MEASLES IN SOUTH AFRICA: A COMMUNITY HEALTH INTERPRETATION OF THE DATA

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ABSTRACT

This dissertation reviews the international literature and South African literature and data on the epidemiology of measles. The main purpose is to contribute to an understanding of the local epidemiology of the disease that will further the development of more targeted efforts for its control.

Published literature on measles from 1960-1989 listed in the Index Medicus was initially selected for review. Further references were obtained from the bibliographies of the published literature initially selected and from informal correspondence with people working in this field.

In the case of South African literature, the review concentrated on studies of data quality and disease incidence, prevalence and mortality and their possible determinants. Notification (1980-1989), death certification (1970-1986), active surveillance and hospital data were explored.

Much of the existing data had not been previously subjected to statistical analysis. Whenever appropriate, but particularly for determining trends over time, these data were converted from absolute numbers to rates and the rates were submitted to further statistical analysis. Simple linear regression was applied to the data in order to identify statistically significant changes in trends. The analysis was stratified by age and population groups.

Up to the late 1980s the incidence of measles was still high in South Africa. Although incidence over time showed a downward trend this was not statistically significant. This indicated that, at least for the past decade, control efforts have failed to reduce morbidity due to measles. Peak incidence was in the 5-9 year age group.

Notwithstanding data limitations, it is apparent that there exist defined areas of high measles morbidity, such as the Transkei, Lebowa, Kwazulu and the Eastern Cape, with seasonal patterns varying from region to region.

Before 1979-1980 measles mortality rate dropped for Asians and Coloureds. Since 1979 the rate has decreased significantly only for Coloureds. This probably reflects the already low levels of reported mortality for Asians and Whites and successful measles mortality control efforts amongst Coloureds during the last decade.

It is not possible to comment on the mortality trends for Blacks. Although the trend is an upward one it has not reached statistical significance. This could reflect better case ascertainment, better reporting, or a truly high level of measles mortality, that is still on the increase. It probably reflects all three.

In analysing the mortality trend per age group, different patterns were found for each population group. It was

increasing for Coloured infants, for 1-4 years old Asian children and for 5-9 years old Black children. It decreased for 1-4 years old Coloured children and for Whites over the age of 20 years.

The CFR has been high and rising, although this was not statistically significant. The highest CFRs and age specific mortality rates occurred in infants.

This review and analysis of the South African data allows one to advance recommendations in three areas: measures to improve data; measures to reduce the incidence of measles; and measures to reduce complications and mortality.

The dissertation ends with a word of caution and a call for action. Measles control requires commitment at the highest levels and ongoing consistent efforts. Secondly, even if peri-urban areas appear to be at high risk for severe measles, still over half of all the measles cases are reported from the homelands, mostly rural areas, where cases, as in the urban slums, are grossly under-notified. The data from this report suggest that resources should be directed at areas such as the Eastern Cape, Kwazulu, Lebowa and Ciskei, as well as at the urban poor.

DECLARATION

I declare that this dissertation is my own, unaided work. It is being submitted for a Master of Medicine (Community Health) at the University of the Witwatersrand. It has not been submitted for any other degree or examination to the aforesaid, or any other University. The work involved in this dissertation did not, in any way, involve the interviewing of or experimentation with human or animal subjects.

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ABBREVIATIONS

BOP	Bophuthatswana
CFR	case fatality rate
DWT	diphtheria, whooping cough and tetanus.
EC	Eastern Cape
et al	and others
etc	etcetera
fig	figure
GAZ	Gazankulu
ICU	intensive care unit
LEB	Lebowa
NT	Northern Transvaal
OFS	Orange Free State
PHC	primary health care
SSPE	subacute sclerosing panencephalitis
TBVC	Transkei, Bophuthatswana, Venda, Ciskei
VAC	vaccination
vs	versus
WHO	World Health Organization
Y	years
Yrs	years

Abbreviations used in the Bibliography section are not included in this list.

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1 INTRODUCTION

Measles is an airborne infection caused by a RNA virus of the family Paramyxoviridae, genus Morbillivirus. Only one antigenic type is known. Susceptibles of all ages are affected and are left with lifelong immunity after infection[1]. Measles is rarely sub-clinical. It is still a significant cause of morbidity and mortality amongst young children in the developing world, including South Africa[2,3].

The fact that only one antigenic type of measles is known has facilitated the development of live vaccines with a high efficacy, if proper attention is paid to cold-chain maintenance. The current vaccine strain for mass immunization is the Schwartz strain*. Infants acquire immunity transplacentally from mothers who have had measles or measles immunization. This immunity is usually complete for the first four to six months of life and wanes thereafter at a variable rate. Although maternal antibody levels in infancy are generally undetectable after nine months of age, some protection persists which may interfere with immunization administered prior to 15 months of age. In developing countries, where a significant number of cases occur under the age of one

*In the recent past there has been some enthusiasm for the high titre Edmonston-Zagreb vaccine against measles. Since this dissertation was submitted the high titre vaccine has been discontinued because of probable vaccine-induced mortality.

year, it is usually recommended that measles vaccine be given as a single dose at nine months of age[1].

The many similarities in the biologic features of measles and smallpox suggest that measles eradication is a possibility. These features are: a distinctive rash; no animal reservoir; no vector; seasonal occurrence with disease free periods; no transmittable latent virus: one serotype; and one effective vaccine[1]. Mankind will probably succeed in controlling and eradicating this killer of young children[4,5,6]. This could happen sooner rather than later if more resources were allocated to the task and there was a greater commitment towards gaining a clearer understanding of the behaviour of the virus in human communities. Such understanding would enable control programmes to set targets towards which to direct their efforts.

This dissertation analyses and interprets the local data on measles, against the background of international knowledge of its epidemiology, with the aim of developing recommendations for locally targeted efforts for measles control and its possible eradication.

Determinants of acute and delayed morbidity and mortality from measles have been identified, including:

a Health policy factors, such as the practice of

vaccination[7,8] and admission and treatment policies;

b Personal characteristics of the cases, such as age[9,10,11,12] and sex[13];

c Geographical and environmental determinants, such as latitude[14] and season[14], whether or not it is the index case in the household[15-18], the nature of the human settlement[19], the type of family (nuclear or extended, monogamous or polygamous)[15,17,20], population mobility[11] and the degree of household overcrowding[11,16,18,21-25];

d Nutritional status, including the degree of protein energy malnutrition[9,10] and vitamin A deficiency[26,27];

e Traditional customs, such as perceptions of the disease as a normal phase in the development of a child[28,29]; and

f Genetic background, although it is not particularly significant, there is an association of some histocompatibility leucocyte antigens with severe measles[30].

The basic epidemiological patterns of measles are outlined below, with a description of the key determinants of its morbidity and mortality and of its complications.

1.1 EPIDEMIOLOGICAL PATTERNS

The epidemiological patterns associated with measles have

been classified by the level of socioeconomic development, type of human settlement and degree of population mobility[14,19]. These classifications are not useful as the determinants of the incidence and severity of disease are common to all epidemiological patterns. Also, the difference between urban and rural settlements is becoming more and more blurred with the development of peri-urban sprawls, where the rural becomes more urban, and the urban more rural. It seems more useful, therefore, to classify the behaviour of the virus broadly as creating demic or epidemic patterns. There are variations within the patterns, as well as overlaps between them.

Endemic measles is characteristic of large settlements with a high population density, overcrowding and an ongoing increase in the pool of new susceptibles. The latter is a result of high fertility rates or of the immigration of susceptibles, particularly young children. In this pattern measles transmission is perennial. The age of infection tends to be low and the case fatality rate high[14,19].

Epidemic measles is characteristic of small settlements with a low housing density. Measles transmission is characterized by regular outbreaks at variable intervals. Sporadic massive outbreaks occur in isolated communities, affecting all susceptibles regardless of age[14,19].

1.2 KEY DETERMINANTS OF MEASLES INCIDENCE AND MORTALITY

The key determinants of measles incidence and mortality namely, vaccination, age, overcrowding, nutrition and geography, are reviewed in this section.

1.2.1 THE IMPACT OF VACCINATION ON THE EPIDEMIOLOGICAL PATTERN

In both epidemic and endemic epidemiological patterns, vaccination results in a lower incidence of measles, a lower case fatality rate (CFR), a higher mean age at infection and a widened inter-epidemic interval[31-44].

In the endemic pattern, vaccination can change the situation from one of perennial measles to that of small, regular or isolated outbreaks amongst segments of the community that are difficult to reach. Thus, even where there is high vaccination coverage (>90%), frequently islands of poorly vaccinated individuals remain. This usually occurs in areas of low education and poor economic status. Religious practice forbidding vaccination can also lead to clusters of susceptible individuals prone to sporadic outbreaks of measles[41].

1.2.2 MEASLES AND AGE, OVERCROWDING AND NUTRITION: THE ROLE OF INFECTIVE DOSE

Recent developments in the understanding of measles suggest that, although vitamin A deficiency, genetic background and vaccination policies have an independent impact on measles severity and outcome, other factors act through the common pathway of a higher infective dose of measles virus[11,22-25].

The importance of this new understanding is that strategies to correct the impact of measles in a community should not address confounders of the true pathway, but should rather take measures to overcome exposure to a heavy dose of infection. This could be achieved by: improved vaccination coverage; reduction of overcrowding through the increased provision of basic housing; and effective fertility control[22]. The changes in the epidemiology of measles that occurred in developed countries before the advent of vaccination are consistent with this understanding[16,18,20]. It also suggests that measles-specific policies would be more effective, in reducing childhood mortality in underdeveloped communities than is usually acknowledged in the literature[36,37,45-47]. Until recently it has been argued that the isolated prevention of measles would only result in deaths from other childhood illnesses

associated with malnutrition or from other diseases[36,47].

This new understanding suggests that measles mortality is relatively independent of protein-energy malnutrition. If correct, it could mean that measles vaccination would, in fact, reduce childhood mortality independent of efforts to reduce protein-energy malnutrition. Some support for this was obtained from a case-control study in rural Bangladesh. Even though the study had a number of methodological problems that do limit its generalisability they do not invalidate the conclusion. This has suggested an association between measles vaccination and a reduced rate of measles and overall mortality[48]. In line with this, community studies from Guinea-Bissau*, Senegal and Haiti have reported higher survival rates among children vaccinated against measles compared with other children in the community[31].

* Also in Guinea-Bissau it was observed that, long after a severe measles epidemic, deaths seemed to cluster in the same houses in which measles had occurred. Children exposed to measles during the first 6 months of life experienced excess mortality, even if they did not develop clinical measles. These observations suggest that vaccinations against measles may reduce mortality by more than the share of deaths usually attributed to measles (Aaby P, Bukh J, Kronborg D, Lisse IM, Da Silva MC. American Journal of Epidemiology 1990; 132: 211-219).

1.2.3 GEOGRAPHICAL VARIATIONS

In both endemic and epidemic measles, there are seasonal variations in the incidence of measles. In Africa, it appears that incidence rates decrease as one moves away from the equator, while at the same time the difference in incidence by month increases[14]. This is related to: changes in temperature and humidity at various times of the year; to cultural and social practices; population movement; and possibly variations in patterns of human settlement, particularly in so far as they relate to population density and overcrowding[11,14,19]. Different combinations of these factors probably explain some of the differences in the incidence of measles across the continent. For example, in West and Central Africa measles tends to peak from November to January and has its lowest incidence in May; whereas the peak in East Africa is in April and the trough in December[49,50]. Throughout Africa the peak incidence of measles seems to occur in both dry and wet seasons[11,51].

1.3 COMPLICATIONS OF MEASLES

Measles morbidity and mortality is aggravated during and after the acute phase of the illness by complications such as respiratory infections[4], diarrhoea[52], blindness[53,54],

chronic lung problems[55], neurological problems[4,56-60], tuberculosis[4], myocarditis[4] and diffuse intra-vascular coagulation[61,62].

The Bangladesh study referred to earlier suggests a reduction in mortality due to measles, diarrhoea, respiratory illness and malnutrition that is sustained for at least two years after measles vaccination[48]. There is also evidence that clinical or subclinical measles may be related to diseases which present many years later, including multiple sclerosis, systemic lupus erythematosus, Paget's disease, a number of sebaceous skin diseases and certain tumours[63].

1.4 CONCLUSION

Control of measles will have a significant impact on morbidity and mortality and on the incidence of the long-term complications associated with the disease. Measles vaccination, a key determinant in control, not only prevents measles mortality, but may lead to a decrease in overall mortality as well. The impact of vaccination is enhanced if overcrowding and other factors that affect the occurrence and/or severity of measles are also addressed.

There has been no recent comprehensive review of the epidemiology of measles in South Africa. This

dissertation reviews and explores local measles data and examines the local epidemiology against the explanations of severity presented in section 1.2 of this first chapter. The dissertation also identifies factors and trends which will enhance public health practice by facilitating locally targeted interventions.

After describing the methodological approach (Chapter Two), South African data on measles and measles related morbidity and mortality are analyzed (Chapters Three, Four and Five). Arising from the main conclusions (Chapter Six) recommendations for locally targeted interventions are presented (Chapter Seven).

2 METHODS

This chapter outlines the criteria for literature selection, analyses literature quality and explains how problems related to poor quality were addressed. The rationale for further statistical analysis of the morbidity and mortality data from notification reports and death certifications is also explained.

2.1 SOURCES OF DATA

Published literature on measles from 1960-1989 listed in the Index Medicus was initially selected for review. Further references were obtained from the bibliographies of the published literature initially selected and from informal correspondence with people working in this field. For the international literature, the focus was on theoretical and empirical explanations of determinants of the incidence of measles and of mortality due to measles. In the case of South African literature, the review concentrated on studies of data quality and disease incidence, prevalence and mortality and their possible determinants. Notification, death certification, active surveillance and hospital data were explored.

2.2 QUALITY OF THE DATA

2.2.1 NOTIFICATIONS

Measles has been a notifiable disease in South Africa since August 1979[65]. Measles and deaths from measles are notifiable as separate occurrences. Notifications emanate from many sources. Although a standardised process of collection and collation is followed[66] there are biases associated with it.

The major bias is under-notification of cases[67], resulting from a lack of procedural awareness amongst notifying officers and from differences in disease severity and access to health care for different age and population groups. Several reports have addressed the problem of under-notification.

In an epidemic in Lindley, Orange Free State, only 60% of the children with measles were notified[68]. Most were white* children. In a similar report from

*Frequently, reference is made to Asians, Blacks, Coloureds and Whites. The use of these expressions is adopted for this dissertation since vital statistics and the social, economic and political institutions in South Africa have been structured along these legally defined "racial" categories[1,2,3]. There is a significant body of literature on the lack of scientific justification for the use of racial expressions. However, in South Africa, racial classification has been one of the determinants of political power, social class, economic experience, environmental exposure and access to health care. As such, it is justifiable to look at measles in the different "races" legally defined in South Africa[1,2,3]. The use of these racial expressions does not imply that their legitimacy is accepted.

Jacobsdal, also in the Orange Free State, 24% of measles cases were not notified[69].

Kettles reports that 60% of 59 general practitioners did not know that measles was notifiable[70]. He goes on to report that, under optimal conditions, only 28% of the known cases of measles in a Stellenbosch outbreak were notified[70].

It is estimated that less than 20% of all measles cases in Cape Town are notified. Approximately 50% of all its notifications come from the City Hospital which only admits children with acute complicated measles[74,75].

In Alexandra, Transvaal, it became apparent during a recent measles epidemic that many clinicians did not know that measles was a notifiable disease[76].

From January to July 1987, 85% of children admitted to Baragwanath Hospital, in Soweto, with measles were not notified[77].

A survey of outpatient doctors in Durban confirmed that few of them notified measles[78].

It seems, therefore, that the process of notification of measles is hampered by the failure

of notifying officers, i.e. doctors and nurses, to fulfil their statutory duty. It is clear from the literature that ignorance of their obligations is a factor; but possibly other reasons such as laziness, lack of concern and failure to understand the importance of notifications, also play a part.

As notification is done by health personnel, it is likely to be biased against communities with poor access to health care. This is exemplified by the observation that, in the Cape, 50% of notifications originated from the single hospital for infectious diseases[74,75], while in Natal-Kwazulu the Clairwood Infectious Diseases Hospital accounted for 40% of all notifications[78]. In Johannesburg, 59% of notifications originated from hospitals, 13% from general practitioners and 3% from local authorities[79]. This notification bias is likely to lead to underestimates of disease in rural communities and the urban poor. Some of the measles cases from these areas may be reflected in the health service statistics of better served contiguous areas.

It seems that nurses have usually, but not always, been less likely than doctors to notify the disease, possibly because of a lack of awareness of their statutory duty to do so. A report from Gazankulu

stated that "measles cases that were treated at clinics and health centres* were not notified. We have now designed forms for clinics and health centres to notify measles", suggesting that absence of notification forms was also a factor in the lack of notifications[80].

Notification is also more likely to occur in more serious cases, i.e. those in need of hospital care. As these are usually young infants, underestimates would have been greater in older children.

The data on notification of deaths are suspect for the same reasons as notification of disease. Wittenberg reported that, in two academic hospitals in Natal-Kwazulu, only 83 (23%) of 356 deaths due to measles were notified[78].

2.2.2 DEATH CERTIFICATION

Death certification in South Africa, as distinct from death notification, has also displayed many problems, especially for Blacks[81,82,83]. Again, problems have been due to different rates of accessibility to, and the inadequate skills of, certifying officers (health workers and police officers). This

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*Entirely staffed by nurses.

has lead to under-reporting and misclassification of causes of death.

There were no changes in the classification of measles in the International Disease Classification, so this could not have affected mortality statistics.

A serious problem in interpreting death certificate derived mortality statistics is the under-reporting of an estimated 50% of deaths amongst Blacks. This has been a more serious problem in rural areas[82,83].

Notified measles deaths in South Africa have represented between 3% (1980) and 44% (1983) of measles certified deaths registered with the central statistical services in Pretoria[69] (Fig. 1), suggesting that certified deaths provide a more complete data set than notified deaths.

2.2.3 ACTIVE SURVEILLANCE DATA

There is only one report of active surveillance of measles in South Africa. The study was based on active follow up of notified cases of measles and their contacts in Johannesburg and Benoni[19]. It provides one of the most detailed data sets on the disease in South Africa. Unfortunately, the process

of data validation, was not described.

2.2.4 HOSPITAL DATA

Data on hospital inpatients and outpatients with measles, or dying from it, have never been rigorously assessed and reported. What is available is limited and its quality is difficult to assess. The reports reviewed here have been found in the correspondence columns of the South African Medical Journal.

Important biases of these hospital data sets were the young age profile, severe disease, high mortality and better access to health care of those admitted.

2.2.5 MULTIPLE DATA SOURCES

One report, based on a mix of methodologies, is also reviewed. Loening and Coovadia reported on surveys of communities and health centres in the Natal-Kwazulu area, postulating a relationship between urbanisation and measles[84]. Although debate on the limitations of their methods appears in the literature[85,86], the case for this relationship remains strong.

2.3 STATISTICAL ANALYSIS

Much of the existing data had not been previously subjected to statistical analysis. Whenever appropriate, but particularly for determining trends over time, these data were converted from absolute numbers to rates and the rates were submitted to further statistical analysis.

The raw measles notification and death certification data used to calculate the rates and trends are tabulated in Table I. This dissertation focuses on data from 1980 to 1989 for notifications and from 1970 to 1986 for death certifications (certification data were only available until 1986)[*].

Notification rates per 100 000 population were calculated using population figures extrapolated from the 1985 census[57] and notification figures reported by the Directorate of Epidemiology of the Department of National Health and Population Development[88].

The denominators used to calculate the rates had limitations. Firstly, there was under-counting, particularly of the Black population group, although this

[*]Minimal information is provided for death certification of measles after 1986 as the data were not available when the analysis for this dissertation was undertaken. Civil unrest and major shifts in the demography and economy have occurred since, which probably has an appreciable impact on measles epidemiology in South Africa. As more recent data become available, the possibility of updating the trends reported upon will be considered.

had, to some extent, been corrected in the population figures used for this dissertation[87]. Secondly, with the granting of "independence" to the TBVC countries (Transkei, Bophuthatswana, Venda, Ciskei), a significant proportion of cases and of individuals at high risk for measles were removed from the numerator and the denominator, leading to reduced national rates. Thirdly, some areas of South Africa were home to refugees from local or foreign civil wars. These refugees were not counted in the denominator, yet they were a high risk group who were likely to be represented in the numerator.

One of the simplest models for data on two variables is a straight line. The technique for fitting straight lines to data and checking how well the lines describe the data is called simple linear regression*. One of the variables (X) is considered the predictor of the other variable (Y), the outcome variable.

The linear regression model assumes that the relationship between X and Y can be summarised as a straight line graph and represented mathematically by two numbers, the intercept (where the line crosses the Y axis) and the slope, as represented by the following equation:

predicted outcome (Y) = intercept + slope x predictor value(X).

*Data were analyzed using simple linear regression methods on BMDP software (Dixon WY. BMDP Statistical Software. Berkeley, California; University of California Press, 1981).

Simple linear regression was applied to the data in order to identify statistically significant changes in trends, as described by the slope of the regression equation. If the slope was significantly different from a horizontal line ($p<0,05$), the regression equation and the degree of correlation (r) between the variables X and Y were reported upon (r also describes the goodness of the fit of the linear model to the data). When trends were significantly different from a horizontal line for data sets of more than one population group, they were compared by multiple linear regression.

Death certification data were similarly analyzed for the time periods up to 1979 and thereafter (1980-1986) or alternatively, up to 1978 and thereafter (1979-1986), according to a piecewise linear model. The reasons for the 1978 cut off point included the fact that mortality data for Blacks were not available before 1979 and the perception, after looking at the data, that major gains in reducing mortality took place until 1978, but not thereafter and therefore, that part of the data seemed to lie along one line, and part along another line. The main reason for the 1979 cut off point was the desire to assess the gains for the 1980 decade, as opposed to preceding years.

The analysis was stratified by population group. The rates for different population groups were compared with

one another using the same regression methods described above For each population group, the data for death certificates were grouped into five age groups: 0-<1 year; 1-<5 years: 5-<10 years; 10-<20 years and 20 years and over. These groups were chosen because they represent risk clusters and they compensate for the small numbers after 10 years of age. The proportion of the total represented by each age group was analyzed. Trends for these proportions were then plotted and analyzed, again using linear and multiple linear regression methods.

2.4 LIMITATIONS

It was apparent that the study of the epidemiology of measles in South Africa would have to be based on incomplete data sets (notified data and certified mortality), or data sets of dubious quality (hospital reports). Changes in trends may reflect changes in the factors determining under-reporting rather than in the incidence of the morbidity or mortality of measles. Still, the qualitative and quantitative nature of some of the biases were known, and could be and were taken into account when analysing, speculating, concluding or making recommendations. Despite the limitations of the data described above, they still have the potential to display trends and patterns that could be useful for local targeting of control interventions.

As the quality of the reports reviewed was extremely variable, more attention was paid to studies that took the following steps to address possible biases: identifying the extent of under-reporting[70]; ensuring a study design that strives to minimize the extent of under-reporting, e.g. using a prospective study design[79]; paying particular attention to studies carried out in areas where the surveillance systems were known to be functioning well[100,102]; or stratifying the data for analysis according to some of the factors responsible for under-reporting (age, accessibility to health care facilities, urban/rural residence and population group). For one's own analysis it was decided to stratify the data per age and population groups.

3 MEASLES IN SOUTH AFRICA: INCIDENCE, MORTALITY AND RELATIONSHIPS TO AGE, POPULATION GROUP, SEX AND GEOGRAPHICAL DISTRIBUTION

In South Africa, measles has a long recorded history of devastating effects, with reports of outbreaks dating back to 1807[89], 1852 and 1899-1901[90]. The severity of the disease amongst Black rural children has long been acknowledged[91]. In 1960, over 1 000 Black miners required hospital admission for measles[92]. Since the 1970s, there has been an increasing interest in the disease, culminating recently in a number of publications[1-3,7,93-95]. The reasons for this interest are related to the perception that measles is still a common cause of morbidity and mortality, new developments in vaccine technology[96] and several high level official statements about measles control in South Africa[64].

In this chapter, patterns of the occurrence of the disease in South Africa are described, and morbidity and mortality data in relation to age, sex, population group and geographical distribution are studied, in order to evaluate the extent to which these patterns are compatible with the current understanding of measles, as described in Chapter 1.

3.1 ENDEMICITY AND SEASONAL DISTRIBUTION

In South Africa, measles has been a perennial disease with national peaks of notification in late winter and early spring. Notification rates have been highest in the dry season, i.e. in September, followed by October and then August[88]. The lowest rates have occurred in February, followed by January and December[88].

The local pattern reported for Soweto coincided with the national one, although the lowest rates occurred in March[97]. However, in other areas of South Africa the local seasonal variation has differed from the national picture.

In the peri-urban areas of the Western Cape the pattern has varied from winter peaks (June, July and August respectively for 1981, 1982 and 1983) to a spring peak (November 1984), summer peaks (January 1985 to 1987), an autumn peak (May 1988) and, again, a winter peak (August 1989) (PM Barron, personal communication, September 1990). Although the reasons for these shifting trends are not clear, they probably reflect major population movements into the Western Cape[98] in the summers of 1984 to 1987, following the relaxation of influx control legislation, with the population again stabilising towards the end of the decade. More recently, the seasonal peaks have occurred against a constant

background of five or more measles notifications per week (PM Barron, personal communication, August 1990).

Data for Durban for 1985 reflected a winter peak (June), with the lowest incidence in summer (January)[99].

In Alexandra, an urban slum near Johannesburg, there have been no notified cases outside of the yearly outbreaks that start in August, peak in September, and drop slowly, with the last cases occurring in January (unpublished observations).

In Gelukspan, a rural area in central Bophuthatswana with high vaccination coverage, outbreaks have recently occurred in typical two to three year cycles, affecting mostly school-going children[100].

In the Glen Grey Hospital of Transkei, data from July 1985 to June 1986 show measles peaking in July, followed in prevalence by May and June, with the lowest rates occurring in November[101].

In Letaba Hospital, Gazankulu, measles admissions during 1985 peaked in August with prevalence next highest in June and July[102].

The national seasonality clearly hides regional patterns, the reasons for which are not always apparent. It is

possible that some of this seasonal variation could be explained by climate and population mobility. What is apparent is that the patterns described fit with the notion of measles being endemic in South Africa, with levels of endemicity and epidemiological patterns varying from region to region. The seasonal distribution of cases seems more similar to the West and Central African pattern than to that of East Africa[49,50].

3.2 MEASLES INCIDENCE IN SOUTH AFRICA

The review of measles incidence is based on data from notifications, active surveillance systems and hospital reports.

3.2.1 NOTIFIED INCIDENCE

Notification data, after taking account of the biases (see pages 12-15), provide valuable insights into the epidemiology of measles.

After measles notification was introduced[65], the absolute number of notifications remained low for 1979 because of low reporting, but increased sharply in 1980 and has remained high (Table I). It is the second most commonly notified disease in South Africa, and is the most common for children under five years of age[88].

Between 1980 and 1989, the number of notifications varied from 300 to 2 000 per month (excluding the TBVC countries), averaging 14 292 per year and 1 190 per month[88,103,104]. For the period 1980 to 1989, the incidence of notifications per 100 000 population varied between 78,9 (1987) and 27,4 (1989) (Fig. 2). The notified incidence has shown an overall downward trend, but this does not reach statistical significance. The average annual incidence rate for the past decade (1980 to 1989) is 52,5 per 100 000 population per annum.

3.2.1.1 RACIAL DIFFERENTIALS

The rates for the notification of measles show clear differences according to race, the highest rates being for Blacks, followed by Coloureds[88]. Most of the notified cases have been for Blacks.

3.2.1.2 GEOGRAPHICAL DIFFERENTIALS

The seven health regions of the Republic of South Africa (i.e. excluding the homelands) have averaged 2,4 (excluding tuberculosis) or 3,1 times (including tuberculosis) more notifications each year of the diseases targeted by the Expanded Programme on Immunization than all ten homelands (i.e. including TBVC countries) combined. In view of their roughly

equal total populations, Ijsselmuiden and Gear concluded that there was a higher notified incidence in the Republic of South Africa, and under-notification in the homelands[105]. This profile has not been true for measles. The 1985 census found that 43% of the South African population were based in the homelands while 60%[88], 77%[103] and 72%[106] of measles notifications in 1986, 1989 and 1990 respectively came from these areas.

Data from the National Directorate for Epidemiology suggest that, in relation to their share of the population, there has been a consistent excess of notifications from the Eastern Cape, Kwazulu and Lebowa. There is no reason to believe that case ascertainment and notification have been better in these areas than in the rest of the country, suggesting that these excesses in notification reflect higher prevalence rates of measles.

Data from Lebowa[107], Kangwane, Bophuthatswana, Kwandebele, Gazankulu, Northern Transvaal, the Orange Free State and the Eastern Cape are presented in Tables II and III[40]. It is again apparent that Blacks in the Eastern Cape and Lebowa stand out as the groups at greater risk of measles.

The proportional population distribution (1970) of

the four independent homelands, Bophuthatswana, Ciskei, Transkei and Venda, was 26%, 15%, 51% and 8% respectively, while their share of measles notifications was 0,4%, 1,5%, 79,1% and 18,9% in 1989[103], and 0,1%, 2,9%, 95,2% and 1,[illegible] in 1990[106]; showing a clear excess of cases for Transkei (1989 and 1990) and Venda (1989). Personal knowledge of some of the areas, and contact with professionals working in the others, allows speculation that notification in Venda and the Transkei has not been better than in the other homelands. To what extent the figures reflect true geographical differentials, rather than different phases in the epidemic cycle, is not clear. However, the consistently low notification rate for Bophuthatswana is in keeping with the high vaccination uptake reported for different regions of the homeland[100,108-117].

For the recent past, the Ciskei has reported significant success in promoting universal vaccination to reduce measles morbidity[118]. This has been associated with a concurrent reduction in notifications. The extent to which this is a cyclical variation in the epidemic cycle or a real success in controlling the illness is not yet clear.

Data for the Transkei (1987), presented in Table IV[119], reflect the magnitude of the problem in a

region where vaccination services have reportedly not been functioning well[120].

3.2.2 ACTIVE SURVEILLANCE DATA

The findings of an active surveillance system in Benoni and Johannesburg revealed a rate of measles notification in Benoni fifteen times that of Johannesburg[79]. The reason for this difference was not entirely clear, but it could be related to the fact that Benoni's data included surveillance of squatter populations.

3.2.3 HOSPITAL DATA

Hospital reports on the number of measles admissions are difficult to evaluate because of a lack of definition of denominators, and a number of other difficulties previously discussed (see page 17). What is apparent is that some hospitals have been reporting a decreasing workload because of fewer measles admissions, while others have been reporting the reverse. The reasons for this are explored here.

A review of admissions to the City Hospital for Infectious Diseases in Cape Town revealed an enormous increase in the number of measles admissions between 1950 and 1973[121]. This upward

trend has continued. In 1950, there were less than 100 measles admissions[121]. These increased to 357 in 1973[121], 554 in 1985[75], and 527 in 1986[75].

At Baragwanath in Soweto, a 1978 report failed to identify measles as a major cause of morbidity in children[122], but increases in its proportion of the inpatient load were reported from 1981 to 1987[77].

At Ga-Rankuwa Hospital, measles accounted for 4,8% of all paediatric admissions in 1986, 9,6% in 1987, 4,5% in 1988 and 1,2% in 1989[123].

In Natal-Kwazulu, Wittenberg reported that 2428 cases of measles were admitted to two Natal Hospitals in 1985[78,99].

At Bophelong Community Hospital in Bophuthatswana, measles accounted for 2% of paediatric admissions during the autumn of 1985 and for 1% between August and July of that year[124].

At Moroka Community Hospital, also in Bophuthatswana, there were 597 measles admissions in 1978, 183 in 1979, 460 in 1980, 128 in 1981 and 117 in 1982. More than half of the cases had been admitted from contiguous farm areas, that were not legally part of Bophuthatswana. These areas were

without health services for the Black population[125].

At Elim Hospital, in Gazankulu, the proportion of admissions to the children's wards attributable to measles dropped from approximately 10% of total paediatric admissions in 1976-1981 to 1% in 1986[126,127].

Figures for a two week survey of all 8 684 patients in 12 hospitals in Transkei and Ciskei in the summer of 1972 showed that 22% of males and 18% of females had a communicable disease. About 15% of the patients in this category had measles[128].

In many areas, hospitals are the only sources of notification data. A summary of these data is presented in Tables II and IV.

3.2.4 THE RELEVANCE OF INCIDENCE DATA FOR THE PLANNING OF MEASLES CONTROL

The measles morbidity data suggest that there have been recognisable areas of high incidence. These, as most other areas in South Africa, are likely to represent areas where notifications have also been a fraction of the true occurrence and, as discussed later, where vaccine coverage has been poor (pages 34 and 70).

Notification data for measles should, therefore, not be collapsed into one national data set, but reported by separate health districts. If this was done timeously, it would allow for energetic and prompt action to be directed at areas of high notification. A possible mechanism for this would be the deployment of a Health Information Officer in each (or selected) health district, to provide managers with information of relevance to the operations of PHC services. One of the duties of this officer would be to ensure compliance with notification regulations, to analyze notification data as they were reported, and to raise the alert before a threshold number was reached and for which the full strength of public health interventions would need to be mobilized.

Hospital data are not easily interpreted. They are dependent on the nature of the hospital (e.g. academic, infectious diseases), particular interests of staff, the number of hospitals serving one community, measles admission policies and the total lack of health care facilities in some rural communities. The latter results in admissions to hospitals in contiguous health wards. What the data tell us is that in those hospitals for which data were available, measles patients accounted for a significant workload. Assuming that the hospital

data on trends can be generalised, it is apparent that downward shifts have been associated with successful vaccination efforts in the communities served by those hospitals (Ga-Rankuwa and Elim Hospitals), while upward shifts seem to be associated with a large and growing urban poor sector (City Hospital in Cape Town).

Very little is known about measles amongst farm workers. These data are likely to be hidden amongst the figures reported by hospitals not directly serving the farm worker population, because of the absence of local health services. This has been the experience of the author in the Gelukspan Health Ward of Bophuthatswana, and it is also reflected in the reports of Moroka hospital.

3.3 MEASLES MORTALITY RATE AND CASE FATALITY RATE IN SOUTH AFRICA

Death notifications, death certifications, reports of outbreak investigations, infant mortality studies, active surveillance systems and hospital reports provide the data from which mortality rates, CFRs and trends of these rates are calculated. Data on deaths certified as due to measles were obtained from the Central Statistical Services via the Centre for Epidemiological Research in Southern Africa. Mortality rates are calculated using

population figures, based on the 1985 census, after correcting for under-counts and after taking into account population growth[67].

Because of the post-acute complications of measles, the definition of measles mortality remains difficult. One accepted in the literature is "any death occurring within one month of the onset of measles symptoms"[42-44,48]. This definition could not be applied here as the raw data did not allow it. For the purpose of this analysis a case fatality is any measles death reported as such either in the literature or on death notification or certification forms. As these sources are more likely to include only deaths in the acute stage of the disease, the true CFR will probably be under-represented.

3.3.1 NOTIFIED MORTALITY

Between 1980 and 1989, the absolute number of notified deaths varied between 171 (1981) and 494 (1983), averaging 303 per annum (Table I)[33,103,104]. For the same period, CFRs based on notifications varied between 1,3% (1981) and 3,8% (1989), with an overall rate of 2,2%[33,103,104]. The slope of the regression line has shown a statistically insignificant upward trend (Fig 3). This is in contradistinction to the notified mortality rate per 100 000 population, which has not changed (Fig 4). The average yearly

notified mortality rate for the past decade was 1,10 per 100 000[88,103,104]. During 1990, measles was the fourth most common cause of notified deaths[6].

3.3.1.1 RACIAL DIFFERENTIALS

No report could be found of notified mortality data or rates stratified by race.

3.3.1.2 GEOGRAPHICAL DIFFERENTIALS

Regional data are reflected in Tables II, IV and V. Blacks in the Northern Transvaal and the Eastern Cape had the highest CFRs. Most CFRs have not shown any apparent trend, except for the consistent downward trends in Gazankulu and the Eastern Cape.

3.3.2 CERTIFIED MORTALITY

In this section, death certificate data on measles mortality obtained from the Central Statistical Services are analyzed by calendar year and racial group for 1970 to 1986 (Figs 5-14). Data for Blacks have been available only since 1979.

Figure 1 shows that notified measles deaths have usually been less than a quarter of the certified deaths. The overlap between the two data sets has

never been studied. In 1983, a year with an excessively high CFR (3,43), the number of notified deaths represented over 45% of the certified deaths. The reason for this variation is not clear to the author.

The certified mortality rate per 100 000 population (all population groups) (Fig 5) reflects a negative trend before 1980 and a positive trend afterwards. The figures for Blacks are available and included only from 1979.

3.3.2.1 RACIAL DIFFERENTIALS

For certified measles deaths, there has been a racial differential. Most of the measles deaths certified since 1979 were for Blacks (Fig 6). Measles mortality rates for Blacks have been higher than for all other racial groups, despite the fact that Black causes of death were more likely to remain undefined[81-83]. The mortality rates have been low for Asians and Whites in the 1980s, although in the early 1970s the Asian mortality rate was much higher than for Whites (Fig 7). The rate for Coloureds has been between that for Blacks and for Asians and Whites.

All population groups, except Blacks, have shown

statistically significant decreases in their measles mortality rates for the period 1970-1986 (Figs 8,9,10). For Blacks, although the regression slope has been positive, the trend was not statistically significant.

The data are further analyzed for the periods 1970-1978 and 1979-1986, or 1970-1979 and 1980-1986 for the reasons outlined in page 20. During the seventies, statistically significant increases were observed for Whites and decreases for Asians and Coloureds (Figs 11-13(B)).

For the eighties, all the trends were negative, with the exception of the Black mortality rate which increased. The only regression slope reflecting statistically significant trends was the one showing a decrease in Coloured mortality rates for the period 1979-1986 (Fig 14).

When mortality rates for Coloureds vs Whites, Asians vs Whites and Asians vs Coloureds were compared for the period 1970-1986, the mortality trends differed statistically from each other ($p < 0,001$). For the period 1970-1978 the negative trends in mortality rates for Coloureds and Asians were statistically different from the positive trend for Whites ($p < 0,001$).

The Coloureds are the only population group for whom the mortality trend of the earlier period differed significantly from the latter (1970-1978 vs 1979-1986) ($p < 0,001$) reflecting more significant improvements in the Coloured rates for the second period when compared with the first.

In summary, the absolute number and the rate per 100 000 population of measles certified deaths seem to be on the increase for Blacks, although none of the trends were statistically significant. Besides reflecting a true increase, these trends are also likely to reflect better case ascertainment and/or more diligent reporting of deaths.

Before 1979, measles mortality rates for Whites were increasing, but already decreasing for Asians,and Coloureds (no data for Blacks). Since 1979, the only population group showing a significant decrease in mortality has been the Coloureds with Blacks not showing even a downward trend.

Coloured mortality, high in the 1960s and 1970s, dropped sharply between 1977 and 1979, and has since been decreasing slowly.

The above trends are seen to be similar to those reported by Wyndham on age specific mortalities for

selected magisterial districts. Using death certificate data, he ranked measles in 1970 as the sixth and seventh leading causes of death in Coloured and Black South African children respectively. He calculated the mortality rates as 125,7 per 100 000 for Coloureds and 108,5 per 100 000 for Blacks[129]. He remarked that, in the ten years from 1968 to 1977, the mortality rates due to measles decreased significantly in Asians but not in Whites (similar to the findings of the analysis of certification data presented in this dissertation), or Coloureds (in contrast to the findings of the analysis of certification data presented in this dissertation). On the basis of his analysis he suggested that the campaign to reduce mortality in Coloureds was largely unsuccessful[130]. Moodie reported an improvement of the Coloured rate to 6,3 per 100 000 for the period 1978 to 1982[131] (similar to the findings of this dissertation).

Bradshaw et al., again using mortality data supplied by the Central Statistical Services, analyzed the measles share of deaths in different age groups (Table VI). In all age groups, measles had a greater share of the deaths in Blacks than in the other population groups. For all except Asians, the share was highest in the age group 1-4 years[81].

3.3.2.2 GEOGRAPHICAL DIFFERENTIALS

Geographical differentials for certified mortality are not available.

3.3.3 INFANT MORTALITY REPORTS

Infant mortality as an indicator of health in general, and child health in particular has received a lot of attention[132-138]. Three infant mortality studies have analyzed infant deaths by cause[132,135,137].

One of the studies ranked measles as the second commonest cause of death of Coloured infants in 1983. The study also showed that the infant mortality rate for measles in Coloured children had varied from 16,9 per 1 000 live births in 1938, to 9,6 in 1950, 12,6 in 1960, 20,8 in 1970, 8,2 in 1980 and 6,9 in 1983[135]. For White infants the rates were 4,1 in 1929, 3,8 in 1938, 0,5 in 1950, 1,2 in 1960 and 0,0 in 1970, 1980 and 1983[135]. A report from Cape Town identified measles as accounting for 0% of post-neonatal mortality in White children and 2,8% in Coloured children[137].

3.3.4 ACTIVE SURVEILLANCE DATA

The overall CFR for the cases studied in the

Johannesburg-Benoni surveillance system was 3%, a figure comparable to the CFR for notifications, but much lower than that reported from hospitals[79].

3.3.5 OUTBREAK INVESTIGATIONS

Two of three outbreak investigations did not report on mortality data[68,69], while in Port Elizabeth there was a CFR of 6% for Whites, 12% for Coloureds and 16% for Blacks[139].

3.3.6 HOSPITAL DATA

Most available hospital statistics have not provided a breakdown by population group.

In City Hospital, the infectious diseases hospital of Cape Town, the 1973 CFR was 8%, comprising 9% for Coloureds, 6% for Blacks and 0% for Whites. The overall rate was 8%[121]. For 1985 to 1986 the CFR was 4%[75].

In 1981, at Baragwanath, in Soweto, measles had a CFR of 2,6%, which dropped to 1,4% in 1983 and increased to 5,8% in 1987[77].

At Ga-Rankuwa Hospital measles was amongst the five major causes of death every year from 1986 to 1989.

Measles accounted for 16,9% of paediatric deaths in 1986 with a CFR of 8,1%; 17,9% of paediatric deaths in 1987 with a CFR of 7,1%; 10,8% of paediatric deaths and a CFR of 6,6% in 1988; and 15,1% of paediatric deaths and a CFR of 10,2% in 1989[123].

In Natal-Kwazulu, a CFR of 15% was calculated for the 2 428 cases of measles admitted to two Natal hospitals in 1985[99].

The Jane Furse Memorial Hospital reported that in the 1960s 352 consecutive admissions of measles carried a CFR of 7%[92]. The inpatient CFR at the community hospital of the Praktiseer district of Lebowa varied from 1% to 3% for the period 1984-1988 (Table II)[107].

In Transkei (1987) the CFRs for 22 hospitals varied from 0,2% to 25,5% (overall 6,3%). The high rate observed for Umtata Hospital could reflect its status as a referral hospital, or its proximity to an overcrowded urban area (Table IV)[119]. Another hospital report from the Transkei mentioned that, over a period of one year (1985-1986), of the 122 post-neonatal infants admitted with measles 7 (6%) died. In this hospital, measles accounted for 17% of all post-neonatal infant admissions and for 7% of all deaths in this age group[101].

At the Gelukspan Community Hospital, Bophuthatswana, several reviews of hospital deaths failed to identify measles as a cause of childhood mortality[108,109,110,140]. A 1983 review of hospital deaths from Moroka, in Bophuthatswana, reported one death from measles out of 237 deaths in the age group 0 to 2 years[125(a)].

At Elim Hospi[illegible] in Gazankulu, measles accounted for 13% of all paediatric inpatient deaths (1976-1981) (CFR = 8%)[127]. However, in 1985-1986, there were none, apparently as a result of improved vaccination coverage[127]. In 1985-1986 at Letaba Hospital, also in Gazankulu, the [illegible] was 8%, as 9 out of 109 measles admissions di[illegible][127].

Reynolds reported a CFR of 36% for cases with measles and its complications admitted to an intensive care unit (ICU)[141]. Other reports on cases requiring ICU admission mention a CFR of 32% in 1973[121] and 40% in 1987[77].

3.3.7 THE RELEVANCE OF MEASLES MORTALITY DATA FOR THE DEVELOPMENT OF CONTROL STRATEGIES

While the notified incidence of measles has shown a consistent, although not statistically significant, drop since 1980 the mortality data have not been so

encouraging. Measles has carried a high CFR in South Africa. National data, based on notifications, have shown an upward trend in the CFR (not statistically significant), although some regional data have shown clear and consistent downward trends.

The notified mortality rate has remained around 1 per 100 000, with occasional peaks. This pattern is very similar to that of certified death rates, with rates beginning to decrease in the 1970s, and then stabilizing in the 1980s. This indicates that the 1970s was a decade of substantial gains for Asians and Coloureds, but not for Whites (no data for Blacks), in terms of measles mortality, although each group started from different baselines, high for Coloureds and Asians and low for Whites. The 1980s represented a period of further gains for Coloureds, stabilisation for Asians, reduced mortality for Whites and a worrying, although not statistically significant, upward trend for Blacks.

Measles deaths as a share of the infant mortality rate seem to be on the decrease.

Common to most hospital reports have been CFRs in excess of 5%, but these data are difficult to interpret. The factors mentioned under hospital-related morbidity (pages 33-34) are again important

in interpreting mortality.

One hospital has reported CFRs decreasing in the presence of increasing admissions for measles (City Hospital in Cape Town). This could reflect more liberal admission policies or changes in treatment protocols. Another one has reported variable but high CFRs in the presence of dwindling numbers of admissions (Ga-Rankuwa Hospital), possibly reflecting more stringent admission criteria. Decreases in both admissions and mortality are likely to reflect a successful immunisation strategy in the community served by the hospital, with little influx of non-vaccinated residents.

In summary, measles was found to be a less frequent disease than in the past. However, it has remained an unjustifiably frequent disease in the Coloured and Black population groups, with a severe prognosis, particularly for Blacks. In Lebowa and the Eastern Cape, measles has remained a common disease with an unacceptably high CFR.

Although the data are limited to a few urban hospitals, it seems that measles amongst the urban poor carries an increasingly more severe prognosis.

In Africa, measles has been particularly severe,

with CFRs often exceeding 10%. In the 1960s, hospital CFRs in West Africa averaged 12% and in East Africa 6%[49,50]. The South African pattern is similar to the high CFRs reported from East Africa in the 1960s[50].

All these data suggest that measles control strategies should put a strong emphasis on limiting mortality.

4 MEASLES RELATED MORBIDITY AND MORTALITY IN SOUTH AFRICA

Several immediate and delayed complications of measles can extend measles related mortality for up to 12 months after the acute illness[46]. Data on this are limited worldwide. In South Africa delayed complications are not reflected on notification forms and are unlikely to be reported in death certificates.

In Johannesburg-Benoni although 42% of the cases were admitted to hospital, only 17% of children with measles had either acute or post acute complications (bronchopneumonia, dehydration, encephalitis and otitis media)[79]. This high admission rate in the absence of explicit complications could reflect either liberal admission policies or under-reporting of complications.

In South Africa, hospital reports have shown protein-energy-malnutrition[142-150], diarrhoea[142,146,147] and respiratory infections[55,79,121,142-151] as related to post-measles morbidity and mortality. One report identified measles diarrhoea in 68% of patients during the acute illness, with a CFR of 14%, rather than the CFR of 6% reported for the uncomplicated cases. In 1986, diarrhoea was the main reason given for admission in nearly one quarter of the children admitted for post-measles complaints within four

weeks of the acute illness. However, as a symptom, diarrhoea was present in 54%. The hospital stay of the post-measles cases with diarrhoea was 50% longer than that of children without diarrhoea[142].

It has been argued that measles vaccination, in developing countries, is such an important measure to control diarrhoea that a significant proportion of cases could be prevented by effective vaccination[52]. If it is assumed that, in 1984, only 30% of measles cases were notified, then the expected true incidence for South Africa was 50 000. If, again, it is assumed that 40% had diarrhoea and that 15% of these died[78], then 20 000 episodes and 3 000 diarrhoeal deaths were associated with measles. Using prevalence and mortality predictions for diarrhoeal diseases in South Africa[152], control of measles could prevent 20%-25% of all diarrhoeal deaths, and about 5% of all diarrhoeal episodes.

Some post-measles complications are related to the prevalence of vitamin A deficiency in the community. In several areas of South Africa vitamin A deficiencies have been reported as common[146,147,153-159]. In these areas, measles may have had serious consequences for children in terms of high mortality and blindness. Around the Elim Hospital in Gazankulu, over the period 1976-1979, health workers found a prevalence of blindness in all age groups of 15 to 20 per 1 000 inhabitants, which was far beyond the

WHO's norm of 1 to 3 per 1 000 population. The prevalence of corneal scarring, mostly due to vitamin A deficiency (with or without concomitant measles infection), was five times higher than the level at which the WHO considers the problem to be a public health priority. Blindness present in children in the Elim Health ward was in 41% of cases associated with measles and malnutrition and with malnutrition alone in 15%[157-159].

A rare complication of measles that, for no obvious reason, has been relatively common in the Cape is subacute sclerosing panencephalitis(SSPE)[131,160-167].

4.1 CONCLUSION

In South Africa, when searched for, measles related morbidity is common both in the acute stage and after the acute episode. Although limited data are available, mortality related to acute stage measles complications and to post-measles morbidity has clearly been underestimated. The available data from South Africa and elsewhere suggest that morbidity and mortality may have been associated with vitamin A deficiency, and that supplementation during an attack of measles would not only reduce mortality, but actually re. ce the severity of the complications.

5 OTHER FACTORS INFLUENCING MEASLES INCIDENCE AND MORTALITY IN SOUTH AFRICA: PROTEIN-ENERGY MALNUTRITION, AGE AT INFECTION, URBANISATION, SOCIOECONOMIC STATUS AND HEALTH CARE.

Thus far, this review has shown that measles remains a common problem in South Africa. It has also presented data to show that "race", vaccination coverage, poverty, urbanisation and vitamin A deficiency may be important factors in understanding its incidence and mortality.

As indicated in the introduction, the literature[168] explores several other factors considered as possible determinants of the incidence and severity of measles. Because of their importance and the availability of South African data, the influence that protein-energy malnutrition, age at infection, urbanisation, socioeconomic circumstance and availability of health care have on incidence and mortality of measles will be examined. This is done in order to determine the extent to which these factors explain the epidemiological patterns observed in South Africa.

While the data presented in the previous chapters were less controversial, this chapter speculates on issues

vital to a greater in-depth understanding of the local measles epidemiology. This understanding could contribute to the further development of an appropriate research agenda and facilitate locally targeted interventions to reduce measles morbidity and mortality.

5.1 PROTEIN-ENERGY MALNUTRITION AND MEASLES

There are South African reports that have identified protein-energy malnutrition as an important determinant of measles severity[142-149]. However, these reports are hospital-based, and failed to control for factors such as overcrowding and micronutrient deficiencies (such as vitamin A), which, in view of the current knowledge of measles and its determinants, were more likely to be the relevant determinants in cases from socioeconomic environments where malnutrition was also common[11,12,15,17,21-25]. As vitamin A deficiency has been reported as common in South Africa[146,147,154,155,157-159], the high measles CFR observed could, to some extent, be due to this.

The degree of pre-morbid protein-energy malnutrition and its contribution to the severity of measles in South Africa needs to be reviewed, taking into account pre-morbid vitamin A status and several of the factors mentioned in Chapter 1, particularly those related to the dose of infection.

5.2 AGE AT INFECTION

Measles data (case notifications, death notifications, death certifications, active surveillance, outbreak investigations, hospital data and community surveys) are analyzed by age and population group.

Although the age structure for each population group in South Africa varies, the overall national age structure is that of a developing country, with less than 5% of the population under one year of age, 20% below 5 years, 15% from 5-9 years, 15% from 10-14 years and 50% below 15 years.

5.2.1 INCIDENCE BY AGE DISTRIBUTION AND POPULATION GROUP

5.2.1.1 NOTIFICATIONS

For 1985-1987 a significant (chi-square, $p < 0.0001$) proportion of measles notified in the non-White population groups (26% for Coloureds, 22% for Blacks and 20% for Asians vs 7% for Whites) occurred in children under one year of age (Fig 15)[88]. A similar age pattern was found for the period 1988 to 1990. Although younger age is likely to lead to greater chances of notification due to increased hospitalization and mortality, the non-White proportions are unacceptably high. The age specific

incidence shows that measles has been six times more common for Black and five times more common for Coloured than for both Asian and White infants[88].

Of notified disease, children 1-4 years of age have accounted for 46% of cases in Blacks, 36% in Asians, 31% in Coloureds and 22% in Whites[88] (Fig 15). In this age group, measles has been four times as common for Black, and twice as common for Coloured than for White children. Asian children had the lowest 1-4 years age specific incidence[88].

In the 5-9 year old age group, Blacks had the highest age specific incidence, with Coloureds and Asians having lower rates than Whites[88]. In children over ten years of age, Whites had the highest incidence rates[88].

Regional reports from the Cape indicate trends similar to the national one. In Cape Town, during 1986, the notification rate was 60 per 100 000, with a rate in children under one year of age of 816 per 100 000[70]. The Cape data also suggested that urbanised children were more likely to get infected at a younger age than rural children[70].

Other regional data on age distribution are presented in Table VII. In Kangwane and

Bophuthatswana, peak age-specific notification rates have occurred amongst 5-9 year old children. In all the other areas excess notification occurred in the first year of life[80].

Notification data have also demonstrated that, in the population groups with an average household size of greater than five (Asians, Blacks and Coloureds), the risk of measles infection amongst younger children was three to six times that of older ones[86].

5.2.1.2 ACTIVE SURVEILLANCE DATA

In Johannesburg-Benoni, the median age for all notified measles cases was 37 months. Ten percent of the cases were younger than 9 months and 18% in Johannesburg and 19% in Benoni were under 12 months of age[79].

5.2.1.3 OUTBREAK INVESTIGATIONS

The 1983 Port Elizabeth epidemic confirmed that measles occurs at the youngest ages in the most overcrowded shanty camps [135].

5.2.1.4 HOSPITAL DATA

During 1985-1986, 72% of children admitted with acute complicated measles to the City Hospital, Cape Town, were aged 15 months and younger[75]. Similar figures were found in 1973, when 90% of measles cases were below three years of age, 50% were younger than 15 months, and 25% were less than ten months old[121].

A report from the ICU at the Red Cross War Memorial Children's Hospital, in Cape Town, identified the median age of children admitted with measles as nine months, of whom 9% were younger than six months (January 1985 to April 1986)[141].

In Clairwood and King Edward Hospitals in Natal, 28% of 111 measles cases were below nine months of age (August 1986)[99]. In Baragwanath, in 1987, 72% of measles admissions were under two years of age, and 36% were nine months or younger[77]. In Letaba, Gazankulu, 41% of 109 measles admissions during 1985-1986 were under five years of age[102].

In some rural areas there has been a noteworthy trend for measles admissions to occur in older age groups, probably because of successful efforts to achieve adequate vaccination coverage[100,110,115,116,169].

Crisp et al. reported that the average age of measles admissions in the Elim Health Ward of Gazankulu increased from 29,7 months in 1976, to 60,9 months in 1986[127]. In Venda, in 1985, an epidemic of measles involved children of an age group "older than norral"[169]. In the Gelukspan Health Ward of Bophuthatswana, measles has no longer been a problem in "under-five" children[115]. Recent outbreaks have occurred in primary school children (Sutton, C. 1988, personal communication).

5.2.1.5 MULTIPLE DATA SOURCES

Loening and Coovadia used an urban community sample, and samples of patients attending several health facilities (2 urban hospitals, 1 peri-urban hospital and 3 rural hospitals) to study urban-rural incidence and mortality differentials from 1978 and 1981. The most important findings of this study were that over 25% of children in the urban environment contracted measles at a very young age, and that the proportion decreased as the population became more rural. An unexpected finding was that the proportion of measles contracted by children aged eight months and younger was higher in the urban community, than in some of the hospitals. This was despite the fact that the largely community-based urban sample was more likely to be biased towards older children than

the peri-urban and rural samples, which were both more hospital- and clinic-based[84].

5.2.1.6 SUMMARY

In summary, measles occurs at younger ages for Blacks and for the urbanised, particularly those living in overcrowded shanty conditions and in areas where vaccination coverage is poor.

Age trends, as reflected in hospital admission figures, seem conflicting. Some areas, particularly the rapidly urbanising, e.g. Cape Town, report a decreasing age at infection with measles. Other areas, apparently those with high vaccination coverage, report the reverse.

5.2.2 MORTALITY BY AGE DISTRIBUTION AND POPULATION GROUP

5.2.2.1 NOTIFICATIONS

The author was unable to find any report of the age breakdown of notified deaths.

5.2.2.2 DEATH CERTIFICATION

Bradshaw et al., using death certificate data supplied by the Central Statistical Services,

analyzed measles' share of deaths in different age groups (Table VII)[81]. In all age groups, measles comprised the greatest share of the deaths of Blacks. For all population groups, except Asians, the share was highest in the 1-4 years age group.

The age pattern of certified measles deaths has been very similar to the pattern of notified deaths.

For 1985, the age specific mortality rates for each population group was calculated using census data as denominators. For children younger than 15 years of age, Blacks had the highest age-specific mortality rates, followed by Coloureds, Asians and Whites. Amongst 15-19 years age group, the highest mortality rate was in Coloureds, then Whites, Blacks and Asians. For those 20 years of age or older, the mortality rate was similar for Blacks and Coloureds (Table VIII).

For Blacks and Coloureds, infants accounted for over 35% of certified deaths and children under 5 years of age for more than 90%. For Asians, infants accounted for 44% of the deaths, but only 80% were under 5 years of age. For Whites, the infant share of deaths was stable at 30%, with 78% of all deaths occurring under the age of 5 years (Table IX). The percentage of infant deaths in all non-White

population groups seemed to be on the increase, although the increase was statistically significant only for Coloureds (1968-1986) (Fig 16). This change was accompanied by a significant decrease of the percentage of deaths occurring in children 1-4 years of age (1968 to 1986). The trends of the two age groups differed significantly, $p<0,001$.

The only statistically significant trend for Asians was a decreasing percentage of deaths occurring in the 1-4 years age group (1968-1986) (Fig 17), for Blacks an increasing percentage occurring in the 5-9 years age group (1979 to 1986) (Figs 18(A) and 18(B)) and for Whites a decreasing percentage after 1980 in the 20 years and older age group (Fig 19).

5.2.2.3 OUTBREAK INVESTIGATIONS

During the 1983 Port Elizabeth epidemic, mortality was found to be particularly high in the socioeconomically poor squatter areas. Fifty four percent of Blacks and 62% of Coloured deaths were under 1 year of age (84% and 79% respectively were under 2 years of age). The highest age-specific CFR was in children under 6 months of age (44%)[139].

5.2.2.4 HOSPITAL DATA

Age breakdown has not been reported frequently in hospital data. Data from the City and Red Cross War Memorial Children's Hospitals in Cape Town illustrate the high measles mortality load in children younger than five years, particularly in infants[75,121,141]. In King Edward Hospital, mortality in the very young was very high, with a CFR of 26% for infants under 8 months[78,99,142].

5.2.2.5 SUMMARY

It is vital to remember that all incidence data are defective in terms of under-ascertainment of measles and in under-estimates of some of the denominators. Also, considering gradients among population groups, we must not ignore that underlying them were gradients in data quality, with the highest at risk having the worst quality data set. Therefore, the differentials are minimum estimates rather than precise values.

The incidence and mortality trends described are consistent with the lowest socioeconomic status for Blacks and Coloureds, an intermediary situation for the Asians, and the best status for the Whites. It is difficult to speculate any further on the cause

of the differences, as data on other variables that might account for differences, such as vaccination coverage and mean age at infection, are not available for each population group.

What has become apparent from comparing disease notification with death certification data, is that infants, and to a lesser extent children under 5 years, have had a share of mortality far in excess of their share of the disease (Table X).

The age-specific mortality trends have varied among population groups. The share of deaths has shown a significant increase in the age group 1-4 years for Asians, 5-9 years for Blacks and under 1 year for Coloureds. It has shown a significant decrease for the age group 1-4 years for Coloureds and 20 years or older for Whites. For all population groups except Asians, the measles share of age-specific mortality has been highest for the 1-4 year group. CFRs and age-specific mortality rates have been highest in infants.

5.2.3 THE RELEVANCE OF AGE AT INFECTION AND OF POPULATION GROUP AS DE' 'NANTS OF MEASLES INCIDENCE AND MORTALITY

The importance of age at infection as a determinant of measles incidence and mortality is not easy to interpret[39-41,170]. The following explanation of the relevance of age as a determinant of incidence and mortality is based on a blending of the South African data with current understandings of measles epidemiology, as reflected in the international literature and reviewed in chapter 1.

In unvaccinated or low vaccination coverage populations, particularly in rural settlements, higher mean age at infection is more likely to be associated with an epidemic pattern of the disease. Thus, the presence of the virus in the community will be short-lived, but high in concentration. All those susceptible, independent of age, will be infected, frequently with multiple cases in the same household. In these circumstances, the high dose of infection is likely to result in more severe infection, and a higher overall CFR, particularly in the young.

In contrast, in the endemic situation, mostly in large, densely populated, urban areas, measles

occurs the year round and there are no epidemics. Thus, as the measles virus is at a constant low level, siblings will tend to be infected in different years, and there will be no accumulation of susceptibles in the older ages. Therefore, younger children will be infected with a lower infective dose. Although this will still result in a high CFR, it will be lower than that of the epidemic situation, where, inter alia, multiple household cases would result in increased mortality[23].

To be able to advance a logical explanation of what has been happening at the community level in South Africa it would be necessary to know the overall mean age at infection, the overall CFR, the age-specific CFR, vaccination coverage and the endemic or epidemic nature of the disease. No single community in South Africa has had all these indices studied. Nevertheless, all the South African data reviewed so far in this dissertation are compatible with the above speculation. South African data point to the fact that CFRs have been highest for the younger infant, particularly those from peri-urban settlements and particularly from the population group with the highest level of overcrowding. As vaccination rates increased, as in Gelukspan and Elim, the mean age at infection also increased and the CFRs decreased.

From this, it is suggested that the South African data are compatible with the explanation that higher incidence and mortality is associated with factors that predispose to a higher dose of infection. Further examination of the measles data in relation to differentials in urbanisation and socioeconomic status will support, but will not necessarily prove, this interpretation.

5.3 URBANISATION

Urbanisation represents all the processes of social change in the urban environment. The most urbanised are undergoing the least change, and the least urbanised the most change. Being urbanised and undergoing urbanisation represent realities that differ according to socio-economic status, political enfranchisement, environmental conditions and culture.

The relationship between urbanisation as a reflection of the nature of a settlement and measles is not a simple one[39,170]. Whites have had a lower incidence of notifications and lower CFR than both Asians, who are more urbanised, and Coloureds and Blacks, who are less urbanised. Also, Kettles' analysis of notifications in the Western Cape showed that more urbanised Blacks had a higher incidence and background rate of notifications, and more frequent measles peaks than the least

urbanised[70]. This work was consistent with another study that showed that measles incidence was highest in urban Blacks and lowest in rural Blacks[84]. The mean age at infection was for urban Blacks lower than for Black rural residents[84]. The limited data available also suggested that hospital CFRs have been higher for urban Black residents (Table IV)[119].

An obvious conclusion is that, in South Africa, urbanisation under poor socioeconomic conditions has been associated with a higher incidence of measles at a younger age and with a higher fatality rate. The reverse seems to apply to urbanisation under good socioeconomic conditions. The operative factor, therefore in the epidemiology of measles and urbanisation is likely to be socioeconomic status.

5.4 SOCIOECONOMIC STATUS

Socioeconomic data have never been recorded on notification forms. However, in South Africa, "race" is well known to be closely related to socioeconomic status, overcrowding, the degree of urbanisation and accessibility to health care. Therefore, using "race" as a proxy for socioeconomic status, it becomes apparent that the poorest, least urbanised, most overcrowded group who have the least access to health care, i.e. Blacks, have the highest incidence of and CFR for measles.

This was specifically illustrated during the 1982-1983 measles epidemic in Port Elizabeth. During the epidemic, 88% of notifications and 91% of all deaths occurred amongst Blacks, who accounted for only 50% of the population. This was in comparison to only 1% of cases, and 0.3% of deaths amongst Whites, who comprised 26% of the population. The areas most affected were the most socioeconomically deprived, with the most overcrowding and the worst housing[139].

In Cape Town, the overall notification rate for 1986 was 60 per 100 000, but for Blacks it was 179. Seventy five percent of cases admitted to the City Hospital with complicated acute measles were Blacks from the Peninsula, an area where Blacks account for only a small fraction of the population[75].

Health workers from Durban reported an average of 9,4 persons living in Black households which experienced cases of measles, while the overall average for Blacks in South Africa was 5,9, and for Whites 3,6[171]. Notification data demonstrated that, in the population groups with an average household size greater than 5 (Asians, Blacks and Coloureds), the risk of measles infection amongst the under-ones was 3 to 6 times that of older children[88]. Eighty percent of cases of measles studied in Johannesburg-Benoni, in 1988, stayed in houses with a crowding index of more than 2,5 persons per sleeping

room[79].

South African data as reviewed here are compatible with explanations of heavy dose of infection as the main determinant of measles severity. It was previously suggested that the dose of infection is most commonly related to the degree of overcrowding in the community, particularly overcrowding of children. The overcrowding in the poor urban areas with poor housing and ventilation would then contribute to a high dose of infection with the virus, and be one of the reasons behind high rates of severe measles in South Africa. In South Africa, the poor and urbanised Blacks would be, because of overcrowding and poor ventilation, the group at highest risk for high dose of infection. This risk could be successfully counteracted by high vaccination coverage, but the urban poor, particularly the Black urban poor, have also been a politically deprived group whose health care has been neglected.

5.4.1 HEALTH CARE PROVISION

Health care is an important modelling factor of measles morbidity and mortality. Three important factors, for which there are South African data, will be discussed: health care facilities as a source of measles; vaccination against measles; and care of the sick child with measles.

5.4.1.1 HEALTH CARE FACILITIES AS SOURCES OF MEASLES

It is ironic that the development of clinics and hospitals has brought susceptible children into contact with measles cases. Thus, health care facilities have became important vectors in the spread of measles in developing countries[173-178] and in South Africa[76,99,141,179-181]. Wittenberg, in a survey of 111 new measles admissions, found that 59 (53%) had attended a clinic or hospital 7 to 15 days before the onset of illness[99]. Reynolds reported that 25% of all cases of life-threatening measles in Cape Town acquired the disease in hospital or as outpatients[141]. A three month survey of measles admissions to the City Hospital for Infectious Diseases again found that 20 (32%) of 61 admissions had had a contact with a health care facility 10-14 days before the onset of disease[181].

The prognosis for hospital-acquired measles is particularly severe[141,175-177]. Observations of children with measles, some of them dating back to the past century, have related a severe infection to a history of or concurrent diseases[18]. The poorer prognosis for hospital-acquired infection could be related to this phenomenon but the extent to which this could be explained in terms of the dose of infection model needs to be explored.

The above facts have resulted in recommendations to protect children coming into contact with health facilities with vaccination or immunoglobulin[176,179]. The current public health importance of such measures again became apparent during a recent survey of health facilities in the Western Cape, when it was found that these recommendations are still not enforced with the necessary rigour[7].

5.4.1.2 PREVENTIVE HEALTH CARE: VACCINATION AND MEASLES

Vaccination remains central to the control of measles. The falling incidence and CFR of measles in areas with successful vaccination services has already been mentioned. In South Africa, there have been vast areas in the homelands, White farm areas and peri-urban squatter camps where health care has been absent, or where preventive care has never been aggressively promoted.

Table XI reviews data from areas where there are morbidity and/or mortality data and data on vaccination coverage. It is apparent that, with the exception of Malamulele in Gazankulu[186], vaccination coverage above 70% has been associated with a lower incidence rate for measles[106,107,111,114,116,117,156,157].

The data from Malamulele[186] can be explained in six

ways: they could be a reflection of the methodology used (Buch E, personal communication, 1991); high vaccination coverage in children between 12 and 23 months of age, but much lower in older children with an outbreak of disease in the older children; cold chain failure with concomitant failure to impart immunity to children; above average notification services when compared with other areas, although this is unlikely (Buch E, personal communication, 1991); the prevalence of poor environmental and socioeconomic conditions in Malamulele, with extreme clustering of children not vaccinated, allowing for outbreaks of disease in the presence of high vaccination coverage; or lastly, high density housing associated with the occurrence of measles before younger children had been vaccinated. It is not possible to speculate on the factors at play here as the local conditions were not known and essential information, such as the mean age at infection, were not specified.

The existing vaccination surveys show that measles vaccine coverage has not usually been higher than for diphtheria/whooping cough/poliomyelitis (DWT) vaccine3[114,116,188-194] (Table XII). There are, therefore, no apparent operational advantages in recent recommendations to shift the age of vaccination to six months[93,94], although these recommendations were

not entirely based on operational factors.

5.4.1.3 CLINICAL CARE OF THE SICK CHILD WITH MEASLES

The treatment of measles has been, up to very recently, of a supportive nature. In uncomplicated measles, the emphasis has been on control of pyrexia, nutritional support and hygiene[9].

The development of complicated measles has always been followed by treatment specific to the complication.

On the basis of a small randomised control trial[195], which did not achieve statistical significance for any of its findings, the WHO has been promoting the use of vitamin A supplementation to reduce mortality in areas with CFRs equal to or in excess of 1%[26,27*]. More recently, a randomised control trial in Cape Town found strong supportive evidence for the routine use of vitamin A in children with measles complicated by pneumonia, diarrhoea or croup, who were admitted to hospital within five days of the onset of the rash. The Cape Town trial found twice as many deaths in the control group as in children

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*This recommendation was first mentioned and ignored in 1932[196]. Morley has, for more than 20 years, advocated the use of vitamin A supplementation in areas known to be deficient in this nutrient, to prevent the blinding complications of measles[9].

given a high dose of vitamin A. The treatment group also had a more rapid recovery from pneumonia and diarrhoea, less croup and shorter periods of hospitalisation. Vitamin A supplementation in severe measles has, therefore, the potential to halve mortality and reduce morbidity by one-third[146,147].

What has not yet been proven, and requires urgent investigation, is the value of vitamin A in preventing severe measles by supplementation before the onset of complications.

5.5 CHAPTER CONCLUSION

Certain factors are clearly linked to the incidence of and mortality from measles. What the analysis in this chapter has tried to demonstrate is that factors which are commonly assumed to be directly associated with measles severity and mortality may be confounders of a causative pathway in which dose of infection may be the most important operative factor. The South African data are not very conclusive, but are certainly compatible with this explanation.

6. CONCLUSIONS

This analysis and review of the South African data has yielded potentially valuable insights on measles epidemiology, against the background of the current concepts reviewed from the international literature. It allows conclusions to be drawn that will assist local targeting of control efforts. These conclusions pertain to data, morbidity, mortality and the determinants of morbidity and mortality.

6.1 DATA

The available information on measles has been limited by the inadequacies in the surveillance system and the poor quality of data analysis and reporting. The data reported in this dissertation are biased due to a systematic lack of data from high prevalence areas, especially because of poor service cover and, for political reasons, the lack of information from the independent homelands.

The system for routine measles surveillance has been limited to notifications of disease and death due to measles. Death certification is another potentially useful surveillance data base that has not been utilised to plan and monitor health care delivery.

The poor surveillance has resulted in under-counting.

This under-counting reflects inadequate patient accessibility to the reporting officers and, to some extent, to health care facilities. This is particularly so in peri-urban areas and some rural health wards. The ignorance of the notifying officers compounds the problem of under-reporting of measles. The lack of a management culture that uses information to plan and monitor health care results in data that are not analyzed or are submitted only to limited analysis and are not timeously reported.

Despite the poor quality of the data available, it has been possible to identify reports that try to correct for under-reporting, either at the data collection stage or at the analysis stage. From these, and from the analysis of death certificates and of notified data carried out for this dissertation, it has been possible to gain useful insights into the trends and possible determinants of measles morbidity and mortality in South Africa.

6.2 INCIDENCE

Up to the late 1980s the incidence of measles was still high in South Africa. Although incidence over time showed a downward trend this was not statistically significant. This trend indicated that, at least for the past decade, control efforts have failed to reduce morbidity due to measles. Peak incidence was in the 5-9 year age group.

Notwithstanding data limitations it is apparent that there exist defined areas of high measles morbidity, such as the Transkei, Lebowa, Kwazulu and the Eastern Cape, presenting seasonal patterns that vary from region to region. These seasonal patterns seemed to have been greatly influenced by the population shift that occurred when influx control was relaxed.

Vaccination coverage of 70% or higher seems to have been associated with lower infection rates. The South African data did not support the contention that shifting the age of vaccination against measles to coincide with the third dose of DWT would increase vaccination coverage, although this recommendation must also be viewed against the background of other factors affecting vaccine efficacy*.

6.3 MORTALITY

Before 1979-1980 measles mortality rate dropped for Asians and Coloureds. Since 1979 the rate has decreased significantly only for Coloureds.

It is not possible to comment on the mortality trends for Blacks. Although the trend is an upward one it has not reached statistical significance. This could reflect

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*As mentioned in the introduction, two important considerations to take into account are the rate at which maternal antibodies are lost and the age of occurrence of measles in the epidemiological pattern prevalent in a community.

better case ascertainment, better reporting, or a truly high level of measles mortality, that is still on the increase. It probably reflects all three.

In analysing the mortality trend per age group, different patterns were found for each population group. It was increasing for Coloured infants, for 1-4 year old Asian children and for 5-9 year old Black children. It decreased for 1-4 year old Coloured children and for Whites over the age of 20 years.

The CFR has been high and rising, although this was not statistically significant. The highest CFRs and age specific mortality rates occurred in infants. Mortality in the very young was particularly high.

The South African literature, in line with the international literature, indicate that measles acquired in health facilities has a particularly high mortality rate. Vitamin A supplementation has the potential to reduce morbidity and mortality in children with severe measles if administered early in the acute phase of the disease.

Over and above acute measles deaths, it seems likely that a significant proportion of deaths due to diarrhoeal diseases in South Africa could have been prevented by measles vaccine.

6.4 DETERMINANTS OF THE INCIDENCE AND MORTALITY OF MEASLES

Even if not conclusive, the data reviewed and analyzed support explanations of increasing severity of measles and higher mortality with an increased infective dose. The observed epidemiological patterns seem dependent on vaccination coverage and overcrowding, both major determinants of the infective dose.

In South Africa, Blacks in general, and peri-urban Blacks in particular, have been the group at highest risk for a high dose of infection because of overcrowding, poor ventilation and low vaccination coverage . Measles therefore, has been more common, has occurred earlier and resulted in more deaths amongst Blacks and the urban poor.

6.5 OVERALL CONCLUSION

The data presently available provides a picture of country-wide endemic measles, that still has a high CFR, particularly amongst the urban poor and in some rural areas. Overcrowding, a common problem in South Africa, has predisposed the urban poor to high doses of infection and more severe measles. Poor vaccination coverage has also been an important determinant of incidence.

7. RECOMMENDATIONS

This review and analysis of the South African data allows one to advance recommendations in three areas: measures to improve data; measures to reduce the incidence of measles; and measures to reduce complications and mortality. At times some of the recommendations seem to state the obvious; but the obvious has not been implemented. As such, it still needs to be recommended for inclusion in a comprehensive strategy for measles control.

7.1 MEASURES TO IMPROVE DATA

Despite the fact that there have been systems for regular collection of measles morbidity and mortality data, it is obvious that significant gaps remain in our knowledge about measles in South Africa. A number of measures are required to overcome these gaps. These include infra-structural development, the will to overcome artificial political barriers, the pooling of essential health data collected in a standardised way and better regional reporting of data obtained from death certificates and notifications.

This could be addressed by careful utilisation of a mix of systems of surveillance that blend improved notification and death certification with more innovative

systems, like the use of sentinel sites, active and detailed outbreak investigations and selected epidemiological studies.

It would be essential to pay particular attention to the different steps involved in data collection, starting with accessibility to reporting officers and ongoing auditing of data quality and completeness. All data should be monitored and evaluated regularly, according to standard definitions and methods of collection and reporting. Reporting officers should be given further training. Both doctors and nurses need to be repeatedly reminded of their statutory duty to notify measles. Notification books should be available at all clinics, health centres and hospitals, even when these are staffed entirely by nurses.

It is important to create a position of Health Information Officer in the health service personnel structure. This person would assume responsibility for the supervision of data collection, analysis and reporting and for initiating locally public health measures to address the problems identified.

As routine surveillance systems do not always provide a reliable data base or, when reliable, may not allow for the collection of some of the data that are so useful for health planning and monitoring purposes, it is important

to complement the data from these routine systems with data from sentinel sites, outbreak investigations and community surveys. These should be done using standardized methods[105,198,199]. Nevertheless, in situations where the denominators and numerators can be reliably approximated, vaccination coverage can be reliably calculated[200] using methodologies already described[201].

Sentinel sites should be chosen to include communities, health centres and hospitals so as to access data from community, primary care and hospital levels. These data could be used to guide health service development. This approach was successfully experimented with in the Ivory Coast[197] and seems practical for South Africa.

Outbreaks of disease should be actively investigated along the lines of the surveillance system implemented in Johannesburg[79], or using methods reported in the literature[105].

This mix of methods would increase the availability of information currently not available but important for planning and evaluation of the control programmes, namely mean age at infection, age distribution of cases and deaths, overall and age specific CFR, nature of the settlement from which the cases derive and data on key risk factors, such as overcrowding.

7.2 MEASURES TO REDUCE THE INCIDENCE OF THE DISEASE

Any policies aimed at correcting the inequities in the disease load of different population groups must immediately address the problem of poor vaccination coverage. More medium to long term strategies would prioritize the promotion of anti-crowding measures.

Vaccination remains the most important means of measles control. Determinants of vaccination coverage in South Africa have been studied in a number of surveys. The essential ingredient, which was still missing as late as 1989, was the commitment of political and state structures to measles eradication. The levels of notified measles, since the "measles campaign" was initiated in the late 1980s, are a clear indication that effective vaccination coverage remains inadequate. It is important that all services be galvanised into a consistent, on-going, outreach-based programme to increase vaccination coverage to above 90% nationwide, and to maintain it at that level.

What is needed is: a national policy with national, regional and local objectives; policies and programmes for training of personnel; allocation of resources (including time and transport); an efficient vaccine supply system; a functioning cold chain; and a system of measles and vaccination surveillance.

The observation that peak incidence is in the 5-9 years age group suggests that vaccination at primary school entry should be added to vaccination currently given at nine months*. Despite the fact that for many years academics[178] and the department of health[202] have recommended that children in contact with health facilities be immunised against measles, this has not taken root, as shown in a recent survey of health facilities in the western Cape[7]. It is therefore essential to keep re-emphasizing the importance of this measure to reduce the incidence of severe measles.

Anti-crowding measures would involve more caring housing policies and improved housing conditions. The urban poor are already, and will remain, a priority to be specifically targeted. Family spacing methods would complement this by reducing the pool of susceptible siblings in the same household, thereby reducing virus concentration in the domestic micro-environment.

7.3 MEASURES TO REDUCE MORTALITY

Vaccination procedures recommended today in South Africa, if adequately implemented, would reduce incidence and increase the mean age of infection.

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*The case for this type of two dose policy has been defended convincingly in a recent article in the Bulletin of the WHO 1993; 71: 93-103.

The impact of improved vaccination coverage on mortality would probably be greater than could be expected solely from the elimination of measles. The excess mortality that may occur for months after the acute attack would be reduced.

Vaccination may also, indirectly, reduce mortality among un-immunised children still exposed to measles by reducing clustering and in-house transmission of the virus, thereby reducing their infective dose.

Guidelines for clinicians along the lines of those developed by Morley, for East and West Africa, might help to improve patient care and reduce mortality.

The WHO recommends that, in the presence of CFRs in excess of 1%, vitamin A be routinely prescribed to all children with measles. In view of local data on vitamin A deficiency, and following the results of the Cape Town study, it seems wise to implement this policy, while waiting for more detailed epidemiological data.

One must end with a word of caution and a call for action. Firstly, although measles is eminently controllable, its control requires commitment at the highest levels and an ongoing consistent effort. In South Africa, the disease is still far from controlled and the children of this country need more than press releases,

policy statements and vaccination surveys, which to date seem to have been the core of the "measles campaign". Secondly, even if peri-urban areas are at high risk for severe measles, over half of the cases are still reported from the rural homelands in spite of gross under-notification. The data from this report suggest that extra resources and effort should be directed at Eastern Cape, Kwazulu, Lebowa and Ciskei, as well as at the urban poor. The resources should be used to strengthen the PHC infra-structure and to support a sustained programme of vaccination and appropriate monitoring. The PHC infra-structure would provide the base from which to launch inter-sectoral collaboration and the spirit of community development, from where initiatives to support appropriate housing could be developed.

TABLES

TABLE II. INCIDENCE RATE FOR, CASE FATALITY RATE OF, AND VACCINATION COVERAGE AGAINST MEASLES IN LEBOWA.

YEAR	INCIDENCE/100 000		CFR (%)
	PRAKTISEER*	LEBOWA	PRAKTISEER*
1984	97	-	1,0
1985	103	127	2,9
1986	60	49	1,4
1987	109	93	1,9
1988	88	77	1,2

* PRAKTISEER - ONE OF THE HEALTH WARDS OF LEBOWA.

TABLE III. MEASLES INCIDENCE RATE PER 100 000 BLACK POPULATION IN SELECTED AREAS.

YEAR	KANGWANE	KWANDEBELE	BOP*	GAZ*	LEB*	NT*	OFS*	EC*
1985	35,6	27,0	26,7	99,8	122,0	42,3	32,5	88,7
1986	16,1	17,8	19,7	14,4	48,6	8,0	15,4	198,8
1987	138,8	53,9	34,7	178,7	93,3	20,1	75,0	170,5
1988	112,6	27,4	19,2	88,8	76,6	16,2	31,4	91,7
1989	21,7	18,8	1,9	30,3	86,9	17,7	3,8	72,9

*BOP=BOPHUTHATSWANA, GAZ=GAZANKULU, LEB=LEBOWA, NT=NORTHERN TRANSVAAL, OFS=ORANGE FREE STATE, EC=EASTERN CAPE

TABLE IV.REPORTED MEASLES CASES AND CASE FATALITY RATES (CFR) IN TRANSKEI HOSPITALS, 1987.

HOSPITAL	CASES	DEATHS	CFR(%)
All Saints	916	118	12,9
Bambisana	334	9	2,7
Butterworth	119	1	0,8
Cala	147	10	6,8
Canzibe	294	25	8,5
Cofimvaba	173	9	5,2
Empilisweni	181	2	1,1
Glen Grey	442	25	5,7
Greenville	90	1	0,1
Holy Cross	539	36	6,7
Madwaleni	433	15	3,5
Mary Terese	325	2	0,6
Nessie Knight	174	24	13,8
Sipeto	132	3	1,7
St Barnabas	115	10	8,7
St Elizabeth	620	28	4,5
St Lucy's	322	2	0,6
St Patric	159	4	2,5
Tayler Bequest	373	10	2,7
Umlami	474	1	0,2
Umtata*	325	83	25,5
Zitulele	342	21	6,1
TOTAL	7029	439	6,3

BASED ON REFERENCE 119
NOTE: The following hospitals are not included in the table as no deaths were reported: Isilimela, Mt Ayliff, Rietvlei, St Margaret's, Tafalofefe.

* LARGEST URBAN AREA IN TRANSKEI

TABLE V. BLACK CASE FATALITY RATES FOR NOTIFIED MEASLES IN SELECTED AREAS.

YEAR	KANGWANE	KWA-NDEBELE	GAZAN-KULU	LEBOWA	NORTH. TRANSVAAL	OFS*	EASTERN CAPE
1985	0,6%	0,0	5,3	0,0	5,1	0,0	11,1
1986	6,2%	1,9	5,4	0,0	0,0	0,0	7,5
1987	0,6%	1,2	1,5	0,8	7,9	1,0	9,5
1988	0,5%	0,0	1,5	2,2	1,5	1,5	6,4
1989	2,6%	1,6	1,0	1,6	5,3	2,7	4,7
AVERAGE	1,0	0,9	2,5	0,9	4,7	0,9	8,0

* OFS=ORANGE FREE STATE

TABLE VI. MEASLES AS A PERCENTAGE OF ALL DEATHS IN SPECIFIED AGE GROUPS, 1984.

AGE	ASIANS	BLACKS	COLOUREDS	WHITES
0-1m	0,0	0,0	0,0	0,0
1-11m	1,0	3,6	1,4	0,7
1-4y	0,0	7,7	3,9	1,3
5-14y	0,0	2,7	0,7	0,6

CERTIFIED MORTALITY. BASED ON REFERENCE 81

TABLE VII. AGE-SPECIFIC MEASLES INCIDENCE RATES PER 100 000 BLACKS IN SELECTED AREAS, 1988.

AGE YEARS	KANGWANE	KWA NDEBELE	BOPHUTA-TSWANA	GAZAN-KULU	LEBOWA	NORTHERN TRANSVAAL	OFS	EASTERN CAPE
0-1	120,6	100,2	45,6	313,8	262,5	71,8	107,6	975,8
1-4	247,7	80,8	50,0	259,3	219,0	45,3	91,0	303,5
5-9	334,5	85,9	61,1	210,5	217,5	29,7	75,2	99,9
10-14	193,7	13,4	23,1	96,7	78,2	20,2	36,1	47,6
15y +	13,5	0,5	0,7	9,6	3,3	2,6	1,6	2,8

NOTIFICATION DATA. ADAPTED FROM REFERENCE 80

TABLE VIII. AGE-SPECIFIC MEASLES MORTALITY RATE PER 100 000 POPULATION (CERTIFIED MORTALITY, 1985)

AGE GROUP	POPULATION GROUP ASIAN	BLACK	COLOURED	WHITE
0-4 YEARS	7,9	38,0	28,9	0,0
5-9 YEARS	2,2	2,4	1,3	0,0
10-14 YRS	0,0	1,1	0,3	0,0
15-19 YRS	0,0	0,1	0,3	0,2
20 OR +	0,0	0,1	0,1	0,0

TABLE V. BLACK CASE FATALITY RATES FOR NOTIFIED MEASLES IN SELECTED AREAS.

YEAR	KANGWANE	KWA-NDEBELE	GAZAN-KULU	LEBOWA	NORTH. TRANSVAAL	OFS*	EASTERN CAPE
1985	0,6%	0,0	5,3	0,0	5,1	0,0	11,1
1986	6,2%	1,9	5,4	0,0	0,0	0,0	7,5
1987	0,6%	1,2	1,5	0,8	7,9	1,0	9,5
1988	0,5%	0,0	1,5	2,2	1,5	1,5	6,4
1989	2,6%	1,6	1,0	1,6	5,3	2,7	4,7
AVERAGE	1,0	0,9	2,5	0,9	4,7	0,9	8,0

* OFS=ORANGE FREE STATE

TABLE VI. MEASLES AS A PERCENTAGE OF ALL DEATHS IN SPECIFIED AGE GROUPS, 1984.

AGE	ASIANS	BLACKS	COLOUREDS	WHITES
0-1m	0,0	0,0	0,0	0,0
1-11m	1,0	3,6	1,4	0,7
1-4y	0,0	7,7	3,9	1,3
5-14y	0,0	2,7	0,7	0,6

CERTIFIED MORTALITY. BASED ON REFERENCE 81

TABLE VII. AGE-SPECIFIC MEASLES INCIDENCE RATES PER 100 000 BLACKS IN SELECTED AREAS, 1988.

AGE YEARS	KANGWANE	KWA NDEBELE	BOPHUTA-TSWANA	GAZAN-KULU	LEBOWA	NORTHERN TRANSVAAL	OFS	EASTERN CAPE
0-1	120,6	100,2	45,6	313,8	262,5	71,8	107,6	975,8
1-4	247,7	80,8	50,0	259,3	219,0	45,3	91,0	303,5
5-9	334,5	85,9	61,1	210,5	217,5	29,7	75,2	99,9
10-14	193,7	13,4	23,1	96,7	78,2	20,2	36,1	47,6
15y +	13,5	0,5	0,7	9,6	3,3	2,6	1,6	2,8

NOTIFICATION DATA. ADAPTED FROM REFERENCE 80

TABLE VIII. AGE-SPECIFIC MEASLES MORTALITY RATE PER 100 000 POPULATION (CERTIFIED MORTALITY, 1985)

AGE GROUP	POPULATION GROUP ASIAN	BLACK	COLOURED	WHITE
0-4 YEARS	7,9	38,0	28,9	0,0
5-9 YEARS	2,2	2,4	1,3	0,0
10-14 YRS	0,0	1,1	0,3	0,0
15-19 YRS	0,0	0,1	0,3	0,2
20 OR +	0,0	0,1	0,1	0,0

TABLE IX. PERCENTAGE PER POPULATION GROUP OF CERTIFIED MEASLES DEATHS UNDER 1 OR UNDER 5 YEARS.

AGE	POPULATION GROUP	YEARS 1968-1971	1972-1976	1977-1981	1982-1986
<1y of age	AFRICANS*	NA#	NA	41%	43%
	ASIANS	20%	35%	16%	44%
	COLOUREDS	34%	35%	42%	43%
	WHITES	24%	30%	33%	30%
<5y of age	AFRICANS*	NA	NA	95%	92%
	ASIANS	94%	95%	82%	80%
	COLOUREDS	96%	96%	95%	95%
	WHITES	77%	79%	82%	78%

* FOR THE BLACK POPULATION DATA ARE ONLY AVAILABLE FROM 1979 TO 1986. THESE DATA ARE THEREFORE REPORTED IN TWO 4 YEARS GROUPS, 1979-1982 AND 1983-1986

NA=NOT AVAILABLE

TABLE X. PERCENTAGE PER POPULATION GROUP OF MEASLES CASES AND DEATHS OF CHILDREN UNDER 1 OR UNDER 5 YEARS.

AGE	POP. GROUP	NOTIFIED MORBIDITY*	CERTIFIED MORTALITY**
< 1 year	BLACKS	23%	43%
	ASIANS	21%	44%
	COLOUREDS	31%	43%
	WHITES	8%	30%
< 5 years	BLACKS	69%	92%
	ASIANS	59%	80%
	COLOUREDS	66%	95%
	WHITES	32%	78%

* AS MEASURED BY NOTIFICATIONS FOR THE PERIOD

** AS MEASURED BY CERTIFIED DEATHS FOR THE PERIOD

TABLE XI. NOTIFIED MEASLES INCIDENCE PER 100 000 POPULATION IN AREAS WITH KNOWN VACCINATION COVERAGE.

AREA	INCIDENCE/100000 (year)	VAC. COVERAGE (year)
BOTSHABELO	19,5 (1988)	31% (1989)
PRAKTISEER	88,0 (1988)	48% (1989)
MOLOPO	40,4 (1988)	50% (1989)
NSIKAZI	42,5 (1988)	56% (1988)
ALEXANDRA	30,0 (1988)	67% (1988)
ALEXANDRA	25,0 (1989)	78% (1990)
ODI	0,0 (1988)	78% (1989)
TAUNG	14,5 (1988)	81% (1985)
MALAMULELE	121,7 (1988)	82% (1987)
GELUKSPAN	1,9 (1988)	84% (1988)
ELIM	25,7 (1988)	85% (1985)

TABLE XII. COMPARISON OF VACCINATION COVERAGE WITH THE THIRD DOSE OF DWT AND THE FIRST DOSE OF MEASLES.

AREA	YEAR	DWT3 % COVERAGE	MEASLES % COVERAGE
DWT3 coverage greater than MEASLES coverage			
MOLOPO	1984	75%	59%
ELIM (RITAVI)	1985	94%	85%
MALAMULELE	1987	83%	58%
BERSTERUS	1989	92%	90%
HILLBROW	1989	75%	70%
LAUDIUM	1989	97%	87%
ODI (12/12)	1989	81%	78%
PRETORIA	1989	95%	93%
DWT3 coverage less or equal to MEASLES coverage			
GELUKSPAN	1985	81%	81%
TAUNG	1985	65%	81%
BETHESDA (UBOMBO)	1985	58%	58%
GELUKSPAN	1985/86	83%	88%
INGWAVUMA (MOSVOLD)	1986	56%	56%
ALEXANDRA	1988	66%	67%
GELUKSPAN	1988	82%	84%
NSIKAZI	1988	50%	56%
BOTSHABELO	1989	31%	31%
KAYELITSHA (Site C)	1989	57%	63%
PRAKTISEER	1989	46%	48%

FIGURES

FIGURE.1 MEASLES – NOTIFIED DEATHS AS A PERCENTAGE OF CERTIFIED DEATHS

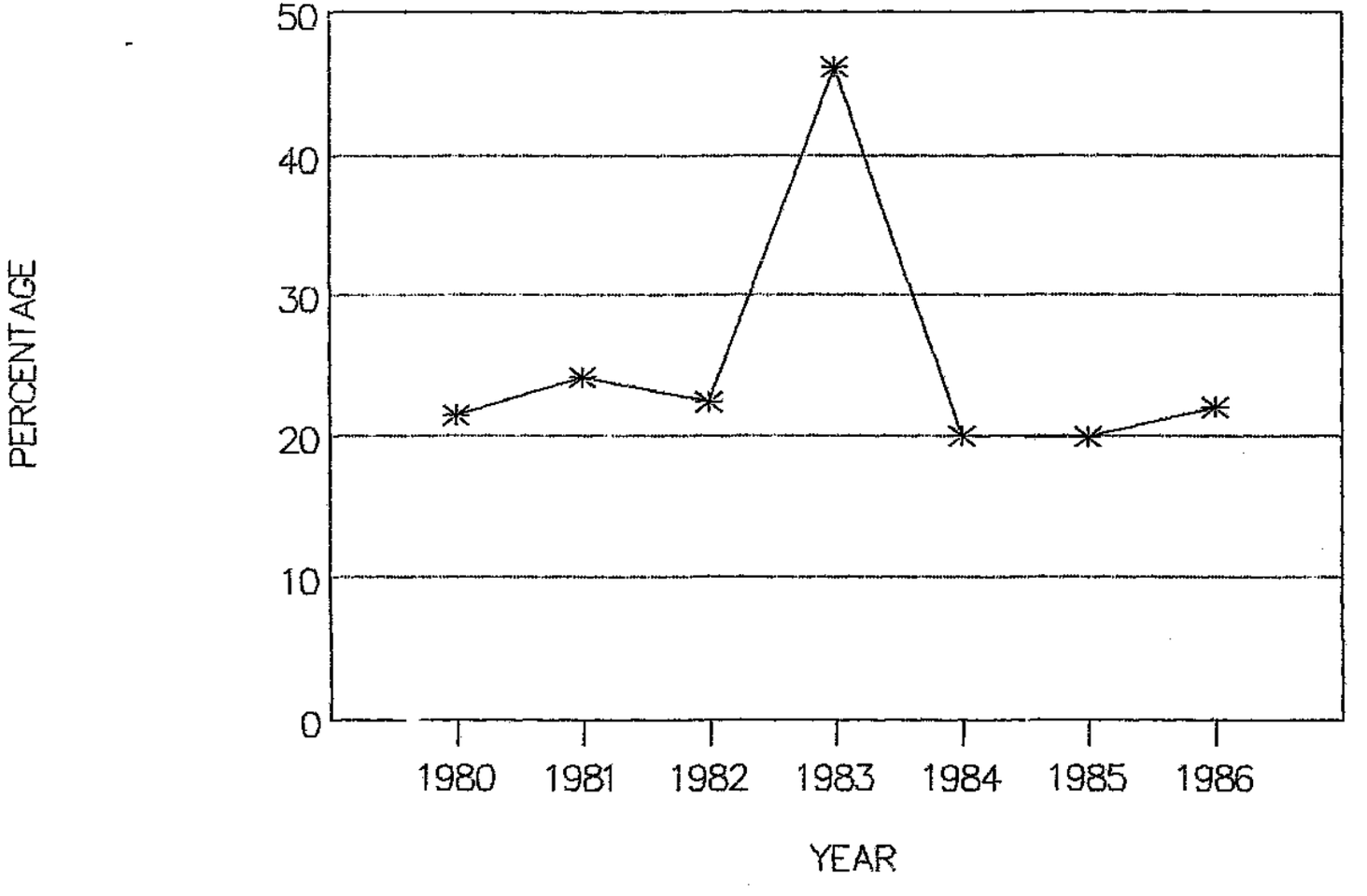


FIGURE. 2 NOTIFIED INCIDENCE OF MEASLES PER 100 000 POPULATION

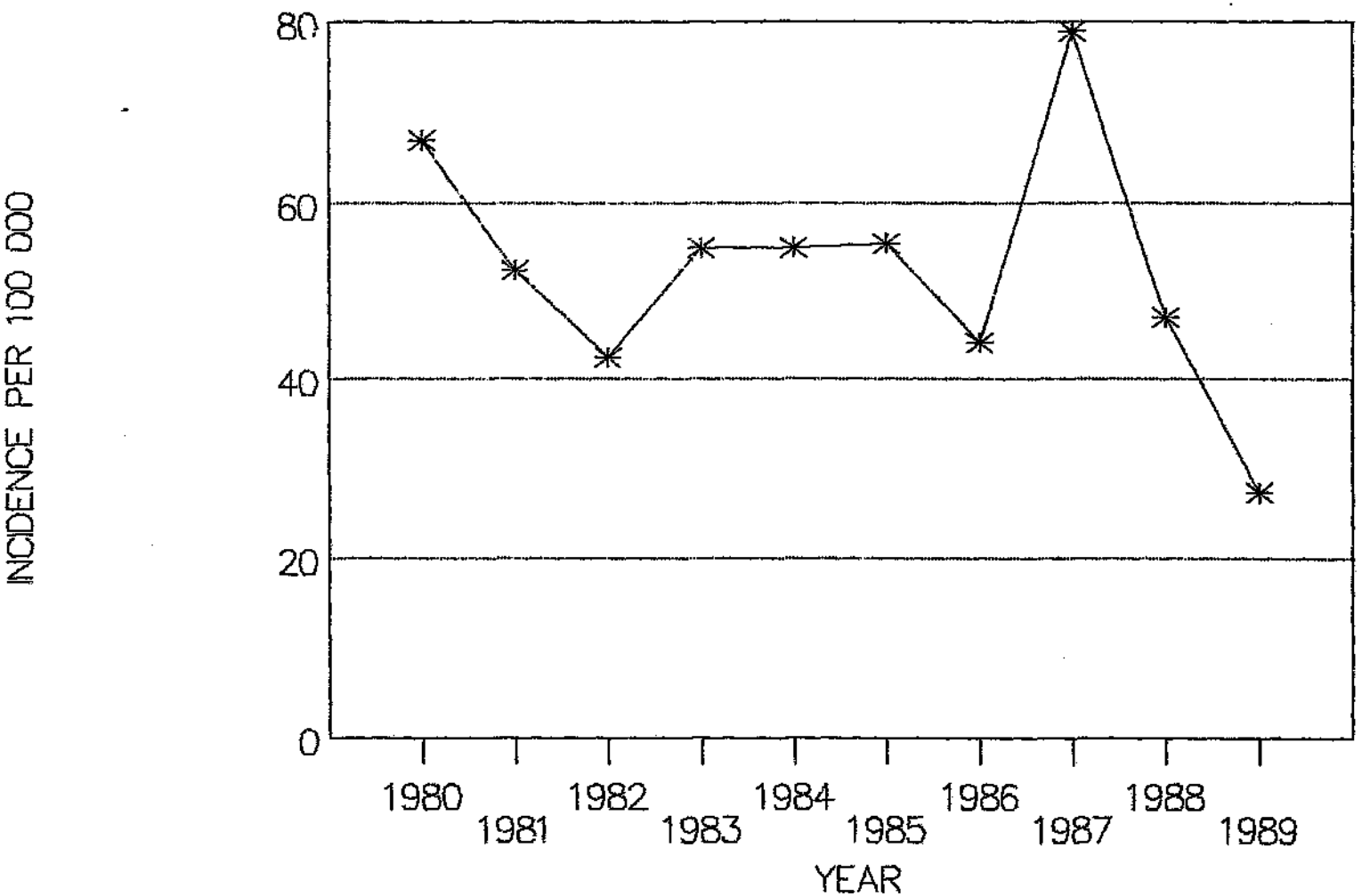


FIGURE. 3 MEASLES CASE FATALITY PER 100 MEASLES NOTIFICATIONS

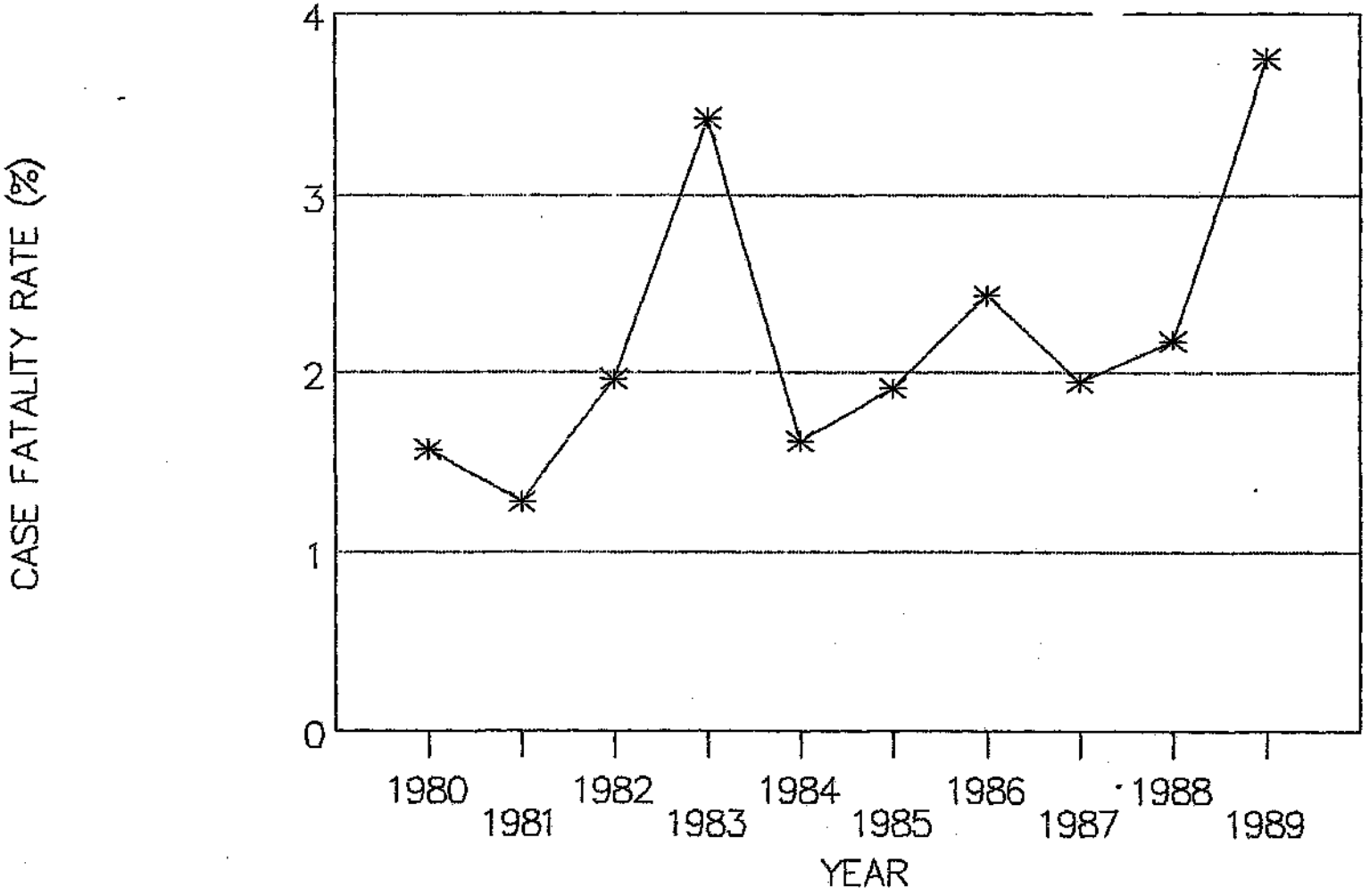


FIGURE.4 NOTIFIED MEASLES DEATHS PER 100 000 POPULATION

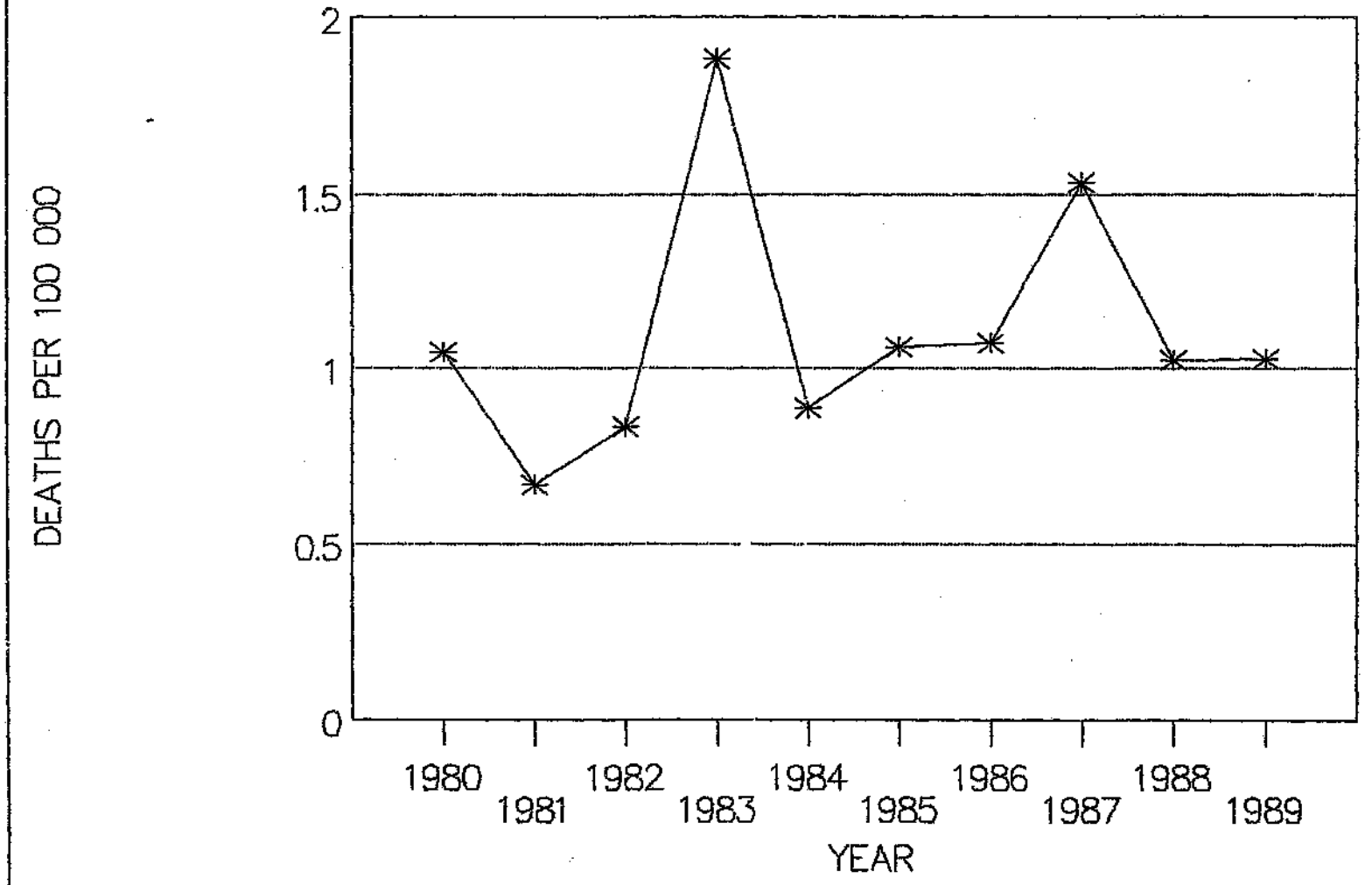


FIG. 5 CERTIFIED MEASLES DEATHS PER 100 000 POPULATION (ALL "RACES")

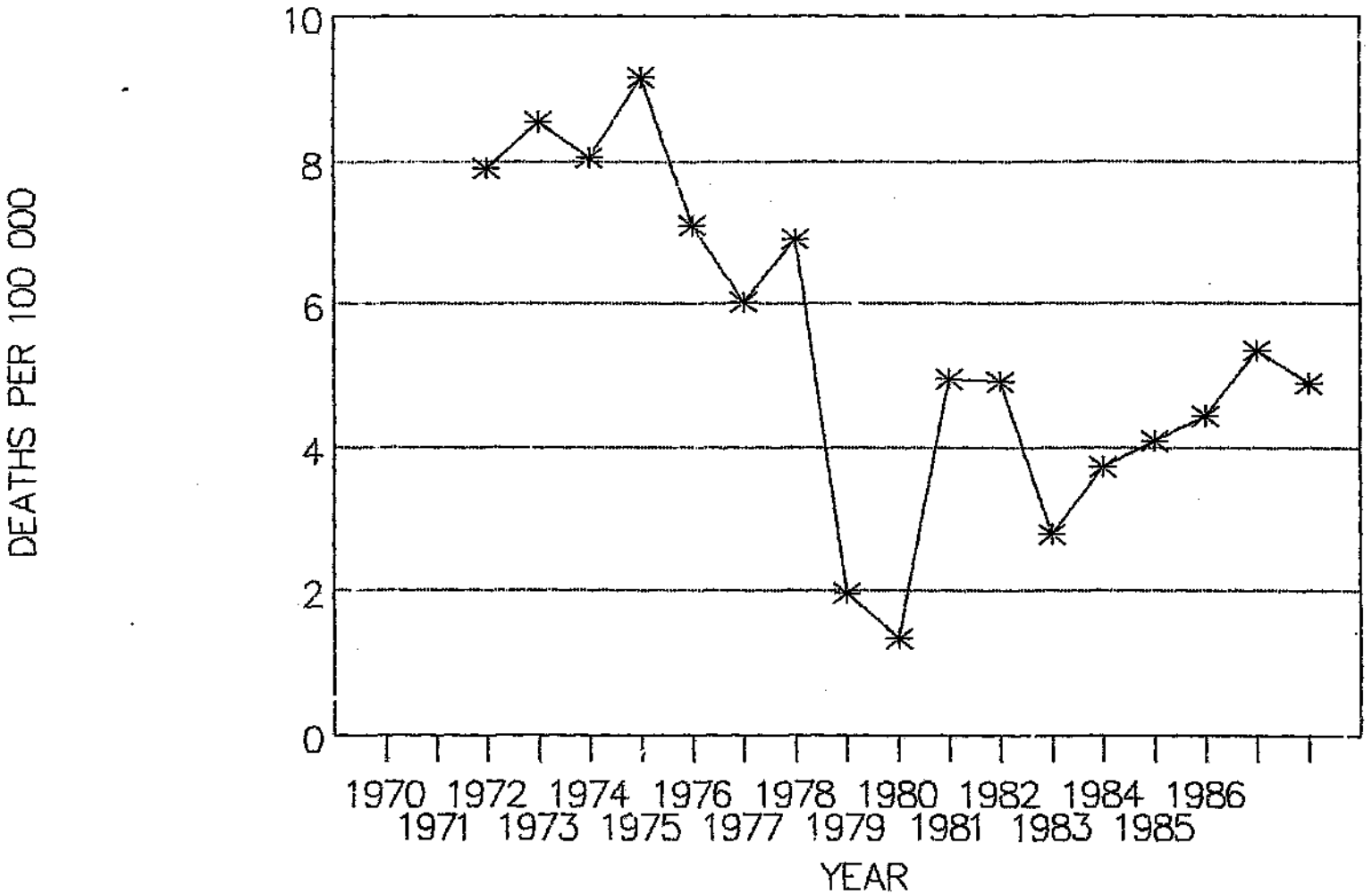


FIGURE.6 NUMBER OF CERTIFIED MEASLES DEATHS, 1968 – 1986

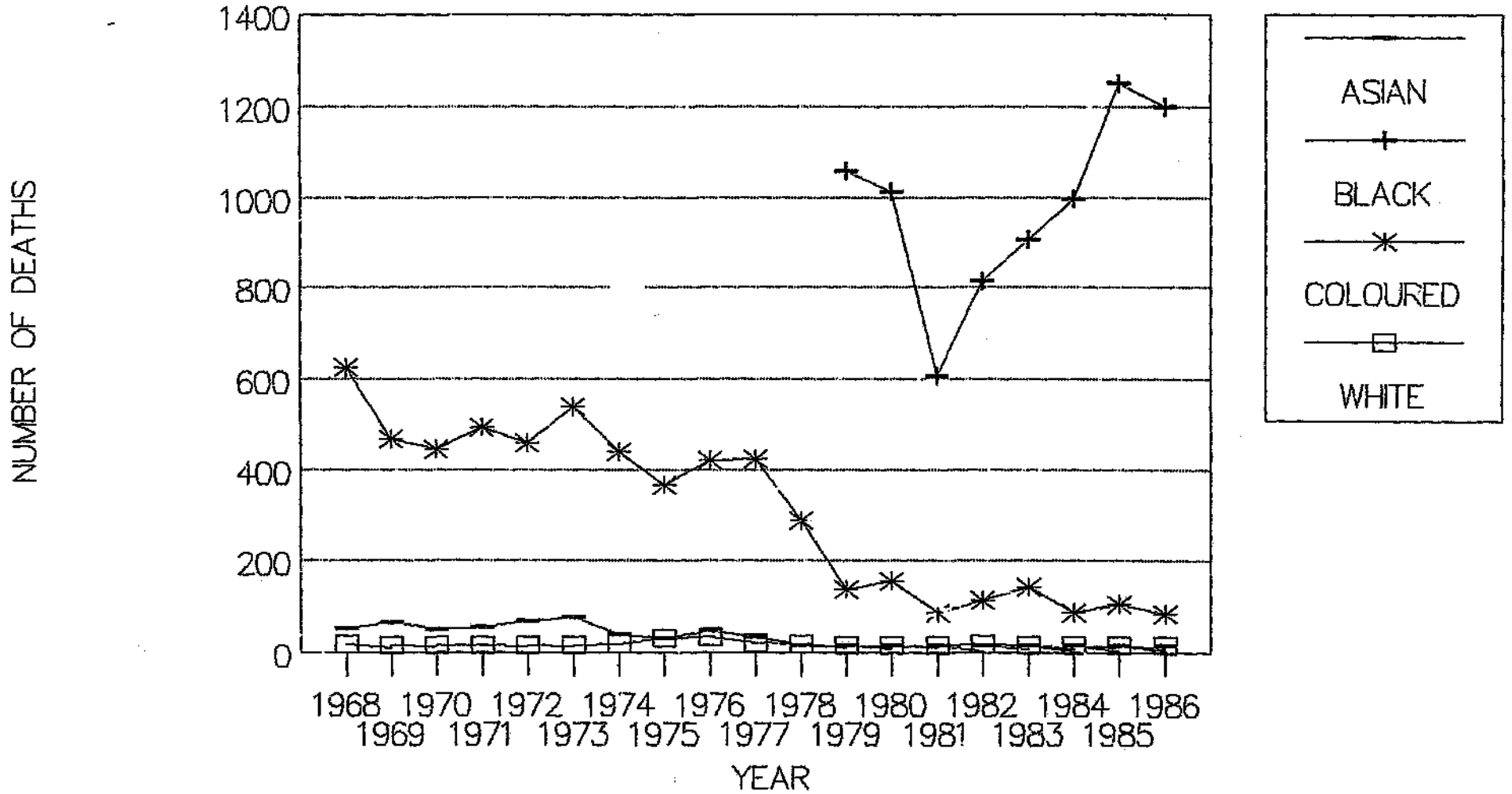
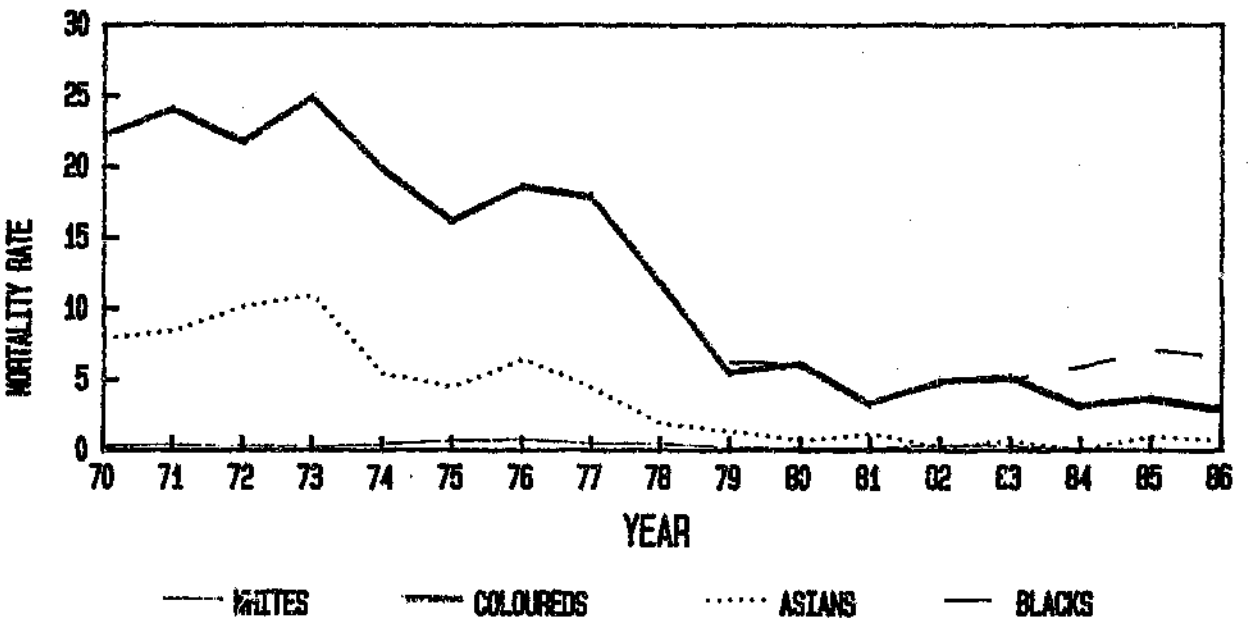
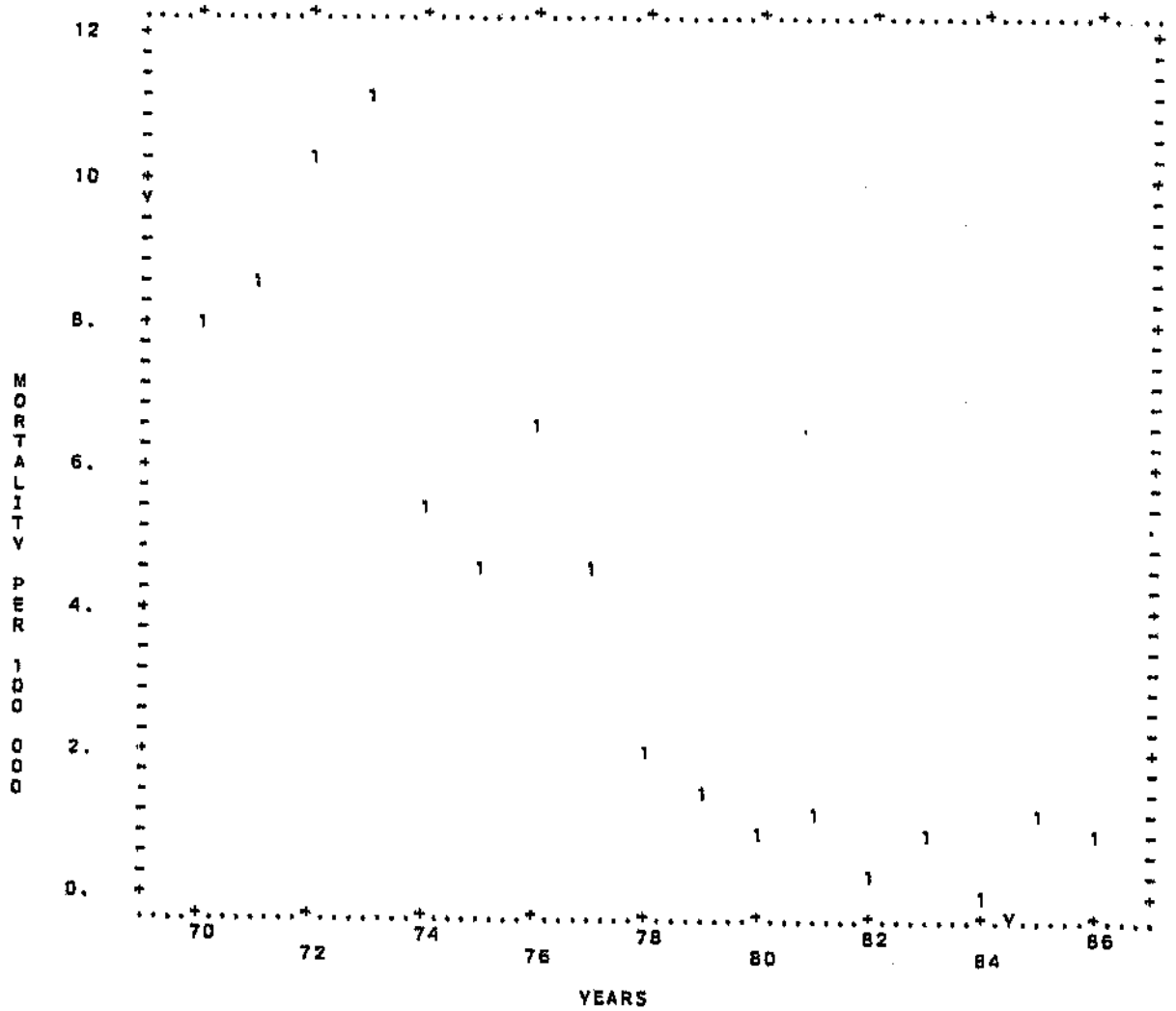


FIGURE. 7 POPULATION GROUP SPECIFIC CERTIFIED MEASLES DEATHS PER 100 000 POPULATION, 1970 – 1986



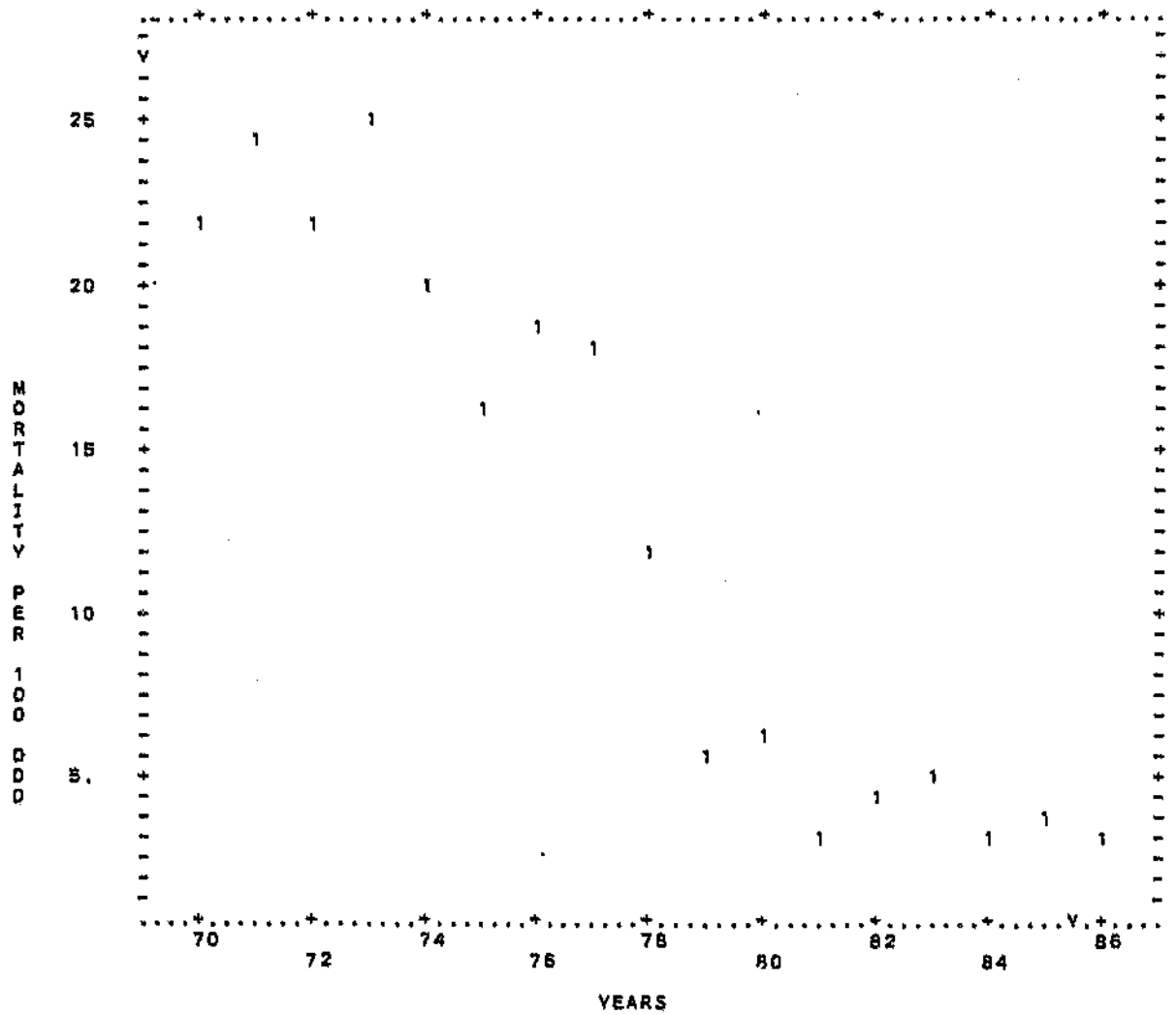
* 1970-1978 there is no data for BLACKS
TBVC countries are excluded

FIGURE 8 SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE ASIAN POPULATION, 1970 - 1986



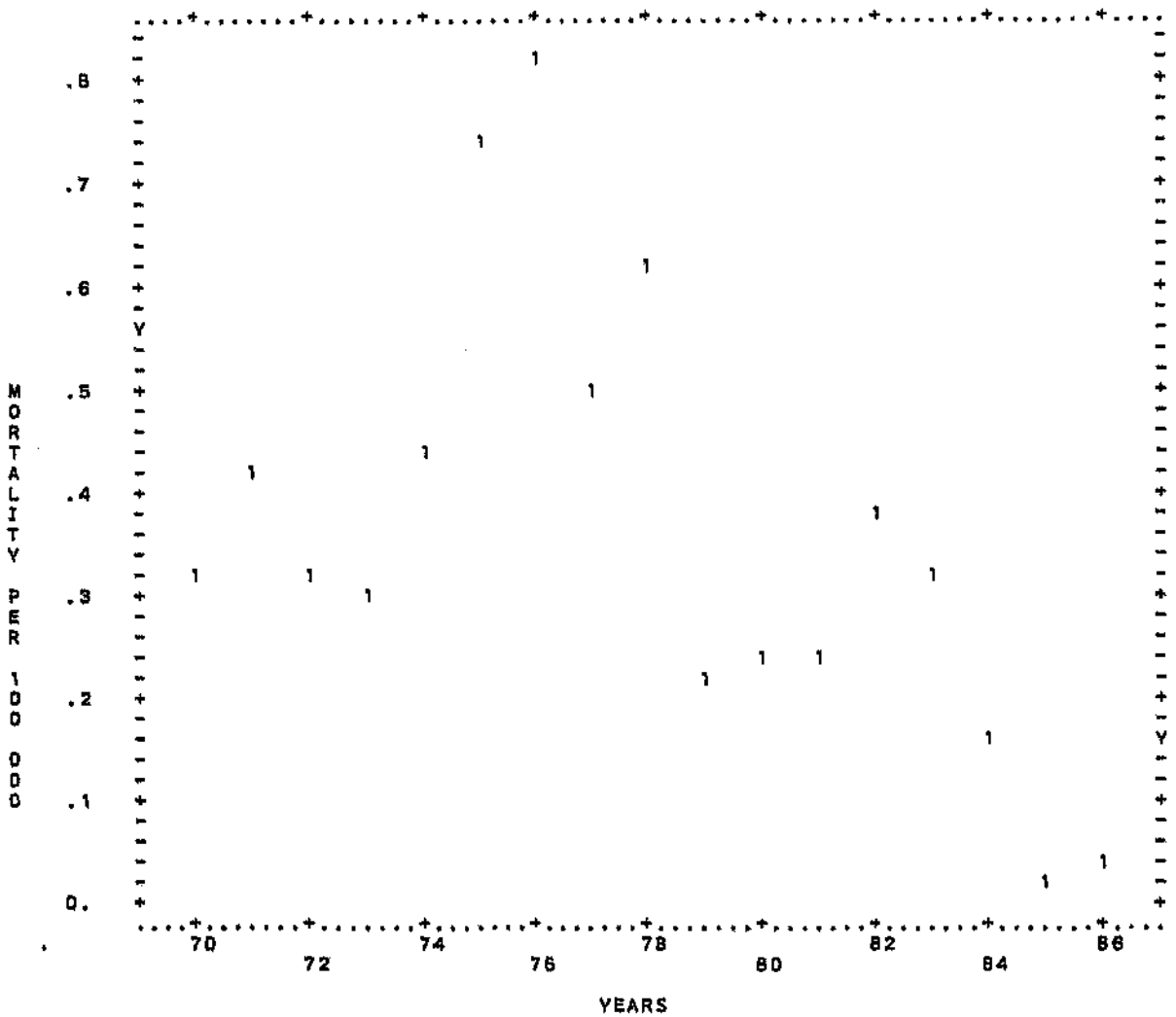
R =-0,890
P < 0,001
REGRESSION EQUATION Y = 54,848 - 0,55269*X

FIGURE 9 SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE COLOURED POPULATION, 1970 – 1985



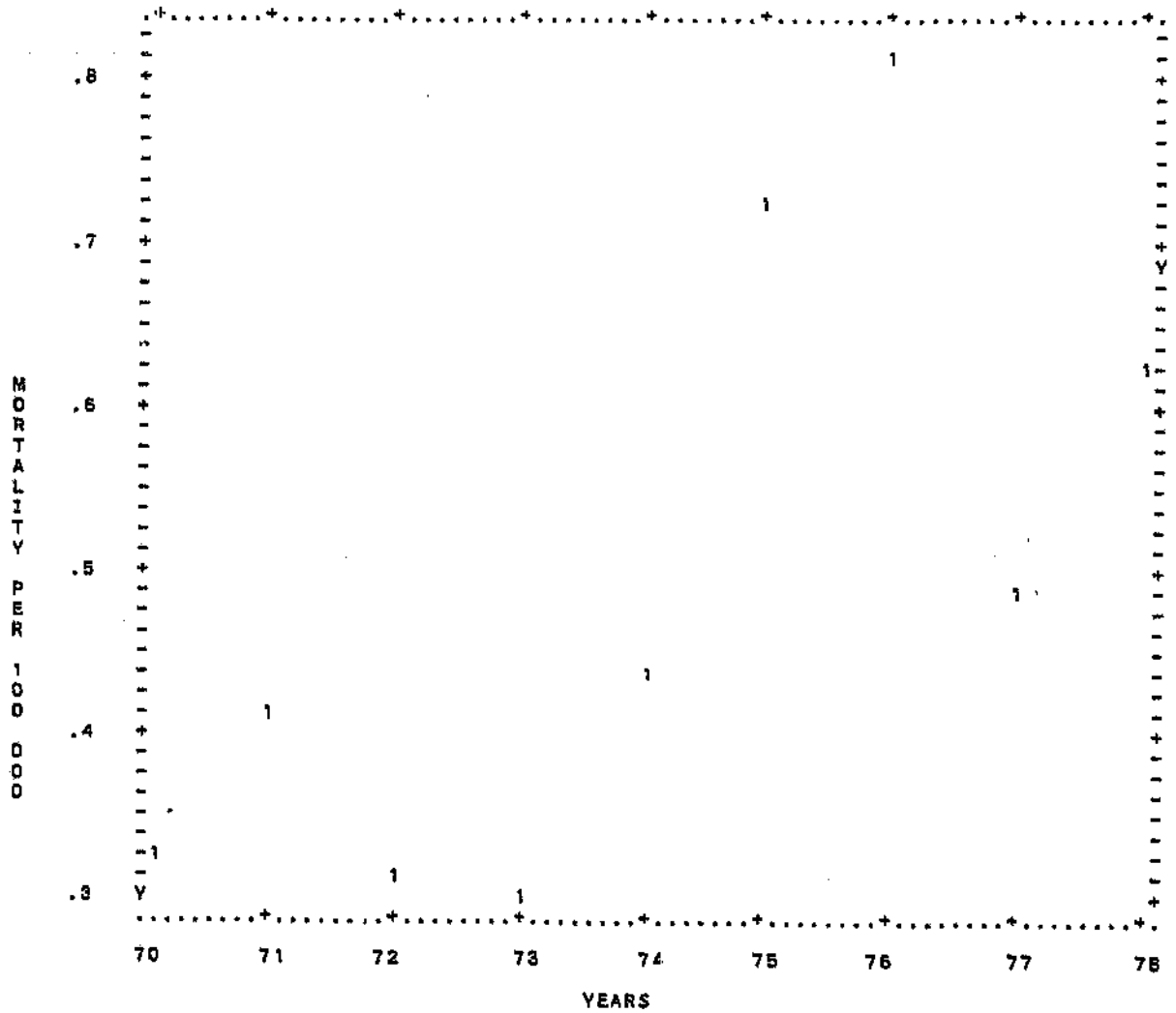
R = – 0,941
P < 0,001
REGRESSION EQUATION Y = 135,08 – 1,585 1X

FIGURE 10 SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE WHITE POPULATION, 1970 – 1985



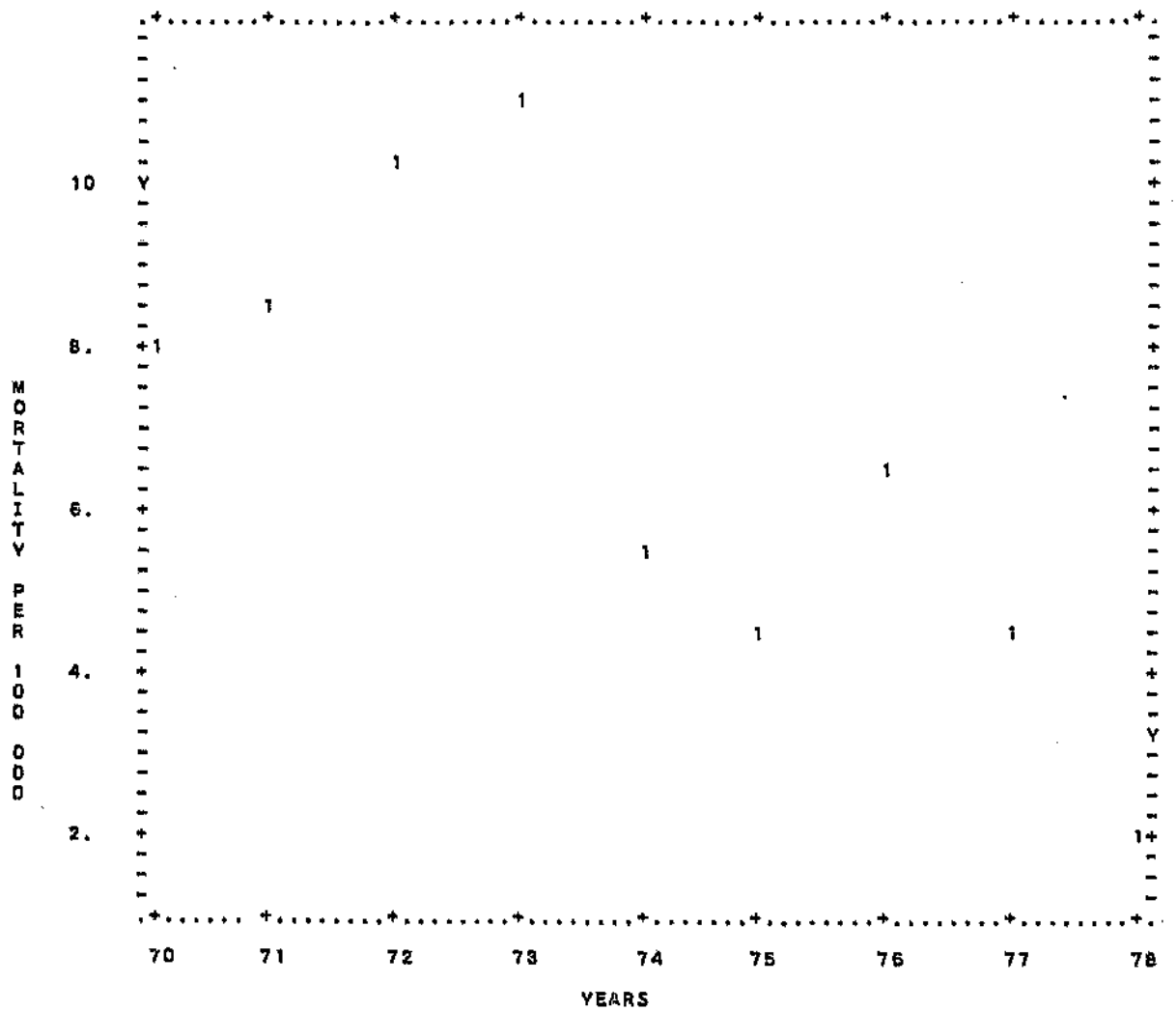
R = – 0,511
P = 0,035
REGRESSION EQUATION Y = 2,0544 – 0,0219XX

FIGURE 11 SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE WHITE POPULATION, 1970 – 1978



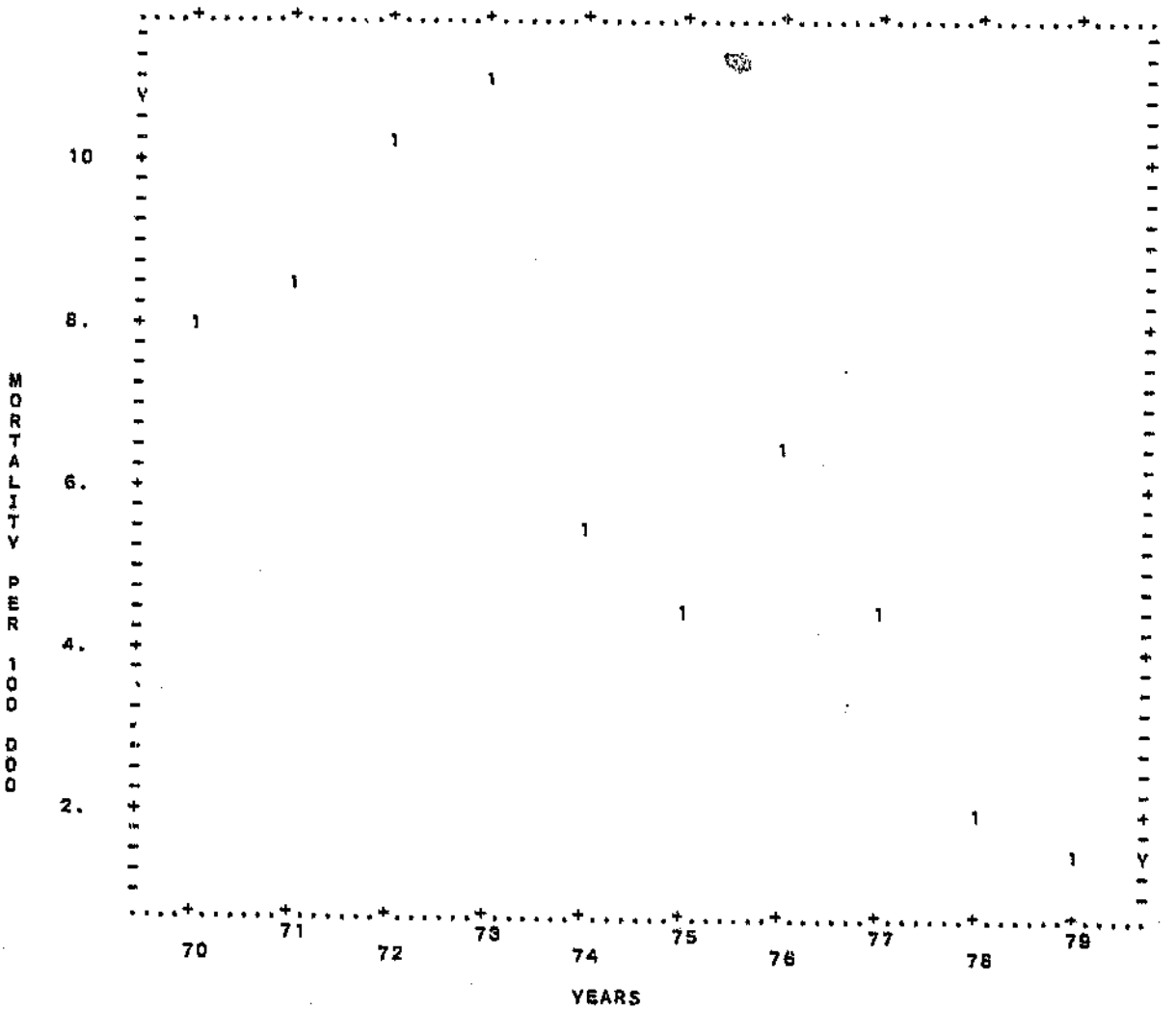
R = 0,595
P = 0,035
REGRESSION EQUATION Y = −3,0474 + 0,047833X

FIGURE 12(A) SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE ASIAN POPULATION, 1970 – 1978



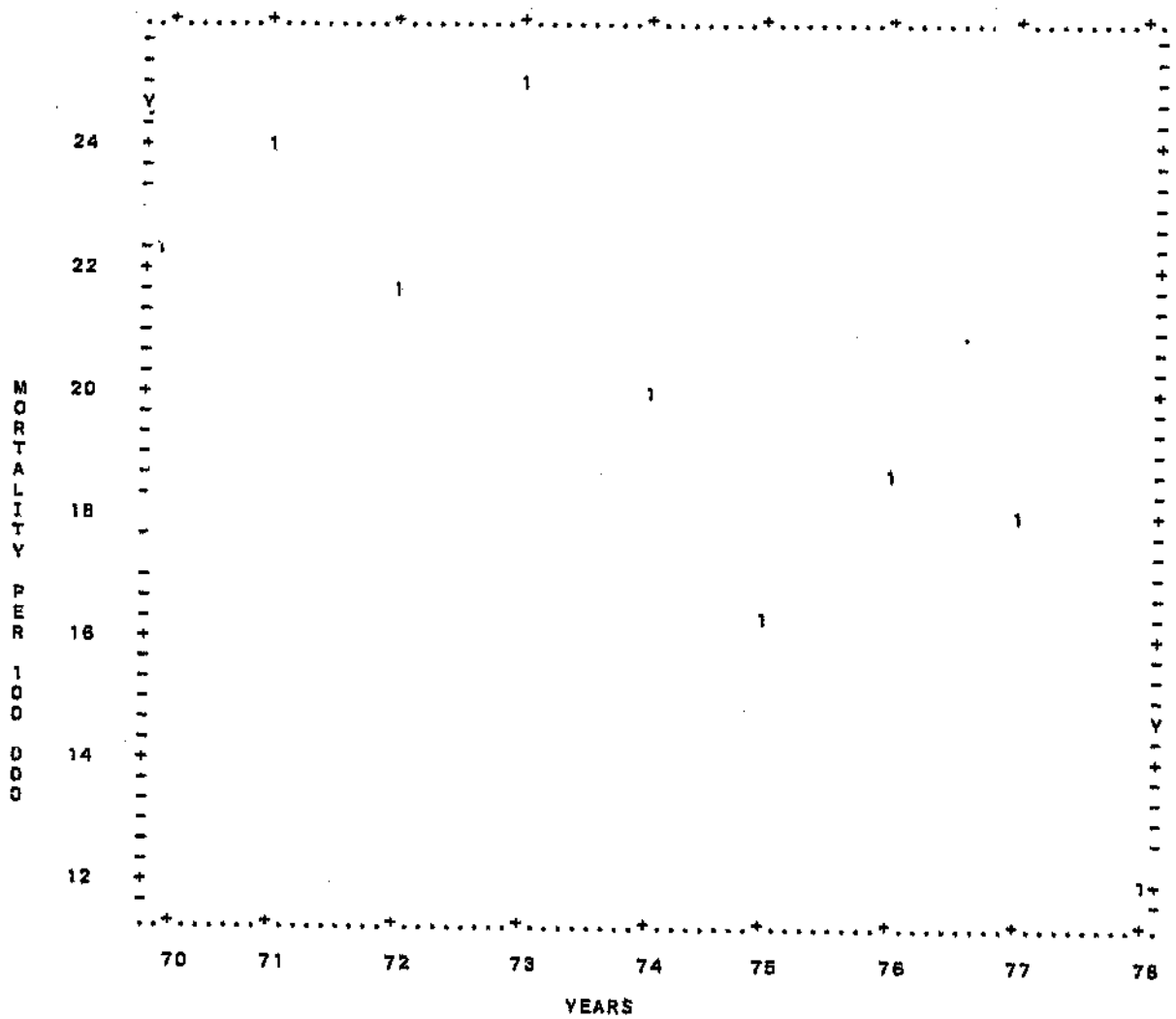
R = – 0,773
P = 0,012
REGRESSION EQUATION Y = 58,259 – 0,83150XX

FIGURE 12(B) SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE ASIAN POPULATION, 1970 – 1979



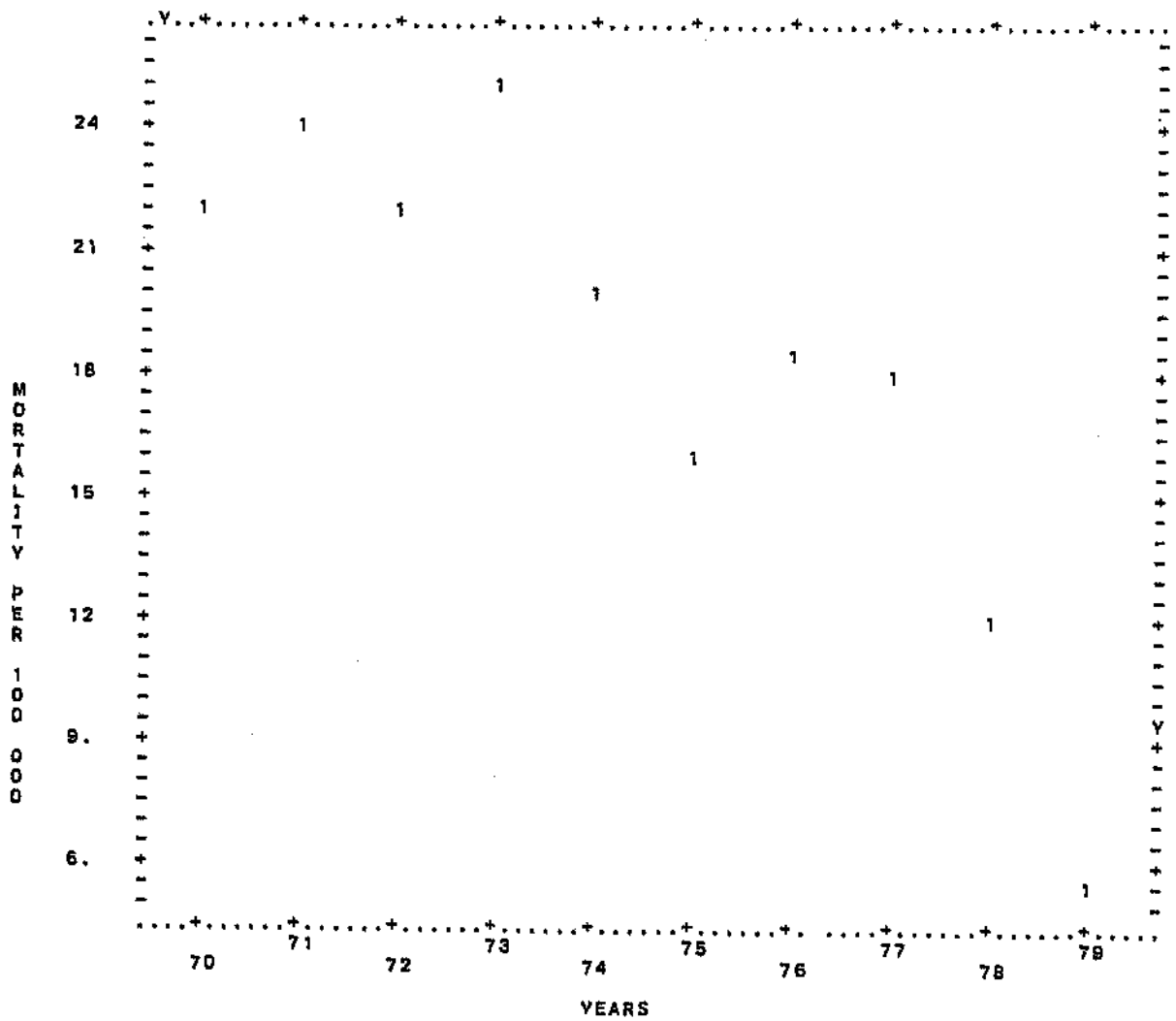
R = – 0,835
P = 0,001
REGRESSION EQUATION Y = 72,778 – 0,89870X

FIGURE 13(A) SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE COLOURED POPULATION, 1970 – 1978



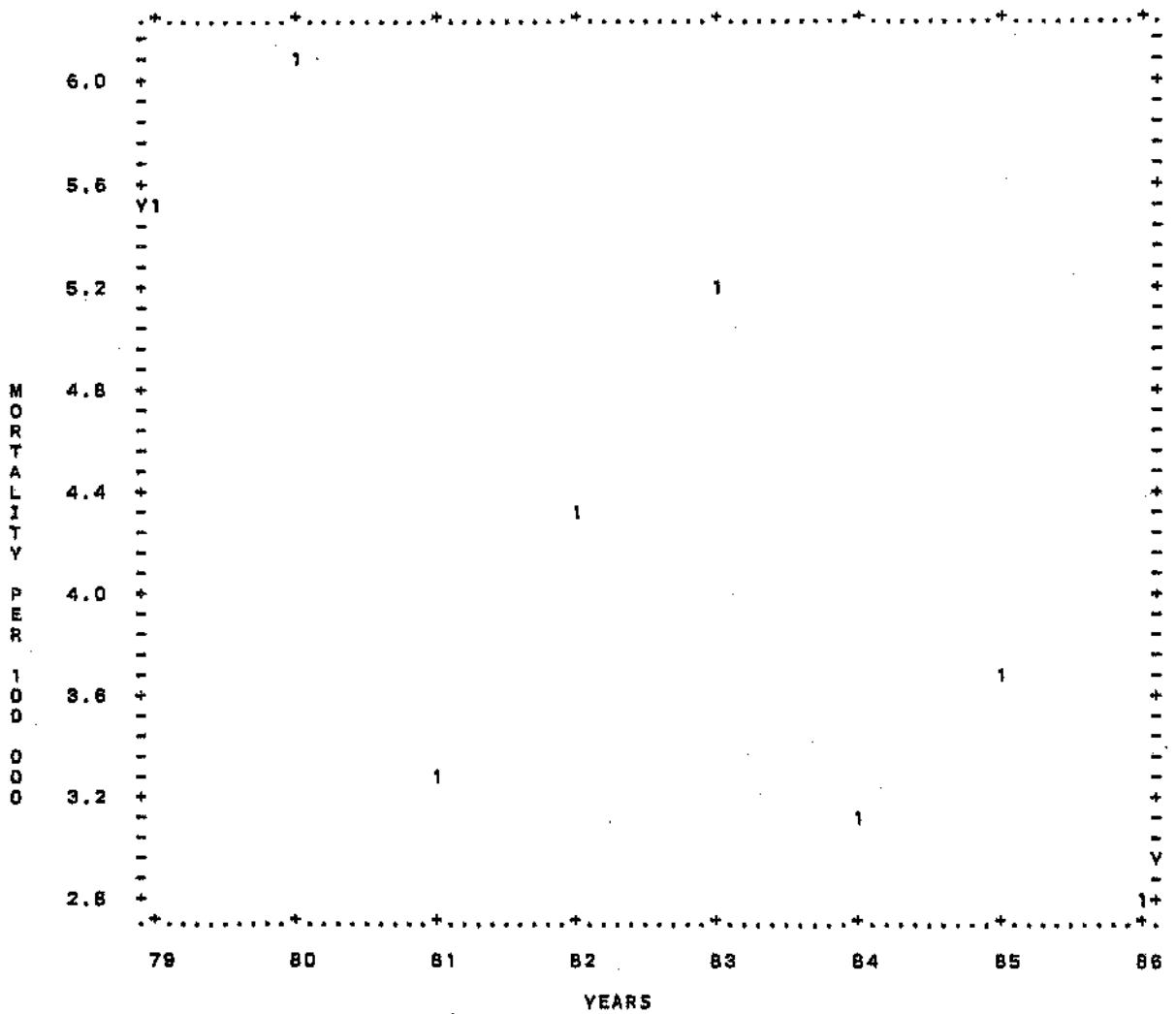
R = – 0,832
P = 0,005
REGRESSION EQUATION Y = 112,19 – 1,249XX

FIGURE 13(B) SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE COLOURED POPULATION, 1970 – 1979



R = – 0,859
P < 0,001
REGRESSION EQUATION Y = 143,84 – 1,852XX

FIGURE 14 SIMPLE LINEAR REGRESSION ANALYSIS OF CERTIFIED MEASLES DEATHS PER 100 000 OF THE COLOURED POPULATION, 1979 – 1986



R = – 0,727
P = 0,089
REGRESSION EQUATION Y = 33,941 – 0,35988XX

FIGURE 15. NOTIFIED MEASLES PER AGE AND POPULATION GROUPS, 1985–1987

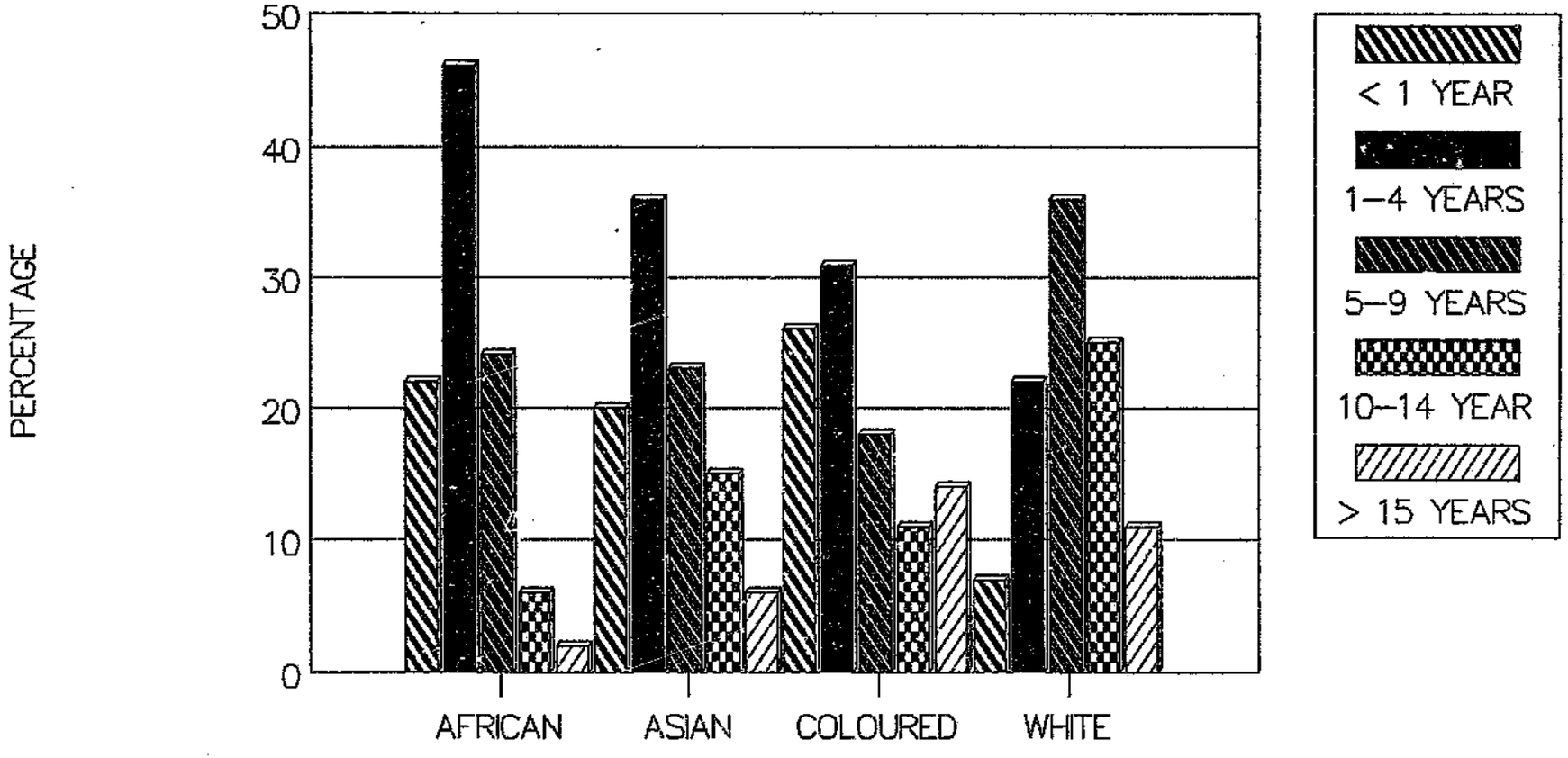
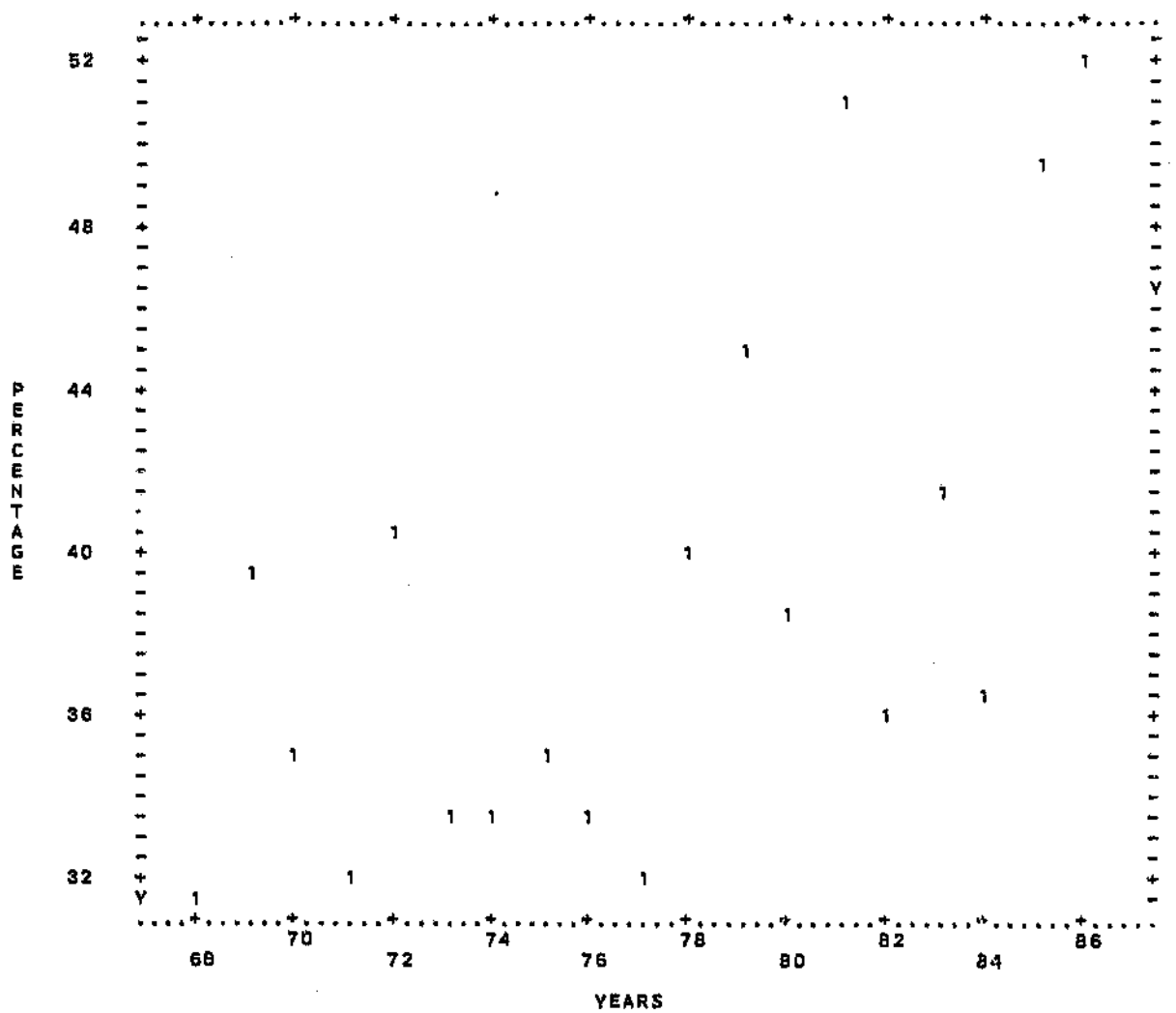
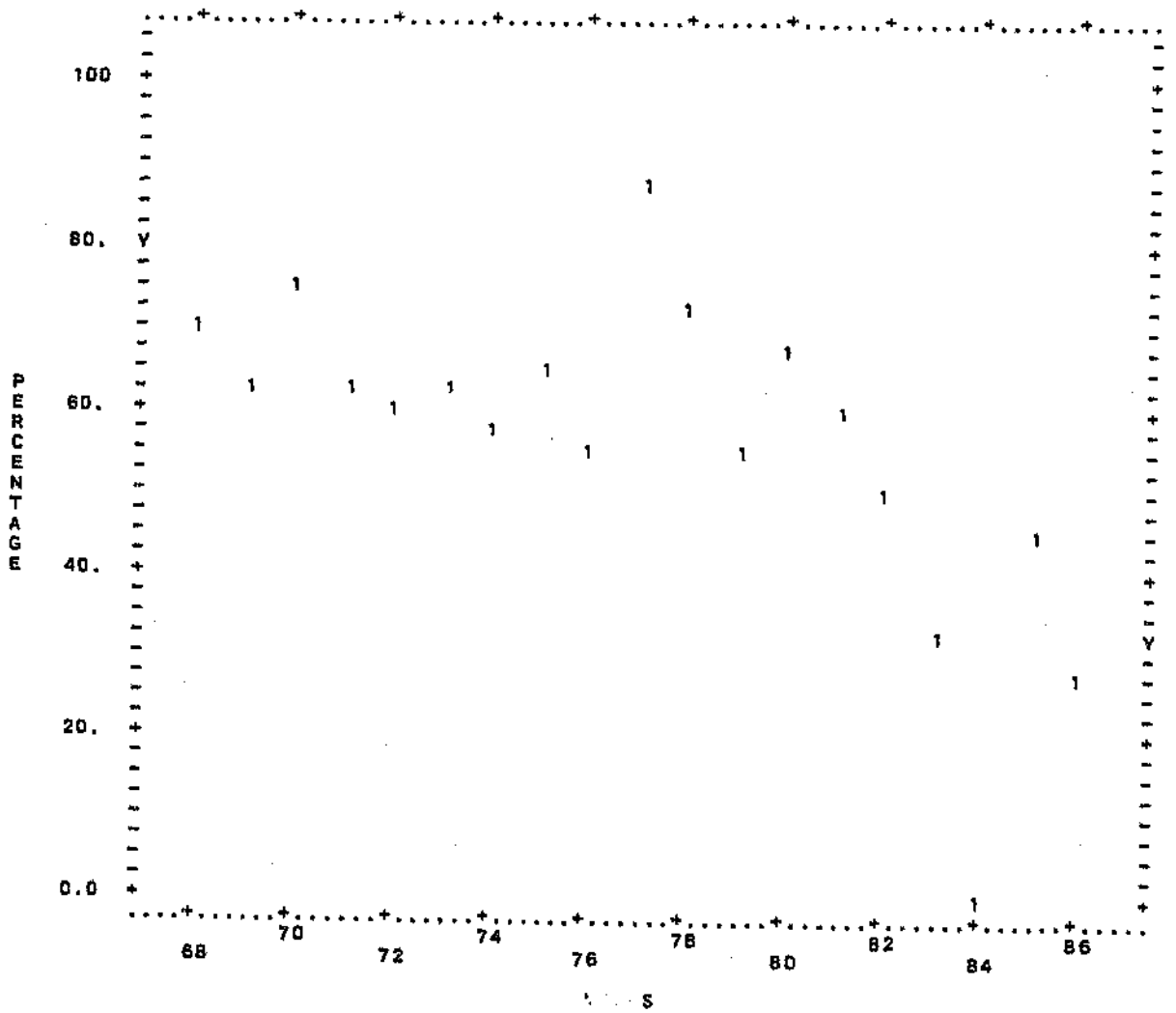


FIGURE 15 SIMPLE LINEAR REGRESSION ANALYSIS OF PERCENTAGE OF CERTIFIED MEASLES DEATHS AMONGST COLOUREDS, FOR INFANTS UNDER ONE YEAR, 1968 – 1986



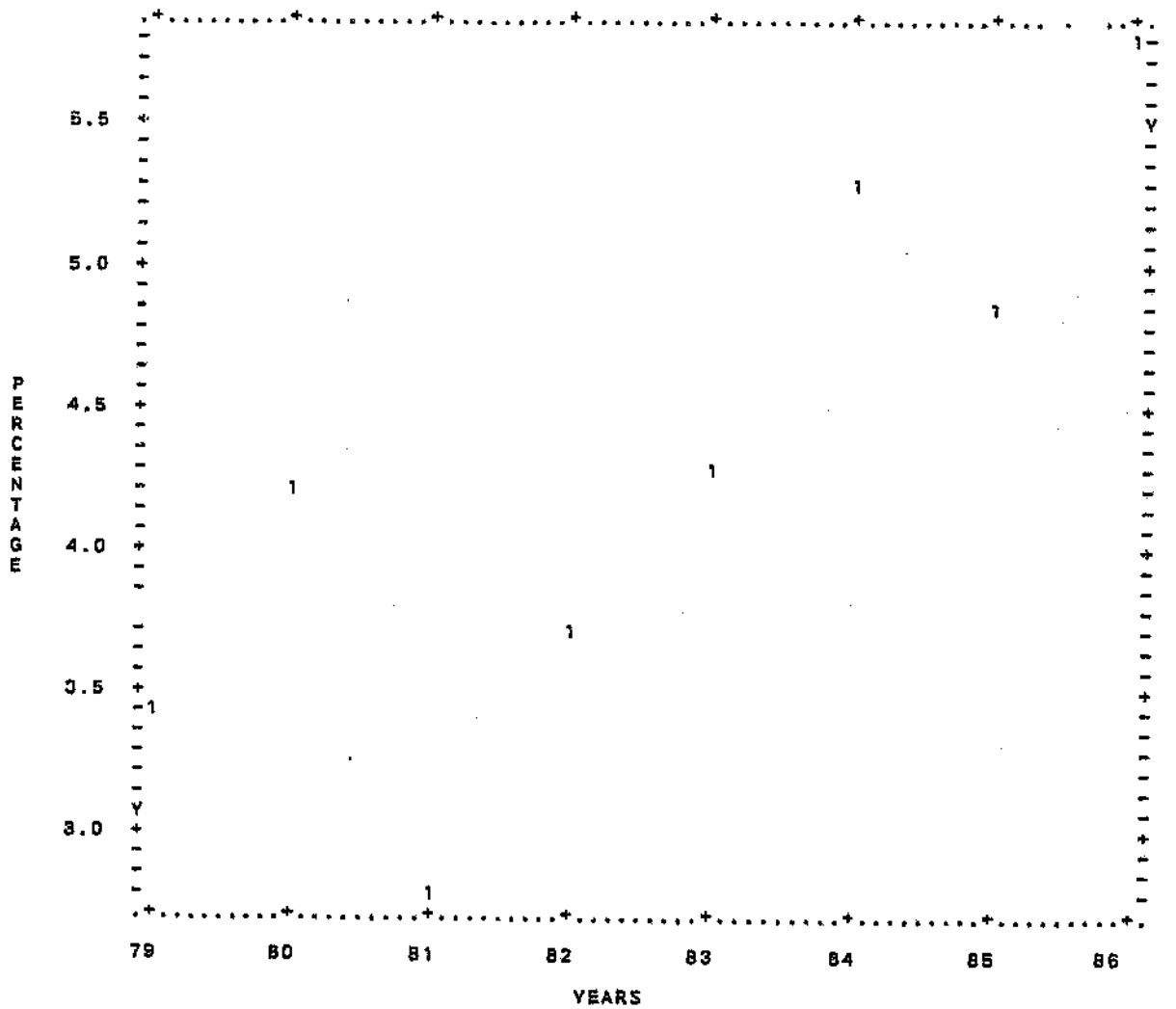
R = 0,640
P = 0,002
REGRESSION LINE Y = 18,050 + 0,737453X

FIGURE 17 SIMPLE LINEAR REGRESSION ANALYSIS OF PERCENTAGE OF CERTIFIED MEASLES DEATHS AMONGST ASIANS, FOR CHILDREN 1-4 YEARS, 1968 - 1986



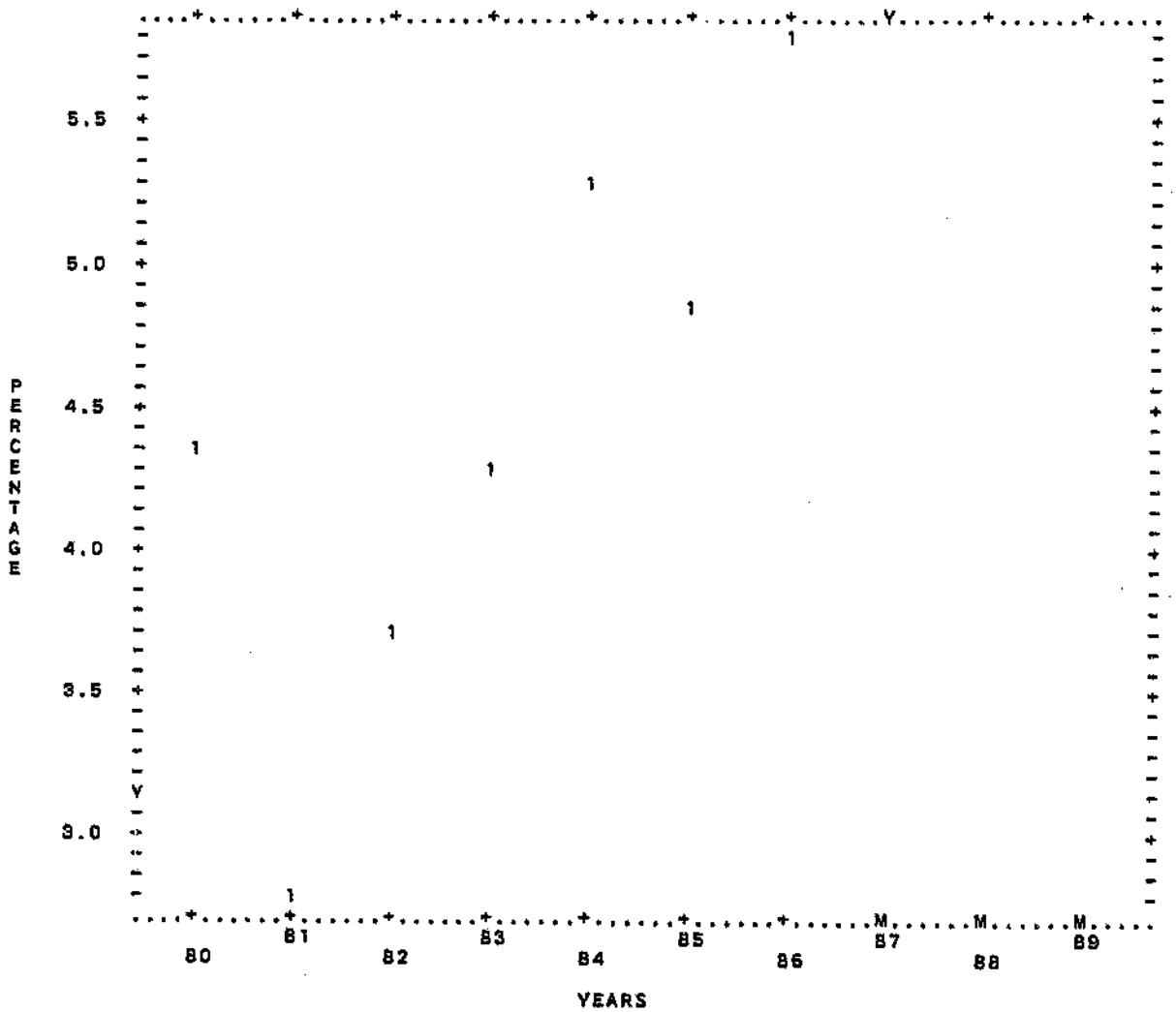
R = -0,540
P = 0,002
REGRESSION EQUATION Y= 227,35 -2,2208*X

FIGURE 18(A) SIMPLE LINEAR REGRESSION ANALYSIS OF PERCENTAGE OF CERTIFIED MEASLES DEATHS AMONGST BLACKS, FOR CHILDREN 5–9 YEARS, 1979 – 1986



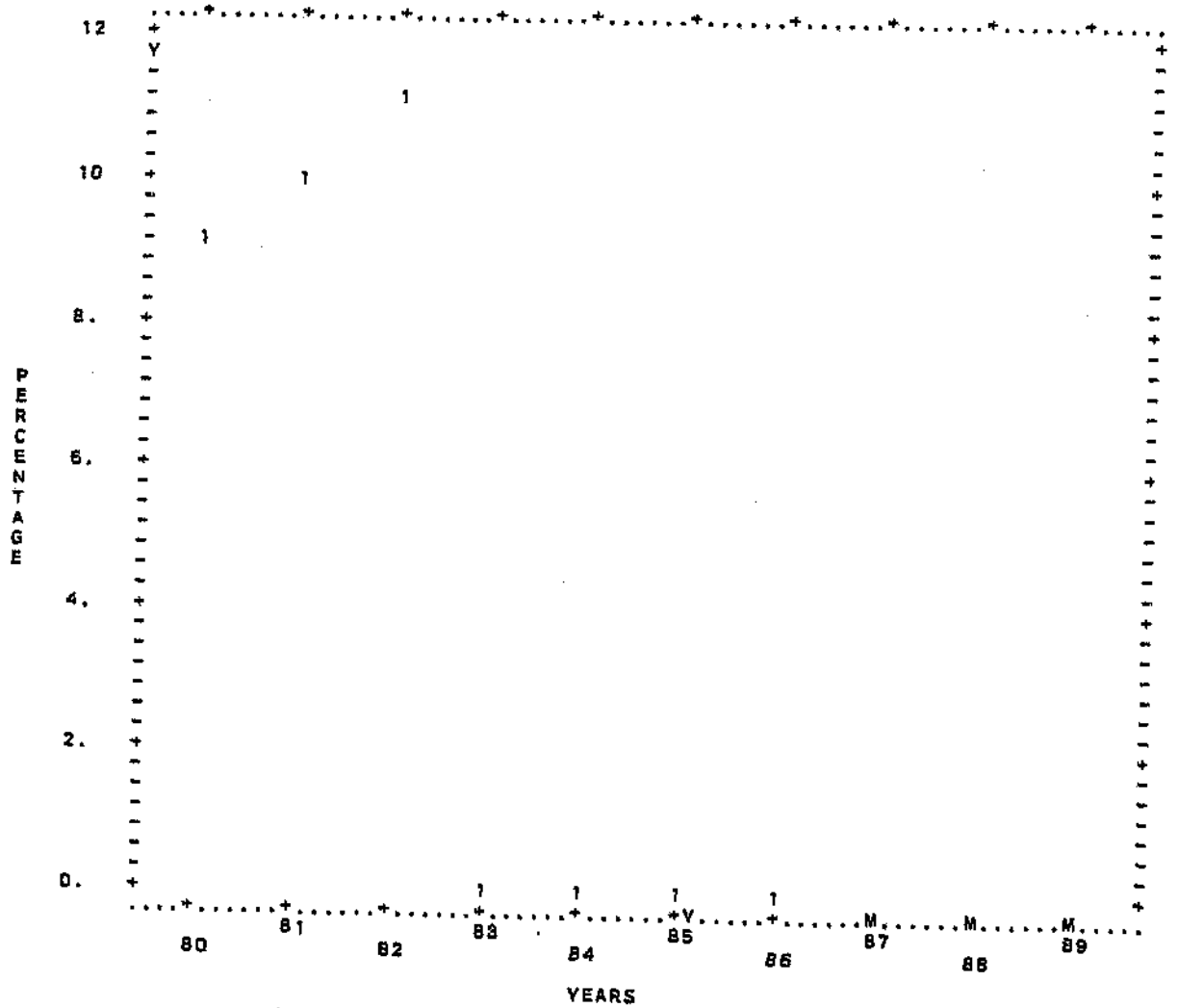
R = 0,821
P = 0,010
REGRESSION EQUATION Y = -23,107 + 0,33214*X

FIGURE 18(B) SIMPLE LINEAR REGRESSION ANALYSIS OF PERCENTAGE OF CERTIFIED MEASLES DEATHS AMONGST BLACKS, FOR CHILDREN 5–9 YEARS, 1980 – 1985



R = 0,778
P = 0,068
REGRESSION EQUATION Y = −25,170 + 0,35579XX

FIGURE 19 SIMPLE LINEAR REGRESSION ANALYSIS OF PERCENTAGE OF CERTIFIED MEASLES DEATHS AMONGST WHITES, FOR THE AGE GROUP 20 YEARS OR OLDER, 1980 – 1986



R = – 0,832
P = 0,017
REGRESSION EQUATION Y = 177,37 – 2,0850XX

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