1	1.120	50.25	8.91	3.81	31
all	not preg	834	13.87	1.58	2.58	2.080	26.50	10.10	3.98	269
	unbooked	211	12.13	1.23	1.63	2.740	51.21	8.90	3.70	138
	booked	76	5.05	1.30	1.15	1.470	63.50	8.77	4.09	59

Table 5A-8. Table of mean density of Plasmodium falciparum by urine positivity for 4-aminoquinolines, parity and stage of gestation

		DILL-GLAZKO URINE TEST POSITIVITY			
PARITY	GESTATION	NEGATIVE mean	N	POSITIVE mean	N
0	20-	16.0	7	2.6	14
	30-	33.2	8	5.6	19
1	20-	5.8	11	0.0	7
	30-	3.7	11	2.7	17
2+	20-	8.7	22	2.7	25
	30-	1.2	29	2.9	47

Table 6A-1. Details of newborns not seen until after 120 hour age

HOURS	SEQUENTIAL BABY NUMBER	WEIGHT AT VISIT	POSSIBLE REASON FOR DELAY
576	3	2.4	setting up phase
288	5	2.9	setting up phase
600	6	2.6	setting up phase
168	7	2.6	setting up phase
336	10	2.4	setting up phase
744	1	?	setting up phase
136	2	2.1	setting up phase
171	104	2.8	unknown (parity 9)
154	38	3.1	husband died during pregnancy
162	44	3.0	unknown
175	47	2.2	delivered 2 weeks after neonatal death in same village
150	139	2.4	delivered in provinical capital, Wewak
127	86	3.6	husband away; perineal tear

Table 6A-2. Mean birthweight by gestation of infant (refer figure 6-3)

GESTATION	BIRTHWEIGHT N	MEAN	STD
<37	5	2.04	0.321
37-	4	2.20	0.365
38-	33	2.48	0.325
39-	49	2.60	0.365
40+	30	2.62	0.298
ALL	121	2.54	0.358

Table 6A-3. Mean birthweight by parity group

PARITY GROUP	BIRTHWEIGHT N	MEAN	STD
0	23	2.43	0.305
1	23	2.58	0.411
2-3	30	2.61	0.352
4+	45	2.53	0.356
ALL	121	2.54	0.358

Table 6A-4. Mean birthweight by number of times attending antenatal clinic

times attended antenatal clinic	birthweight n	mean
1	8	2.325
2	36	2.492
3	37	2.557
4	31	2.574
5	9	2.700

Table 6A-5. Mean birthweight by postnatal assessment of chloroquine compliance

COMPLIANCE ASSESSMENT	N	MEAN	STD
poor understanding, poor compliance	8	2.29	0.514
good understanding, poor compliance	31	2.49	0.366
good compliance reported	70	2.57	0.340
good understanding, unknown compliance	2	2.50	0.000
erroneous regime	3	2.43	0.351
ALL	114	2.53	0.362

Table 6A-6. Mean birthweight by health, level of work in week prior to delivery and haemoglobin at first visit

	BIRTHWEIGHT		
	N	MEAN	STD
HEALTH			
well	109	2.56	0.354
fever	8	2.30	0.378
other illness	4	2.40	0.327
ALL	121	2.54	0.358
WORK			
rested	18	2.47	0.371
light work	61	2.59	0.350
heavy work	42	2.48	0.359
ALL	121	2.54	0.358
HAEMOGLOBIN (GM/L)			
0-	5	2.36	0.40
6-	24	2.49	0.42
8-	78	2.56	0.35
10-	12	2.57	0.29
ALL	119	2.53	0.36

Table 6A-7. Mean maternal head circumference in relation to birthweight

	maternal head circumference		
	n	mean	std
weight group (kg)			
<2.5	41	51.5	1.10
2.5+	68	51.7	1.27
all	109	51.6	1.21

Table 6A-8. Mean haemoglobin by stage of gestation at first visit

gestation	N	HB MEAN
1st quarter	1	10.40
2 quarter	34	8.83
3 quarter	47	8.41
4 quarter	37	8.69

Table 6A-9. Results of multiple linear regression on birthweight Generalised linear model procedure/SASPC, using stage of gestation at booking as a categorical ("class") variable at 3 levels (missing values n=2)

Dependent Variable: ponderal index

Source	DF	Sum of Squares	Mean Square	F Value	Pr > F
Model	3	0.61390672	0.20463557	4.23	0.0071
Error	115	5.56334195	0.04837689		
Corrected Total	118	6.17724868			

R-Square	C.V.	Root MSE	PI Mean
0.099382	8.8635146	0.21994746	2.48149265

Source	DF	Type I SS	Mean Square	F Value	Pr > F
booking stage	2	0.46438818	0.23219409	4.80	0.0100
haemoglobin at booking	1	0.14951854	0.14951854	3.09	0.0814

Source	DF	Type III SS	Mean Square	F Value	Pr > F
booking stage	2	0.37198588	0.18599294	3.84	0.0242
haemoglobin at booking	1	0.14951854	0.14951854	3.09	0.0814

Table 7A-1. Mid-study data review of fevers and umbilical cord sepsis in newborns (30 July, 1987)

SEEN AT 0-5 DAYS	well	(38)	febrile/cord infection	(16)
FOLLOW-UP	no morbidity recorded	(38)	died	(1)
			definite neonatal infection	(4)
			possible neonatal infection	(1)

Table 7A-2. Umbilicial cord care packs issued, and components used in village births (unknown =2)

	ISSUED		
USED	no pack	razor	clamp
traditional	57	3	1
razor		7	5
razor + spirit		22	3
clamp + razor + spirit			17
clamp + razor			7
ALL	57	32	33

Table 7A-3. Infants who developed definite and probable neonatal infections: condition at postnatal examination, and subsequent morbid episodes

NEONATAL SEPSIS CLASSIF.	ID	POSTNATAL: CORD CARE (membrane rupture)[a]	POSTNATAL: CORD STATE	POSTNATAL: TEMPERATURE	ILLNESS: AGE	ILLNESS: CLINICAL[b]	ILLNESS: TEMP	ILLNESS: FACILITY[c]
DEFINITE	32	trad (<9)	no comment	36.6	6	septic arthritis and N.I.	NR	Ma
	38	trad (<2)	infected	38.7	7	infected cord, sores in groin	NR	Ku
	43	trad(<0.5)	no comment	34.8	6	moist umbilicus, fever, vomiting	37.8	Ku
					23	fever, irritable, tender ankle	37.9	Ku
					27	fever, swollen breast, inflamed ankle joint (N.S.)	37.9	Ku
	45	trad (<1)	2mm dry	37.5	21	unwell, irritable, twitching (N.S.)	39.2	Ku/PG
	50	trad (<1)	flare	36.5	20	unwell, irritable, floppy (N.S.)	37.3	Ka/PG
	94	used new razor (<0.5)	12mm, clean	36.7	28	moderate pneumonia	NR	Ka
PROBABLE	24	trad (<0.5)	4mm	37.9	6	DIED; off feeds, diarrhoea	not used	
	37	trad(<0.5)	0mm, dry	38.7	5	fever, pustules on eyelid	36.0	Ku
	53	trad (<4)	flare	37.2	19	fever	38.9	Ka

Trad = traditional methods used to cut the umbilical cord

[a] Time in hours in brackets after cord care classification is the time in hours from reported membrane rupture to delivery. <0.5 is 0-29 minutes, and <9 is 8-8.9 hours

[b] N.S. indicates a diagnosis of neonatal sepsis by the clinician

[c] Ma = Maprik; Ku = Kunjingini; Ka = Kaugia; /PG indicates the infant was seen by PG

Table 9A-1. Mean birthweight of deliveries in Maprik Hospital 1982-7 and reported obstetric complications

complication	N	mean birth-weight (kg)	standard error
nil	1324	2.82	0.014
retained placenta	51	2.80	0.064
stillbirth	24	2.34	0.167
delay 2nd stage	10	3.07	0.163
breech	14	2.51	0.169
pph	16	2.90	0.112
twins	52	2.18	0.070
other	8	2.36	0.273
ALL	1499	2.79	0.014

Table 9A-2. Mean birthweight of live singleton births at Maprik Hospital by year of birth

year	n	mean	standard error
1984	314	2.86	0.029
1985	364	2.81	0.026
1986	371	2.79	0.025
1987	374	2.82	0.026
ALL	1423	2.82	0.013

Table 9A-3. Mean birthweight by parity group and maternal residence

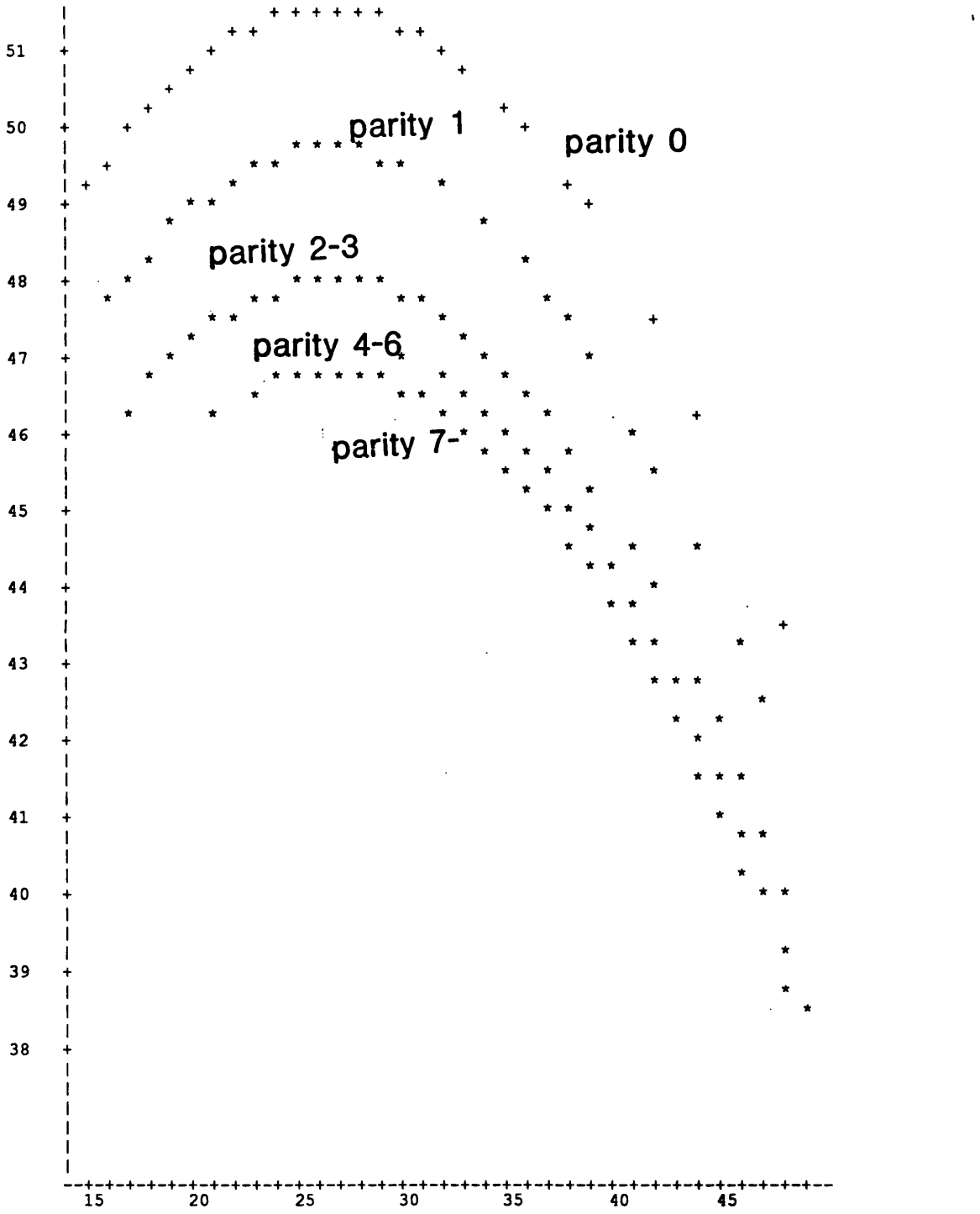
PARITY GROUP	AREA OF RESIDENCE											
	WOSERA			MAPRIK TOWN			OUTSIDE DISTRICT			RURAL MAPRIK		
	N	MEAN	SE	N	MEAN	SE	N	MEAN	SE	N	MEAN	SE
0-	36	2.55	0.076	108	2.66	0.045	31	2.61	0.085	194	2.54	0.031
1-	21	2.68	0.095	94	2.88	0.049	15	2.98	0.155	110	2.88	0.047
2-	18	2.79	0.112	70	2.89	0.053	17	3.12	0.091	94	2.89	0.044
3+	42	2.74	0.084	225	2.96	0.034	28	3.09	0.100	278	2.92	0.029
ALL	117	2.68	0.045	497	2.87	0.022	91	2.92	0.056	676	2.80	0.019

SE = standard error

appendix 2

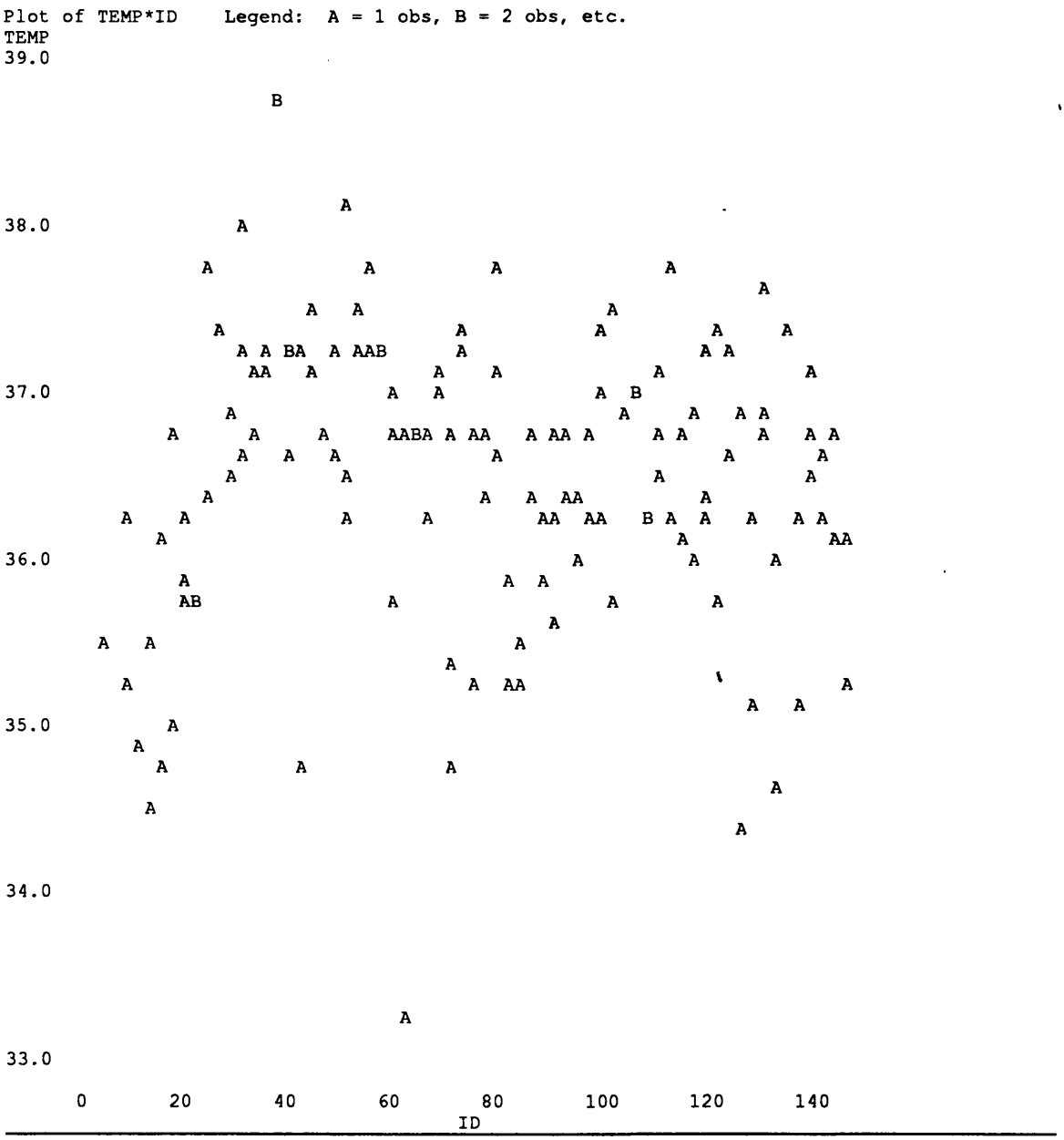
FIGURES

Figure 4A-1. Plot of predicted weight * against age for different parity groups



* predicted weights derived from a generalised linear model procedure in SAS/STAT using age as a continuous variable and parity group as a categorical variable.

Figure 6A-2. Plot of infant body temperature (corrected) by newborn sequential id number



appendix 3

VALIDATION STUDIES

1 THE INTENSIVE STUDY OF WEIGHT CHANGE IN THE FIRST 7 DAYS OF LIFE

Method: In order to examine the validity of infant weight measurements 1-5 days as proxy measures of birthweight, an intensive follow-up study was carried out on 27 consecutively born study infants. Each infant was seen four times:

first visit:	as soon postnatally as reasonably possible
second visit:	next day
third visit:	baby born before midday : date of birth + 3 baby born after midday : date of birth + 4
fourth visit:	baby born before midday : date of birth + 6 baby born after midday : date of birth + 7

Results: All first visits were carried out within 24 hours of birth. The table below shows mean weight by age of the infant. The mean weight varied little except at day 7 when an increment was evident.

Table of mean weights of the infants in the intensive follow-up study

AGE	N	MEAN WEIGHT (kg)
within 24 hours	25	2.56
weight aged 2 days	25	2.54
weight aged 4 days	24	2.55
weight aged 7 days	23	2.63

Multivariate analysis of variance showed that the differences between weights made on different days for the same individual was not a chance effect ($F=10.9$, $df=(3,19)$, $p<0.0002$). However, the effect is sufficiently small for use of any weight taken in the first 7 days as a proxy of birthweight. In particular, weights taken during the first 5 days were more or less identical.

2 DUBOWITZ GESTATIONAL AGE VALIDATION

Method: Dr and Professor Dubowitz visited the study in order to validate the Dubowitz scores carried out by the staff on the project. This was done formally by examinining babies together at the base hospital in Wewak (n=5); in the villages around Wosera (n=10); and in the hospital at Maprik (n=10).The main observer scored a number of infants which were then subsequently scored by Dr Dubowitz. There were 10 such infants, 2 seen in Wewak and 8 in the villages around the Wosera. The second observer, MEB, scored a number of babies with Dr Dubowitz who regarded his scoring as equivalent to that of the main observer, and quite accurate, and no formal comparison of this observer separate to the first was regarded as necessary.

Results of comparison between observers: The scores were remarkably similar for the 10 infants scored both by Dr Dubowitz and PG. The mean score for Dr L Dubowitz (LD) for the 10 infants was 38.75 weeks, and for PG it was 39.02 weeks, due to an overall mean score difference of 1.1 points.

The score difference ranged from -2.5 to +0.5 againest LD with a standard deviation of 0.994 giving a repeatability measure of 0.7 on 9 degrees of freedom (Michael Hills, LSHTM lecture notes). This measure depends on the units and the value of the measure, and is therefore best expressed as a coefficient of variation: 0.013. The correlation coefficient between the two scores was 0.98. These measures indicate low variation in the difference between LD and PG, and a high correlation between the two observers despite the range of values being quite narrow.

Discussion and comparison of scoring suggested that on 2 criteria PG was overscoring, and on 1, underscoring. The maximum error was approximately 2 points (about 0.5 weeks).

Results from Dr Dubowitz assessments of Sepik newborns: The gestation of the total of 25 infants examined by LD, which included hospital and village deliveries, was similar to that found in the main study. The minimum gestation was one infant of 36.9 weeks, and the maximum one of 40.7 weeks, with a mean of 39.1 weeks. This compares to the mean of 39.3 (n=121) from the main study, and the difference is not significant.

The table below shows the means of the other anthropometric parameters of the 25 infants examined by LD, excluding one infant diagnosed as suffering from an interventricular haemorrage.

Table of measurements from PNG infants seen by Dr L Dubowitz (n=25)

parameter	mean	sd
gestation	39.1	0.92
birthweight	2.79	0.472
length	43.1	2.71
head circumference	33.6	1.36
ponderal index	2.52	0.233

The overall length, head circumference and infant weight were slightly higher than those of the main Wosera study. A t-test performed on the birthweight of the 2 groups showed this difference was significant ($t=2.995$ on df 144, $p<0.005$). This probably reflects the higher mean weight seen in hospital deliveries compared with those in the village, which was also shown comparing the study mean weight with the mean weight of Wosera infants born in Maprik Hospital 1984-87 (chapter 7).

3 MEAN GESTATION BY THE 2 MAIN EXAMINERS

Mean gestation of the 37 babies examined by MEB was 39.6 weeks, and of the 81 examined by PG the mean score was 39.1. This difference (while significant statistically after birthweight of the infant has been accounted for in a generalised linear model: $f=6.9$ $p=0.0098$) it probably reflects that PG was more likely to examine babies reported ill or small.

Appendix 4

QUESTIONNAIRES

WOSERA FERTILE FEMALE SURVEY

PAPUA NEW GUINEA INSTITUTE OF MEDICAL RESEARCH, MADANG

1. ID.....

2. DATE SEEN(DD/MM/YY).....

3. VILLAGE.....

4. NAME_____ 5.MAN_____

6. FATHER_____ 7.MOTHER_____

8. AGE (YRS).....QUALITY.....

9. AS PLES MERI_____.....

10 YU YET BIN KARIM HAMAS PIKININI NAU OL ISTAP?.....

11. YU YET BIN KARIM SAMPELA NAU OL IDAI LONG BEL?.....

12. YU BIN KARIM SAMPELA NAU OL IDAI TAIM EM NUPELA YET?.....

13. YU BIN KARIM SAMPELA NAU OL I DAI BEHIN?.....

14. YU BIN KARIM TWINS?.....

15. if there is any suggestion of infertility, ask and record:

16. YU BIN PINISIM WANEM GRED LONG SKUL?.....

17. HAMAS YIA YU BIN SKUL KATAKIST?.....

18. NAU LONG OL SISTA BILONG YU, IGAT WANPELA EM I BIKPELA PINIS NAU EM IDAI?

no	=0,
yes, yanpela nogat pikinini	=1,
PREGNANT OR BABY NUPELA	=2,
child-bearing age only	=3,
yes, lapune pinis	=4.....

if (2), record NAME_____MAN_____

FATHER_____MOTHER_____

any details_____

_____

19. YEAR OF DEATH.....

20. YU TUPELA IGAT WANPELA PAPA OR NOGAT?.....

WHO TO INTERVIEW_____LIVING WHERE_____

FERTILE FEMALE SURVEY page 2

21. HEIGHT.....

22. WEIGHT.....

23. MUAC.....

24. TSF.....

25. SSF.....

26. examiner tsf/ssf.....

27. HB.....

28. PCV.....

29. FEP.....

30. BS.....

36. YU GAT BEL? (NO=0,YES=1,?=9).....

37. SPLEEN HACKETTS.....

CM.....

examiner.....

IF PREGNANT, OR POSSIBLY PREGNANT, NOW FILL IN THE
PREGNANCY SUPPLEMENT FORM
IF NOT PREGNANT WRITE HERE TREATMENT OR ADVICE GIVEN
GIVE ALL WOMEN A CENSUS CARD (DATE, ID , NAME, PLES, WHETHER PREGN)
16 January, 1987

WOSERA PREGNANCY FORM

PAPUA NEW GUINEA INSTITUTE OF MEDICAL RESEARCH, MADANG

PLEASE ENSURE FERTILE FEMALE FORM HAS BEEN FILLED IN, OR THAT THE WOMAN SHOWS YOU OUR CARD GIVEN AT THE FF SURVEY

USE THE SAME ID AS THE FF SURVEY

1. ID.....

2. DATE SEEN (DD/MM/YY).....

3. VILLAGE.....

4. NAME_____ 5. MAN_____

6. FATHER_____ 6. MOTHER_____

8. WANEM MUN YU NO BIN LUKIM SIK MUN INAP NAU?.....

9. MIPELA LAIK STORI LONG OL NARAPELA TAIM YU BIN IGAT BEL

order	name	dob	sex m=1 f=2	ples=1 HC=2	complications (twins, APH, PPH, etc)	born alive=1 still=2	now alive=1 dead=2	age *at death & cause
1								
2								
3								
4								
5								
6								
7								
8								
9								
10								
11								

twins: use a separate line for each and bracket together
*put units (y-ears m-onths; d-ays)

pregnancy form page 2

PREVIOUS ILLNESS

10. TIBI?.....

11. DOKTA KATIM BEL?.....

12. KISIM BLUT LONG PLASTIK? (blood = 1; imferon IV =2).....

13. NARAPELA BIKPELA SIK? detail.....

14. YU SAVE SMUK LONG OLGETA DEI?
no=0 sampela dei=1 olgeta dei=2.....

15. (a) YU SAVE SMUK WANEM SAMTING?

	nil	sampela dei	olgeta dei
Brus			
Stik			
Spia			
Sigaret			

15 (b) YU SAVE SMUK LONG WANEM?

pipe=1 leaf=2 niuspepa=3 (main method)

16 ASDE YU BIN SMUK? MONING?.....

no=0 LONG APINUN?.....

yes=1 LONG NITE?.....

GERAP LONG BIKNITE TU?.....

17 YU SAVE KAIKAI BUAI? LONG MONING?.....

LONG APINUN?.....

LONG NITE?.....

GERAP LONG BIKNITE TU?.....

FOR POSTNATAL WOMEN NOW GO STRAIGHT TO THE POSTNATAL FORM

pregnancy form page 3

18. YU BIN GO LONG KLINIK NAU SISTA SKELIM BEL BILONG YU, OR INO YET?

no=0, yes, usual MCH=2_____

state clinic if usual MHC_____

19. HAMAS TAIM YU BIN GO?.....

20. LONG DISPELA TAIM EM SKELIM MERISIN LONG YU OR NOGAT?_____

_____

_____

(not attended=0; attended, drug not given=1; drugs given as a single dose=2; drugs supplied for prophylaxis=3_____

21. WANEM KAIN MERISIN?

(find out types, what the women think they are for, what they have taken in the last week/month, and how many they take each time)

22. YU BIN SIK NAU LONG DISPLEA WIK? specify_____

_____

23. YU BIN KISIM SKIN HAT LONG DISPELA WIK?_____

_____

24. YU BIN KISIM MERISIN LONG HATIPOS(1), HAUS SIK(2), MERISIN MERI(3), OR YU HOLIM SAMPELA LONG HAUS NAU YU DAUNIM(4)?.....

25. estimate tablets of chloroquine ingested.....

26. FROM CLINIC RECORDS

ANTENATAL ATTENDANCES THIS PREGNANCY

date	observations	fundal height	clinical anaemia	treatment prescribed

antenatal records found ( ) checked clinic records and not found ( )

ANTENATAL FOLLOW UP FORM

VILLAGE: MOTHERS NAME: ID:

date	date
visit	visit
complaints	complaints
temp	temp
systolic	systolic
diastolic	diastolic
cuff	cuff
FMF	FMF
fundus	fundus
spleen (cm)	spleen (cm)
obs exam	obs exam
oedema	oedema
observer	observer
weight	weight
machine	machine
MUAC	MUAC
TSF	TSF
SSF	SSF
observer	observer
HB	HB
PCV	PCV
FEP	FEP
URINE	URINE
Rx returned chloro folate iron	Rx returned chloro folate iron
Rx supplied chloro folate iron	Rx supplied chloro folate iron
Rx other	Rx other
advise	advise
days last chloro	days last chloro

POSTNATAL VISIT FORM

PAPUA NEW GUINEA INSTITUTE OF MEDICAL RESEARCH

1. ID.....

2. DATE SEEN (DD/MM/YY).....

3. VILLAGE.....

HAMLET_____

4. NAME_____ 5. MAN_____

6. FATHER_____ 6. MOTHER_____

7. MIPELA BIN LUKIM YU LONG SURVEY? CHECK SURVEY CARD
SHE HAS NOT BEEN SEEN: FILL IN FERTILE FEMALE FORM ( )
PREGNANCY FORM ( )
POSTNATAL FORM ( )

SEEN AT FERTILE FEMALE SURVEY, BUT NOT PREGNANT:
FILL IN PREGNANCY FORM ( )
POSTNATAL FORM ( )

SEEN BY US ONCE OR MORE ANTENATALLY : FILL IN POSTNATAL FORM ( )

8. YU BIN GO LONG KLINIK NAU SISTA SKELIM BEL BILONG YU?

no=0; yes, PNGIMR clinic=1; yes, usual MCH= 2.....

state clinic if usual MCH_____

9. HAMAS TAIM YU BIN GO?.....

10. FROM CLINIC RECORDS

ANTENATAL ATTENDANCES THIS PREGNANCY

date	observations	fundal height	clinical anaemia	treatment prescribed

antenatal records found ( ) checked clinic records and not found ( )

postnatal form page 2

11. LONG DISPELA TAIM EM SKELIM MERISIN LONG YU OR NOGAT?_____

_____

_____

(not attended=0; attended, drug not given=1; drugs given as a single dose=2; drugs supplied for prophylaxis=3_____

12. WANEM KAIN MERISIN?

(find out types, what the women think they are for, what they have taken in the last week/month, and how many they take each time)

13. YU BIN SIK NAU LONG DISPLEA WIK? specify_____

_____

14. YU BIN KISIM SKIN HAT LONG DISPELA WIK?_____

_____

15. YU BIN KISIM MERISIN LONG HATIPOS(1), HAUS SIK(2), MERISIN MERI(3), OR YU HOLIM SAMPELA LONG HAUS NAU YU DAUNIM(4)?.....

16. estimate tablets of chloroquine ingested.....

17. TAIM YU GAT BEL, SAMPELA SIK IKAMAP?_____

_____

	yes/no	when
18. skin hat?		
traut?		
sotwin?		
pekpekwara?		
ai raun?		
leg solap?		
lusim blut?		
pen taim yu pispis?		
baksait pen?		

postnatal form page 3

19. WANEM DEI YU KARIM?_____

20. NARAPELA WIK YU BIN STAP GUT KAM INAP NAU? (specify)_____

_____

21. YU BIN WOKIM WANEM SAMTING?_____

_____

22. LONG DEI BEFOR PEN LONG KARIM STAT YU BIN STAP GUT?_____

_____

23. YU BIN WOKIM WANEM SAMTING?_____

_____
(garden, saksak, collecting firewood, stayed at home, etc)

24. TAIM YU BIN KARIM, HUSAIT BIN STAP WANTAIM YU?_____

name_____relationship_____
(mother=1, sister=2, aunty=3, inlaw=4)

25. WANEM TAIM BELPEN LONG KARIM ISTAT? dei_____ taim_____

26. WAMEM TAIM WARA IBRUK? dei_____ taim_____

27. WANEM TAIM YU BIN KARIM? dei_____ taim_____

28. HET IKAM PAS OR LEK? (vertex=1. breech=2, twins=3)_____

29. DELIVERY: IF PROBLEMS WITH LABOUR, SPECIFY_____

_____

30. INFANT_____
(alive=1, stillbirth=2, born alive then died=3)

31. BILUM IKAM HARIAP?_____

_____

32. YU BIN LUSIM PLENTI BLUT?_____
(no=0, plenti liklik=1, obvious pph=3)

33. YU BIN KARIM WE?_____
(house=1, garden=2, health facility=3, on the way=4)

34. IF DELIVERED IN A HEALTH FACILITY, WHY DID THEY GO?_____

_____

35. IF ADMITTED AFTER LABOUR, WHY?_____

_____

36. HOW MANY DAYS AFTER?_____

37. IF A HEALTH FACILITY, WHERE DID THEY FINALLY REACH?_____

INTERVIEWER.....

postnatal form: MATERNAL EXAMINATION

ID.....

NAME_____

COMPLAINTS

EXAMINATION well or sick?

ANAEMIA

OEDEMA

DIASTOLIC.....

SYSTOLIC.....

CUFF.....

TEMP.....

SPLEEN (CM).....

EXAMINER.....

WEIGHT.....

MUAC.....

TSF.....

SSF.....

examiner tsf/ssf.....

HB.....

PCV.....

FEP.....

BS.....

TREATMENT RETURNED:

CHLOROQUINE

FOLATE

IRON

TREATMENT GIVEN:

TOTAL MEDICINES SUPPLIED:

OTHER ADVICE GIVEN:

16 January, 1987

postnatal form BABY EXAM

CHILD ID.....

DATE SEEN.....

MOTHERS NAME.....

MOTHERS ID.....

TIME BIRTH_____DATE OF BIRTH_____

TIME EXAM_____DATE OF EXAM _____

BABY HOW MANY HOURS OLD?_____

SEX.....

SINGLETON/TWIN (single =0, twin 1=1, twin 2=2).....

BABY ALIVE=1 DEAD=2.....

sign	score	comment	sign	score	comment
oedema 0-2			posture 0-4		
skin texture 0-4			square window 0-4		
skin colour 0-3			ankle 0-4 dorsiflexion		
skin opacity 0-4			arm recoil 0-2		
lanugo 0-4			leg recoil 0-2		
plantar creases 0-4			popliteal angle 0-5		
nipple form. 0-3			heel to ear 0-4		
breast size 0-3			scarf sign 0-3		
ear form 0-3			head lag 0-3		
ear firmness 0-3			ventral suspension 0-4		
genitalia 0-2			TOTAL_____		

postnatal form page 8- BABY EXAM

GENERAL EXAMINATION child well or sick?

CORD (not tied=0, tied=1).....

CORD LENGTH (cm).....

TEMPERATURE .....

HEAD CIRCUMFERENCE .....

WEIGHT .....

LENGTH .....

CROWN HEEL .....

HB .....

BS .....

EXAMINER .....

(added later)

ISSUED WITH CORD PACK?

PACK USED? WHICH COMPONENTS?

Addendum

REVIEW OF RESULTS IN RELATION TO EXISTING LITERATURE

This addendum was written in response to a request by the MD thesis examiners in which "the findings of the study are reviewed in relation to knowledge available from previous studies in more developed and less developed countries".

1. NEONATAL HEALTH

1.1. Characteristics of newborns

Birthweight: This study showed that the mean birthweight (2.56 kg) and the proportion of LBW infants (40%) in the Wosera population was low, even when compared to other developing country populations. Some of the lowest birthweight values reported have been from populations in India, where a mean birthweight of 2.5 kg has been reported (Kramer 1987); however, mean values of 2.8 kg for term infants is more typical for India (for example, Ghosh et al. 1971).

The mean birthweight estimate derived from this study is compared with other PNG studies in table 6-3. This study shows the mean birthweight derived is similar to other estimates in the Sepik area. It is not as low as that reported from the West Sepik in 1969 (Wark and Malcom). However, their data are from hospital deliveries occurring in the 1960's in a deprived population prior to any significant economic development.

Population based birthweight data from developing countries in areas where most deliveries occur in the village (equivalent to the data from this study) are uncommon. The population-based WHO study conducted in four countries examined birthweight data by birthweight categories. In rural populations, the percentage of newborns less than 2.5 kg varied from 8.2% (Thailand) to 19.9% (India); thus the proportion of LBW babies in the Wosera population was much greater than in the 4 countries where the WHO work was carried out (Perera and Lwin 1984). Greenwood et al (1989) in a rural study of village deliveries determined a mean birthweight of 2.7 kg in a group of pregnant primiparous Gambian women in a placebo group as part of a *Maloprim* prophylaxis trial, but the mean birthweight was higher in women having a subsequent child.

Most birthweight data from developing countries is derived from hospital delivery statistics. In many developing countries women living in rural areas deliver at home, and those that deliver in hospital may be of higher socioeconomic status. This bias may result in the mean birthweight of hospital births being higher than the mean of births in the community.

In fact, this study showed that women attending hospital for delivery appeared privileged, and that the mean birthweight of hospital deliveries of women from the study area was greater than that of women delivering in the village (chapters 6 and 8). This suggests that hospital birthweight data in areas where many births occur at home need to be interpreted with caution, as they may be an overestimate of the true population birthweight mean.

themselves almost certainly exert an effect on the risk of death in infancy: birthweight reflects this risk, rather than determining it (Sterky and Mellander 1978). Thus mortality in this population is not inflated by a large number of newborns falling into the residual proportion of the birthweight distribution (Wilcox and Russell 1986).

The follow-up through the neonatal period demonstrated that bacterial sepsis was common, and often related to umbilical cord care at delivery (chapter 7). This risk, it can be surmised, is not specific to particular birthweight groups. In order to reduce neonatal mortality in the Wosera, it is necessary to apply a set of interventions that reduce the entire birthweight specific mortality curve (Wilcox and Russell 1986).

2. MATERNAL HEALTH

2.1. Maternal depletion syndrome

The definition of the "maternal depletion syndrome" is muddled in published literature. Originally, it referred to the synergistic, negative effects of repeated childbearing, short birth intervals, poor nutrition and heavy physical activity on maternal health (Winikoff and Castle 1987). Later, the term was used as an explanation of poor pregnancy outcome. There is debate as to whether nutritional status in women reflects their socioeconomic status and physical work load rather than being an outcome of their reproductive history. As Winikoff and Castle note, "it is difficult to distinguish between the effects of repeated pregnancy and lactation on nutrition from the effects of a lifetime of poverty". Studies have not confirmed a specific, lasting effect of childbearing on maternal nutrition. For example, a study in the Philippines showed that prolonged lactation contributed to short term depletion of maternal energy reserves, but this effect was not long term: in fact, there was a positive association between parity and maternal weight (Adair, submitted manuscript). Cross-sectional studies from India, Iran, Lebanon, Philippines and Turkey showed little or no relationship between ponderal index of women and age and parity (Omran et al 1976). In contrast, a longitudinal study of 2000 women in Bangladesh showed that maternal weight, controlling for height, was lower for older, higher parity women (Huffman et al 1985). However, their study was unable to control for the effect of age on nutritional status, and was therefore unable to show an independent effect of parity.

This study, however, showed a clear significant independent relationship between maternal nutritional status (measured by body mass index, mid upper arm circumference and subscapular skinfolds thickness) and parity after age had been taken into account (chapter 4, section 3.7.2). The effect was striking, and marked in the parity 0 and 1 groups aged 15-29 years. Non-pregnant women of parity 1 in this age group are likely to be lactating, which would contribute to their poorer nutritional status when compared with nulliparous women; however, the apparent trend through subsequent parity and age groups suggest that recovery of nutritional status between pregnancies does not occur.

This data, in common with other studies, is cross-sectional and can be criticised as not demonstrating maternal depletion because of confounding by socioeconomic status, as women of high parity may come from poorer circumstances (Graham and Danso-Manu 1988).

However, longitudinal data from the pregnancy study provides indirect evidence of an effect of childbearing on maternal nutritional status. The study showed that the estimated mean weight gain in pregnancy was 5 kg (chapter 6, section 4.8), indicating the weight gain from fat deposition, increase in blood volume and extra-cellular volume expansion averaged 650 g (footnote, page 90). The data on nutritional status at 6 months postpartum showed that, on average, maternal weight declined from the immediate postpartum value. Other studies in PNG have shown some 5.6 kg of fat may be used during the first 12 months of lactation (Orr-Ewing, quoted in Groos and Garner 1988). Therefore, even if the average Wosera maternal gain of 650 g were all fat, this would be insufficient to offset lactational loss. Most Wosera infants are exclusively breast fed up to 8 months, and breast feeding is generally continued up to 2 years (Heywood et al 1986). Thus this study, combined with other work concerning maternal nutritional and lactation, provides some evidence for repeated childbearing resulting in a sustained deterioration of maternal nutritional status.

2.2. Determinants of maternal mortality

It has already been documented in PNG that maternal mortality is high in comparison to many developing countries, estimated 20/1000 live births in rural areas (National Health Plan 1986). It is known that postpartum haemorrhage appears to be a major cause of maternal death in the lowlands of PNG, and some sources have noted the high incidence of placental retention postpartum in village deliveries (Garner and Giddings 1985). Some authors suggest that postpartum haemorrhage is more common in the lowlands because of the high prevalence of maternal anaemia, and that the anaemia in addition contributes to the likelihood of maternal death when haemorrhage occurs (Brabin 1990).

This study shows that the third stage of labour was markedly delayed (longer than one hour) in 8% of village births (chapter 6, section 3.2.7). The hospital obstetric admissions analysis confirmed that one of the most common reasons for obstetric admission was a retained placenta (chapter 9). Further, the study showed that a large proportion of maternal deaths in the area were related to retained placentae or postpartum haemorrhage (chapter 5, section 4.11). It is known that at village births, the cord is not cut until the placenta is delivered (chapter 3, section 1.4), and breast feeding is not initiated until the third stage is complete and the mother washed.

These results suggest that delay in the third stage of labour is probably contributing to the risk of maternal mortality at delivery in the villages. They indicate that intervention to reduce maternal mortality may include: encouraging early attendance at antenatal clinic so that anaemia can be treated and managed appropriately; provision of easily accessible obstetric services supervised by health workers (chapter 10, section 2.3); and encouraging breast feeding immediately after the second stage, to promote uterine contraction and placental expulsion.

3. HEALTH SERVICE INTERVENTION

Chapter 10 discusses in detail the implications of the study for health service managers in the Wosera district, national health policy, and for further applied research. This section highlights findings of the study in relation to maternal health service planning in developing countries in a more global context.

3.1. Interventions and birthweight

The study emphasises that the birthweight characteristics of a population reflect a variety of economic, nutritional, environmental and disease factors present in the community; and that these factors themselves are likely to have an independent effect on neonatal or infant mortality risk. Thus health service interventions designed specifically to increase the birthweight of babies in order to reduce neonatal or infant mortality are unlikely to be successful. Thus the prediction of a 26% fall in infant mortality if the incidence of LBW were reduced by 15% through food supplementation in pregnancy (Ashworth and Feachem 1985), for example, may not be valid. In fact, it can be argued that in the absence of good obstetric care, perinatal mortality may be increased if mean birthweight is raised (Offringa and Boersma 1987). This thesis suggests a refocusing of attention on maternal health in pregnancy, rather than specifically concentrating on factors that may influence fetal growth, is an appropriate way forward (chapter 10).

3.2. Malaria control in pregnancy

This study confirms the increased susceptibility to malaria in early pregnant primigravidae, and demonstrates the problems of implementing antenatal drug prophylaxis regimens. Difficulties implementing prophylaxis programmes has been observed in other studies, such as the Saradidi Project in Kenya (Kaseje et al 1987) and in Malawi (Heymann et al 1990). Even if maternal compliance was good, chloroquine resistance of *Plasmodium falciparum* would be likely to reduce the efficacy of the prophylaxis.

3.3. Neonatal sepsis

This study demonstrated that neonatal sepsis was common, and that use of delivery packs distributed through antenatal clinics appeared to reduce the risk of neonatal infection. The effect of delivery procedures affecting neonatal outcome is comparable with studies of neonatal tetanus. It has been shown that use of bamboo compared with scissors is associated with a higher incidence of neonatal tetanus (Yusuf 1986). The low incidence of neonatal tetanus in the Wosera study may reflect the widespread coverage with tetanus toxoid immunization (chapter 7); however, traditional birth practices have remained unchanged, thus the risk of bacterial invasion remains. Further studies at provincial level, distributing cord care packs through antenatal clinics province wide, are suggested before deciding whether to scale up the intervention nationwide (chapter 10, section 4.4). Nevertheless, in areas where neonatal sepsis is common, and many births occur at home, distribution of cord care packs along with information on their use through antenatal clinics or by traditional birth attends can be recommended.