THE EPIDEMIOLOGY OF MEASLES IN BAUCHI STATE NIGERIA

BY

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DEDICATION

To my two little ones, Yila and Mela

for escaping the ravages of this "Inevitable

trauma of Childhood - Measles".

DECLARATION

I declare that this work is wholly the author's and has not been part of any presentation for any other qualification.

Signed:.....

Date: 3RD SEPTEMBER 1987

CERTIFICATION

This Thesis entitled "The Epidemiology of Measles in Bauchi State, Nigeria" by David Nadeba Bukbuk meets the regulations governing the award of the degree of Masters in Microbiology of Ahmadu Bello University, and is approved for its contribution to knowledge and literally presentation.

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ABSTRACT

Measles in Nigeria and most developing- countries is endemic and major canse of childhood morbidity and mortality, where malnutrition is high compled with inadequate vaccination activities, The disease is known to bring a treumetic experience to children the world over. It is with this background together with the daily pathetic situations in most clinice in Bauchi State, Nigeria, of Mothers bringing in their children and some drying from clinical cases of measles and its complications that prompted this study.

The study is therefore, and attempt to analyse the impact of the massvascination activities before and after the lunching of the expanded programme on Immunisation in 1979 and the current revised E.P.I. Programme Launched in 1985 in the State, on the overran incidence of Measles and deaths due to measles. It also became pertinet to access the Immunity status, perticipations in the Immunisation Programme, and response (Seroconversion) to Measles Vaccines used during the two compaigne.

Monthly Measles cases, deaths and also vaccination data retrieved from medical records between 1977 and 1984 were analysesd. A total of 32,395 measles cases and 1966 deaths due to measles were notified, with 675,169 doses-of the vaccines adminis tered during the same period, A considerable decline in the overall incidences of Measles Cases during the precampaign period from 6,960,661 cases increased steadily to over 10,000 cases in 1983, even after the launching of the mass Vaccination compaign. The age distribution of those cases revealed the 1-4 year age group being mostly afflicted, followed by the 5 - 1 4 year olds. The cases were less in the under 1 year and over 15 year old age groups. The transmission of Measles was continous throughout the period of observation, 1977 - 1984. With cases reported monthly. There were two patterns of epidemic transmissions,

One major and other minor, with incidences peaking up from February to May annually, and between August to November respectively. Urban centres contributed 76% of the total cases of Measles in the area, which could be seen as foci of transmission to the rural areas, with the movements of young children and Mothers during the agricultural cycles and theopening/closing of schools and the low relative humidity in the dry seasons which enhances the spread of the measles Virus being speculated, as of determinants of the seasonality of Measles in the area.

A Serological study carried out on 439 randomly collected serumsamples in the state indicated that 54% had Measles HAI titer greater than 1:8, which indicates protective Immunity. There was no significant difference in the prevalence rates of Measles antibody between Males/females, however, antibody levels in Females were marginally higher than in Males. Amongst residents of urban-rural Centres a significant diff-eehce ($p<o.Ol$) in Measles immunity status was observed. There was also a significant difference, ($P<0.Ol$) in the prevalence rate of Measles antibody between the pre-school and the school aged children in the area, with more children in the later showing evidence of immunity to Mealses. The prevalence rate of antibody in the lower social class is higher (62%) as against 38% in the higher social class and is highly significant ($P<O.0l$). However, more of the pre-school and school aged children in the hgiher social group showed a higher antibody prevalence rate relative to some in the lower social group.

The national Measles Vaccination Programme has not low-ed ' the number of individuals sussceptible to the disease due to low vaccination coverage. Hence the EPI campaign launched in 1979, showed little impact on Measles Cases, its epidemiology, deaths due to the diseass and the overall immunity state of the people, therefore, for the present revised FPI mass compaign to be successful, it should take into account the sessonality of the Measles epidemics as it

relates to the epidemiology and human behavioral patterns in changing the incidence of measles in the community, A continous serological surveillance to monitor the immunity status of the people and Seroconversion to the Measles vaccines is necessary.

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ABBREVIATIONS

ADH	Antidiuretic hormone
CI	Confidence interval
CNS	Central nervous System
CPE	Cytopathic effect
DI	Defective interference
DNA	Deoxyribonucleic acid
EPI	Expanded Programme on Immunization
HA	Haemagglutinin
HAI	Haemagglutination inhibition
HL	Hemolysin
HLA	Human Leucocyte anti gen
IGA	Immunoglobulin A
IgG I	Immunoglobulin G
MOH	Ministry of Health
Mol.wt.	Molecular weight
MRC	Medical research Council
MS	Multiple sclerosis
PHA	Phytohaemagglutinin
PMN	Polymorphonuclear Neutrophil leucocyte
RNA	Ribonucleic acid
SLE	Systemic lupus eryth·matosus
SSPE	Subacute sclerosing panecephalitis
ts	Temperature sensitive
U.S.A.	United states of America
WHO	World Health Organization.

INTRODUCTION

Measles has long been recognised as one of the "inevitable traumas of childhood " (ter Meulen, 1974).

The World Health Organisation estimates that each year approximately 1.5 million children die from measles and its complications around the world.

This is approximately one death from measles every 20 seconds (Hinman, 1983). The current global status of measles has been described very succintly by Himan (1983) to be high with low immunization coupled with the non-existent surveillance and outbreak investigation in many parts of the world. Infact, the WHO estimates that only 20 percent of eligible children receive measles vaccine. Presently, immunization coverage levels in England is less than 50 percent. In France, it is less than 20 percent , in Italy less than 10 per cent and in Denmark the vaccine has not been used on a routine basis.

The most recent WHO estimate of measles vaccine coverage by region in dicates anestimated coverage ranging from 0.2 percent in young children in south east Asia region and 63 percent in the European region (Hinman 1983). Measles occurs at a younger age in developing countries than in industrialized countries (Anonymous, 1976). In some developing countries 20-30 percent of measles cases occur in children under 1 year of age, and few cases before 6 months of age (Grigsby and Adetosoye, 1973; Ministry of Health, Kenya and WHO 1977).

Measles is endemic in Nigeria, where Malnutrition is High, and is also a Major cause of morbidity and mortality in young children (Gans, 1961) Grigsby and Adetosoye 1973: Abdurrahman, 1979). Despite the first application of the measles vaccine in Africa in the village of Imesi (South Western Nigeria) by David morley and Magret Woodland (1963). The disease remains one of the problems of child health in Nigeria and other developing Countries. In these developing nations, up to 10 percent of measles infected children often die, with the highest death-to-case ratio-accuring in infants, (puffer and Serano, 1973; Morley 1975).

Reports on the incidance and problems associated with measles infection in Nigeria children have neen well documented (Morley and Macwilliam, 1961; Morley, 1962, Morley et at 1963 and Katz et al 1962).

The strategy of measles control, is to protect a large number of children by immunizing them before they are exposed to natural infection in the community, and subsequently to reduce the influx of the young susceptibles into the population.

The General Assembly of the WHO passed a resolution that encourages all member nations to develop vaccination programmes that would immunise at least 90 percent of all children against vaccine preventable disease by the year 1990,(Creesse and Henderson, (1980). In 1974, a special expanded programme on Immunization (EPI) Unit was set at the WHO Headquarters, Geneva, to assist member states in achieving this goal, A national plan of action was therefore, designed which will be part of an already ongoin health care system to ensure that each infant received vaccinations.

In Nigeria. the EPI was embarked upon in 1979. In Bauchi State, EPI was launched in 1979, in the Dass and Tafawa Balewa Local Government areas. Prior to 1979 some immunization had been going on in Bauchi. The current revised EPI Programme was lauched in Bauchi on the 19th February, 1985.

The present study is an attempt at analyzing the changes in immunity patterns to measles in Bauchi State and to access the impact of the EPI since its establisnment in 1979. To achieve this objective records of all monthly and annual measles deaths, vaccinations and cases in the state, with the sex age, location, history of previous immunization against measles was retrieved from medical records at the State Epidemiological Unit, and analysed. The second part of the work was a serological survey of the general population for measles haemagglutination inhibition antibody. Serum samples were randomly collected from the general population with useful data recordeded and analysed. Such a data included the age, sex and stage of pregnancy in case of pregnant females.

CHAPTER 2

LITERATURE REVIEW

2.1. Historical Background

Measles was first recognised as a diseaseim the 11th century by an Arabian physician called Rhazes (Abu Becr, 1948). Rhazes providdd the earliest description of measles when he quoted other authors as Al Yehudi who lived in the Seventh Century. Rhazes looked on measles as a relctively severe disease and considered it "more to be dreaded than smallpox". Measles has been known by several linguistic evidences measles (English); Hasbah (Arabic); rubeola and morbilla (Latin); Masern (German); Mislingar (Icelandic), (Black, 1976). In the Roman Empire there were massive epidemics of measles starting in 165 and 251 A.D., and two similar epidemics in china in 162 and 310 A.D. (McNeil, 1976).

Kenneth Maxcy, (1948) once remarked that the simplest of all infections diseases is measles. This relative simplicity makes measles and ideal model for the study of infections diseases epidemiology. In Northern Europe, Sydenham (1922) first described measles and considdred it infections, Home in 1958 attempted a procedure of immunization with measles similar to variolation. Peter Panum in 1846 went to the Faroe Islands to give helpduring a measles epidemic, which led him to conclude that the source of this disease was solely through contagion. He was also able to define the 14-day incubation period and to show that infection conveyed a life long immunity. Hirsch (1883) concluded that an epidemic persists, so long as there are susceptible individuals. Hamer in 1906, gave a modern concept of the epidemiology involving imput of new births and output of immunes to maintain a fluct - tuating population of susceptibles within stable limits.

In the 20th century, measles was adapted as a good model for the development of an epidemic theory, when Enders and Peebles (1954) first isolated the measles virus and also demonstrated immune reactions by neutralisation and complement fixation tests. These test coupled with the vaccines from attenuated measles virus isolates helped to delineate the changing patterns of measles disease in any society.

2.2 STRUCTURE OF MEASLES VIRIONS

Measles virions are composed of an outer lipoprotein envelope, 10 to 20nm thick and contains short projections (Spikes) and an internal, helical nucleocapsid. The virus particles are roughly spherical in shape but are mostly pleomorphic and range in size from 120 to 470nm in diameter, with larger particles occassionally being observe (Waterson, 1965; Nakai and Imagawa 1969). Infectious virions have been reported to have a bouyant density of 1.23 to 1.25g/cm^3 (Norrby et al 1964a; Hall and Martin, 1973). A linear, helical necleocapsid that contains the genomic RNA forms the major component of the virion. The nucleocapsid has an RNA content of about 5% and a bouyant density of 1.28 to 1.30g/cm^3 (Hall and Martin 1973. Water and Bussell, 1974) Under the electron microscope these nucleocapsids appear as coiled rods with an outer diameter of 17 to 18nm and a central, hollow channel measuring about 5nm in diameter (Norrby and Hammarskjeld,1972 Waters and Bussel,

1974). Isolated nucleocapsids obtained without enz enzymatic treatment appears loosely coiled and flexible but when enzymatic treatment is employed, these nucleocapsids becomes rigid and tightly coiled (Waters and Bussell, 1974).

These changes have been correlated with alterations in the nucleocapsid protein. The nucleocapsids are fragile and fragment readily, these breakages make nucleocapsids to sediment heterogeneously between 200g and 300g in sucrose density gradients (Hall and Martin, 1973). Some circular and elongated forms of nucleocapsids have also been reported (Thorne and Dermott, 1976). The presence of RNA within measles virus particles was first reported in 1964 (Norrby, et et al.; 1964b St. Geme 1964). This RNA is a single stranded molecule with a mol. wt. of $6.2x10^6$ and sedimentation coefficient of approximately 52S in sucrose density gradient (Hall and Martin, 1973). Measles virus RNA issimilar to other paramy xoviruses in having a base composition that is rich in uracil (Yeh, 1973, Waters and Bussell, 1974). Polyacrilamide

gel electrophoresis studies have demonstrated that measles virions are composed of six polypeptides whose approximate mol. wts. range between 38,000 and 79,000. These gel patterns resembles those of canine distemper and rinderpest, and they also exhibit similarities to the other paramyxoviruses (Waters and Bussell, 1973; Underwood and Brown, 1974). The nucleocapsid polypeptide forms the major structural protein mol. wt. 60,000 (Waters and Bussell 1973). The smallest polypeptide, mol. wt. 38,000 may be analogous to the membrane (M) protein of the other paramyxoviruses (Mount castle and Choppin, 1977). This protein is beneath the lipid bilayer of the virus envelope and probably functions in maintaining the intergrity of the virus particle (Choppin and Compass, 1975). The second largest polypeptide mol. wt. 70,000 is associated with the nucleocapsid and it has been proposed that this polypeptide may be part of the transcriptase complex (Mount castle and Choppin, 1977). The identity of the remaining polypeptides is uncertain.

The envelope is the source of antigens which are involved in the hemagglutinating (HA), hemolytic (HL), and cell fusion properties of the virion (Waterson, 1965). There is evidence that the HA and HL activities of measles virus may be associated with two surface glycoproteins that form the spikes (Hall and Martin 1973). The HL component is more sensitive than the HA to thermal inactivation and treatment with formalin, other, or Tween 80-ether (Norrby and Falksveden 1964); and HA, HL are antigenically distinct (Ehrnst, 1975; Norrby 1975).

Measles virus is antigenically stable. A single exposure to measles infection confers life long immunity in the host and this was thought to indicate that the heterogenous antibody formed in the infected individual reacts with a virus that exists in only one invariant form. However, Birrer, et al (1981) reported that antigenic variants of measles arise spontaneously following selection in the presence of a neutralizing monoclonal antibody directed to the HA of Edmonston measles virus. The variant viruses are not neutralized by the monoclone used for selection, while the s econd monoclone still

neutralizes. This limited analysis showed one of these variants to be antigenically similar to a low passage "street" virus isolate of measles. Thus the possibility arises of antigenic variation of measles virus outside the laboratory, and the authors speculated whether antigenic variants arising in nature can play a role in the persistent infections of man with measles - like virus.

2.3 MEASLES VIRUS REPLICATION

There are many variables which show significant influence on the severity of measles infection, the length of time required for the appearance of progeny virions, and the amount of virus ultimately produced (Dejong and Winkler, 1970, Portner and Bussell, 1973). These variables are the virus strain itself, host cell, and the multiplicity of infection. The replication cycle of the measles virus can be divided into three major events which are, the adsorption penetration and uncoating; synthesis of virus-specific macromolecules and finally, the assembly and release of progeny virus. The virions attach to cellular receptors, by means of the spikes on the virus envelope. One such receptor for measles virus is a glycoprotein that contains only low levels of sialic acid (Duffy and Howe, 1976). It is thought that the virions enter the cell either by "Viropexis" (Phagocytosis) (Compans et al., 1966) or by fusion of the viral membrane with the cellular membrane (Morgan and Home, 1968) as seen in other paramyxoviruses. Either of these

presesses is ultimately followed by the release of nucleocapsids into the cytoplasm (uncoating) and it is likely that both occur during infection (Choppin and Compans, 1975). Virus RNA synthesis is initiated shortly after infection (Portner and Bussell, 1973); synthesis of virus proteins and genomic RNA, which is encapsidated into progony nucleocapsids then occurs. All of these events appear to take place within the cytoplasm of infected cells. Though it has been reported that measles RNA can be isolated from the nuclear fraction of infected cell extracts (Schluederberg and Chavanich, 1974). The measles virus genome, is negative-stranded hence cannot function as a message; the synthesis of smaller, complementary RNAs (plus strands) is necessary. Although enzymatic activity has not been reported for measles virus, other paramyxoruses contain a virion-associated RNA polymerase (transcriptase) which is associated with the nucleocapsid (Robinson, 1971). Virus-specific RNA transcripts, in association with five different size classes of polyribosomes have been detected in measles virus infected cells (Yeh, 1973). These RNAs

are complementary to genemic RNA (Yeh 1973). Therefore at least a portion of the complementary RNA functions are message for the synthesis of virus-specific proteins. At one stage of the replication cycle, the virus must transcribe complete 52S (olus) strands to serve as templates for the f ormation of 52S (minus strands) genomes. It is not known whether the enzyme that is necessary for genomic RNA replication (replicase) represents an alternative function of the transcriptase or whether a separate enzyme or enzyme submit is required (Kingsbury, 1974). It is also apparent that the progeny molecules are rapidly encapsidated, since free paramyxovirus RNA has not been observed in infected cells (Kingsbury, 1974). Studies using metabolic inhibitors have shown protein synthesis can be detected within 3 hours after infection and that it reaches a peak long before the appearance of infectious virions (Portner and Bussell, 1973). The development of virus specific antigens within infected cells has been studies with immunofluorescence techniques

Within a few hours after infection, virus-specific antigen can be detected in the cytoplasm. The fluorescence first appears as fine dots in the perinuclear region, and as the infection proceeds and the intensity of staining increases, this fluorescence often completely encircles the nucleus. Ultimately, the nucleocapsid antigens become dispersed throught the cytoplasm (Norrby, 1972). In the nuclei of some cells nucleopasid antigens can also be detected; with nuclear staining being most prevalent late in infection; and the antigens are associated with nuclear inclusion bodies (Enders and Peebles, 1954, Norrby 1972).

The envelope antigen (HA and HL) first appear in the cytoplasm at one pole of the nucleus, producing a meshwork of fine threads of fluorescence (Norrby, 1972) These antigens may become dispersed later in infection, throughout the cytoplasm and can accumulate in the cytoplasmic membrane (Norrby, 1972 Ehrnt et al 1974). The budding of nucleocapsids to form infectious virions occurs at those sites on the membrane where virus specific antigens are present (Dubois/and Dalcq Barbosa, 1973).

The assembly and release of progeny virions the final step in virus replication starts when the cell membrane becomes altered in appearance to more closely resemble the virus envelope; it becomes thicker and more electron dense, and spikes appear. The nucleocapsids then assemble beneath these areas of the cellular membrane and align in parallel arrays. The membrane then protrudes (buds) to form progeny virions (Nakai and Imagawa 1969, Dubois Dalcq and Barbosa, 1973).

2.4 MEASLES VIRUS-CELL INTERACTIONS

There are two aspects of measles virus-cell interactions that are of immense interest to virologist. These are the cytopathic effects on measles virus-infected cells and interactions of hemagglutinin (HA) with certain erythrocytes (Perics and Chamy, 1960; Rosen, 1961). It has been shown that measles virus can be grown in a variaty of primary and continous cell lines, although the best results are obtained with cells of human and simiam origin (Matumoto, 1966). Enders and Peebles (1954) first described measles virus infections which usually produces very distinctive CPE that are characterized by the formation of multinucleated giant cells (Syncytia) formed by fusion of mononucleated cells. The chromatin assumes a marginal position and is replaced by intranuclear inclusions with eosinophilic inclusions developing in the cytoplasm. In addition, chromosomal aberrations and breakage have been observed in measles virus infected cells (Csonka, et al., 1975).

The formation of syncytia is dependent upon the presence of a virus-specific protein on the surfaces of infected cells. This "fusion factor" may be identical also to the hemolysin (Cascado and Karzon 1965, Choppins and Compans, 1975). This is supported by data obtained from ts mutants of measles virus. Some of these mutants fail to produced syncytia at the non-permissive temperature, and the infected cells do not contain detectable amounts of the HL polypeptide, as determined on immunofluorecence techniques (Haspel et al., 1975). Syncytia formation can occur within 5 hours when cells are infected at high multiplicities (Cascado and Karzon 1965). Non-infectious measles virus particles, such as ultraviolet-irradiated virions (Toyoshima et al 1960) and the "Small HA particles" (Schluederberg, 1962) can also produce syncytia. Therefore, syncytia can occur even in the absence of virus replication.

A second type of measles virus CPE (straind-forming) has also been described. This is charactirized by the appearance of spindle-shaped or stellate cells that have long and irregular cytoplasmic processes (Oddo, et al, 1961). Straind forming CPE often occurs when cells are infected with undiluted, passaged inoculum, and infectious yields are often greatly reduced (Oddo, et al 1961). Measles virus is known to agglutinate only monkey or baboon erythrocytes by interaction with a different cell receptor from that used by orthomyxoviruses. This observation was first reported by Paries and Chamy (1960). Unlike haemagglutination by other paramyxoviruses, that by measles virus is maximal at 37°c and, because of the lack of neuraminidase there is no spontaneous elution of the virions. Because of the successful interaction at 37°c Higher titers are obtained in the hemadsorption test at 37°c than at 4°c, and since the virus does not elute from the erythrocytes at the higher temperature, 37°c is used for these tests. The haemagglutination

2.5 PERSISTENT AND LATENT MEASLES VIRUS INFECTIONS

Measles virus is known to share with other paramyxoviruses the ability to establish persistent infections in-vitro (Choppin and Compans, 1975). Though "measles infections usually produces widespread cellular destruction, but when the surviving cells are selected and subcultured, they often show evidence of being persistently infected. A common feature of most persistent infections is that the cultured cells are usually refractory to superinfection with the measles virus, but are readily infected with other, unrelated viruses (Rustigian, 1966, Norrby 1967; Menna et al, 1975). The type of persistent infections produced by measles virus varies considerably from " carrier culture" in which low levels of infectious virus are continously shed into the medium (Rustigian, 1966; Norrby, 1967) to a more latent form in that the cultures fail to produce infectious virus, that is non-producer cultures. These type of culture

contain virus-specific intracellular antigens (Knight et al; 1972, Rustigian 1966) and all size classes of virus-specific RNA (Winston et al, 1973) Persistent measles infections can be established by growing infected cells in the presence of specific antisera (Joseph and Oldstone, 1975) and there is no evidence they can be "cured" by continued growth in the presence of antisera (Norrby 1967, Gould and Linton, 1975).

However, it should be noted that the distinctions between these two types of persistent infections are not absolute for example, by subculturing carrier cultures of Hela cells in the presence of antiserum, Rustigian (1966) was able to isolate clones that failed to release infectious virus. Conversely, persistently infected hamster ombryo fibroblasts that did not produce infectious virus began to spontaneously release low levels of infectious virus, when the cells were repeatedly passaged (Haspel et al 1973).

There are possible [illegible] involved in the establishment and maintenance of persistent infection of measles virus. The possible role of defecting interfering (DI) particles; temperature sensitive (ts) mutants and the intergration of virus genome have been exploited (Huang and Baltimore, 1970, Preble and Youngner, 1975). The genetic information of non-oncogenic RNA viruses may become intergrated into the cellular genome. There have been reports that DNA isolated from cells persistently infected with measles virus contains sequences that are homologous to measles genomic RNA (Zhdanov, 1975). In studies with another paramyxovirus, respi atory syncitial virus, DNA extracted from persistently infected cultures was used to infect susceptible cells, which subsequently released infections virus (Simpson and Tinuma, 1975). It has been suggested that defective interfering particles may be associated with persistent infections in-vitro and with certain sub-acute or latent infections in-vivo (Huang and Baltimore, 1970). These particles arise during serial passage of undiluted virus stocks.

These are particles that contain only part of the virus genome. The presence of defective measles Virus particles was first suspected when species of RNA smaller than 52S genomic RNA were detected in preparations of measles virions (Winston et al., 1973). In addition smaller nucleocapsids (200S) which contain cells (Kiley and Payne, 1974). Those smaller RNAs and nucleocapsids can be eliminated when virus stocks are plaque purified and passaged at low multiplicifies (Kiley et al., 1974). It has been reported that undiluted passage of measles virus results in strand-forming CPE that is associated with a decrease in infections virus yields, and that different types of virus-specific products, "non-infectious HA" are produced (Oddo et al., 1961). There is a growing body of evidence which suggests that (ts) mutants may participate in the establishment and/or maintenance of persistent infections and that they may be selected for during the course of a persistent infection (Preble and Youngner, 1975). There have been two reports of the isolation of (ts) mutants of

measles virus. Knight et al., (1972) established a "non-yield" culture of hamster embryo cells which began to spontaneously produce small amount of virus after 18 passages at 37°c. Greater yields of virus were obtained when the culture were incubated at 33.5°c, and the virus released was (ts) (Haspel et al., 1973). Gould and Linton (1975) also isolated a (ts) mutant from HEP-2 cells persistently infected with measles. When shifted to 39°c the cultures appeared to be "cured" based upon the loss of detectable intracellular antigens. However, the antigens reappeared when the cells were shifted back to 37°c and subcultured. Both (ts) mutants and defective interfering particles may act in a fashion analogous to DI particles by interfering with the replication of wild-type virus. This suggests that (ts) mutants may have some type of selective advantage in persistent infections, since there is no evidence for the subsequent isolation of wild-type revertants (Youngner and Quagliano, 1976). These observations have not been detected in persistent measles virus with certainty since ts mutant of measles virus was lost from persistently infected hamster cells during further subculture and was replaced with a virus that replicated like the wild type virus.

2.6 MEASLES DISEASE AND ITS COMPLICATIONS

Measles disease is charactirized by a persistent fever of 41°c hacking, dry cough and delirium photophobia, darkening of the skin due to increased blood flow into the rash. The virus is highly infectious spreads by droplet infection, and initiates infection in the upper respiratory tract of susceptible individuals. The clinical course of the infection has been well documented (Katz, 1972). The clinical consequences that follow the immuno-suppression induced by measles virus infection vary widely. Pneumonia, gastroenteritis and otitis media are frequent complications of severe measles and play a major part in producing the high mortality of this infection noted in many tropical areas (Morley, 1969; Anonymous, 1976). Post-measles pneumonia is sometimes due to infection with organisms of low virulence and it is likely that the severe impairment of the cellular and humoral immunity that accompanies measles, predisposes to this serious

complications. Diarrhoea is also a common manifestation of measles with the effect of reducing the intake of food by the child, hence dehydration and desquamation of the skin due to a negative nitrogen balance.

The association between measles and diarrhoea is quite obvious in developing countries today, as there was in the developed world in earlier times (Morley et al,, 1963). The role of immunological factors that may account for the association between measles and diarrhoea have been reviewed (Greenwood and Whittle, 1981). Measles at the pro-dromal stage can lead to appendicitis caused probably by defects in immune responsiveness as a result of infection of the immunocompetent lymphocytes by the measles virus (Lonbet et al., 1982). Ogunye and Gbadebo (1981) remarked that measles and malarial infection in children often leads to an inappropriate secretion of antidiuretic hormone (ADH). Measles virus infection can also cause a brief remission in children with juvenile rheumatoid arthritis (Yoshioka et al., 1981). Severe measles

virus infection can reactivate a latent herpes simplex infection characterized by deep eroding oral ulcers and severe corneal losions (Whittle et a;., 1979). Severe anaemia a complication not reported before in measles was observed by Effiong (1975) in 12% of severe measles cases from Nigeria, possibly due to malnutrition, poverty and delay in between onset of illness and the admission of patients.

Symptoms of neurological involvement can develop in many patients during measles virus infections. Encephalomyelitis, myelitis, hemiplegia, optic neuritis, Bell's palsy, guillain-Barre' syndrome are often associated with measles virus infection, (Miller et al., 1956, Tyler, 1957; Katz 1972), Pollack, et al 1975, and Guerrin et al, 1981)

Subacute sclerosing paneucephalitis (SSPE) a rare, slow, chronic neurological disease is associated with measles virus infection. It is charactirized by mental impairment, myoclonous and progressive mental deterioration which lead to death within 1 - 3 years from the onset of the illness (Agnarsdottir, 1977). The association of SSPE to a viral origin was first

proposed by Dawson, (1933). The persistence of measles virus within the CNS of SSPE patients were also observed (Connolly et al., 1967; Payne et al., 1969). Epidemiological data shows that SSPE is more likely to develop when natural measles occurs before the age of 2 years than in older children; with a clinical onset beginning at the mean age of 10 years (Sever et al 1974). Hence SSPE will be common in those areas of the tropics where early measles infection is the rule. Burnet (1968) remarked that, since SSPE is contracted early in life, an immature immune system or a heightened susceptibility to a measles virus variant could be contributing factors. Available data tend to support the proposal that SSPE may be due to variants of measles virus, possibly defective variants unable to produce mature virions. Such variants may be selected within the host during measles infections (Payne and Banblis 1973).

Another chronic neurological disease often associated with a persistent measles virus infection is multiple sclerosis (MS). This disease is common in developed than in developing countries. This disease is seen in young adults following a relapse and remission over a period of years. MS is common in developed countries possibly because there are always an incidence of measles towards the older age brackets in these countries. MS is characterized by demyelination and fibrosis gliosis (Lumsden 1972). The demyelination in MS may be due to an autoimmune response, since antimyelin antibodies can be demonstrated (Paterson, 1971). Some genetic factors may pre-dispose to MS, since a high proportion of MS patients with HL-A3 AND HL-A7 tissue antigens are seen.

Other autoimmune diseases are also associated with measles virus infection, such as rheumatoid arthritis (Laitinin et al, 1972) Polymyositis (Chon, 1967), Scleroderma and Goodpasture's syndrome (Norton, 1969); Sjogren's syndrome (Shearn et al., 1970); Reitor's Syndrome (Philips and Christian, 1970) and Systemic lupus erythomatosus (SLE). The association between

measles virus and these commective tissue disorders is evidenced by either the detection of high measles antibodies in serum and other body fluids without any viral etiology and also the detection of measles virus antigens in infected cells or biopsies. Tubular structures resembling paramyxovirus nucleocapsids or measles virus antigen have been defected in both SLE cells and tissue biopsies (Kawano et al 1969, and Alekberova et al 1975). Also elevated titers of measles virus antibodies and DNA from SLE tissues contained sequences which hybridized with measles RNA (Alekberova et al., 1975).

2.7 IMMUNITY AND IMMUNOPATHOLOGY OF MEASLES VIRUS INFECTION

The first clinical features of measles which appear after an incubation period of 10 to 14 days are fever, conjunctivitis, a runny nose and small papular lesions in the mouth (Koplik's spots). Virus is present in circulating lymphocytes, Koplik's spots and the epithelium of the respiratory and gastrointestinal tract. Three days after, rashes may appear and become confluent, haemorrhagic and darkened (Morley, 1969). One week after the onset of rash, extensive desquamation of the skin may occur in severe cases. Measles antibodies may be first detected from the first to the third day of the rash; they reach their maximum titer by the twenty-fifth day after the onset of illness (Enders-Ruckle, 1965) a short drop in titer may be noticed in about 8 - 18 months. These antibodies can persist for life with a slow decline for more than 30 years after the illness. Dekking (1965) observed measles antibodies even at

80 years of age in 15% of individuals.

It is a known fact from the classic studies of Panum (1847) and of Black and Rosen (1962) that following natural measles, infection, immunity is completely solid and lasts at least 65 years, even in the absence of reinfection. This life long immunity might be due to persistent measles infection which can help to maintain immunity by continued stimulation of the immune system. There is a possibility of having a reinfection by the same virus or cross-reacting viruses. The continual re-exposure of adults to infected children is also likely to play an important part in maintaining effective protective immunity (Enders-Ruckle, 1965). There are clear evidence that measles antibodies are neither the only nor the most important mechanism i involved in immunity. Even patients with immunoglobulin defiency handle measles infection normally (Good and Zak, 1956), while those with defects in cellular immunity suffer serious con sequences from measles infection. There is giant cell preumonia without any rash and often

accompanied by the involvement ofT-cells, (Valdimarsson et al., 1975). The rash may be a generalized delayed hypersensitivity reaction, Coombs and Gell Type 2 (Coombs & Gell 1975). On the other hand, histological and immunofluorescent studies of the skin show measles antigen and features suggestive of an Arthurs reaction Type 3 (Kimura, et al., 1975); Olding-stenkvist and Bjorvatn, 1976). Antibody and complement can lysc measles-infected cells (Kibler and Meulen, 1975). These rash reach its maximum at the time that free antibody appears in the serum. Antibody - independent cyto-toxicity to measles infected cells is at a maximum during the time of the height of the rash (Whittle and Wer blinska, 1980). However, the immunopathology of the rash of measles is thus difficult to classify, it may be a type II, III or IV reaction or a combination of all three.

T-lymphocyte (HLA) restricted killing of measles virus infected cells was noted in an invitro study (Wright and Levy, 1979) but this phenomenon was never n noted during natural measles infections in man. This killing can only be achieved when the target

FAECAL EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc	Tabulated t t tab(0.05)	
Shredded paper	4099.20 ± 137.14	3.91	2.31	P<0.05
Saw dust	2000.60 ±237.47	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust. df = 8

TISSUE EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc)	Tabulated t t tab(0.05)	
Shredded paper	17521.00 ±632.92	3.85	3.21	P<0.05
Sawdust	23041.60 ± 1295.07	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust. df = 8.

FAECAL EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc	Tabulated t t tab(0.05)	
Shredded paper	4099.20 ± 197.14	3.91	2.31	P<0.05
Saw dust	2000.00 ±237.47	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust, df = 8

TISSUE EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc)	Tabulated t t tab(0.05)	
Shredded paper	17501.00 ±632.92	3.85	3.21	P<0.05
Sawdust	23041.60 ± 1295.07	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust, df = 8.

(host infected cells) and effector cells (killer T-cells) share common major histocompatibility antigens. The mechanism of this restricted killing is not completely understood but possibly due to the fact that cyto-toxio T-cells can only recognise the new antigenic dterminants produced by the combination of host cell surface antigens with those of the invading microorganisms which become susceptible to attack by the antibody (Greenwood and Whittle, 1981).

Both cellular and humoral immunity are depressed in children with measles (Whittle et al., 1973). The defect in cell-mediated immunity in children with this condition is complex. Delayed hypersensitivity skin reactions are lost. This was first noted by Von Pirquet in 1908. Lymphocyte counts, especially T-cells are reduced (Coovadia et al., 1977). Diminished response of lymphocyte transformation and leukocyte migration inhibition to tuberculin and candida antigens are also noticed (Kantor, 1975; Whittle et al, 1978). Lymphocytes stimulated with PHA failed to produce lymphokines (Joffe and Rabson, 1976) and chemotaxis by PMN was also impaired (Anderson et al., 1976).

Infact a potent immunosuppressive factor which contributes to most of the defects in cellular immunity are produced in acute phase sera from measles infected children (Whittle et al., 1978). Because of this severe impairment of the immune system, there is a predisposition to some of the serious complications associated with severe measles infection.

FAECAL EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc	Tabulated t t tab(0.05)	
Shredded paper	4099.20 ± 197.14	3.91	2.31	P<0.05
Saw dust	2000.60 ±237.47	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust. df = 8

TISSUE EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc)	Tabulated t t tab(0.05)	
Shredded paper	17501.80 ±632.92	3.85	3.21	P<0.05
Sawdust	23041.60 ± 1295.07	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust. df = 8.

2.8 EPIDEMIOLOGY OF MEASLES

Since the discovery of measles by Rhazes, it has become a favourite in most epidemiological studies, moreso, because of its high incidence, regular occurance throughout the world and clinical severity particularly among malnourished children (Barkin 1975; Dossefor et al 1977). The regular recurrence of epidemics in large unvaccinated populations is one of the impressive epidemiological features of measles; a reflection of a dynamic implication of the constant influx of susceptible children into the population (Soper, 1929). These susceptible vary from place to place. In Nigerian and Sudanse communities, studies by Obio (1979) and Elkarim (1981) respectively showed that the 1 - 3 year age were most susceptible to measles infection. In West and Central Africa, several studies has shown that almost all cases of measles occur before the sixth birthday (Morley et al 1964; 1966; McGregor, 1964; McBean, 1972. Hence all children six months to six years

of age are at risk of contacting measles disease.

Therefore, mass vaccination campaign should attempt at immunizing - these age groups. The changing incidence of measles in any community depends on the immune status of the population as it relates to its size, density and composition by age (Fraser and Martin, 1978). Black (1976) suggested that all persons are equally susceptible to measles initially and when a susceptible is infected he remains infectious for a limited period and then through death or acquired immunity, withdraws from any further participation in the epidemic cycle. The severity of epidemics being a function of the number of susceptibles in the population.

Measles has a temporal seasonal distribution pattern. Dejong and Winkler (1964) found that measles virus is more stable when the relative humidity is low. The favourable effect of dry air on measles virus would enhance spread in the hot dry season of Persia (Abu Beer, 1748) or Sahelian Africa (Meyer, 1965). Hence the relatively high incidence of measles during these seasons.

Also the effect of climate is indirect, through its effect on human habits, since humans tend to congregate most, in these inclement seasons.

There is no difference between the sexes in either the incidence or the severity of measles. However, antibody titers in women, are marginally higher than in men (Black, 1976).

Occupation and institutions affect the overall incidence of measles; the age at which measles is usually contacted and also the rapidity of its spread by isolating or congregating susceptible individuals (Black, 1976). Black and Davis in 1968 noted that social setting exerts a clear influence on the age at which measles is contacted. The changes in national social structure have resulted in a highly immune population at the time of induction. For example, in 1962, at least 98.8% of recruits entering the U.S Army had measles artibody. Nevertheless, 0.1% of the military population developed measles annually (Black 1964). Socioeconomic factors often show a correlation to age at which measles infection occurs (Black, 1976).

The pattern of measles transmission in the tropics is quite variable, with three distinct patterns in the vaccinated and unvaccinated tropical population; one each in urban, rural and remote areas (Robert Davis, 1982). In densely populated urban areas, with sufficient non-immunes, measles is continously transmitted and also strikes very early in life (Rosenbloom, 1968). Little is known about intra-urban transmission (Robert Davis, 1982) but nosocomial transmission occurs within neighbourhoods and household contacts (Guyer, 1976). Measles transmission is discontinous in unvaccinated rural populations with interepidemic intervals of varying length (Agboton, 1969). Robert Davis (1982) indicated three theoretically possible patterns of urban to rural transmission. Measles can spread from village to village, independent of urban transmission, or it can also spread from urban areas to villages, with a secondary spread between villages. Finally, measles spreads from urban areas to villages, without secondary spread between villages. Therefore, the periodicity

and age distribution of measles distinguishes a rural from an urban measles transmission. In unvaccinated remote areas measles transmission can be viewed from the classic work of Panum (1940) on the Faroe Islands. The smaller and less accessible the community, the lower is the risk that it will be struck by measles in any given year. When measles strikes, it affects all susceptibles, with higher case-fatality rates among young children, and declines strongly with age. In remote communities, measles spreads rapidly and then dies out, when introduced to the whole population, (Black, et al., 1974; Black 1975; Adel and Gajdnsek, 1963). Geographical and ethnic factors determines the epidemiological pattern of measles virus infections between and within island communities (Rosen, 1962; Black, 1966; and Gould et al., 1971). These factors such as crowding in densely populated unvaccinated cities promotes close personal contact between susceptibles and contagious persons. The large population then, assume constant replenishment of the pool of susceptibles (Robert Davis 1982)

FAECAL EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc)	Tabulated t t tab(0.05)	
Shredded paper	4099.20 ± 137.14	3.91	2.31	P<0.05
Saw dust	2000.80 ±237.47	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust. df = 8

TISSUE EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc)	Tabulated t t tab(0.05)	
Shredded paper	17501.00 ±632.92	3.85	3.21	P<0.05
Sawdust	23041.60 ± 1295.07	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust. df = 8.

Indular communities are too remote to be constantly reinfected from outside. Introduction of the virus produces an explosive outbreak, but the number of births is not large enough to sustain transmission once local susceptables have been infected . The behaviour of measles in most rural communities is intermediate between those in insular and urban communities , indicated by marked seasonality.

The incidence of measles is vaccinated urban communities, however, in very low with a high vaccination coverage as a result of easy access. There is also a lower unit cost and an easier cold chain conservation, Observed declines in incidence and mortality of measles after mass immu nization have been demonstrated (Guerrere et al., 1974 WHO, 1978). Heymann (1980) observed a decline in number of declared measles cases and a shift in the age distribution of urban measles cases towards higher age brackets in Yaounde - Cameroun. Similar observations were reported in CZechoslovakia and the U.S.A. (Segda 1979, Himman. et al, 1980, Pol, 1983) but not in other urban areas of the developing world (Heyman, 1980). Following the introduction of mass measles vaccination campaign in several rural areas of West Africa, the incidence of measles decline from 15 per 100,000, to only 4 per 100,000 (Robert Davis, 1982). These cases of measles reached its lowest point, about eight months later, it returned to its pre-vaccination programme levels (Forge, 1971).

In The Gambia, in 1967, a mass measles campaign exercise was launched which reached 92.5% of the rural children aged Six months to four years (Foster and Pifer, 1971). The impact of immunization on remote and insular populations suches the Pacific Atoll (Ebeye and Marshall Islands)

FAECAL EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc	Tabulated t t tab(0.05)	
Shredded paper	4899.20 ± 197.14	3.51	2.31	P<0.05
Saw dust	2800.80 ±237.47	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust, df = 8

TISSUE EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc)	Tabulated t t tab(0.05)	
Shredded paper	17501.80 ±632.92	3.85	3.21	P<0.05
Sawdust	23041.60 ± 1295.07	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust, df = 8.

resulted in achieving a high vaccine efficacy of 83.5% without any decrease in immunity in the population several year after immunization (McIntyre, et al 1982.

In Africa the epidemiology of measles is quite variable in both its seasonal pattern of antibody development and the modal age for the peak incidence of the disease. In the tropics, measles occurs at an earlier stage of life than in temperate areas and earlier still in crowded urban communities than in scattered rural settlements (Morley, 1962). In Nigeria, the median age at which measles is acquired varies from 16.5 months in northern to 21.5 months in eastern Nigeria and is 24.7 months in Ghana, 29.7 months in Zambia, Rhodesia and South Africa, (Morley, 1969). Serological surveys for measles antibodies carried out using serum collected from various areas indicated a difference in patterns of antibody development. In isolated areas, the presence of antibodies corresponded with the history of the disease in the community. In measles endemic areas most persons in the older age brackets had antibody but the age at which they were acquired varied considerably in different countries (Black 1975). The pattern of antibody development in a community coincides with the age-specific attact rate. Antibodies are acquired more rapidly in large families, but there

is no relationship with some socioeconomic factors. Measles antibodies cross the placenta and protect the infant during the first six months of life. In some, maternal antibody may persist for almost a year. Vaccination with the live virus fails to 'take' during this period because of the neutralizing effect of maternal antibody (Greenwood and Whittle, 1981). In the tropics, maternally derived measles antibody disappears more rapidly than it does in developed countries, perhaps because of a higher rate of immunoglobulin turnover in infants exposed to many infectious and most antibodies have decayed by the time an infant reaches the age of 7 - 8 months (Greenwood and Whittle, 1981; Abdurrahman, et al., 1982).

The seasonal pattern of measles in some African Countries are well documented. These patterns shows a lot of seasonal variation depending on the location of the area. In Sudan, measles shows a peak during the dry period, from April to June (Elkarim and Salih, 1981). The Niger Republic and Sierra Leone show a

rising incidence from May to June, (Wetseason) but in Nigeria and Ghana, the lowest incidence occurs during the wet seasons (Morley et al., 1967a). Hence in West Africa, the peak incidence occurs in the dry season, January to March. In East Africa, in the equatorial countries of Uganda, Kenya and Tanzania, the peak month is April. In Zambia, Rhodesia and Malawi the peak months are November, December and January, (Morley et al., 1967b; WHO, 1962). Gans et al (1961) observed the epidemiology of measles based on data collected from widely separated places in Nigeria (Vom - Northern Nigeria, Ilesha and Lagos-South Western Nigeria). They showed that measles occurs in annual outbreaks with the greatest intensity in the months before the rains. The variation in the annual incidence in areas of high infection pressure (large town - Lagos) is low, whereas in rural areas (Vom) it is m uch higher.

Hence there are high infection pressures in larger towns than in the rural areas. In rural or semi-rural areas the seasonal activity on the farms may influence

the readiness with which mothers bring their children to hospital, as well as the spread of the disease (Morley and MacWilliam, 1961). The development of the epidemics in the dry seasons in West Africa was noted by Morley, (1962) to be due to the fact that agricultural work comes to a standstill and, by tradition, the people gather together for festivals, to which children are brought on their mothers' backs and are exposed to droplet infection. While, the decline in measles cases with the start of the rains in Nigeria, West Africa may be associated with the dispersal of a considerable proportion of the community to their farms, (Morley and MacWilliam, 1961).

2.9 THE CONTROL OF MEASLES DISEASE

The prevention of measles can only be possible on the basis of stimulating both the humoral and cell-mediated immunity (Beale, 1977). Since the introduction of the live attenuated measles virus vaccine in 1963; it has been estimated in 1976 that 24 million cases of measles and 2,400 deaths have been prevented (Bass et al., 1976). Most of these vaccines presently in use were derived from one of the viruses isolated by Enders and Peebles (1954) which has been designated the Edmonston strain. The other vaccine against measles to be used were killed whole virus preparations introduced in 1965 and later more potent and purified preparations were made. Split vaccines based on the hemagglutinating component were again developed (Karzon et al 1965; Norrby, 1967; MacMcrine and Parisins, 1968). The development of killed measles vaccines became necessary as a result of infection of the CNS caused by living

vaccines. Also the protection of children under one year old who are prone to serious complications can be effected by using killed vaccines given early in life (Beale, 1977). To simplify the administration of vaccines, killed measles vaccines can safely be combined with other killed vaccines against diseases such as diptheria, tetanus, pertusis and poliomyelitis (Dick et al., 1966). There is a limitation however, to the use of killed measles vaccines, since they are associated with serious hypersensitivity reactions (Fulginiti et al., 1967) probably an acute immune complex disease with bronchiolitis and petechial hemorrhages in the skin. The vaccine is known to induce only a short-lived immunity, but sensitized individuals could be reinfected and produce very large quantities of antibody directed against the viral hemagglutinin (Karzon et al., 1965). In addition, atypical often severe measles infections have been reported in some children who were vaccinated with the killed

vaccines and subsequently exposed to measles (Anonymous. 1979). In some instances especially when children were revaccinated with live vaccines, delayed hypersensitivity reactions have occured (Buser, 1967). There are several possible explanations for such hypersensitivity reactions and failures caused by the killed vaccine; such as the failure to stimulate IgA at the portal of entry of the measles virus in the respiratory tract upon parental administration, the virus then multiplies locally producing sufficient antigen to create circulating immune complexes (Beale, 1977). This explanation may be wrong, since other killed virus vaccines, (polio or influenza, do not confer solid local immunity and are not associated with similar hypersensitivity. Another explanation is that hypersensitivity can occur to some sensitizing non-viral components which can be removed by purification of the antigen and omission of the adjuvant (Lennon et al., 1967; Wilson et al., 1968). A more plausible explanation is that. the killed vaccine lacks an important antigen

required for protection, or fails to present it properly to the immune system; since low levels of antibody produced by natural measles infection confer protection to infected hosts when administered passively, while a larger quantity of antibody raised by killed vaccine are required for protection (Karzon et al., 1965). Natural measles in sensitized individuals is the trigger which causes the hypersensitivity reaction, so that it is very unlikely that any tissue culture components are the cause (Beale, 1977). There are marked differences in the response against killed and living measles vaccines. Norrby. (1975) has shown that antibodies which inhibit haemolysis, are present after natural measles or living vaccine but not after killed vaccine. Another possible difference is the presence of cytotoxic antibody not adsorbed by haemagglutinin. This antibody appears not to be induced by killed vaccine (Kibler and ter Meuten, 1975).

There are two living attenuated measles vaccines most widely used today, Schwartz and Moraten .. strains, both derived by continued passage of the Enders - Edmonston strain in chick embryo fibroblasts at reduced temperatures (Dudgeon, 1969). Other strains such as the Sugiyama strain grown in calf kidney cells (Mirchamsky, et al., 1974) have been widely used. There are two major problems associated with living measles vaccines as it relates to adverse reactions both short term and long term and also the duration and degree of immunity. The Schwartz strain which was used in a controlled trial by the Medical Research Council (MRC, 1966) showed less cases of febrile convulsions following measles vaccine (1.9/1000) than following natural measles (7.7/1000) also the rate was reduced following prior administration of killed vaccine. Living measles vaccine is not recommended for use in children with leukaemia or other malignant diseases, nor in those with a deficiency of cellular immunity. In such people

measles and measles vaccine cause a generalized infection with a characteristic giant cell formation such as giant cell pneumonia (Martin et al., 1982). Natural measles confers a life long immunity even in the absence of reinfection, while living measles vaccine remains effective at a high level (MRC, 1966) for about 10 years post vaccination.

Unlike natural measles where second attacks are very rare, a significant number of attacks of clinical measles do occur in vaccinated individuals (Beale, 1977). Krugman, (1971) studied the response of children given, living measles vaccines and showed that many children developed low levels of antibody with an anmnestic response achieved by subclinical reinfection rather than clinical infection on exposure to measles virus. The increasing occurence of measles in previously immunized children have been reported by a number of investigators (Wyll and Witte, 1971; Plotkin, 1973; Rawls et al., 1975).

In Nigeria, field surveys have indicated the incidence of measles in recipients of the vaccines (Morley, 1962; Henderickse and Sherman, 1965; Fabiyi and Tomori, 1973). The factors possibly responsible for these vaccine failures are as a result of the exclusive use of killed measles virus vaccine which affords only short-term immunity (Krugman, 1971; Norrby, 1975). The usual and expected failure of a serological response is 2.5% of persons given the vaccine under proper conditions (Lerman and Gold, 1971).

The use of live measles vaccine of low or no potency, owing to improper handling, high storage temperatures and over-exposure to light (Nagler et al., 1965; Arbeteretal, 1972; Eghafona, 1978) have been noted. Also the neutralization of the live virus in the vaccine by maternal antibodies in infants less than one year of age (Lepow and Nankervis 1969; Weiner et al 1977; Greenwood and Whittle, 1981) have been observed.

In the tropics maternally derived antibody disappears more rapidly mostly at the age of 7 - 8 months (Abdurrahiman et al, 1981) than it does in developed countries, perhaps as a result of the higher rate of immunoglobulin turnover in infants exposed to many infections. Hence the optimum age for measles vaccination in different areas can be determined, when the age distribution of clinical measles cases and the rate of disappearance of maternally-derived measles antibody are known. Measles vaccine is relatively expensive, beyond the financial resources of poorer developing countries which have less money to spend per head on health projects.

The impact of measles immunization in some developed countries such as Czechoslovakia and the USA clearly shows that measles immunization can reduced the incidence of measles very greatly (Winman, 1983). In developing countries, however, many factors intervene to reduce the overall impact of a measles immunization programme. These limitations which includes maintenance of vaccine potency, achieving high coverage

FAECAL EGG COUNT

Bedding	Mean ± SE	Calculated t. (t calc	Tabulated t t tab(0.05)	
Shredded paper	4099.20 ± 197.14	3.91	2.31	P<0.05
Saw dust	2000.00 ±237.47	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust, df = 8

TISSUE EGG COUNT

Bedding	Mean ± SE	Calculated t (t calc)	Tabulated t t tab(0.05)	
Shredded paper	17501.00 ±632.92	3.85	3.21	P<0.05
Sawdust	23041.60 ± 1295.07	-	-	-

Note: Mean for shredded paper was compared with mean for sawdust, df = 8.

of children within the target age range, typically 9 - 11 months and operational costs must be accounted for (Feachem and Koblinsky, 1982).

In the Cameroun, Heyman et al., (1982) carried out field trials of a more heat-stable measles vaccine with some degree of success. Intranasal immunization has been tried with live attenuated measles vaccine (Zahradnik et al., 1978) and quite recently a new measles vaccine (Edmonston-Zagreb vaccine) that can be given at 4 - 6 months old intranasally, has been developed and tried in the Gambia (Whittle et al, 1984. This vaccine offers certain advantages. The vaccine grows well in lung fibroblasts and maternal (1gG) antibodies do not reach the lung epithelial surface to neutralize the vaccine.

To define the correct age at which measles vaccine should be administered is necessary to safeguard against vaccine wastage. In the U.S.A. measles vaccine is given at 15 month and 12 months in Canada. For the African child, the age is not well defined.

It is 6 - 9 months in Nigeria (Harry and Ogunmekan, 1981; Ogunmekan and Harry, 1981, Abdurrahman; et al., 1982, Heyman, 1983), and 7½ months in Kenyan Children (M.O.H Kenya and WHO, 1977). The 9 months minimum age seems to be widely adapted in most parts of tropical Africa. The question of identify-ing the target population is also necessary. In West and Central Africa almost all cases of measles occur before the sixth birth day (Morley et al., 1966, McGregor, 1964; McBean, 1972). Thus, the initial attack phase of mass measles campaigns carried out in these areas should attempt to immunize all children six months to six years of age. For an effective measles elimination strategy, attention should be focussed on an improved surveillance system; aggressive outbreak control, national will to apply resources; increased professional education; a fully functional immunization programme with total geographical

coverage for maintenance of a high level of immunisation. Others includes the necessity to ensure an effective cold-chain for maintenance of vaccine potency; the identification of a correct age limit; the enactment and enforcement of school immunization laws requiring children from kindergarten to a higher grade show proof of measles immunity and finally, national immunization programmes should emphasize control in urban settings since they serve as reserveirs of the measles infection (Rey 1969)

CHAPTER 3

RESEARCH PROCEDURES

3.1 MATERIALS AND REAGENTS

3.1.1 Microtitre Apparatus

The hemagglutination inhibition test was performed in Linbro/Titertek microtitration multi-well plates (No.76-221-05) with conical "V" bottom wells (1.0x0.[illegible]cm approximately) with each well having a capacity of [illegible].25ml approximately. The plates were obtained from Linbro Scientific, Inc. a Subsidiary of Flow Laboratories Inc., Handem, Conn. 06517, U.S.A. Microdiluters and micro-dropper pipettes obtained from the same source were used.

3.1.2 Measles HA antigen (Behringwerke)

Hemagglutinin obtained by splitting measles virus (Strain 1977, Dr. G. Enders) with Tween -80-ether and then lyophilised.

3.1.3 Measles control serum (human), positive, lyophilised (BehringWerke); prepared from a pool of measles immune sera (this positive control serum inhibits hemagglutinating activity of measles HA antigen and has a titer of 1:1024).

3.1.4.Measles control serum (rabbit), negative, lyophilised (Behringwerke), prepared from pool of measles - susceptible children sera (this does not inhibit hemagglutinating activity of measles HA antigen).

3.1.5. Kaolin

The kaolin used to absorb all the non-speccific antibodies that might be present in the sera was obtained from Sherman Chemicals Limited, London, and prepared as follows:-

25.0g of kaolin powder was added to 50ml of normal physiological saline, mixed thoroughly and made up to 100ml.

3.1.6. Normal Physiological saline

8.5og Nacl was mixed in 500ml of distilled water and made up to 1 liter, and sterilised by autoclaving for 15 minutes at 120°C.

3.1.7 Alsever's solution (Composition and preparation)

Glucose	20.8g
Nacl	4.2g
Citric acid	0.55g
Sodium citrate	8.0g

The salts were disolved in 500ml distilled water and made up to 1 liter. This was followed by
and made to 1 liter. This was followed by sterilisation in an autoclave for 30 minutes. The solution was then stored at 4^{o}c until needed.

3.1.8 Collection of Monkey Erythrocytes for HA1 Test

The red blood cells of a one year old patas monkey (Erythrocebus patas) was collected on four occasions by cardiac puncture and on two occasions through the jugular veins without sedation as performed by an experienced small animal veterinary surgeon. In either of these methods, the blood were withdrawn and placed in four times its own volume of Alsever's solution.

The monkey erythrocytes were washed in physiological saline for at least three times and the buffy cost removed. The packed erythrocytes were then stored at 4°c for not more than one week. A one percent suspension was made from the stock, whenever needed for the hemagglutination inhibition test.

3.2. MATERIALS & MATHODS

3.2.2. The study Area

Bauchi State is located between latitude 9 30^{0}N to 12 30^{0}N and longitude 8 36^{0} East to 11 58^{0} East approximately. Bauchi state has a population of about 2.4 million (1963) census and an area of 66.5 million square kilometers with Bauchi as the capital. The state is composed of sixteen local Government areas and is berdered in the East by Gongola State, to the North East corner by Borno State, to the South by Plateay State and to the West and South West by Kano and Kaduna states respectively. The state has mean daily maximun Temperature of 32.4^{0}C. The average annual rainfall of 109cm. The hottest month is April (40.6^{0}C) with December (6.1^{0}C) and January (7.2^{0}C) as the coldest months. Health care in Bauchi State is delivered through nine functional general hospitals, one leprosarium, two comprehensive health Centres, seven primary health centres three urban maternities, several health Clinics,

a specialist hospital at the State Caiptal, four private medical clinics and a few industrial, police and Military clinics.

3.2.2 Collection and Analysis of Data from Medical Records

Measles immunization data for the EPI mass campaign exercise were extracted from the records of the Bauchi State Epidemiological Unit covering the periods 1980 through 1983. These immunization activities were carried out by the mobile team of the Epidemiological Unit and by centres attached to some hospitals within the State.

Cases of measles infection and deaths notified as due to measles disease were recorded daily and reported monthly to the State Epidemiological Unit from hospitals, dispensaries, child health centres, and schools within the State. The diagnosis were made by experienced nurses and doctors. Name, age, sex, residential area, history of previous immunizations were reported for each of the children diagnosed to have measles.

3.2.3 Collection of Blood Samples and Serological A /sis

The existing health care establishments in the State such as hospitals, health centres, and clinics were used as centres for the collectinn of blood specimens in each locality. This is because, it was easier to reach, or find a sizeable number of individuals in these places. Questionnaires were designed which included the following information (Appendix 3).

(a) Name and date of survey

(b) Age

(c) Sex

(d) Addresses

(e) Occupation

(f) Medically important data such as pregnancy, if female, duration of pregnancy, number of pregnancies, miscarriages and abortions.

(g) Income, education or school attended

(h) Socioeconomic status

(i) Means of livelihood and dietary habits.

(j) Type of water supply

(k) Toilet facilities

T The consent of subjects in this study was obtained and about 10mls of blood from adults and adolescent and 5mls from infants were drawn by veneous puncture. The blood was immediately transferred to a sterile bijou bottle kept in a sloping position at room temperature and the blood allowed to clot. The serum was gently decanted into a sterile centrifuge tube and centrifuged for 10 minutes at about 1,000 rpm to pack any red blood cells present. This clear serum was again transferred to another sterile bijou bottle and properly labelled. All samples collected from the field, were transported in dry ice to the immunology laboratory of the Specialist Hospital Bauchi and stored in a deep freezer at - 20oc. When enough serum samples were collected during the survey which started in November 1982 to early 1984, transportation was effected in dry ice to the virology laboratory of the Department of Microbiology ABU Zaria. The samples were again stored at -20°c. until used. In this laboratory, the serum samples were thawed only once for the hemagglutination inhibition test, that was conducted in batches representing each of the age groups surveyed.

3.2.4 Treatment of Sera to Remove Non-Specific antibodies

Since some human sera (as well as those from o r species) contain agglutinins for monkey red blood cells. It is therefore advisable to adsorb each serum at 4°c overnigh with 0.1ml of a 50% monkey erythrocytes (Kunita et al., 1963). This also applied to the measles positive and negative control sera after each had been reconstituted with 1.0ml distilled water. The removal of non-specific inhibitors of agglutination was carried out by teatment of each serum samples with 25 percent kaolin (Monath et al., 1970; Inouye and Kono, 1972; Kono and Milgrom; 1977).

Serum in 0.1ml volume was added to each 0.3ml of 25 percent kaolin. The mixture was allowed to stand at room temperature for about 20 minutes. After which the mixture was centrifuged at 1,000 rpm. to pack the kaolin. Then, into each of the treated serum, a drop of 50 percent monkey erythrocytes was added, and this mixture was kept at 4°c overnight.

to remove the non-specific inhibitors. The following day, the mixture was contrifuged at about 1,000 r.p.m. for 10 minutes to pack the crythrocytes. The supernatant (which now represented 1:4 serum dilution) was removed carefully, with a sterilo pastour pipetto into a sterile test tube.

3.2.5 Titration of Antigen:

The philised measles HA antigen was reconstituted with 1ml sterile distilled water as recommended by the manufacturer (Behringwerke). A preliminary 1:4 dilution of the antigen was prepared in a test tube by adding 0.1ml of reconstituted antigen to 05ml of sterile saline. 0.025ml of sterile saline was added to wells 2 to 9 in wach of three rows of wells in a V-type microtitration plate and 0.05ml to 3 additional wells. 0.05ml of the 1:4 dilution of antigen was added to the first well in each of the three rows with the aid of a diluting loop. 0.025ml was transfered from the first well to the second well in each row and contained in series to the end of each row. The solution was mixed thoroughly, after each transfer and 0.025ml from the last well of

each row was discarded. The dilutions of antigen now ranged from 1:4 in well 1 to 1:1024 in well 9, with a 2 fold difference between wells 0.025ml of saline was added to each well of diluted antigen. 0.025ml of a 1 percent suspension of monkey crythrocytes was added to all wells. The plate was tapped gently to ensure an even distribution of erythrocites. The plate was then sealed to prevent evaporation and incubated for 1 hour at 37^{0}C. After which the highest dilution of antigen which caused HA was taken as the antigen titer. This represented one haemaglutination unit (1 HA unit). This 1HA unit was then multiplied by 4, to obtain a 4HA unit. The 4HA unit of antigen was used in the HA1 test.

3.2.6 The Hemmaglutination Inhibition Test

Enders-Ruckle (1965) described the HA1 test as the most sensitive. Simple and accurate test of choise in measuring the humoral antibody to measles virus.

Hence, the adoption of the test for the present sorological survey. 0.025ml sterile saline was added to wells 2 to 12 of a microtitration plate, using a microdropper popette. 0.025ml of the already treated serum was added to well No.1 and 0.025ml of the same serum into well 12 (serum control). Using a microdiluter 0.025ml of treated serum was transfered from wells 1 to 2; from 2 into 3 to the 11th well and 0.025ml was then discarded. The same procedure was also adopted for both the positive and negative control sera. Then the 4HA antigen in 0.025ml volumes were added to all the wells except the last well, the serum control well. The mixtures was incubated at room temperature for 1 hour. After which 0.025ml of the 1 percent monkey erythrocytes were added into all the wells from 1 to 12. The mixtures were incubated for a further 1 hour at 37^{o}c and titers read. The highest dilution of serum giving a well - settled red blood cell pattern (inhibition of hemagglutination) was taken as the serum titer. Any small degree of hemagglutination was taken as lack of inhibition.

3.2.7 Controls: Each test contained the following controls

a. Antigen Control

To check that 4HAU were actually used in the test, 0.025ml of steriled saline was added to wells 2 to 5 in each of 3 rows. The first well of each row was left empty.

0.025ml of the antigen dilution (4HAU) was then added to the first and second wells of each row. With the aid of diluting loops 0.025ml was transfered from the second well to the third well of each row. After mixing, 0.025ml was transferred from the third well to the fourth and so on to the fifth well. The wells in each row now contain 4, 2, 1, ½ and ¼ HAU of antigen respectively. 0.025ml of saline was added to each well, followed by 0.025ml of 1 percent monkey red cells, the plate was then tapped gently to ensure even distribution of cells. The plate was covered and incubated with the test for 1 hour at 37°c. Complete agglutination occurred in wells 1, 2 and 3; indicating that 4 HAU of antigen were used in the test.

b. Serum Controls: Both positive and negative serum controls were included in each test. Each was treated appropriately with kaolin and monkey erythrocytes. The final titer of the positive control corresponded to the one stated on the label (1:1024) and indicates the highest dilution which completely inhibits agglutination.

C. Red Blood Cell controls:

0.025ml sterile saline was added to two wells. At the time of adding the erythrocytes to the remaining test samples 0.025ml of the same 1 percent erythrocyte suspension were also added and incubated with the remainder of the test

CHAPTER 4

RESULTS

4.1. RESULTS OF ANALYSIS OF DATA FROM MEDICAL RECORDS

4.1.1. PREVALENCE OF MEASLES BEFORE AND AFTER THE MASS IMMUNISATION CAMPAIGN IN BAUCHI STATE NIGERIA

Figure 1, gives details of the vaccinations given during the measles immunisation exercise in Bauchi state (1977-1984). Over 675,000 doses of meacles vaccines were administered to Children during the same period.. At the height of the active phase of the EPI (1980-1984),473,761 doses of the vaccines were administered, which represented more than half the total number of measles vaccine doses given for the study period (1977-1984).

Meanwhile, the vaccine doses were doubled in 1980/81 to over 135,000 and 169,000 doses respectively. From 1982 it started declining drastically to a level below the initial stages of the precampaign period.

There was a considerable decline in the overall incidence of measles cases during the EPI precampaign period upto 1980, a decline from 6,960 cases in 1977 to only 667 cases in 1980. Thereafter, from 1981 to 1983, the measles cases increased steadily even after the mass campaign was launched to a total of 22,427 cases a level which more than doubled the precampaign period level of 9,968 cases.

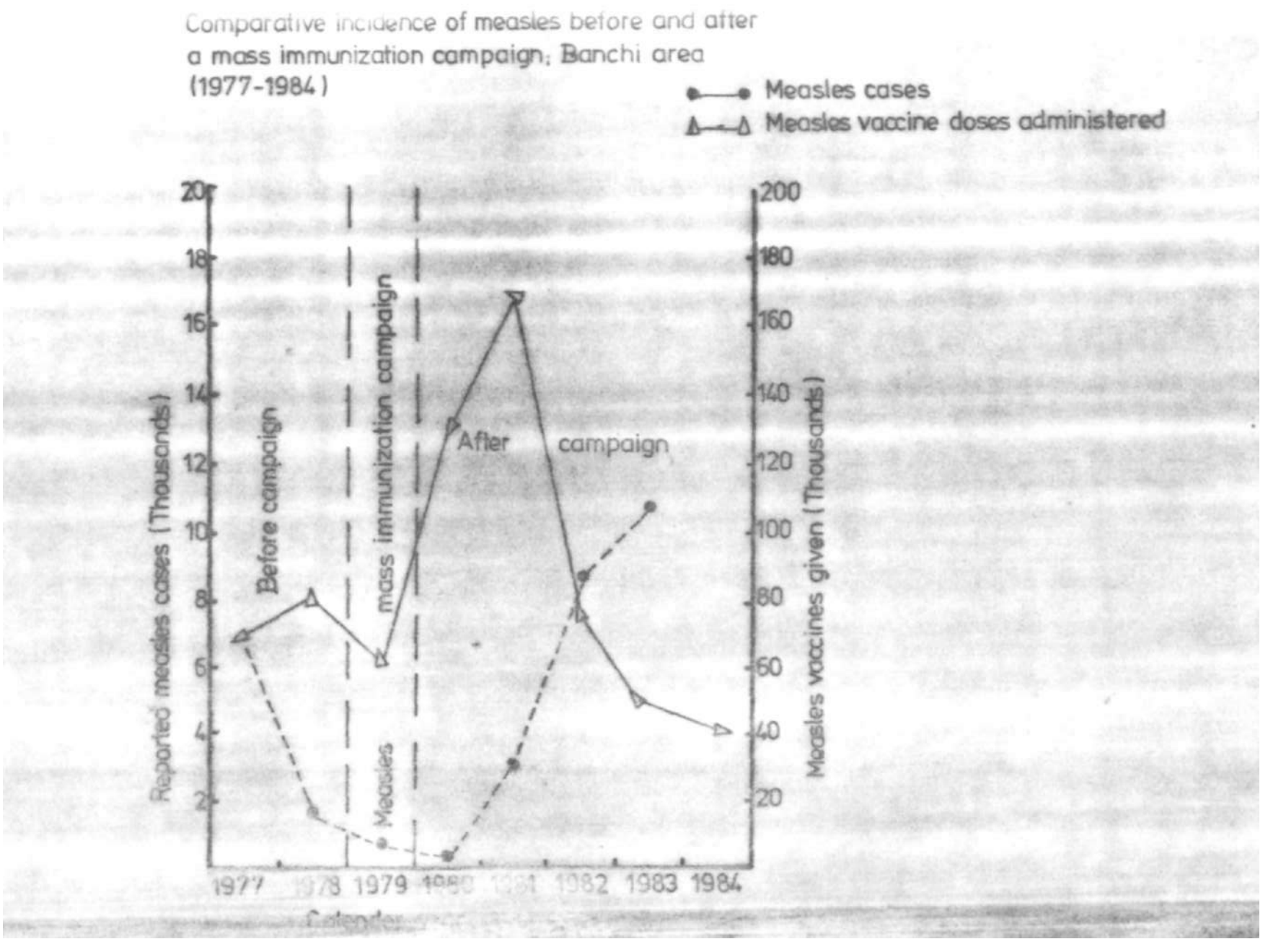
4.1.2. MONTHLY REPORT OF MEASLES CASES CASES IN BAUCHI STATE, NIGERIA

Measles transmission was centinous throughout the entire period of observation (1977 - 1984) with cases reported in every month. Figure 2 shows the distribution of reported cases of measles by month and year for period 1981 - 1983. Annually, measles opidemics begin in November of the one year and centinous to May of the following year. This is followed by a decline in measles incidence between June/July. Two paterns of measles epidemics one major the other minor, were observe during the study period. During the major epidemics measles incidence peaks up from February to M to May each year, with minor epidemic occuring between August to November. The peak incidences of measles in February and May of 1983 was significantly higher than those observed for the other years. These peak incidences more than double the peak incidences of the previous years.

4.1.3 Age Distribution of Measles Cases:

An analysis of the age distribution of notified measles cases in Bauchi State, shows that 59% of the 22,427 cases occur in the 1-4 year group. This in creased steadily throughout the period surveyed (1981-1983). This was closely followed by 5-14 year old age group with 22%, but there was a noticeable drop later in the number of cases in 1983. Those children under 1 year also showed a steady increase throughout the period. Similar to 1-4 year tage group but dropped considerably below it, with only 18% of the total cases of measles between 1981 and 1983. Measles cases in the 15 year older (c group) @@ was only 1% the lowest throughout the period (fig.3).

Comparative incidence of measles before and after a mass immunization campaign; Bianchi area (1977-1984)



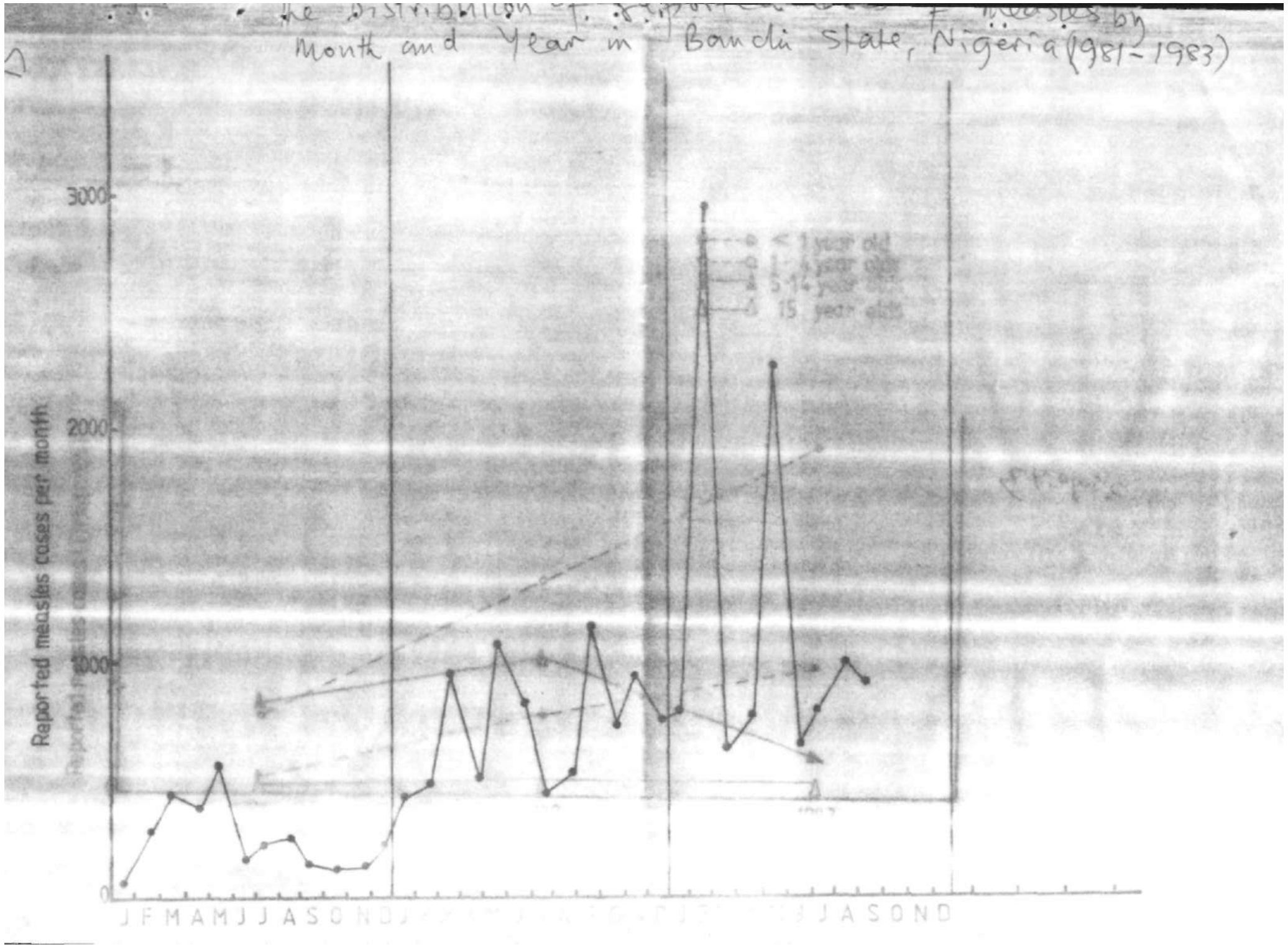
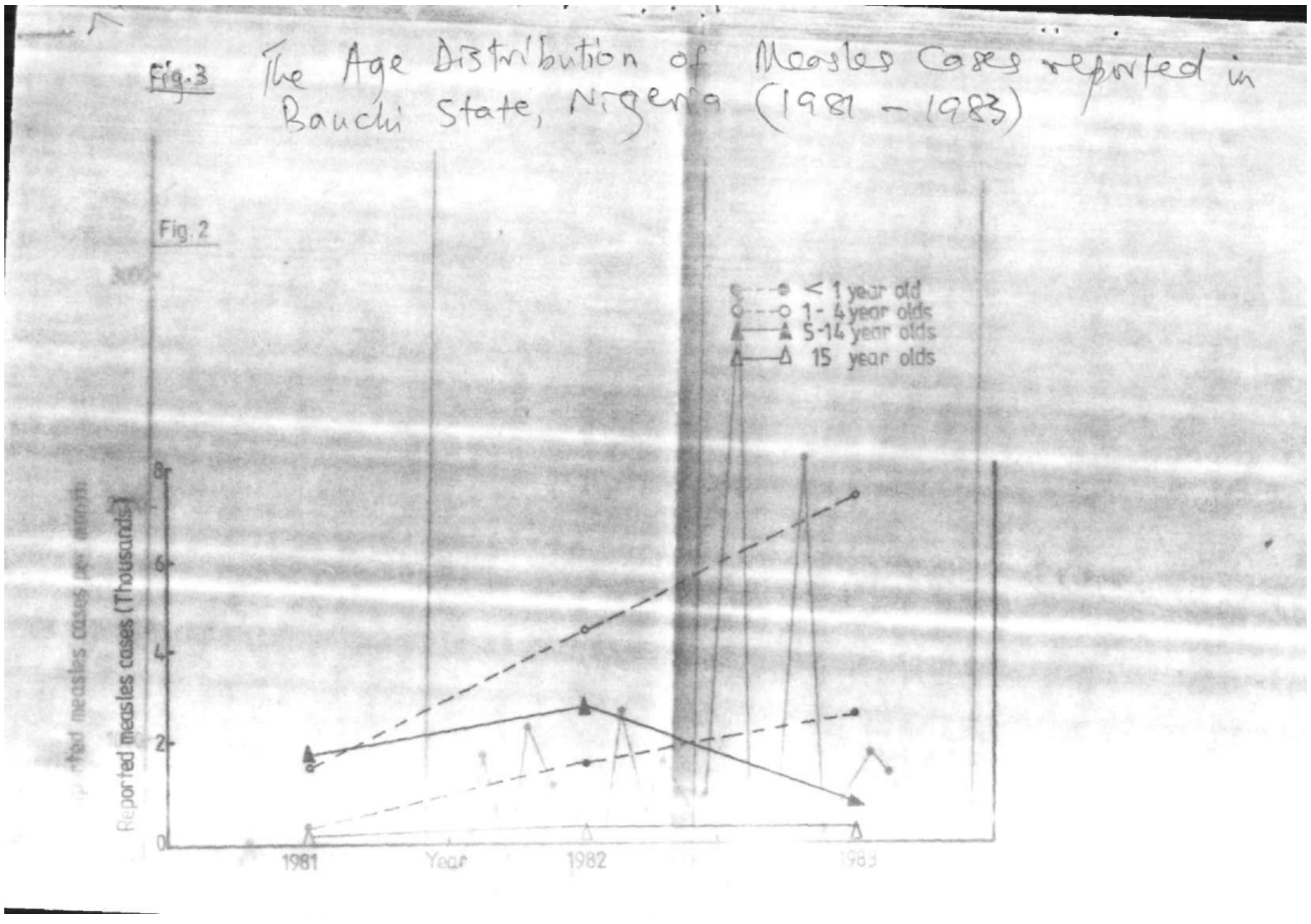

Month and Year in
Bauchi State, Nigeria (1981-1983)
3000
2000
1000
0
Reported measles cases per month
< 1 year old
1 - 4 year old
5-14 year old
15 year old
J F M A M J J A S O N D
J A S O N D

Fig. 3 The Age Distribution of Measles Cases reported in Bauchi State, Nigeria (1981 – 1983)



4.1.4 Type of Community and Measles cases:

Cases of Measles in all the urban areas of Bauchi State Nigeria was 12.5% in the population between, 1981, to 1983. While all the rural arears put together had only 2.1% of the cases: (table 4.1)

4.1.5. Mortality due to measles in Bauchi State, Nigeria

Deaths due to measles was reported every month during the entire period of observation (1980-1984), Figure 4, and every year for the period 1977 to 1984 Figure 5.

The overall mortality rate due to measles deaths t tended to correspond with the beginning of the annual epidemic period in November. There was no correlation however, between the peak incidence of measles during the major epidemics and the deaths to measles during the same period. Similary, there was no correlation during the minor epidemics except in 1982 when a large number of deaths occurred in August-September.

When measles cases were high, deaths due to measles tended to increase, there was no correlation between the overall annual incidence of deaths due to measles and the number of new borns who were brough into the population. (Fig 5).

4.2. RESULTS OF SEROLOGICAL ANAYSIS OF BLOOD SAMPLES

4.2.1. Measles HAI antibody titer in Bauchi State NIGERIA

Table 4.2 shows the prevelence of the measles HAI antibody in the general population. It can be observed that antibodies to measles was present throughout the population, and in all groups sampled. This

gave a total prevalence of about 74.5%. Measles antibodies were lowest (54%) in the under 4 year old age group. In the other age groups 5-14 and 15+ there was a high prevalence of antibodies to measles virus, which showed a prevalence rate of 75% and 77% respectively.

The measles antibody titer ranges from 1:4 to 1:1 024 in the 0:4 and 5-14 age groups. While it indicated a higher prevalence range of 1:4 to 1:4096 in the older age group.

4.2.2 Distribution of Meales HAI antibody in children and Pregnant Women

Table 4.3 shows 54% of the pre-school age children (0-4years) with antibodies to measles virus, while 74% of the school aged children (5-19years) had antibodies to the virus. The antibody prevalence rate between the two age grouping is signicantly different ($P<0.01$) . The antibody titer prevalence range is higher in the school aged and pregnant women of all age group (1:4-1:4096) as compared to the pre-school age children, with a range from 1:4 to 1:4096.

Table 4.1:

The relationship between type of community and measles cases in Bauchi State, Nigeria (1981 - 1983).

Local government Area	Type of Community	Population (1963 Census)	No Cases Notified	%Cases in the Population
Alkaleri	rural	207,121	1,175	0.57
Darazo	rural	263,603	922	0.35
Gamawa	rural	212,069	706	0.33
Ningi	rural	136,981	1,510	0.52
Tangale Waja	rural	290,031	1,058	0.36
Total	rural	1,109,895	5,371	2.13
Akko	Urban	439,159	1,845	0.43
Bauchi	Urban	190,099	6,606	3.47.
Gombe	Urban	298,239	1,819	0.77
Jama'are	Urban	55,416	3,980	7.18
Katagum(Azare)	Urban	290,406	1,274	0.44
Misau	Urban	245,239	1,532	0.62
Total	Urban	1,418,558	17,056	12.51.

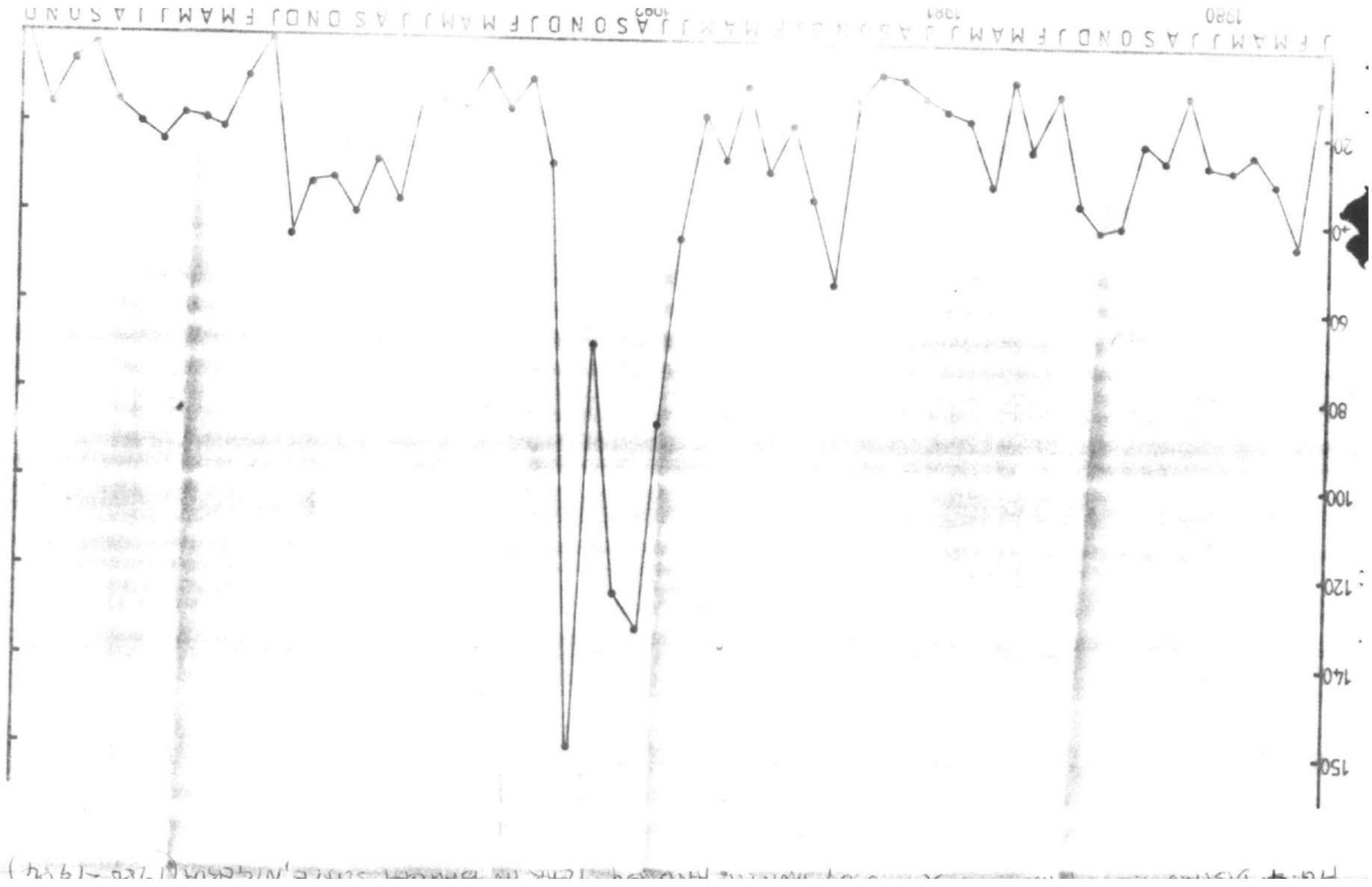

1980
1981
1982
20
40
60
80
100
120
140
150

Fig.5 Estimated number of newborns and deaths due to measles in Banchi state, Nigeria (1977-1984)

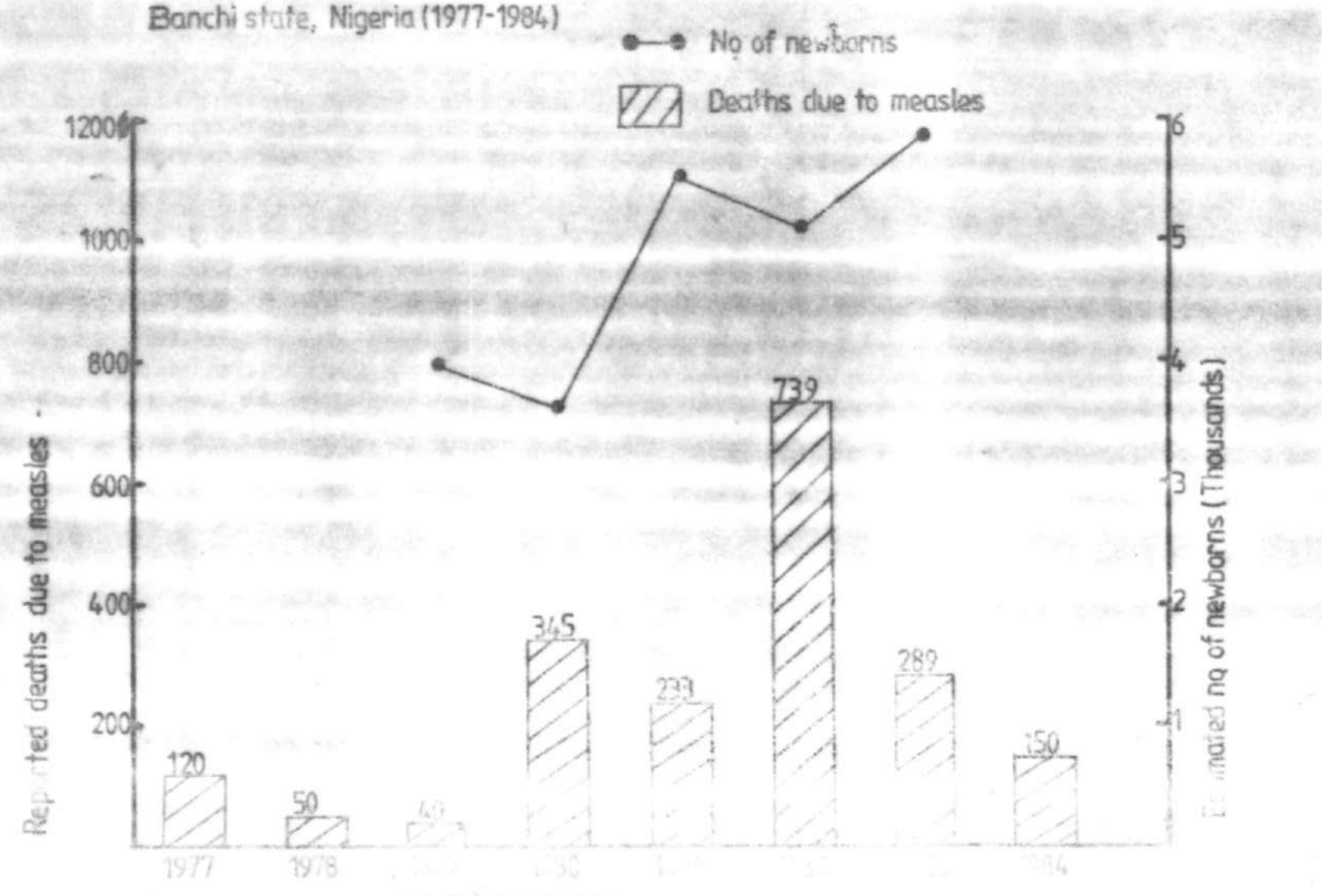


Table 4.2 Prevalence of measles HAI antibody in Bauchi State, Nigeria:

Age gropp	No Sample tested	No.Sample Positive	% Positive	HAI anti-body titer range
0 - 4	54	29	54	1:4 to 1:1024
5 - 14	38	30	79	1:4 to 1:1024
15+	347	268	77	1:4 to 1:4096
Total (0-15+)	439	327	74:5	

HAI - Hemagglutination inhibition.

Table 4.3

Distribution of Measles HAI antibody titer in children and pregnant Women:

Age Group	No. sample tested	no. sample positive	% Positive	HAI antibody titer range
Pre-School(0-4)	54	29	54	1:4 to 1:1024
School age(5-19)	105	78	74	1:4 to 1:4096
Pregnant women	94	63	67	1:4 to 1:4096
Total	253	170	67	

HAI - Hemagglutination inhibition

(0-4

4.2.3. Distribution of Measle HAI antibody titers According to sex

71.9% females and 78.2% males had detectable measles HAI antibody even at high titers (Table 4.4.) Women had marginally higher antibody titers than men, Fig.6 but the difference in the acquisition of measles antibody between the two sexes was not statistically significant ($X2=2.33$, $P>0.01$) Also a cohort analysis showed a 95% C.I. of 1 indicating no significant difference between the sexes in antibody titer.

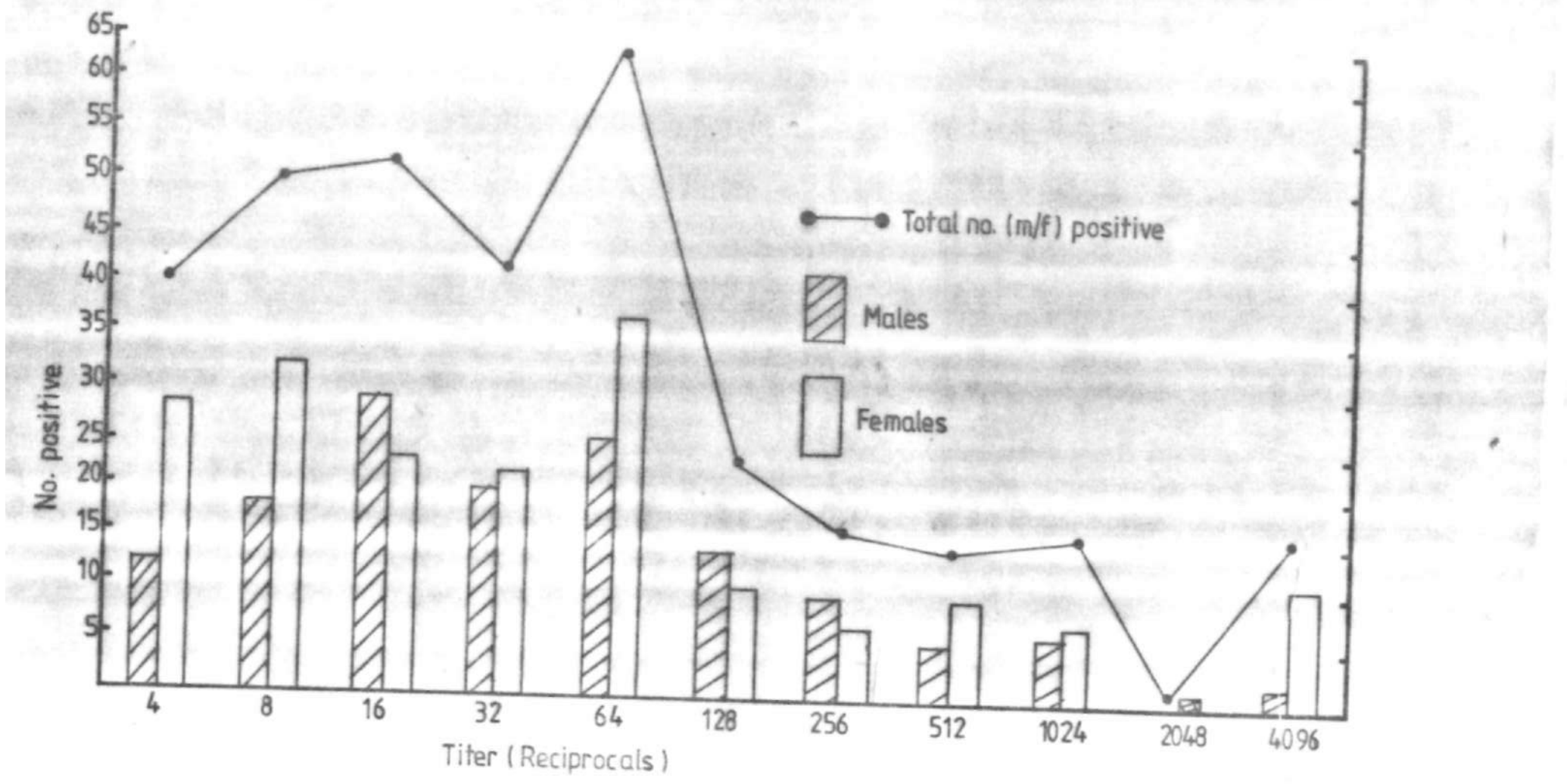
Table 4.4
Distribution of Measles HAI Antibldy titres According to Sex:

Sex	NO Samples Tested	No positive	Positive	Hai Antibody titre 0	4	8	16	32	64	128	256	512	1024	2048	4096
Females	260	187	71.9	73	28	32	23	22	37	10	7	9	7	1	11
Males	179	140	78.2	39	12	18	29	20	26	13	9	5	6	-	2
Total	439	327	75.5	12	40	50	52	42	63	23	16	14	13	1	13

X^2 1.d.f = 2.33, P>0.05: 95% CI<1, hence not significant

HAI - Hemagglutination inhibition

Fig. 6. Sex distribution of measles H A I antibody titre



4.2.4 TYPE OF COMMUNITY AND MEASLES IMMUNITY

Table 4.5, shows the relationship between some randomly selected communities and measles immunity in the State. It can be seen that the immunity status was quite variable in the surveyed areas, ranging from 65% at the State headquarter (Bauchi-Urban) to 95% in Ningi (Rural) and 100% in Darazo (rural) and Dass (rural) local government areas respectively. The prevalence of measles antibody in the rural areas was 83% compared to the 70% in the Urban centres. There was a statistically significant difference ($P<0.01$) between the two communities ~~in the prevalence~~ of two communities in the prevalence of measles HAI antibody titer.

Table 4.5

The relationship between type of community and Measles immunity in Bauchi state, Nigeria

Local Government Area	Type Community	No. Samples tested	No. Samples Positive	No. Samples Positive
Alkaleri	rural	4	3	75
Darazo	rural	9	9	100
Gamawa	rural	4	3	75
Ningi	rural	38	30	95
Tangale Maja	rural	46	38	82
Toro	rural	15	13	87
Dukku	rural	6	4	67
Tafawa Balewa	rural	22	18	82
Dass	rural	9	9	100
Total	rural	153	127	83
Akko	Urban	21	18	86
Bauchi	Urban	155	100	65
Gombe	Urban	94	71	75.5
Misau	Urban	10	7	70
Katagum (Azare)	Urban	6	4	67
Total	Urban	286	200	69.9

* X^2 1.d.f. = 9.63 $P<0.01$, 95% CI>1

highly significant.

4.2.5. MEASLES HAI ANTIBODY AND STAGE OF PREGNANCY

Out of 94 pregnant women at various stages of their pregnancy 63(67%) had detectable measles HAI antibody in their sera, while 31 (33%) gave no antibody response. 46(73%) of these women with detectable Measles antibodies were in their second trimester pregnancy, 15(23%) and 2(3%) in the third and first trimesters respectively. Those in the last two trimesters had detectable antibodies occuring at higher titers.

4.2.6. MEASLES HAI ANTIBODY AND SOCIOECONIMIC STATUS

Table 4.6, depicts the prevalence rate of measles HAI antibody in the two social classes, in the study area. The prevalence rate of antibody in the lower social class is higher (62%) than it is in the higher social class (38%). This difference is highly significant (P o.01). However, more of the pre-school and school-aged children in the higher class indicated a higher antibody prevalence rate relative to the same group in the lower social class.

Meanwhile, adults in the lower social class had higher measles antibody than in the high social class.

Table 4.6.

Measles HAI antibody and socioeconomic status:

Age group	No. Samples Tested	No Samples Positive	Social status low	%	high	%
Pre-School age (0-4)	54	29	11	20	18	33
School-age (5-19)	105	78	15	14	63	60
Adults (above 20)	280	220	176	63	44	16
Total	439	327	202	62	125	38

$X^2=26.237$, $P<0.01$, highly significant.

CHAPTER 5

DISCUSSION

The analyses of data from medical records indicated a high preponderance of notified measles cases in the 1-4 year age group. This increased steadily throughout the period under survey, 1981-1983. A similar observation was reported by Bartlett in 1957 in the U.S.A. He indicated that the attack rate of measles increases sharply in the 1-4 years old, after which there is a gradual decrease until it approaches zero at about 12-15 years of age. Our observed in crease in cases in the 1-4 year age group could probably be due to the increasing socialization of the child couple with the low proportion of immune individuals since most of them have had no previous exposure to the measles virus,

In a similar tropical setting of Norther Nigeria. Morley in 1969 observed that measles is acquired at a median age of 16.5 monthswhich falls within the 1-4 year group indicated in the present study.. Measles transmission was continous with cases reported in every month for which surveillance data were available. Annual seasonal epidemics begins in November to May and declines as the rains reach their heaviest in the subsequent months. These annual epidemics begins when there was a large number of susceptibles in the population. The declining incidence of the annual epidemics corresponds with the time (July-October) when people leave the cities to cultivate their crops in the villages. This is further compounded by the increase departure of susceptible urban young children to the villages with their mothers. In July, children also leave school which facilitates further Movements of the family at this time. In October-November crops planted with the rairs are harvested and the long dry season set in. Also schools re-open bringing children into the cities and the sudden build up of susceptibles

facilitates an out break, hence the observed increase in measles cases at the time. In December, schools close and families are free to travel for holidays only to reopen in January bringing in cases into the population, hence the sudden increase in number of measles cases furing the January months each year.

The vaccination activities by the mobile team of the Epidemiological Unit and by centres attached to some hospitals within the state seemed to have been intensive and changed the pattern of measles transmission prior to the launching of the mass immunization campaign in 1979. The period after the mass campaign experienced a rise in measles incidence Despite large doses of vaccine that was administered, particularly between 1980 and 1981. In 1982 onwards there was a reduction in the quantity of vaccines administered. The are possible reasons for such vaccine failures. The correct age for vaccine administration was not identified, hence vaccine wastage. Also loss of vaccine potency due to poor storage facilities are contributory factors (Eghafona, 1978). Actually, the identification of the correct minimal age for measles vaccine administration is crucial, since trans-placentally acquired passive immunity decays very fast in the African childr 1981; (M.O.H. Kenya and WHO, 1977; Harry and Ogunmekan, 1981; Abdurrahman etal, 1982.

Greenwood and whittle, (1981) reported that the rapid disappearance of maternally-derived measles antibody in the tropics than in developed countries are perhaps as a result of the higher rate of immunoglobulin turn over in infants exposed to many infections in the tropics. Hence, when the age distribution of cases of clinical measles and the rate of disappearance of maternally-derived measles antibody are

known, it becomes possible to determine the optimum age for measles vaccination in any community.

Cases of measles in all the urban areas of Bauchi state was 12.5% in the population between 1981 and 1983 while all the rural areas combined has only 2.1% of all the cases. It has been noted that crowded urban communities enhance the changing incidence of measles in any community (Morley , 1962). This probably might be as a result of the influx of susceptibles into the urban areas from the rural population. The child mortality rate due to measles in Bauchi sate is high with the high rates corresponding with the onset of the epidemics in November. Mortality rate increased with increased cases. This may be attributed to the fall in vaccination activity from (1982 to 1984). The EPI campaign did not appear to have an impact on the overall incidence of deaths due to measles in Bauchi State.

Results of Serological analysis of blood samples collected from the general population indicated an incidence of 75%. This is low, showing that the EPI campaign exercise had not been successful, However, there were variations in the incidence of measles HAI antibody. It was 54% in the under 4 years old and over 70% in the 5-15 year age group. The high levels of antibody in people more than 15 year of age can be attributed to their continous exposure to infected children which enhances the build up of their immunity. There was a noticeable, low antibody response in the under 4 years old, despite the high incidence of measles in the age group, It is also expected that they might have been previously vaccinated in either 1979 or 1980 at the age of either 1 or 2 years, since the 4 years mass immunization campaign in the state was supposedly concentrated on children within this age group. But with the low prevalence of antibody in this age group, it became doubtful that most of them were actually vaccinated

There are possible reasons for such Obserrably low antibody prevalence in the under 4 year age group. Despite the active immunisation campaigns which aims at vaccinating this target ppulation.

First measles vaccines cannot 'take' in the presence maternal antibody, since the antibody neutralises the measles virus in children vaccinated less then one year of age (Greenwood and whittle, 1981). The use of vaccines which may not be immunogenic enough to induce antibody formation as a result of loss of vaccine potency due to poor storage can conttribute to the low antibody response in the under 4 year age group (Eghafona , 1978). Another reason is that some of the children may be suffering from measles without sero-conversion (Lenette, et al, 1961; Cherian, etal, 1984), since inf-ection and disease without antibody response is known to occur occasionally with certain Viruses.

There was no difference between males and females in the distribution of measles antibody in the general population of Bauchi state. However, antibodies were marginally higher in females than in males. This observation is similar to that reported by Black (1976).

The immunity status of people was quite variable depending on the type of community, There were significantly more individuals in the urban areas with measles antibody than those in rural areas. There are two possible reasons for this, firstly, since urban centres serve as foci of measles virus infection (Rey,1969) coupled with the crowded nature of most urban centres, and the high rate of interpersonal contacts, more people are infected with measles and hence develop immunity.

Another reason is that, these groups of urban settlers are nearer the centres of vaccination activities than rural dwellers, hence they are easily accessible by the vaccination teams.

Mealses in pregnancy is known to cause a high rate of spontaneous abortions and premature deliveries without inducing the congenital anomalies characteristic or rubella (Briody, 1974). Investigation of meales HAI antibody in pregnancy revealed that 67% of the pregnant women had protective antibody and 73% of the seropositives were in the second trimester pregnancy. Those in the least two trimesters had detectable antibodies occuring at higher levels, implyig that their children will be fully protected and will acquire enough maternal antibody in utero.

Measles HAI antibody in the two social classes of the State were significantly different. The high seropositivity in the under 20 years old of the high social class does shows a reflection of an enlightened society who must have been vaccinated at one time in life against measles. The high antibody prevalence in the above 20 years old age group of the lower social class indicates that, they had been previously infected by measles virus. There is also the possibility of continous exposure to infected children thus, maintainig a high level of immunity.

The EPI campaign generally had little or not impact on measles cases, its epidemiology, deaths due to measles and the acquisition of protective antibody among the people of Bauchi State Therefore, the strategy of measles control in the state, must take into account the seasenal dynamics that conditions the epidemiology of the disease in different communities and social setting. Bauchi and other urban centres are reservoirs of measles disease and children who are susceptible to measles virus infection. Thus, immunization programmes must stress control in urban settings, which will reduce the potential for exportation of the disease to rural areas. It is necessary to identify an age limit for measles immunization which minimises the rate of vaccine failures and maximises the number of children under 1 year of age who can be protected against measles. Also, it is necessary for a continous sero- epidemiological evaluation to determine immunity levels of the population and response (Seroconversion) to measles vaccine. The current regised expanded programme on immunization inherited and increasing number of measles cases with few doses of vaccines so far administered, The reverse should be aimed at for the programme to be successful.

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MAP OF THE STUDY AREA (BAUCHI STATE)

Key
⊙ Local government capitals
--- Local government areas
Map not drawn to scale



(Appendix 1)

APPENDIX 2

Test of Significance:

(a)

Sex Factor	Measles antibody Present	absent	Row totals
Females	187	73	260
Males	140	39	179
Column totals	327	112	439

Null hypothesis: The difference in measles antibody titers between the two sexes is due to chance (not significant)

Alternative hypo.: There is a real (significant) differene difference between the two sexes

Calculating Pvalue:

$$x^2 = \sum \frac{(\text{observed number} - \text{expected number})^2}{\text{Expected number}}$$

Expected numbers;

$$\frac{327}{439} \times 260 = \frac{85020}{439} = 193.7$$

$$\frac{112}{439} \times 260 = \frac{29120}{439} = 66.3$$

$$\frac{327}{439} \times 179 = \frac{58983}{439} = 134.4$$

$$\frac{112}{439} \times 179 = \frac{20048}{439} = 45.7$$

$$x^2 \text{ i.d.f.} = \frac{(187-193.7)^2}{193.7} + \frac{(73-66.3)^2}{66.3} + \frac{(140-134.4)^2}{134.4} + \frac{(39-45.7)^2}{45.7}$$

$$= \frac{44.89}{193.7} + \frac{59.29}{66.3} + \frac{31.36}{134.4} + \frac{44.89}{45.7} = \underline{\underline{2.33}}$$

Pvalue at i.d.f. is greater than 0.05 and 0.01 hence accept the Null hypothesis. Not significant

P>0.01; P>0.05

Therefore the difference in the acquisition of measles antibody between the two sexes are more due to chance.

(b) Cross-sectional (cohort) analysis

Sex factor	outcome present	condition absent	Row totals
Females	187	73	260
Males	140	39	179
Column totals	327	112	439

The outcome in this study, refers to either of the sexes (male/female) with detectable measles antibody. While the condition in either of the sexes, refers to those without antibody.

setting up 95% CI:

$$\text{Relative Risk (RR)} = \frac{187\ (179)}{140(260} = 0.0919$$

$$\text{Var}\log_e RR = \frac{187(179)}{140\ (260)} = \log_e 0.0919 = -2.3871$$

$$\text{Varlog}_e RR = \frac{1}{187} + \frac{1}{140} - \frac{1}{260} - \frac{1}{179} = 0.003057$$

$$\text{SElog}_e RR = \sqrt{\text{Varlog}_e RR} = \sqrt{0.003057} = 0.05529$$

95% CI = $\log_e RR \pm 1.96(\text{SElog}_e RR)$

$= -2.38705 \pm 0.10836$

$= -2.2787, -2.4954$

antilog (LN) = 0.1024172, 0.082463

hence $0.08246 \leq RR \leq 0.1024172$

$\therefore$ 95% CI < 1, not significant

(c)

Type of Community	Measles antibody present	absent	Row totals
Rural	127	26	153
Urban	200	86	286
column totals	327	112	439

Null hypothesis: The difference in measles antibody titers between the two communities is due to chance (not significant).

Alternative: There is a real or significant difference in measles antibody between the two communities.

Calculating the pvalue

$$x^2 = \sum \frac{(O-E)^2}{E}$$

expected numbers;

$$\frac{327}{439} \times 153 = \frac{50031}{439} = 114 \qquad \frac{112}{439} \times 153 = \frac{17136}{439} = 39.04$$

$$\frac{327}{439} \times 286 = \frac{93522}{439} = 212.9 \qquad \frac{112}{439} \times 286 = \frac{32032}{439} = 72.95$$

$$\therefore x^2 1df. = \frac{(127-114)^2}{114} + \frac{(26-39.04)^2}{39.04} + \frac{(200-212.9)^2}{212.9} + \frac{(86-72.95)^2}{72.95} = \frac{13^2}{114} + \frac{(13.04)^2}{39.04} + \frac{(12.9)^2}{212.9} + \frac{(14.05)^2}{72.95}$$

$$= \frac{169}{114} + \frac{170.04}{39.04} + \frac{166.4}{212.9} + \frac{197.4}{72.95}$$

$$= 1.483 + 4.354 + 0.7818 + 2.707$$

$\therefore x^2$ 1df. = 9.63

Pvalue at 1.d.f. is less than 0.01 (P<0.01) hence Null hypothesis is rejected, therefore. the alternative is accepted.

There is then a highly significant difference in measles antibody titers between the two communities.

(d)

Socioeconomic Group	Measles HAI antibody Present	absent	Row totals
low	202	38	240
High	125	74	199
column totals	327	112	439

Null hypothesis: There is no significant difference between the two Socioeconomic groups in measles HAI antibody titer

alternative: There is a significant difference in measles HAI antibody titer in the two social classes

$$x^2 = \sum \frac{(O - E)^2}{E}$$

Expected numbers:

$\frac{327}{439} \times 240 = 178.7$ $\quad$ $\frac{112}{439} \times 240 = 61.22$

$\frac{327}{439} \times 199 = 148.2$ $\quad$ $\frac{112}{439} \times 199 = 50.77$

$$x^2 \text{ 1.d.f.} = \frac{(202.178.7)^2}{178.7} + \frac{(38-61,22)^2}{61.22} + \frac{(125-14802)^2}{148.2} + \frac{(74-50.77)^2}{50.77}$$

$$= \frac{23.3^2}{178.7} + \frac{23.22^2}{61.22} + \frac{23.2^2}{148.2} + \frac{23.23^2}{50.77}$$

$\therefore x^2$ 1.d.f. = 26.237, Pvalue < 0.01, highly significant

(e) Measles antibody

Pre/school age	present	absent	total
0 - 4	29	25	54
5 - 19	78	27	105
Total	107	52	159

Null hypothesis: There is no significant difference im measles antibody in the two age groups

Alternative hypothesis: There is a real significant difference between the two age groups.

Calculating pralue

$$X2 = \frac{(O-E)^2}{E}$$ O = observed values, E = Expected values

(E) values

$$\frac{107 \times 54}{159} = \frac{5778}{159} = 36.34$$

$$\frac{52 \times 54}{159} = \frac{2808}{159} = 17.66$$

$$\frac{107 \times 105}{159} = \frac{11235}{159} = 70.66$$

$$\frac{52}{159} \times 105 = \frac{5460}{159} = 34.34$$

$$x2 \text{---} \quad \frac{(29-36.34)^2}{36.34} + \frac{(25-17.66)^2}{17.66} + \frac{(78-70-66)^2}{70.66} + \frac{(27-34-34)^2}{34.34}$$

1.d.f.

1.48+0.76+1.57+3.05+6.06

0.01 highly significant, hence the alternative by hypothesis is expected which means that there is a significant difference between the pre-school age and school age antibody pre-valence rate.

Appendix 3.

QUESTIONAIRE USED IN DATA COLLECTION:

1. SAMPLE NO.....DATE OF COLLECTION.....
2. NAME.....SCHOOL.....
3. AGE.....L.G.A.....
4. SIZE OF COMMUNITY, URBAN ( ) RURAL ( )
5. ADDRESS.....TRIBE.....
6. SEX, M ( ) F ( ) MARRITAL STATUS M ( ) S ( )
7. IF FEMALE, WHETHER PREGNANT.....
8. DURATION OF THE PREGNANCY.....
9. TOTAL NO. OF PREGNANCIES.....
10. NUMBER OF DELIVERIES.....
11. NUMBER OF MISCARRIAGE/s (IF ANY) .....
12. OCCUPATION .....EDUCATION .....
13. INCOME.....
14. SOCIOECONOMIC STATUS LOW ( ) MEDIUM ( ) HIGH ( )
    BASED ON:
    (a) TYPE OF HOUSING, RENTED ( ) OWNED ( )
    (b) KITCHEN AND COOKING UTENSILS
        TYPE OF STOVE OR HEARTH.....
        EARTHENWARE POTS.....
        IRON POTS.....
        DRINKING UTENSILS.....
        TYPE OF CUTLERY (a) MANUFACTURED ( )
        (b) LOCALLY MADE ( )
    (c) FOOD STORAGE FACILITIES, KITCHEN ( )
        BARNS ( ) POTS ( ) BASKETS ( )
    (d) HEALTH AND SANITATION, TYPE OF LIGHTING.
        ELECTRICITY ( ) LANTERN ( ) OIL LAMP ( )
    (e) BATHROOM ( ) TOILET, PIT LATRINE ( )
        WATER CLOSET ( ) REMOVABLE TINS ( )
        AVAILABLE BUSH ( )
    (f) REFUSE DISPOSAL DUSTBIN ( ) INCINERATOR ( )
        [illegible] ( )
    (g) WATER SUPPLY (SOURCES) TAP ( ) WELL ( )
        RIVER, POND, STREAMS ( )
    (h) CONDITION OF SURROUNDING. BUSHY ( ) TIDY ( )
15. FOOD AND HEALTH HABITS (a) POPULAR FOOD EATEN ( )
    (b) FARMING METHODS, MECHANISED ( )
        LOCAL EQUIPMENTS ( )
    (c) TYPE OF FOOD PRODUCED .....

Appendix 4

ENUMERATION LISTS FOR THE STUDY POPULATION

The following lists are included to permit reference to individuals studied in the area (Bauchi State).

Sample No.	Name	Sex	Age	Geographical Location	Type of Community	Socioeconomic Group	HAI Titre
01.	Mary Kirmon	F	5 Yrs.	Tafawa Balewa	rural	H	Nil
02.	Kabiru Hashimu	M	3½ "	Bauchi	Urban	L	Nil
03.	Danladi Ahmadu	M	1 "	Bauchi	Urban	L	1 : 4
04.	Saidu Yau	M	1 "	Bauchi	Urban	H	Nil
05.	Audu Inuwa	M	1 "	Bauchi	Urban	H	Nil
06.	Augustine Luka	M	1 "	Bauchi	Urban	H	Nil
07.	Yakubu Mohammed	M	5 mths	Bauchi	Urban	L	Nil
08.	Mohammed Gambo	M	2½ Yrs	Bauchi	Urban	H	Nil
09.	Ahmadu Ibrahim	M	4 Yrs.	Toro	rural	L	1 : 256
010.	Garba Ahmadu	M	1 "	Gombe	Urban	L	1 : 16
011.	Rilwanu Hassan	M	9 "	Bauchi	Urban	H	1 : 8
012.	Tabitha Ali	F	8 "	Tafawa Balewa	rural	H	1 : 16
013.	Juliana James	F	3 "	Bauchi	Urban	H	1 : 32
014.	Rufai Mohammed	M	10 "	Bauchi	Urban	L	1: 64
015.	Mohammed Buhari	M	2 "	Bauchi	Urban	L	1: 8
016.	Mune Mohammed	F	10 "	Jama'are	Urban	L	1: 256
017.	Saidu Salihu	M	2 "	Bauchi	Urban	H	1: 128
018.	Halima Umaru	F	7 mths.	Bauchi	Urban	L	1: 8
019.	Habiba Yakubu	F	2 Yrs.	Bauchi	Urban	L	Nil
020	Maria Joshua	F	2½ "	Bauchi	Urban	H	Nil
021.	Florence Nuhu	F	22 days	Bauchi	Urban	H	1 : 64
022.	Daniel James	M	5 Yrs.	Tafawa Balewa	rural	H	1: 32
023.	Audu Mamuda	M	9 days	Bauchi	urban	L	Nil
024.	Bilyaminu Ibrahim	M	3 Yrs.	Bauchi	Urban	H	Nil
025.	Gberga Jacob	M	2 "	Bauchi	Urban	H	Nil
026.	Sule Maimako	M	1 "	Bauchi	Urban	H	Nil
027.	Lydia Ezekiel	F	1 "	Bauchi	Urban	H	Nil
028.	Shehu Daure	M	1 "	Bauchi	Urban	L	Nil
029.	Adamu Ibrahim	M	8mths.	Bauchi	Urban	L	1: 512
030.	Nuhu Abdullahi	M	4 Yrs.	Bauchi	Urban	H	1: 16
031.	Murtala Yakubu	M	3 "	Bauchi	Urban	H	1: 8
032.	Salihu Idi	M	2½ "	Bauchi	Urban	L	Nil
033.	Sule Usman	M	22 days	Bauchi	Urban	L	1: 8
034.	Hauwa Adamu	F	1 Yrs.	Bauchi	Urban	L	Nil
035.	Aminu Sani	M	1½ Yrs.	Bauchi	Urban	H	1: 8
036.	Sirajo Bala	M	3 "	Bauchi	Urban	L	1:64
037.	Salisu Namman	M	3 "	Bauchi	Urban	H	Nil
038.	Dahiru Abdul	M	1 "	Bauchi	Urban	H	Nil
039.	Adama Isa	F	4 "	Dass	rural	L	Nil
040.	Hajara Adamu	F	4 "	Bauchi	Urban	L	Nil

mple No.	Name	Sex	Age	Geographical Location	Type of Community	Socioeco-nomic Group	HAI -itre
1	Lubi Mohammed	F	2 Yrs.	Bauchi	Urban	L	1:256
2	Hadiza M.	F	1 "	Bauchi	Urban	H	Nil
3	Shehu M.	M	1½ "	Bauchi	Urban	H	1:32
4	Abas Tanko	M	1½ "	Bauchi	Urban	H	1:16
5.	Binta Alhassan	F	7 mths.	Bauchi	Urban	L	Nil
6.	Marka Udoh	F	3 Yrs.	Bauchi	Urban	H	1:4
7.	Mohammed D.	M	9 Mths.	Bauchi	Urban	H	1:8
8.	Mohammed G.	M	2 Yrs	Bauchi	Urban	H	Nil
9.	Rufai Adamu	M	3 "	Bauchi	Urban	H	Nil
0.	Adamu Saleh	M	1½ "	Bauchi	Urban	L	1:128
1.	James Joseph	M	11 Mths.	Bauchi	Urban	H	1:32
2.	Umaru Ibrahim	M	4 "	Gombe	Urban	H	1:1024
3.	Aisha Hassan	F	4 "	Gombe	Urban	H	Nil
4.	Babangida M.	M	6 "	Gombe	Urban	H	1:4
5.	Edward O.	M	3 Yrs.	Gombe	Urban	H	1:512
6.	Aisha Umaru	F	3 "	Akko	Urban	H	1:8
7.	Yusuf M.	M	4 "	Gombe	Urban	H	1:64
8.	Kabiru Ibrahim	M	4 "	Gombe	Urban	H	1:32
9.	Aisha Mohammed	F	4 "	Gombe	Urban	H	1:64
0.	Christopher Emma	M	2 "	Tafawa Balewa	rural	H	1:16
1.	'Bala Garba	M	1 "	Gombe	Urban	L	1:16
2.	Nyarim Ayuba	F	6 "	Bauchi	Urban	L	1:64
3.	Shatu Buba	F	10 "	Gombe	Urban	H	1:8
4.	Maikudi M.	M	6 "	Gombe	Urban	H	Nil
5.	Mairama	F	5 "	Gombe	Urban	L	1:128
6.	Hashiya Moh.	F	8 "	Gombe	Urban	H	1:8
7.	Adamu Sambo	M	9 "	Bauchi	Urban	L	1:8
8.	Josephine John	F	7 "	Gombe	Urban	H	1:64
9.	Hadi Ibrahim	M	9 "	Gombe	Urban	L	1:8
0.	Ai Mohammed	F	8 "	Gombe	Urban	H	1:64
1.	Aishatu Usman	F	5 "	Gombe	Urban	H	Nil
2.	Adama Usman	F	6 "	Bauchi	rural	L	1:64
3.	Rabi Adamu	F	5 "	Gombe	rural	L	1:32
4.	Rabi Ibrahim	F	6 "	Gombe	rural	H	1:8
5.	Bello Sani	M	7 "	Gombe	rural	H	1:1024
6.	Mohammed U.	M	8 "	Gombe	rural	H	1:16
7.	Hamsatu Adamu	F	7 "	Bauchi	rural	H	Nil
8.	Musa Mohammed	M	8 "	Bauchi	rural	H	1:16
9.	Danjuma Garba	M	8 "	Bauchi	rural	L	1:32
0.	Audu Adamu	M	12 "	Bauchi	rural	L	Nil
1.	Salamatu Abdul	F	10 "	Bauchi	rural	H	Nil
2.	Fatu Ibrahim	F	11 "	Bauchi	rural	H	1:8
3.	Hajara Idi	F	13 "	Bauchi	Urban	H	1:4 3 mths. Preg.
4.	Halima Maimako	F	12 "	Bauchi	Urban	H	1:16
5.	Dorothy Anum	F	13 "	Bauchi	Urban	H	1:64
6.	Ladi Isa	F	10 "	Gombe	Urban	H	1:4
7.	Charity E.	F	13 "	Gombe	Urban	H	1:32
8.	Buba Adamu	M	7 "	Gombe	Urban	H	1:1024
9.	Safiya Shaibu	F	11 "	Bauchi	Urban	H	1:128
0.	Mohammed Wase	M	10 "	Bauchi	Urban	H	1:16

ample No.	Name	Sex	Age	Goegraphical Location	Type of Community	Socioeconomic group	HAI Titre
1.	Sani Yerima	M	12 Yrs.	Bauchi	Urban	L	Nil
2.	Zainabu Saidu	F	14 "	Bauchi	Urban	H	1:16
3.	Ngozi U.	F	15 "	Gombe	Urban	H	1:64
4.	Asamau Ahmadu	F	15 "	Gombe	Urban	L	1:64
5.	Sa'adatu Usman	F	15 "	Bauchi [illegible]	rural	L	1:512
6.	Deborah Sarbo	F	15 "	Bauchi	Urban	H	Nil
7.	Talatu Ibrahim	F	14 "	Bauchi	Urban	H	Nil
8.	Alti Tanko	F	15 "	Bauchi	rural	L	1:64
9.	Adama Usman	F	15 "	Bauchi	Urban	L	1:8
10.	Binta Garba	F	15 "	Shira	rural	H	1:32
11.	Dija Haruna	F	[illegible] "	Bauchi Ink l	rural	L	Nil 5mths. preg.
12.	Dan Haruna	M	28 "	Akko-Bara	rural	L	1:16
13.	Adama Ibrahim	F	23 "	Bauchi	Urban	H	Nil 6mths. preg.
14.	Halima Yerima	F	21 "	Tangale Waja	rural	H	1:32
15.	Halima B.	F	23 "	Tafawa Balewa	rural	H	1:256
16.	Patrick Dawaki	M	23 "	Tafawa Balewa	rural	H	1:8
17.	Yusuf Joshua	M	23 "	Alkaleri	rural	L	1:32
18.	Maisaje N.	M	23 "	Tangale Waja	rural	L	Nil
19.	Zakka Zamis	M	20 "	Tafawa Balewa	rural	H	1:32
20.	Mohammed Hanza	M	24 "	Gombe	Urban	H	1:128
21.	Serifatu Abel	F	22 "	Misau	Urban	H	1:8 8mths. preg.
22.	Jamilatu Alkali	F	24 "	Toro	rural	L	1:4
23.	Regina Geyn	F	21 "	Ningi	Urban	H	1:64
24.	Bethy Baba	F	20 "	Tangale Waja	rural	H	1:512
25.	Laraba Shanun	F	20 "	Tangale Waja	rural	L	1:4
26.	Manasseh Bello	M	27 "	Akko	Urban	H	1:128
27.	Rilwanu Abba	M	25 "	Misau	Urban	L	1:32
28.	Aishatu Mda.	F	27 "	Gombe	Urban	L	1:64
29.	Mohammed Ahmed	M	25 "	Gombe	Urban	[illegible]	[illegible]
30.	Jika Kadiri	M	27 "	Gombe	Urban	L	1:4096
31.	Charles M.	M	25 "	Tangale Waja	rural	L	1:128
32.	Ladi Mohammed	F	28 "	Gombe	Urban	H	1:4
33.	Lami Garba	F	27 "	Gombe	Urban	H	1:64 5Mths. Preg.
34.	Tabe Buba	F	28 "	Bauchi	Urban	L	1:32
35.	Veronica S.	F	26 "	Bauchi	Urban	H	1:64 6Mths. Preg.
36.	Maimuna Adamu	F	25 "	Bauchi	Urban	L	1:16
37.	Florence O.	F	25 "	Bauchi	Urban	H	1:8 4Mths. Preg.
38.	Ibrahim A.	M	28 "	Dorroo	rural	L	1:128
39.	Asabe Audu	F	25 "	Bauchi-Zungur	rural	L	1:8 6Mths. Preg.
40.	John Sarauta	M	25 "	Tafawa Balewa	rural	H	1:64
41.	Hussaini S.	M	29 "	T/Waja	rural	H	1:256
42.	No'oni M.	F	25 "	Dass	rural	H	1:256
43.	Abel	M	30 "	Misau	Urban	H	Nil
44.	Aliyu Giwa	M	32 "	Bauchi	Urban	H	Nil
45.	Maikano A.	M	40 "	Akko	Urban	L	1:8
46.	Kalbel Durah	M	41 ""	T/Waja	rural	H	1:16
47.	Bamo Shanum	M	40 "	Akko-Pindiga	rural	L	1:8
48.	Danu Abubakar	F	40 "	Bauchi	Urban	L	1:16
49.	Hanatu Ibrahim	F	40 "	T/Waja	rural	H	1:512
50.	Hanatu Jim	F	40 "	Bauchi	Urban	L	1:512
51.	Ali Mohammed	M	40 "	Bauchi	Urban	L	1:32
52.	Ladi K.	F	40 "	Akko	Urban	L	1:64
53.	Maryamu Ali	F	40 "	Gombe	Urban	L	Nil 5mths. Preg.
54.	Adamu S.	M	37 "	Akko-Pindiga	rural	L	1:16

ple No.	Name	Sex	Age	Geographical Location	Type of Community	Socioeconomic group	HAI Titre
5.	Rahab John	F	35 Yrs	Bauchi-Zungur	rural	H	1:16 6mths.Preg.
6.	Lawal Dutse	M	35 "	Akko-Levanti	rural	L	Nil
7.	Tabava Saleh	F	37 "	Gombe-Dadinkowa	rural	L	Nil
8.	Hassan Yahaya	M	34 "	Akko	Urban	H	1:64
9.	Iya Musa	F	30 "	Bauchi	Urban	H	1:4 6mths. Preg.
0.	Isa Ngaji	M	30 "	Bauchi	Urban	H	1:512
1.	Ladi Sulai	F	32 "	Bauchi	Urban	H	Nil
2.	Zabe Wahala	M	30 "	Bauchi	Urban	H	1:16
3.	Grace O.	F	30 "	Bauchi	Urban	H	1:4 7mths. Preg.
4.	Ado Yusuf	M	22 "	Bauchi	Urban	H	1:128
5.	Nuhu Yusuf	M	20 "	Misau	Urban	L	1:16
6.	Abdul G.	M	21 "	Gamawa	rural	L	1:8
7.	Yakubu Adamu	M	20 "	Gamawa	rural	L	1:256
8.	Yusufu Garba	M	20 "	Gamawa	rural	L	1:256
9.	Musa Zakaria	M	20 "	Misau	Urban	L	1:64
0.	Bappa Ali	M	20 "	Gombe	Urban	L	1:4
1.	Saidu Idi	M	21 "	Misau	Urban	L	1:512
2.	Ibrahim B.	M	22 "	Bauchi	Urban	L	1:256
3.	Hajiya Hajara	F	24 "	Bauchi	Urban	L	1:64
4.	Maryamu Aliyu	F	20 "	Bauchi	Urban	L	1:128
5.	Vivian	F	22 "	Bauchi	Urban	L	1:1024
6.	Celestine A.	F	23 "	Bauchi	Urban	L	1:4 5mths. Preg.
7.	Hadiza D.	F	20 "	Bauchi	Urban	L	1:8 7mths. Preg.
8.	Hauwa Aliyu	F	22 "	Bauchi	Urban	L	1:1024 7½mths. Preg.
9.	Hauwa Isa	F	24 "	Bauchi	Urban	H	Nil 6mths. Preg.
70.	Jummai S.	F	20 "	Bauchi	Urban	L	Nil 7mths. Preg.
71.	Suwaibatu A.	F	20 "	Bauchi	Urban	L	1:4 6mths. Preg.
72.	Hadiza Yahaya	F	20 "	Bauchi	Urban	L	1:8 5mths. Preg.
73.	Clement Bala	M	"	Akko	Urban	H	Nil
74.	A. Mahmood	F	23 "	Bauchi	Urban	H	Nil
75.	Mohammed Idris	M	"	Gombe	Urban	L	1:16
76.	Mavu Balarabe	F	"	Bauchi	Urban	L	1:4 7mths. Preg.
77.	Hussina Sambo	F	"	Bauchi	Urban	L	1:64
78.	Hariratu Yusuf	F	22 "	Bauchi	Urban	L	1:4 8mths. Preg.
79.	Rose Agan	F	"	Bauchi	Urban	H	Nil 7mths. Preg.
80.	Marka Musa	F	20 "	Bauchi	Urban	H	Nil 6mths. Preg.
81.	Ladi Sani	F	20 "	Bauchi	Urban	L	1:512 9mths. Preg.
82.	Ladi Danjuma	F	21 "	Bauchi	Urban	L	1:16 5mths. Preg.
83.	Hajara Abdu	F	20 "	Bauchi	Urban	L	1:64 4mths. Preg.
84.	Mary John	F	20 "	Bauchi	Urban	L	1:4 6mths. Preg.
85.	Zainabu Z.	F	21 "	Bauchi	Urban	L	Nil 6mths. Preg.
86.	Zainabu I.	F	20 "	Bauchi	Urban	L	Nil 4mths. Preg.
87.	A'i Maikano	F	20 "	Bauchi	Urban	L	1:32 5mths. Preg.
88.	Rifkatu Nanu	F	22 "	Bauchi	Urban	H	Nil
89.	Hadiza Adamu	F	22 "	Bauchi	Urban	L	1:128 5mths. Preg.
90.	Lydia Sylvestar	F	20 "	Bauchi	Urban	L	1:64 5mths. Preg.
91.	Sawura Yakubu	F	20 "	Bauchi	Urban	L	1:4 6mths. Preg.
92.	Jummai Shehu	F	22 "	Bauchi	Urban	L	1:256 7mths. Preg.
93.	Alhaji Adamu	M	20 "	Azare	Urban	L	1:4
94.	Regina S.	F	20 "	Bauchi	Urban	L	1:64
95.	Jabbo Yuguda	F	21 "	Gombe	Urban	L	1:32
96.	Fatsuma Adamu	F	27 "	Bauchi	Urban	L	1:512 5mths. Preg.
97.	Philips S.	M	21 "	Alkaleri	rural	L	1:16
98.	Mohammed A.	M	21 "	Gamawa	rural	H	Nil
99.	Mohammed G.	M	20 "	Misau	Urban	L	1:256
00.	Mairiga Gidado	M	22 "	Alkaleri	rural	L	1:16
01.	Abdullahi S.	M	22 "	Bauchi	Urban	L	1:32
02.	Garba Kita	M	20 "	Azare	Urban	L	1:64
03.	Bappayo Jaw	M	22 "	Gombe	Urban	L	1:128

ple No	Name	Sex	Age	Geographical Location	Type of Community	Socio-economic group	HAI Titre
	Fatsuma Audu	F	18 Yrs.	Bauchi	Urban	H	Nil
	Ture Dandeba	F	16 "	Bauchi	Urban	L	1:1024 5½m. Preg.
.	Theresa John	F	18 "	Bauchi	Urban	L	1:64 5m. Preg.
.	Salamatu Isa	F	17 "	Bauchi	Urban	H	Nil 5m. Preg.
.	Precillia O.	F	19 "	Bauchi	Urban	H	Nil 5m. Preg.
.	Alti M.	F	17 "	Bauchi	Urban	H	Nil 6m. Preg.
.	Lucy Joseph	F	18 "	Bauchi	Urban	H	Nil 6m. Preg.
.	Adama Manu	F	18 "	Gombe	Urban	H	Nil 6m. Preg.
.	Adama Usman	F	16 "	Gombe	Urban	L	1:32
.	Zainab Sani	F	19 "	Gombe	Urban	H	1:4096 4m. Preg.
.	Hajara Garba	F	18 "	Bauchi	Urban	H	1:64 6m. Preg.
.	Maimuna G.	F	16 "	Bauchi	Urban	H	1:16 6m. Preg.
.	Mamu Usman	F	18 "	Bauchi	Urban	H	1:4 6m. Preg.
.	Lois Samuel	F	21 "	Bauchi	Urban	H	Nil
.	Lydia O.	F	19 "	Bauchi	Urban	H	Nil
.	Maria Ayuba	F	17 "	Bauchi	Urban	H	1:4096 7m. Preg.
.	Ladi Ayuba	F	17 "	Bauchi	Urban	H	1:16 6m. Preg.
.	Fadimatu M.	F	17 "	Bauchi	Urban	H	Nil 6m. Preg.
	A'i Garbo	F	18 "	Bauchi	Urban	H	1:8 5M. Preg.
	Esther William	F	18 "	Bauchi	Urban	H	1:128 6m. Preg.
.	Maria Idi	F	17 "	Bauchi	Urban	H	1:4 5m. Preg.
.	Mairamu Ali	F	19 "	Bauchi	Urban	H	1:8 6m. Preg.
.	Larai Samaila	F	18 "	Bauchi	Urban	L	Nil 6m. Preg.
.	Uwani H.	F	17 "	Bauchi	Urban	L	Nil 6m. Preg.
	Helen M.	F	18 "	Gombe	Urban	H	Nil
.	Aishatu Moh.	F	17 "	Bauchi	Urban	L	Nil
.	Lami Abubakar	F	18 "	Bauchi	Urban	H	1:16 3m. Preg.
.	Hadiza Ahmadu	F	16 "	Bauchi	Urban	H	1:8 4m. Preg.
.	Roseline I.	F	18 "	Bauchi	Urban	H	1:64
.	Uduoka U.	M	19 "	Bauchi	Urban	H	1:16
.	Umaru Usman	M	19 "	Gombe	Urban	H	1:4
.	Lois Usman	F	19 "	Bauchi	Urban	H	Nil
.	Talatu Adamu	F	18 "	Toro	rural	H	1:64
	Mary Afang	F	19	Toro	rural	H	1:256
	Asmau Garba	F	16 "	T/Waja	rural	L	Nil. 4m. Preg.
	Buba Datti	M	19 "	Dukku	rural	H	1:16
	Helen Boya	F	16 "	T/Waja	rural	H	Nil
	Deborah John	F	18 "	T/Waja	rural	H	1:4
	Dogara Samuel	F	19 "	T/Waja	rural	H	Nil
	Emily Danja	F	18 "	T/Waja	rural	H	1:8
	Elizabeth T.	F	19 "	Ningi	Urban	H	1:128
	Deborah Mamman	F	18 "	Zambuk	rural	H	1:16
	Priscilla M.	F	18 "	Akko	Urban	H	1:1024
	di Ankamah	F	19 "	Akko	Urban	H	1:8
	Martina Silas	F	18 "	T/Waja	rural	H	1:8
	Esther Istifanus	F	19 "	T/Waja	rural	H	1:4
	Juliana Haruna	F	18 "	Toro	rural	H	1:16
	Yelwa Alabura	F	17 "	Bauchi- Zungur	rural	L	Nil 7m. Preg.
	Hamsatu I.	F	18 "	Akko	Urban	H	1:8
	Shaibu Adamu	M	18 "	Malam-sidi	rural	H	1:16
	Paulina Iliya	F	18 "	T/Balewa	rural	H	1:8
	Josephine B.	F	18 "	Dass	rural	H	1:8
	Victoria K.	F	19 "	T/Waja	rural	H	1:8
	Godiya M.	F	50 "	Bauchi	Urban	H	Nil
	Grace B.	F	19 "	T/Waja	rural	H	1:4

ple No.	Name	Sex	Age	Geographical Location	Type of Community	Socioeconomic group	HAI Titre	
.	Baba M.	M	18 Yrs.	Dukku	rural	H	Nil	
.	Talatu Sale	F	19 "	Darazo	rural	H	1:16	
.	Aisha Adamu	F	17 "	Darazo	rural	H	1:8	
.	Aisha M.	F	50 "	Bauchi	Urban	L	Nil	
.	Fatsum A.	F	54 "	Bauchi	Urban	L	1:64	
.	Salamatu A.	F	51 "	Bauchi	Urban	L	Nil	
.	Lantana G.	F	55 "	Bauchi	Urban	L	1: 3	
.	Shuaibu A.	M	50 "	Bauchi	Urban	L	Nil	
.	Jibrin A;iyu	M	60 "	Bauchi	Urban	L	1:64	
.	Salamatu A.	F	51 "	Bauchi	Urban	L	1:128	
.	Salamatu H.	F	60 "	Gombe	Urban	L	1:64	
.	Ahmadu Rufai	M	60 "	Gombe	Urban	L	1:64	
.	Innaji M.	F	52 "	Gombe	Urban	H	1:8	
	Aisha Goggo	F	60 "	Gombe	Urban	H	Nil	
.	Fatsuma M.	F	55 "	Gombe	Urban	H	Nil	
.	Ballo Buba	M	52 "	Gombe	Urban	H	1:32	
	Amina Sule	F	60 "	Gombe	Urban	L	1:256	
	Rahim Adamu	M	50 "	Gombe	Urban	L	1:4096	
.	Mohammed M.	M	60 "	Gombe	Urban	L	1:8	
.	Idi Adamu	M	56 "	Toro	rural	L	Nil	
.	Fatsuma G.	F	55 "	Gombe	Urban	L	1:32	
.	Jebu Moh.	F	60 "	Zambuk	rural	L	1:16	
.	Fatsuma Usman	F	60 "	Wuro	rural	L	1:128	
.	Natte M.	F	60 "	T/Waja	rural	L	1:32	
.	Biba Musa	F	52 "	Kashere	rural	L	1:64	
.	Baba Yari	M	50 "	Toro	rural	L	1:32	
.	Aminu Ibrahim	M	20 "	Bauchi	Urban	L	1:128	
.	Abdul Malam	M	31 "	Angwa	rural	L	1:16	
.	Mairam A.	F	30 "	Lawanti	rural	L	1:64	
.	Dinatu Ayuba	F	30 "	T/Balewa	rural	L 1:64	7m. Preg.	
.	Aisha Audu	F	30 "	Bauchi	Urban	L 1:4	6m. Preg.	
.	Sohan B.	F	34 "	Gombe	Urban	H	Nil	
.	Jummai Adamu	F	30 "	Gombe	Urban	L 1:8	5m. Preg.	
.	Sule Audu	M	30 "	Gombe	Urban	L	1:16	
.	Abrahim A.	M	32 "	Gombe	Urban	L	1:64	
	Aishatu Buba	F	35 "	Akko	Urban	L	Nil	
	Moh.	F	36 "	Bauchi	Urban	H 1:32	7m. Preg.	
	Hadi M.	F	35 "	Bauchi	Urban	H 1:32	6m. Preg.	
.	Fatsuma Ballo	F	36 "	Bauchi	Urban	H 1:1024	7m. Preg.	
.	Dudu Adamu	F	35 "	Bauchi	Urban	L	Nil	
.	Godiya I.	F	35 "	Bauchi	Urban	H	1:8	
.	Lawal Adamu	M	35 "	Bauchi	Urban	H	1:32	
.	Magdalene A.	F	35 "	Bauchi	Urban	H Nil	6m. Preg.	
.	Asabe Manga	F	35 "	Bauchi	Urban	H Nil	6m. Preg.	
	Dauda A.	M	35 "	Gombe	Urban	H	1:16	
	Abigail I.	F	38 "	Zungur	Rural	H 1:4096	6m. Preg.	
	Paulina J.	F	25 "	T/Balewa	rural	L 1:4	6m. Preg.	

le No.	Name	Sex	Age	Geographical Location	Type of Community	Socioeconomic group	HAI Titre
	Joseph Bauda	M	29 Yrs.	T/Waja	rural	H	Nil
	Dija Tata	F	25 "	Akko	Urban	L	1:32
	Jedi Haruna	F	25 "	T/Waja	rural	L	1:128
	Hauwa Hassan	F	25 "	Bojude	rural	L	1:4096
	Khlatu Daniel	F	25 "	Toro	rural	L	1:16
	Asabe Sale	F	27 "	Ningi	Urban	L	1:4
	Bulus Bila	M	26 "	T/Waja	rural	L	1:128
	Manama T.	M	27 "	Dass	rural	L	1:32
	Isa Jeremiah	M	25 "	Shira	rural	L	1:32
	Danladi Ali	M	25 "	T/Waja	rural	L	1:128
	Rosaline E.	F	29 "	toro	rural	L	1:16
	Yakubu Shori	M	25 "	Kangere	rural	L	1:64
	Lamba Yusuf	F	28 "	Maijuju	rural	L	1:32 5m. Preg.
	Rifkatu Bulus	F	25 "	T/Balewa	rural	L	1:8 5m. Preg.
	Litinin Y.	F	25 "	Bauchi	Urban	L	1:1024 6m. Preg.
	Fatsuma M.	F	25 "	Bauchi	Urban	L	Nil 5m. Preg.
	Laraba Usman	F	25 "	Bauchi	Urban	L	Nil 6m. Preg.
	Yelwa Ahmadu	F	25 "	Bauchi	Urban	L	1:4 4m. preg.
	Mary Danladi	F	27 "	Bauchi	Urban	H	Nil 7m. Preg.
	Mary Simon	F	26 "	Gombe	Urban	L	1:64
	Mairama Y.	F	25 "	Bauchi	Urban	L	1:4096 5m. Preg.
	Hlim Salihu	F	25 "	Bauchi	Urban	L	1:16 5m. Preg.
	Maimuna Y.	F	25 "	Bauchi	Urban	H	Nil 6m. Preg.
	Esther Jonah	F	25 "	Bauchi	Urban	L	1:4 8m. Preg.
	Hajo Garba	F	28 "	Bauchi	Urban	L	Nil 6m. Preg.
	Lami Saidu	F	25 "	Bauchi	Urban	L	1:8 5m. preg.
	Hajara Garba	F	28 "	Bauchi	Urban	L	1:2048 5m. preg.
	Aishatu A.	F	25 "	Bauchi	Urban	L	Nil 4m. Preg.
	Theresa E.	F	20 "	T/Balewa	rural	H	Nil
	Annah Ishaku	F	20 "	Ningi	Urban	L	1:4096
	Lois Ishaku	F	20 "	T/Waja	rural	L	1:4096
	Aishatu Bello	F	20½ "	Dukku	rural	L	1:8
	Hassana Ibrahim	F	21 "	Disina	rural	L	1:8
	Innuwa Saleh	M	20 "	Dass	rural	L	1:32
	Hauwa Momoh	F	20 "	Dass	rural	L	1:128
	Momoh Doma	M	20 "	Darazo	rural	L	1:32
	Era Momoh	F	22 "	T/Waja	rural	L	1:16
	Tim Andrew	F	22 "	T/Balewa	rural	H	Nil
	Zep R.	M	20 "	T/Waja	rural	L	1:4
	John Garba	M	20 "	T/Balewa	rural	L	1:64
	Abdu Yakubu	M	20 "	Shira	rural	L	1:64
	Zakariah Y.	M	21 "	Ningi	Urban	L	1:16
	Alex Baraya	M	20 "	T/Balewa	rural	L	1:32
	Dahiru Abu	M	21 "	Darazo	rural	L	1:1024
	Bala Mamuda	M	24 "	Ningi	Urban	L	1:32
	Yakubu Sani	M	21 "	Shira	rural	L	1:64
	Ibrahim Sabo	M	22 "	Darazo	rural	L	1:8
	Shuaibu U.	M	23 "	Toro	rural	L	1:32
	Tukur Abdu	M	22 "	Akko	Urban	L	1:64
	Tukur Usman	M	21 "	Dukku	rural	L	1:8
	Abdu Habu	M	21 "	Urban	Urban	L	1:64
	Emmanuel T.	M	22 "	Ningi	Urban	L	1:256
	Wada Moh.	M	20 "	Darazo	rural	L	1:8
	Nuhu Moh.	M	24 "	Disina	rural	L	1:16
	Saraya David	F	20 "	Toro	rural	L	1:4

Sample No.	Name	Sex	Age	Geographical Location	Type of Community	Socioeconomic group	HAI Titre
361.	Yamie Stephen	F	23 Yrs.	Toro	rural	L	1:8
362.	Rebecca Daniel	F	21 "	T/Waja	rural	L	1:32
363.	Rita George	F	21 "	T/Balewa	rural	L	1:16
364.	Mary N.	F	21 "	T/Waja	rural	L	1:8
365.	Monica O.	F	20 "	Ningi	Urban	L	1:64
366.	Norah Bala	F	21 "	T/Waja	rural	L	1:64
367.	Esther Baba	F	20 "	T/Balewa	rural	L	1:64
368.	Mary Gojere	F	22 "	Dukku	rural	H	Nil
369.	Yasti Toro	F	20 "	T/Waja	rural	H	Nil
370.	Monday Umar	M	21 "	T/Waja	rural	L	1:1024
371.	Hajara S.	F	24 "	Toro	rural	L	1:64
372.	Kawu B.	M	20 "	Dukku	rural	L	1:16
373.	Adamu M.	M	22 "	Shira	rural	L	1:32
374.	Rachael Bello	F	22 "	Dass	rural	L	1:512
375.	John Tal	M	21 "	T/Waja	rural	L	1:16
376.	James Johnson	M	21 "	Yankari	rural	H	Nil
377.	Ila Mohammed	M	20 "	Kangere	rural	L	1:64
378.	Hassana John	F	20 "	T/Waja	rural	L	1:4096
379.	Titus Boyi	M	21 "	T/Waja	rural	L	1:1024
380.	Patience B.	M	22 ""	T/Waja	rural	L	Nil
381.	Mahgaii Usman	M	20 "	Kangere	rural	L	1:64
382.	Mohammed H.	M	22 "	T/Balewa	rural	H	Nil
383.	Ayuba Joseph	M	23 "	Toro	rural	L	1:8
384.	Ibrahim A.	M	20 "	Deba	rural	L	1:64
385.	Auwalu Sulei	M	20 "	Misau	Urban	H	Nil
386.	Abdu Adamu	M	20 "	Azare	Urban	L	1:4
387.	Agnes U.	F	25 "	Bauchi	Urban	L	1:64
388.	Aishatu U.	F	27 "	Bauchi	Urban	L	1:32
389.	Buba Umaru	M	25 "	Gombe	Urban	L	1:8
390.	Huwa Musa	F	29 "	Gombe	Urban	L	Nil
391.	Hansatu A.	F	26 "	GOMBE	Urban	L	1:64 4m. Preg.
392.	Jummai Anbi	F	25 "	Bauchi	Urban	L	1:16 5m. Preg.
393.	Rakiya Musa	F	25 "	Bauchi	Urban	H	Nil 6m. Preg.
394.	Dudu Bello	F	27 "	Bauchi	Urban	L	1:32 5m. Preg.
395.	Hadija Ali	F	25 "	Gombe	Urban	L	1:16 4m. Preg.
396.	Hajara Musa	F	26 "	Gombe	Urban	H	Nil
397.	Musa Marbar	M	20 "	Dass	rural	H	Nil
398.	Buba Kari	M	30 "	Bajoga	rural	L	1:8
399.	Bitrus D.	M	35 "	T/Balewa	rural	L	1:512
400.	Asabe Jahi	F	30 "	Bauchi	Urban	L	Nil 7m. Preg.
401.	Lubo N.	M	30 "	Yamaltu	rural	L	1:64
402.	Lami, Magaji	F	30 "	Gombe	Urban	H	Nil 4m. Preg.
403.	Ardo Yahaya	M	40 "	Gombe	Urban	L	1:512
404.	Yahaya Nuhu	M	21 "	Gombe	Urban	L	1:256
405.	Pala A.	M	23 "	Gombe	Urban	L	1:64
406.	Umar Simon	M	22 "	Gombe	Urban	L	1:64
407.	Yelwa Ahmed	F	20 "	Gombe	Urban	L	1:512
408.	Hassan Yelwa	M	20 "	Azare	Urban	H	Nil
409.	Sale A.	M	17 "	Akko	Urban	H	1:128
410.	Hauwa Yakubu	F	18 "	Bauchi	Urban	H	1:64 6m. Preg.
411.	Hauwa C.	F	18 "	Darazo	rural	H	1:4096
412.	Omolala O.	F	23 "	Bauchi	Urban	H	Nil
413.	James Usman	M	23 "	Bauchi	Urban	L	1:64
414.	Umaru Audu	M	20 "	Gombe	Urban	L	1:32
415.	Haruna Moh.	M	20 "	Gombe	Urban	L	1:4

No.	Name	Sex	Age	Geographical Location	Type of Community	Socioeconomic Group	HAI Titre
	Raihanatu M.	F	21 Yrs.	Bauchi	Urban	H	Nil 3m. Preg.
	Olu O.	M	48 "	Gombe	Urban	H	1:4
	Salamatu M.	F	47 "	Bauchi	Urban	L	1:1024
	Musa Idrisu	M	48 "	Bauchi	Urban	L	1:64
	Ayuba Garba	M	45 "	Bauchi	Urban	H	Nil
	Markus S.	M	49 "	Bauchi	Urban	H	1:16
	Hajaumi Moh.	F	47 "	Toro	rural	L	Nil
	Afiniki Umaru	F	46 "	Gombe	Urban	H	1:4096
	Franklin Kire	M	46½ "	T/Balewa	rural	H	1:128
	Hamsatu Garba	F	48 "	Dass	rural	L	1:4
	Mohammed D.	M	47 "	T/Waja	rural	L	Nil
	Sa'adatu U.	F	46 "	Misau	urban	L	1:512
	Sigari	M	48½ "	Gombe	Urban	H	1:4
	Musa Ahmed	M	45 "	Bauchi	Urban	H	1:4
	Nuhu Yusuf	M	45 "	Toro	rural	L	1:16
	Larai Uma	F	47 "	Darazo	rural	H	1:4096
	Yumana Idris	M	20 "	T/Waja	rural	L	1:64
	Umar Musa	M	49 "	T/Balewa	rural	L	1:256
	Nuhu Musa	M	49 "	Gombe	Urban	L	1:32
	Yakubu Wakili	M	46 "	Bauchi	Urban	L	1:64
	Anna Abdu	F	48 "	Misau	Urban	H	Nil
	Turba Gambo	M	47 "	Ningi	Urban	L	1:128
	Suze Ahmadu	F	45 "	Azare	Urban	H	Nil
	Adama L.	F	45½ "	Azare	Urban	H	1:64

Socioeconomic Status

H → High

L → low

Reported cases of Measles deaths due to measles and immunization given each year in Bauchi state, Nigeria (1977 - 1984).

Year	Vaccinations given	Measles cases	Estimated death
1977	60,898	6,960	120
1978	80,421	1,493	50
1979	60,095	854	40
1980	135,856	661	345
1981	169,280	3,075	233
1982	77,010	8,703	739
1983	51,167	10,649	289
1984	40,448	*	150
Total	675,169	32,395	1,966

* Data not available

(see Fig 1.p9 75)

Appendix 6

MONTHLY MEASLES REPORTS IN BAUCHI STATE, NIGERIA (JAN-SEPT. 1983

Year	Months												Total
	Jan.	Feb.	Mr.	Apr.	May.	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	
1981	89	280	421	393	573	173	246	259	164	109	128	241	3,075
1982	404	474	950	961	1,086	807	433	515	1,157	708	948	725	8,703
1983	769	2944	609	787	2,250	608	782	982	918	*	*	*	10,699
Total	1,262	3,698	1,980	1,676	3,908	1,588	1,461	1,756	2,239	817	1,076	966	22,427

* Data not available

(See fig.2. Pg 76)

Appendix 7

The distribution of notified measles cases by age and year in Bauchi State (1981-1983)

year	Age group 0-1	1-4	5-44	15	Total
1981	71(0.023)	1,362(0.443)	1,609(0.523)	33(0.011)	3,075
1982	1,494(0.172)	4,41(0.507)	2,651(0.305)	142(0.016)	8,703
1983	2,567(0.241)	7,443(o.699)	639(0.06)	0(0)	10,649
Total	4,132(0.436)	13,221(1.649)	4,899(0.888)	175(0.027)	22,427

( ) proportion of cases notified.

(See fig.3 pg 78)