THE PREVALENCE OF ASTHMA IN URBAN AND RURAL BLACK CHILDREN :

AN EPIDEMIOLOGICAL SURVEY

BY

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INDEX

Page

TABLES

Page

FIGURES

Page

Page

SECTION I

INTRODUCTION AND AIMS OF SURVEY

CHAPTER 1 : INTRODUCTION

Respiratory allergic disorders and asthma in particular appear to be a common problem in South Africa. Asthma in children is not a notifiable disorder and has a low fatality rate. It is therefore difficult to establish accurate statistical figures on its true incidence in South Africa.

The incidence or prevalence of asthma in the Black child in South Africa is not known. Wesley, Clyde and Wallace[1] from Durban thought it to be very low, especially in the rural Black population. Shore[2] from Cape Town found a relatively high incidence of 7% in White South African children. Without substantiating information, Ordman[3] from Johannesburg suggested that if the incidence of respiratory allergy in the White population is taken as a standard, the Coloured group has approximately the same incidence. In his opinion, the incidence of asthma in the Black population, and especially the rural Black, was low.

At the Red Cross War Memorial Children's Hospital 228 802 out-patients were seen during 1975. Of these children 20 495 (9,0%) were White, 169 975 (74,3%) were Coloured and 38 342 (16,8%) were Black. Children are only seen at this hospital up to the age of 13 years. Of 3 515 asthmatic children attending the Allergy Clinic, only 13 were Black (0,36%).

This low attendance is out of all proportion to the total number of Black children who attend the out-patients department of this hospital. Only 5 deaths due to asthma out of a total of 2 478 deaths or 5 deaths from 977 980 patient attendances occurred at this hospital during the 5-year period, 1972 - 1976. It is evident that prevalence rates based only on hospital statistics, especially in Black children, could easily result in erroneous conclusions.

The striking observation from Africa, India and tropical countries is that childhood asthma is almost non-existent. Asthma has mainly been studied in adults where the age of onset is in the second or third decade of life.

In contrast, studies from the United Kingdom, United States of America and Australia report an average prevalence rate of between 2 - 6,37%. The age of onset of asthma is usually before the age of 6 years.

Is this disparity between prevalence rates for asthma between children from industrialised western countries and rural less industrialised countries real ? Does it represent a difference in sensitivity to allergic stimuli or a difference in degree and timing of allergenic bombardment to differing lifestyles ?

Seftel[4] recently made a plea for epidemiological research aimed at replacing impressions and suppositions by reliable incidence and prevalence rates for Black populations in urban and rural regions.

The prevalence rates of childhood asthma reported by different investigators

in different populations and different geographic areas vary widely. These variations may represent real differences or they may reflect terminological and methodological differences. The failure to establish or adhere to well-defined criteria, and the inability to define asthma, has impeded epidemiological studies on asthma. Unfortunately there are no clinical pathognomonic or laboratory findings specific for childhood asthma.

The factors responsible for the development of an allergic disorder eg. asthma, have not yet been clearly established. Dietary factors have been incriminated as the major offenders especially early feeding on cow's milk, eggs and wheat. The possible prophylactic effect offered by breastmilk to potentially allergic infants has been suggested. It has also been postulated that the main defect might be a transient IgA deficiency during early infancy which might result in the development of a sensitised population of IgE producing plasma cells following antigen contact.

In an attempt to determine the prevalence of asthma as well as possible responsible factors, I therefore set out to conduct an epidemiological survey amongst representative samples of Black children living in an urban and a rural community.

1. Wesley, A.G., Clyde, J.H.& Wallace, H.L. (1969) Asthma in Durban Children of Three Racial Groups. S.Afr.Med.J., 43, 87-89.

2. Shore, S.C. (1959) Pre-asthma. S.Afr.Med.J., 33, 362.

3. Ordman, D. (1964) The Regional Aspects of Respiratory Allergy in South Africa. S.Afr.Med.J., 38, 369-372.

4. Seftel, H.C. (1977) Diseases in Urban and Rural Black Populations. S.Afr.Med.J., 51, 121-123.

CHAPTER 2 : AIMS OF SURVEY

This survey was undertaken :

1. to provide accurate information on the prevalence rates of asthma in Xhosa children living in an urban westernised community, Guguletu, Cape Town and in a rural traditional community, Tsolo District, Republic of Transkei;
2. to compare the findings of this survey with studies done elsewhere in the world on prevalence rates of asthma among children; and
3. to evaluate and correlate factors that may influence these rates.

To acquire the above, it was necessary :

1. to obtain information on the characteristics and socio-economic status of the family of each child studied;
2. to obtain information on every child relating to exposure to allergens in his immediate home environment;
3. to obtain information on the dietary pattern of every child during infancy and currently;
4. to identify the asthmatic children by subjecting every child to an appropriate exercise stimulus for detection of exercise-induced asthma judged by pulmonary function tests;
5. to conduct a clinical examination of the children. Height, weight and skinfold thickness was also measured;
6. to conduct prick skin testing to assess specific skin sensitivity to common allergens;
7. to conduct laboratory investigations relating to the allergic-immune status of the children;
8. arising out of these findings to relate the prevalence rates of asthma in the 2 samples and to suggest factors influencing the rates.

CHAPTER 3 : TERMINOLOGY

Unless otherwise stated, the terms used throughout this thesis are defined as follows:

ASTHMA is used to signify:

1. the disorder in childhood, as only children from 6 to 9 years were studied
2. a symptom complex in which there are variable paroxysmal bronchial airways obstruction of an episodic and intermittent nature. This bronchial airways obstruction is reversible either spontaneously or by therapeutic means
3. allergic or extrinsic asthma

RATES

The frequency of a disease in a population can be measured or expressed in terms of a rate - incidence or prevalence.

(a) Incidence is defined as "to occur. An expression of the rate at which a certain event occurs, as a number of new cases of a specific disease occurring during a certain period" (Dorland's Illustrated Medical Dictionary, 25th Edition) or "as the number of new cases of a disease in a specific population at risk in a given period of time" (Gordis, 1973).

(b) Prevalence is defined as "the total number of cases of a disease in existence at a certain time in a designated area" (Dorland's Illustrated Medical Dictionary, 25th Edition) or as "the number of cases of a disease existing in a population at risk at a given

point in time" should be well circumscribed to indicate whether a study deals with point prevalence (that is, on a specific date) or period prevalence (that is, during a specific period) or cumulative prevalence (that is, a history of asthma, eg. did your child ever have asthma?)

In this study point prevalence for asthma was determined. When prevalence is used in reference to the samples comprising this study, it will mean point prevalence.

REPUBLIC OF TRANSKEI

At the time of the study, Tsolo District, Transkei was part of the Republic of South Africa with its own government. It has subsequently become an independent state; the Republic of Transkei. The term Republic of Transkei will be used throughout this thesis.

ETHNIC

Terms "Bantu" and "Xhosa" are used in reference to other authors' works. The children under study will be referred to as "Blacks". Bantu and Xhosa people are referred to as "Blacks".

SECTION II

REVIEW OF LITERATURE

CHAPTER 1 : THE PREVALENCE OF CHILDHOOD ASTHMA

A large number of epidemiological surveys have been made in many parts of the world in an attempt to establish the prevalence of asthma. Unfortunately the findings of many of these studies have not always led to a better understanding of the complex problem of this disorder.

The prevalence rates for asthma in children living in westernised, industrialised societies are in striking contrast to those found in children from tropical and less developed societies. Childhood asthma is uncommon in African countries (Johansson, Mellbin & Vahlquist, 1968; Wasunna, 1968; Mitchell, 1970; Godfrey, 1975; Warrell et al, 1975), but asthma is a frequent disease in adults and constitutes a major load on the health services of these countries. The age of onset of asthmatic symptoms was in adults and was usually in the second or third decade (Rees et al, 1974; Cookson & Makoni, 1975; Warrell et al, 1975).

Low frequency rates for asthma are not only confined to children in Africa, but are similar in children living in the area of Patna, India (Viswanathan et al, 1965 and 1966) and in Barbados (Pearson, 1973). Anderson (1974) found childhood asthma to be exceedingly rare in the rural areas of New Guinea.

Tables II.1 to 5 are summaries of studies on the prevalence of asthma in children, grouped under the heading of the country where the study was undertaken.

1. THE PREVALENCE OF ASTHMA IN SOUTH AFRICA (TABLE II.1)

Very few epidemiological studies of childhood asthma have been done in this country. Shore presented a paper at the 3rd Congress of the S.A. Paediatric Association in October, 1958, entitled "Pre-Asthma", of which an abstract was published (Shore, 1959). He estimated the incidence of asthma in the Cape Province to be at least 7% with 70% of children with major allergic disease having a positive family history of allergy. This study was of white children. It was based on the medical records of approximately 70% of all Jewish children (some 3300) born in the Cape Province in the 7 years prior to the study, as well as 600 medical records of children belonging to 200 sequential families of policemen. All were seen in a private referral paediatric practice in Cape Town (Shore, personal communication, 1977). The only valid deduction that can be made from this study is that at least 7% of the children studied in that particular practice suffered from asthma. These findings cannot be extrapolated to the general childhood population.

Wesley, Clyde & Wallace (1969) studied asthmatic children from three racial groups in Durban, their findings being based on hospital admissions over a period of 5 years. As this study represented only hospital admissions, the significance in terms of the prevalence of asthma in the general childhood population is limited. Any sample based on hospital admissions only, tends merely to reflect the more severe cases. This is especially relevant to this study where the authors stated that "Due to the scarcity of non-White beds, criteria for admission to the non-White hospital were more stringent than for the White hospital". The striking

TABLE II.1 THE PREVALENCE OF ASTHMA IN SOUTH AFRICA, INDIA, TANZANIA AND TROPICAL COUNTRIES

AUTHOR	AREA	POPULATION	AGES	PREVALENCE
Shore (1959)	Cape Province Rep.of S.A.	Not stated	Not stated	7% (incidence)
Wesley, Clyde & Wallace (1969)	Durban Rep.of S.A.	33 417 Hospital admissions	Up to 13 years	
		24 731 Bantu		Bantu 0,02% of hospital admissions
		3 373 Indian		Indian 0,77% of hospital admissions
		5 313 White		White 0,79% of hospital admissions
Viswanathan et al (1965)	Patna Urban & Rural India	Rural: 236 children	0-9 years	Rural 0-9 yrs = 0%
		351 children	10-29 years	10-29 yrs = 1,6%
		Urban: 286 children	0-9 years	Urban 0-9 yrs = 0,35%
		374 children	10-29 years	10-29 yrs = 0,53%
				Overall 0,64%
Viswanathan et al (1966)	Patna Urban India	4 413 children	0-9 years	0-9 yrs = 0,18%
		6 189 children	10-29 years	10-29 yrs = 0,71%
				Overall 0,44%
		15 805 Total population	0-70+ yrs	Total population = 1,76%
Mun (1972)	Singapore Malaysia	36 127 school children	N.S.	1,49%
		Kindergarten, primary school, secondary school and university		
Carswell, Meakins & Harland (1976)	Tanzania	128 school children	N.S.	3,3%

finding, however, was the exceedingly low number of Bantu children admitted with asthma; only 5 out of 24 731 admissions (0,02%), while asthmatic children represented 0,79% (42/5313) and 0,77% (26/3373) of the total White and Indian admissions respectively.

In a study of the hazards of carotid body removal for asthma (Louw, 1967) at the Themba Mission Hospital, Eastern Transvaal, asthma was noted to be common among Bantu in that area. The patients consisted of 19 cases, 3 White and 16 Bantu, and the age of onset was strikingly different. In the White group the mean age of onset of asthma was 10,6 years (ages 8, 9 and 15 years), whereas in the Bantu group it was 32,5 years (6, 17 and the rest 22 years and over; range 6 to 55 years).

Lewis, Goldberg & Kew (1976) from Johannesburg studied pulsus paradoxus in Black patients and stated "Bronchial asthma is fairly common in South African Blacks". In only 2 of their 33 cases was the age of onset of asthma before 10 years and the mean age of onset was 32,8 years for the group. Neither of the above authors give figures to support their claims that asthma is common among Blacks.

Ordman of Johannesburg has extensively studied various aspects of allergy in South Africa (Ordman, 1955a, 1955b, 1958a, 1958b, 1964a, 1964b and 1971). He reviewed more than 900 children, from 0 to 20 years of age, referred for skin testing (Ordman, 1958a). The age of onset of bronchial asthma was under 5 years in the majority of children, with 64,4% under the age of 4 years. In the younger children, a male to female preponderance was recorded, and the commonest cause of allergic symptoms in infancy and early childhood was foodstuffs. The races of

the children were not specified. No prevalence rates for asthma were mentioned. In none of these publications, however, can specific prevalence rates for asthma be found, the author merely generalising that the incidence of respiratory allergy was thought to be considerably lower in the Bantu people than in the Caucasians. The incidence in Coloureds was thought to be virtually the same as in the Whites and asthma was more common in Indians than in Whites. No substantiating figures were supplied for any of the 3 racial groups.

These observations follow the same trend as that found in rural communities of tropical countries, especially Africa, where asthma is rare in childhood but common in adult life, with onset in the second or third decade.

2. THE PREVALENCE OF ASTHMA IN AFRICA, INDIA AND TROPICAL COUNTRIES (TABLE II.1)

Asthma has mainly been studied in adults and few studies have been done in childhood populations to establish the prevalence of asthma. Where these have been carried out, the low prevalence rates in the above countries are striking.

Viswanathan et al (1965) undertook a pilot study of the rural and urban area of Patna, India, including all ages of the communities. In the age range 0 - 9 years asthma was virtually absent and exceedingly low even in the higher age range with the overall prevalence rate 0,64%. This pilot study was followed by a study of the total population of urban Patna (Viswanathan et al, 1966) and the prevalence rate for asthma was once again found low for children aged 0 - 9 years ie. 0,18%. The

prevalence for the total population was 1,76%.

Mun (1972) from Singapore studied children from kindergarten to university and found the mean prevalence of asthma to be 1,49%.

Pearson (1973) in Barbados found asthma to be rare in school children aged 5 - 15 years with a prevalence rate of 1,06%.

Similar findings were reported by Anderson (1974) in New Guinea Highlands where asthma in children was extremely uncommon. Based on histories and admissions to a rural health centre, he calculated a prevalence of 0,007%.

A number of studies on various aspects of asthma in adult population groups have been undertaken in different parts of Africa. None of these, however, were epidemiological studies aimed at the determination of the prevalence of the disease and only generalised statements were made without any substantiating figures.

It was the third most common cause of male medical admissions during 1971 to the Ndola Central Hospital, Zambia (Buchanan & Jones, 1972) and the most frequent emergency encountered at the Outpatient Department of the Kenyatta National Hospital, Nairobi, Kenya (Wasunna, 1968). The vast majority of these patients were adults.

An exception to these findings was the report of a 3,3% prevalence of asthma among Tanzanian school children (Carswell, Meakins & Harland, 1976). The validity of the criteria for the diagnosis is questionable (Van

Niekerk et al, 1977). The study was based on 128 school children in a rural area of Tanzania. The identification of asthmatic subjects was a drop of 10% or more in post-exercise peak expiratory flow rate from pre-exercise values. Of the 8 asthmatics identified, 2 had a drop of 10%, 3 had a drop of 11% and one had a drop of 12%. A drop of 15% or more is regarded as indicative of asthma (Silverman & Andrea, 1972; Godfrey, 1974; Fitch, 1975; Kiviloog, 1975; Tinkelman, Cavanaugh & Cooper, 1976).

3. THE PREVALENCE OF ASTHMA IN THE UNITED KINGDOM, AUSTRALIA AND NEW ZEALAND AND THE UNITED STATES OF AMERICA (TABLES II.2 - 4)

A considerable number of studies on the prevalence of asthma have been carried out in these countries. Most workers found a prevalence rate between 2% and 7%, the trend being much the same in all of them.

In the United Kingdom, Graham et al (1967) reviewed the school, medical and hospital records of all children aged 9 to 11 years born in the Isle of Wight between September 1, 1952 and August 31, 1955. Two percent of this group of children were found to be definite asthmatics and another 0,3% were probable asthmatics.

Dawson et al (1969) on the other hand found that 4,8% of children between 10 and 15 years living in the city of Aberdeen were asthmatics. Smith (1961 and 1976) found the prevalence of asthma to be increasing amongst Birmingham children. During 1956-1957 the figure in school children was 1,76% (Smith, 1961), but this had increased to 6,34% by the end of 1975 (Smith, 1976).

TABLE II.2 THE PREVALENCE OF ASTHMA IN THE UNITED KINGDOM

AUTHOR	AREA	POPULATION & SIZE	AGE RANGE	PREVALENCE	
Smith (1961)	Birmingham	49 273 school children	5 - 15 yrs	5-6 yr = 1,84% 10-11yr = 1,83% 13-15yr = 1,58%	mean 1,76%
Graham et al (1967)	Isle of Wight	3 300 children	9 - 11 yrs	Definite 2,0%) Probable 0,3%)	overall 2,3%
Dawson et al (1969)	City of Aberdeen Scotland	2 511 school children	10 - 15 yrs		4,8%
Smith, Harding & Cumming (1971)	Birmingham	20 958 school children	5 - 18 yrs	4-8 yr = 2,3% 9-14 yr = 3,0% 15-18 yr = 1,7%	Definite asthma 2,3% Wheezers 3,2% Overall 5,4%
Hamman, Halil & Holland (1975)	Kent	10 971 school children	5 - 14 yrs	5-8 yr = 3,2% 9-13 yr = 4,4% 14 + yr = 3,9%	Cumulative 3,8%
Smith (1976)	Birmingham	12 733 school children all races	5 - 18 yrs	1966 immigrant children (Indian, Negro) born in England = 2,65% 671 immigrant children (Indian, Negro) born abroad = 1,19%	mean 6,34% all children
	Total	99 746			

TABLE II.3 THE PREVALENCE OF ASTHMA IN AUSTRALIA AND NEW ZEALAND

AUTHOR	AREA	POPULATION & SIZE	AGE RANGE	PREVALENCE
Williams & McNicol (1969)	Melbourne Australia	401 children followed for 3 years from 7 to 10 years		3,7% unequivocal asthma 7,7% doubtful ? wheezy bronchitis 11,4% asthmatic episodes
Milne (1969)	Lower Hutt New Zealand	952 school children	11-13 yrs	7,14% cumulative
Derrick (1973)	Brisbane Australia	Children's Hospital admissions		2,37% of hospital admissions
Turner, Rosman & O'Mahoney (1974)	Busselton Australia	1598 school children	6-17 yrs	6,3% cumulative
	Total	2951		

TABLE II.4 THE PREVALENCE OF ASTHMA IN THE UNITED STATES OF AMERICA

AUTHOR	AREA	POPULATION & SIZE	AGE RANGE	PREVALENCE	
Harris & Schure (1956)	San Francisco	1263 children	6-12 yrs		2%
U.S.P.H.S. (1959)	Whole U.S.A.	Children	0-15 yrs		3,91%
Broder, Barlow & Horton (1962)	Tecumseh Michigan	699 children 1006 children (Total population)	6-9 yrs 10-14 yrs	6-9 yrs = 4,7%) Definite 10-14 yrs = 3,7%) asthma	4,2%
				6-9 yrs = 14,4%) Probable 10-14 yrs = 12,1%) asthma	13,3%
Freeman & Johnson (1964)	Denver	2235 grade 8 - 12 high school children			2,8%
Arbeiter (1967)	Munster Indiana	1842 children	5-15 yrs		4,9%
Myers, Bruyere & Bruyere (1969)	Hawaii	1485 elementary school children		Definite asthma 5,72%	Cumulative 8,2%
Nathanson & Rhyne (1970)	Baltimore City & 28 counties in Maryland	4100 school children	6-11 yrs		6,9%
Broder et al (1974)	Tecumseh Michigan	Total population children		Definite asthma	Cumulative
		571	0-4 yrs	0-4 yrs 4,6%	5,3%
		638	5-9 yrs	5-9 yrs 5,3%	8,9%
		682	10-15 yrs	10-15 yrs 6,0%	10,7%
		1891		5,3%	8,3%

These geographical differences in prevalence rates of asthma in childhood are not confined to the United Kingdom, but are also found in other countries. In Australia, Williams and McNicol (1969) found a prevalence of 3,7% of unequivocally asthmatic children from Melbourne, while Turner, Rosman and O'Mahoney(1974) found a prevalence of 6,3% in children from a small rural town, Bassetton, Australia. Nathanson & Rhyne (1970) in a survey of Maryland (U.S.A.) children aged 6 to 11 years, found the cumulative prevalence of asthmatic symptoms to be 6,9%, while Arbeiter (1967) found a figure of 4,9% for children living in Munster (Indiana), an upper middle class suburb of Chicago.

4. THE PREVALENCE OF ASTHMA IN THE SCANDINAVIAN COUNTRIES (TABLE II.5)

In contrast with the previous group, low prevalence rates for asthma in children ranging from 0,25% to 1,37% have been reported for Scandinavia. These studies involved vast numbers of children. Is this a true reflection of the actual prevalence of asthma in these countries or a difference in definition ? Koivikko (1974) studied all children in Finland aged 0 - 14 years and found a prevalence of 0,25%. This very low prevalence for asthma is questionable. He stated "Finnish people easily understand the word 'attack' as a rather severe situation and cases with mild wheezings were thus omitted". From this, it is likely that many of the milder cases with asthma were excluded. Studying children from 0 - 14 years will result in the inevitable inclusion of children with possible latent asthma, but in whom overt clinical asthma has not yet appeared eg. the child 0 - 2 years. The inclusion of these children in the calculations will lower the prevalence rate.

TABLE II.5

PREVALENCE OF ASTHMA IN

S C A N D I N A V I A

AUTHOR	AREA	POPULATION & SIZE	AGE RANGE	PREVALENCE
Kraepelien (1954)	Whole of Sweden	94 530 Children	7 yr	7 yr = 0,78%
		85 682	9–10 yr	9–10 yr = 0,81%
		55 437	13–14 yr	13–14 yr = 0,53%
		235 649 Total		Overall = 0,73%
	Whole of Sweden	104 550 Children	7 yr	7 yr = 0,77%
		90 938	9–10 yr	9–10 yr = 0,79%
		51 712	13–14 yr	13–14 yr = 0,53%
		247 000 Total		Overall = 0,73%
	City of Stockholm	60 063 Elementary school children	7–14 yr	7 yr = 1,61%
				8 yr = 1,39%
				9 yr = 1,45%
				10 yr = 1,42%
				11 yr = 1,48%
				12 yr = 1,57%
				13 yr = 1,26%
				14 yr = 0,86%
				Overall = 1,37%
	Not stated	712 036 High school children	10–20 yr	1944/45 = 0,39%
				1951/52 = 0,52%
			Overall from	1944–1952 = 0,50%
Peltonen, Kasanen & Peltonen (1954/55)	Turku and towns around Finland	4 832 School children	7–14 yr	0,85%
Frandsen (1958)	Copenhagen	79 944 School children	7–15 yr	0,80%
Koivikko (1974)	Whole of Finland	All children in Finland	0–14 yr	0,25%

M I S C E L L A N E O U S

Edfors-Lubs (1971)	Sweden	7 000 Twin pairs	45–84 yr	3,8% cumulative
Özkarogöz & Çakin (1969)	Anakara, Turkey	1 163 Primary school children	6–13 yr	10,6% cumulative

Kraepelien (1954) studied school children from the City of Stockholm and the whole of Sweden. He introduces his article as follows: "Nowadays bronchial asthma in children is exceedingly common. A children's doctor will find that asthmatics constitute a not inconsiderable proportion of his patients" and continues "these children form a fairly large group of the in-patients in the children's hospitals in Sweden." Despite his statement the prevalence of asthma for children 7 to 14 years from the whole of Sweden was 0,73%, while those from the City of Stockholm was 1,3%. The reason for the inconsistency is not apparent.

Edford-Lubs (1971) obtained history relating to asthma from 7000 twin pairs in Sweden. The cumulative prevalence of asthma for this group of Swedish adults was 3,8%.

The impression is that in spite of a low reported prevalence rate for asthma in the Scandinavian countries, it is not all that uncommon. Many mild cases were excluded from these studies. Other factors, however, may play a role in the low prevalence of asthma in Scandinavian countries as other workers from these countries consistently reported similar low prevalence rates.

Another difficulty in assessing these prevalence rates is that the authors did not define the diagnostic criteria used for identifying the asthmatic subjects.

Although the reported prevalence rates of asthma in children from Tables II.1 - 5 show considerable differences, certain trends are noticeable.

An interesting pattern emerges when the studies in each country are grouped together (Tables II.2 to 5). The prevalence rates in the United Kingdom show an obvious relation and similarity to those in the United States of America, Australia and New Zealand. The mean prevalence rate of asthma for each country calculated from all the studies done in that country is 3,5% for the United Kingdom (1,76% to 6,34%), 4,46% for the United States (range 2% to 6,9%) and 4,87% for Australia and New Zealand (range 2,37% to 7,14%). In striking contrast are the rates reported by workers from Scandinavian countries, where the calculated mean prevalence is 0,75% (range 0,25% to 1,37%). Only cases indicated as "definite" asthma were included in these calculations. The prevalence rates for Scandinavian countries were found to be consistently much lower than for the United Kingdom, United States, Australia and New Zealand. The people of the latter 4 countries all speak the same language and have the same industrialised, westernised way of life, whereas the Scandinavian peoples originated from a different cultural and ethnic group with different languages. Do these differences reflect a different lifestyle and home environment or are they related to methodological differences ?

The mean calculated prevalence of asthma from reports for countries from Africa, India and tropical areas is calculated at 1,38% (range 0,44 - 3,3%). If the study of Carswell, Meakins and Hardland (1976) from Tanzania is excluded from the calculations, the mean calculated prevalence rate for these countries drops to 0,91%. The findings of the study of Shore (1959) and of Wesley, Clyde & Wallace (1969) were excluded from these calculations. Shore's findings were based upon referrals to a paediatric practice (Shore, 1977 personal communication), while the study

of Wesley, Clyde & Wallace (1969) reflects the percentage of asthma among hospital admissions. Neither of the 2 studies relate to the prevalence rates of asthma for the broader childhood population.

CHAPTER 2 : THE EFFECT OF AGE AND LIVING IN AN URBAN OR RURAL COMMUNITY ON THE PREVALENCE RATE OF ASTHMA

The reported differences in prevalence rates of asthma from one country to another have been discussed. There are, however, other aspects of the disorder such as the age of onset and urban and rural communities where differences have been reported.

1. AGE OF ONSET

The onset of asthma generally occurs in the young child and in the majority of cases, the age of onset is under 6 years in the countries with a western way of life. The age of onset, as reported by different authors, is summarised in Table II.6.

From this table it is apparent that the age of onset is related to the age range of the population studied in the United Kingdom, United States of America, Finland and Malaysia. Where the age range of a selected population is 0 - 6 years (A), the mean age of onset of asthma as calculated from Table II.6 will be in the first 5 years of life in 83% of cases. If a student or total community including adults (B) is studied, the age of onset will be expected to be later.

There is, however, a striking difference for African countries (C) where the age of onset is in young adult life. In the studies of Louw (1967) and Lewis, Goldberg & Kew (1976) of South Africa, the age of onset was in the third decade of life for Black adults.

Cookson and Makoni (1975) studied 78 African adults attending Harari

TABLE II.6 AGE OF ONSET OF ASTHMA IN CHILDREN

AUTHOR	COUNTRY	AGE OF ONSET	AGE OF POPULATION SURVEYED	% CASES
A. REPORTS ON CHILDREN ONLY (EXCLUDING AFRICA)				
Fuller (1952)	United Kingdom	First 7 yrs	1-16 yrs	84
Smith (1961)	United Kingdom	First 5 yrs	5-15 yrs	84
Dawson et al (1969)	Scotland	First 5 yrs	10-15 yrs	80
Hamman, Halil & Holland (1975)	United Kingdom	First 5 yrs	5-14 yrs	82
Koivikko (1974)	Finland	First 5 yrs	0-14 yrs	85,4
Mun (1972)	Malaysia	First 5 yrs	1-10 yrs	90
B. REPORTS ON STUDENTS OR TOTAL COMMUNITIES				
Broder, Barlow & Horton (1962)	U.S.A.	First 9 yrs	0-24 yrs	47,8-49,9
Freeman & Johnson (1964)	U.S.A.	First 4 yrs	Grades	68
Blair (1974)	United Kingdom	First 9 yrs	Total Practice	69
C. REPORTS FROM AFRICA				
Louw (1967	Rep. of S.A.	Mean age of onset for White 10,6 yrs for Blacks 32,5 yrs	Not Stated	-
Lewis, Goldberg & Kew (1976)	Rep. of S.A.	Mean 32,8 yrs for Blacks	Adults	-
Mitchell (1970)	Kenya	Teenage or early adult life		
Rees et al (1974)	Kenya	Typical asthmatic is a young adult		
Cookson & Makoni (1975)	Rhodesia	Mean age of onset 26,8 yrs	Adults	-
Godfrey (1975)	Gambia	No cases of asthma found from selected rural or urban children		
Warrell et al (1975)	Nigeria	After 19 yrs	Total Practice	69

Hospital, Salisbury, Rhodesia, for asthma. The mean age of onset was 26,8 years with a range of less than 1 to 56 years. In a study of 106 adult asthmatics in Zaria in the Nigerian savanna region, Warrell et al (1975) commented on the rarity of childhood asthma and in 69% of their patients asthma started after the age of 19 years. Mitchell (1970) from Kenyatta National Hospital, Nairobi, Kenya, found the age of onset of asthma to be in the teenage years or in early adult life without giving definite ages. This delay in the age of onset of asthma was also noted in India (Viswanathan et al, 1966) where the disease more commonly began in the higher age groups than in childhood.

2. URBAN AND RURAL COMMUNITIES

Epidemiological studies of asthma in industrialised countries showed no difference in the prevalence rates for children living in urban as compared with rural communities. Smith & Knowler (1965) studied 1760 rural Iowa families comprising a stable population living on prosperous farms with an abundance of inhalant allergens present. The prevalence of asthma for the total population was 3,7%, which is comparable to that reported elsewhere in the United States. Broder et al (1974) studied the epidemiology of asthma in a total community in Tecumseh, Michigan and found no difference in prevalence for people living in the city or in the country. Hamman, Halil & Holland (1975), in a survey of 10,971 school children in Kent, England, found that the area of residence did not have a significant effect on the prevalence of asthma. The life-style of these people probably does not differ whether they live in rural or urban areas.

Reports from Scandinavian countries are variable. Edfors-Lubs (1971) sent questionnaires to all twins born in Sweden between 1886 and 1925. Of these, 7 000 twin pairs responded. No difference in the prevalence rates for asthma could be found between people living in cities, towns or country. Koivikko (1974) surveyed the total childhood population of Finland and found that 52,2% of patients with asthma came from the country, 18,7% from densely populated areas and 29,4% from towns. The area of residence did not have a large influence on the progress of asthma and he concluded that living conditions had no marked effect on the severity of asthma. In a study of allergic conditions in Finnish school children, Peltonen, Kasanen & Peltonen (1954-55) found that the incidence of allergic disorders was 10,1% for city dwellers (Turku), 8,0% for those in small towns and 5,7% for children living in rural areas. It was not stated whether these differences were significant. The authors, however, attached importance to them and concluded that the lower proportion of allergic conditions amongst the rural population could be partly determined by the way of life.

By contrast, striking differences have been found in the prevalence rates for asthma between communities living in urban and rural environments in Africa and most other tropical and underdeveloped areas. Reports refer mainly to adult asthmatics, as childhood asthma has been found to be rare. The striking finding is the absence of asthma in rural communities (Johansson, Mellbin & Vahlquist, 1968; Woolcock, 1972; Anderson, 1974; Godfrey, 1975; Merrett, Merrett & Cookson, 1976). In South Africa, Wesley, Clyde & Wallace (1969) found only 5 cases of asthma (0,02% of total admissions) among Bantu children admitted to the King Edward VIII Hospital, Durban, during a 5 year period. None of them came from a rural

environment and they exhibited the same features with regard to background as the White and Indian groups. In broad terms, without quoting actual figures, Ordman (1964a) estimated the incidence of asthma in the indigenous Bantu peoples of South Africa to be relatively small.

While these differences in prevalence rates for childhood asthma could in part be due to variations of the prevalence and the pattern of asthma in different communities, they may in part also be due to variations in the methods used in the various studies. There has been no uniformity in the sample selection, definition of asthma, method of examination and follow-up in the various studies. Comparison between studies is therefore very difficult - in fact, well nigh impossible.

Asthma has defied many attempts at definition (cf.p.34) and this may contribute to the apparent differences in the reported prevalence rates. Analysis of epidemiological studies amplifies this shortcoming on the part of many investigators to adhere to a single definition of the disease (which does not exist) or to establish well defined criteria for the acceptance of cases. The variety of criteria or definitions for subject selections in previous studies is astonishing. The following have been applied:

> recurrent dyspnoea of an obstructive type without other demonstrable cause (Dawson et al, 1969)
>
> a condition producing attacks of breathlessness with wheezing. All children who suffered one or more such attacks in the previous years were included (Graham et al, 1967)

asthma defined as a positive response by parents when questioned whether their child ever had asthma (Milne, 1969; Hamman, Halil & Holland, 1975)

manifestations which were either brief or prolonged, a single episode or recurrent ones and occurred either alone or in association with upper or lower respiratory illness (Broder, Barlow & Horton, 1962)

asthmatic child defined as one having a history of attacks of dyspnoea with expiratory wheeze unless such history was explained on some other basis such as cardiac failure, chronic bronchitis, emphysema, bronchiectasis, and so on (Myers, Bruyere & Bruyere, 1969)

Many studies on the epidemiology of asthma do not even attempt to define the condition (Fuller, 1952; Kraepelien, 1954; Frandsen, 1958; Freeman & Johnson, 1964; Wesley, Clyde & Wallace, 1969; Anim & Edoo, 1972; Mun, 1972; Derrick, 1973; Pearson, 1973; Koivikko, 1974; Warrell et al, 1975). The inclusion of patients as asthmatics in these studies was based on a positive parental history, hospital records, school medical examinations or where the general practitioner had made the diagnosis.

From these divergent diagnostic criteria different authors have identified "definite" or "unequivocal" asthma based on their individual criteria (cf. Tables II.1 - 5). Inter study comparison is, therefore, not valid, or at best purely speculative.

Many studies on other aspects of asthma besides epidemiology, for example, diagnostic procedures, exercise-induced asthma and allergens, use the word "asthma" freely without defining it at all (Godfrey et al, 1973; Havnen et al, 1973; Pepys, Roth & Carroll, 1975; Sarsfield et al, 1976; to mention only a few of the many relevant articles).

It is therefore apparent that the inability to define asthma must confuse and impede epidemiological studies. Even if consensus is reached regarding definition, how does one measure or determine the prevalence rate of a chronic disorder which is variable, episodic and intermittent ?

The lack of a single definition of asthma is not the only stumbling block. Another major problem is the lack of distinction made between asthma as a disease entity and asthmatic attacks or wheezy episodes especially when diagnosis depends largely on historical events. Few disorders have so many synonyms which are used interchangeably eg. wheezy bronchitis, asthmoid bronchitis, allergic asthma, bronchial asthma, climatic asthma, asthmatic bronchitis, extrinsic and intrinsic asthma, cat asthma, dog asthma and horse asthma (Rackemann & Edwards, 1952; Ordman, 1955c; Aas, 1969; Williams & McNicol, 1969; Foucard, 1973; Warrell et al, 1975; Davis, 1976).

Any disease has a spectrum of mild, moderate and severe cases. The distinction, if any, between wheezy bronchitis and mild asthma is not clear. How severe should wheezy bronchitis be in terms of number of attacks and duration of symptoms to be labelled mild asthma, and how mild should asthma be before it is regarded as not being asthma any longer ? In an epidemiological study, Williams and McNicol (1969)

examined the prevalence, natural history and relationship between wheezy bronchitis and asthma in children. They concluded that children with wheezy bronchitis and asthma were from the same population, with the same basic underlying disorder. Despite this, they were able to identify a group of children who were "unequivocally" considered to have asthma. The difficulty of distinguishing between wheezy episodes and mild asthma may result in erroneous inclusion or exclusion of cases.

The frequency of a disease in a population can be measured or expressed in terms of a rate - incidence or prevalence. Most epidemiological studies measured the prevalence rate of asthma. Prevalence rate can, however, be measured in terms of time interval, point, period or cumulative prevalence.

Some authors failed to indicate which type of prevalence rates were determined (Tables II.1-5). A further problem is that comparison of surveys studying point prevalence (does the child have asthma at this specific time ?) or cumulative prevalence (did the child ever have asthma ?) is limited.

The difficulty of defining asthma and distinguishing between mild asthma and wheezy episodes has resulted in prevalence rates being expressed as definite asthma cases, probably asthma cases and the combined number of cases. The various authors do not always clearly state whether only definite cases or combined cases were included in establishing the prevalence rate (Tables II.1-5).

Several different methods of sample selection in the study of asthma prevalence rates have been employed. The populations studied varied from total communities including children and adults (Broder, Barlow & Horton, 1962; Viswanathan et al, 1965 & 1966; Blair, 1974; Broder et al, 1974); a total child community (Arbeiter, 1967; Graham et al, 1967; Koivikko, 1974); all school children in an area (Turner, Rosman & O'Mahoney, 1974); a random sample of school children either primary or secondary (Kraepelien, 1954; Peltonen, Kasanen & Peltonen, 1954-55; Smith, 1961; Freeman & Johnson, 1964; Dawson et al, 1969; Mun, 1972) to hospital attendances (Wesley, Clyde & Wallace, 1969; Derrick, 1973; Schmerler & Abramowicz, 1974).

Various ways of identifying the asthmatic subject in a defined population have been used. The problems encountered here relate intimately to the failure to define asthma. The method primarily employed was that of sending a questionnaire to the parents of children in a selected population (Peltonen, Kasanen & Peltonen, 1954-55; Arbeiter, 1967; Milne, 1969; Özkaragöz & Çakin, 1969; Hamman, Halil & Holland, 1975). Other methods were the collection of information from school health visitors, school medical cards, general practitioners and hospital records (Kraepelien, 1954; Frandsen, 1958; Smith, 1961 & 1976; Graham et al, 1967; Dawson et al, 1969; Koivikko, 1974). In these studies, a positive finding was often followed by an interview with the parents. The difference in interpretation as to what constitutes asthma and when it should be diagnosed exists not only for medically trained personnel (doctors, nurses, health visitors) but also for the lay public and here one includes the parents.

To establish whether the reported variations are real in terms of prevalence or only reflect variations in definition, terminology, methodology of sample selection and of local environmental factors that may influence these rates is not possible. The findings of different investigators, as have been pointed out, are unfortunately not comparable as to whether these differences are real.

The reported prevalence rates for childhood asthma show considerable variation. Do these variations reflect true differences in prevalence amongst different populations or do they only reflect methodological differences and differences in terminology ? What is the reason for the apparently extremely low prevalence of childhood asthma among the Bantu peoples of South Africa and other non-industrialised countries, particularly those living in rural environments ? Is it due to racial factors or to a difference in allergic exposure secondary to the environment and a particular lifestyle ?

A change of environment from a rural to an urban community probably entails more than simply a transfer from one house to another. It is likely that the whole lifestyle of the individual changes involving such diverse factors as sleeping on a mat or a bed with mattress or the duration of breastfeeding.

Vaughan & Black as early as 1948 asked whether allergic disease was associated with "civilisation" and whether it was in great measure a product of the artificial environment in which we live. "If so, does this artificiality cause the fundamental intrinsic changes which we term the allergic state, or the allergic tendency, or does such an environment

merely contribute increased and concentrated quantities of the allergic excitant thereby increasing the actual manifestations of an already present or latent predisposition ?"

From the findings of the studies quoted, it would appear that the answer to this question so aptly put in 1948, is that the artificial environment called "civilisation" (Western lifestyle and environment) probably contributes greatly to a latent predisposition.

The probability has been suggested that as people from rural communities become more exposed to a different socio-economic and cultural environment, more will present with asthma and other allergic diseases (Peltonen, Kasanen & Peltonen, 1954-55; Maternowski & Mathews, 1962; Wesley, Clyde & Wallace, 1969; Woolcock, 1972; Smith, 1976). This is not just a change from a rural to an urban community, but implies a change in lifestyle as well.

If the findings of Tables II.1-5 are analysed as a whole, distinct patterns are obvious and the reported differences are probably a true reflection of the prevalence of the disease in different communities and only to a lesser extent differences in methodology.

CHAPTER 3 : IDENTIFICATION AND ASSESSMENT OF THE ASTHMATIC CHILD

INTRODUCTION

To identify and measure the magnitude of a disorder in different population groups, it should be possible to define the disorder accurately. The acceptability of such a definition lies in its exactness for the particular disorder, its simplicity and its reproducibility, so that by applying it any research worker or clinician should, within reason, be able to identify any patient suffering from that disorder. Asthma is a complex disorder and there is no single feature, clinical sign or laboratory finding which is pathognomonic for it (Aas, 1975; Sheldon, Lovell & Mathews, 1967). There is also no universally accepted definition for asthma. The epidemiological investigations into asthma have therefore been bedevilled by problems of definition as to what constitutes asthma.

The term has been used for many years in a clinical application. Willis described the asthmatic attack as early as 1684. In spite of it being recognised and diagnosed as such in clinical medicine, there is at present no single universally accepted definition of asthma. This paradox stems mainly from the differences in opinion as to the clinical entities which constitute asthma. Vaughan and Black in 1948 remarked that "the term bronchial asthma is unfortunate since all true asthmas are immediately bronchial in origin. It represents another of those etymologic failures in medical diction which at times obscure correct perspective". This failure is still unresolved today and, as so aptly phrased, still obscures correct perspective.

Suggestions for a definition of asthma have been offered. During September 1958 a Ciba Guest Symposium was held to see whether a group of British investigators could agree upon provisional definitions, classifications and terminology of chronic pulmonary emphysema and related conditions. No agreement could be reached and it was NOTED that the proposals were provisional and should not be regarded as committing any of the participants to any particular views.

In 1971 the findings of the Ciba Foundation Study Group No. 38 on the Identification of Asthma were published. This group consisted of experts from Great Britain, France, Canada and the United States of America. The final conclusion reached was that "the members of the Study Group on the Identification of Asthma decided that asthma could not be defined on the information at present available".

Attempts have been made to classify and define asthma in terms of the pathological process involved (Sanerkin, 1971). In a disorder with a low fatality rate and where it is difficult to measure these pathological changes, such definitions only add to the confusion.

Although no consensus has been reached on the definition of asthma and what constitutes asthma, many workers and clinicians have used a "working definition" (Ciba Guest Symposium, 1959; American Thoracic Society, 1962; Scadding, 1966; Sheldon, Lovell & Mathews, 1967; Chai et al, 1968; Pappworth, 1971; Scadding, 1971; Ellis, 1975). The main points of this definition are:

(1) A variable narrowing of the bronchial airways resulting in airways obstruction

(2) This variable bronchial airways obstruction is episodic and intermittent

(3) Is reversible or changes its severity either spontaneously or as a result of therapy

This, however, makes a statement about the observed functional abnormality and says nothing about its cause. What degree of variable obstruction and what frequency in terms of episodes and intermittency are necessary to qualify for the diagnosis of asthma ? This remains unanswered.

Since the term "asthma" cannot be satisfactorily defined, how then can the child with asthma be identified and diagnosed as such in clinical practice ?

1. IDENTIFICATION OF THE ASTHMATIC CHILD

(a) The History

The foundation for the identification and management of the asthmatic child in clinical medical practice is a thorough and continuing history (Rapaport, 1972). Asthma is always a dynamic situation because neither the disease nor the status of the patient are static - it is a variable, intermittent, reversible process of airways obstruction. The objectives of the history are to determine (a) whether the child suffers from asthma and (b) what factors are responsible for the symptoms. Questions should be aimed at establishing the nature, pattern, frequency and severity of the symptoms. A detailed enquiry should be made into the environment of the child to establish which factors provoke the asthmatic attack. Emphasis on minute detail is required to establish, in a particular individual's immediate home environment, the causative allergen responsible

for the attack.

In an epidemiological survey where parents are interviewed only once, the extent and accuracy of the history obtained from even a very observant parent has limitations. These can, in some respects, be so severe as to make the history virtually useless. Chai et al (1968) showed that the information given by the majority of mothers reflected past events poorly when compared to other measurements independent of her judgment. They found that the less often histories were taken, the less accurate they were. When memory was required regarding the child's state, even over a 24 hour period, recall was very inaccurate. Parents and patients themselves tend to remember only the more serious episodes. They concluded that any research programme which used only historical data as a measurement in the investigation of asthma would inevitably result in incorrect conclusions most of the time. Hamman, Halil & Holland (1975) found that 6,5% of parents who gave a positive history of asthma for their child, had forgotten that the child had had asthma when questioned again 3 years later, emphasising the unreliability of parental history and reliance on memory. They also found that medical records at school were frequently incomplete. The reliability of histories in epidemiological surveys, whether from questionnaires or direct from parents, is therefore questionable.

In addition, there is the difference in interpretation of what constitutes the disorder asthma. In the grey zone where mild asthma and wheezy bronchitis are not easily differentiated, cases may be erroneously included or excluded.

Where studies are undertaken in a population with a different language and culture necessitating the mandatory use of an interpreter, the limitations of the history become even greater.

(b) The Clinical Examination

The clinical findings on examination may vary tremendously in the asthmatic child. During an attack signs of airtrapping, prolonged expiration with expiratory rhonchi or wheeze may be audible. The same child may, however, show no clinical abnormality between attacks. Even in moderately severe asthma with evidence of small airways disease, no abnormal signs may be found on examination of the chest (Sheldon, Lovell & Mathews, 1967; Jones, 1976). The absence of clinical signs, however, does not rule out severe ventilatory impairment or marked lability of the bronchioles. This applies also to pulmonary function tests eg. Forced Expiratory Volume or Peak Expiratory Flow Rate (Chai et al, 1968). Pulmonary function tests give quantitative information about physiologic normality or impairment of pulmonary function, but will not provide a clinical or pathologic diagnosis (Woods, 1969).

McNicol, Williams & Gillam (1970) studied a group of 276 asthmatic children of all grades from mild to severe. Chronic chest deformities secondary to the asthmatic disorder, eg. barrel chest, pigeon chest and Harrison's sulcus, were found mainly in the groups of severely asthmatic children. The majority of children, however, had mild asthma with almost none of these abnormalities.

The single clinical examination or pulmonary function tests, therefore, have the severe limitation in that clinically no abnormal physical signs may be found between asthmatic attacks. It reflects only the status at

a moment and bears no relationship whatever to the previous 24 hours or to the next hour. It also reflects airways obstruction only when it has reached a certain level and only at the time the examination is undertaken (Chai et al, 1968).

2. EVALUATION OF THE ASTHMATIC CHILD

A number of special diagnostic procedures may be used in the evaluation of the asthmatic child. Skin tests and other special studies can be interpreted only in the light of the information obtained by a careful history and clinical examination (Sheldon, Lovell & Mathews, 1967). There is no pathognomonic clinical picture or single laboratory finding specific for asthma (Aas, 1975) and the assessment and management of the child with asthma in clinical medicine should be the sum of history, clinical examination and special procedures.

Major advances in the field of immunology have led to a better understanding of the processes involved in the type I immediate anaphylactic allergic reaction (Coombs & Gell, 1975). It is now possible to measure the reaginic antibodies involved in this reaction. However, this is unlikely to replace the time-honoured procedures which still play an equally important part in the evaluation of the asthmatic child.

Special procedures may be of great help in evaluation of the asthmatic patient. Each of these has its own shortcomings and no single one by itself is able to provide a final diagnosis.

(a) Prick Skin Testing

Skin tests are widely used to detect and identify reaginic antibodies (Vaughan & Black, 1948; Sheldon, lovell & Mathews, 1967). The prick test, through a drop of allergen on the skin, has been shown to be the most sensitive of the cutaneous tests (Infrajana, Spieksma & Voorhorst, 1971). A positive reaction results in erythema and a wheal maximal at about 12 to 17 minutes (Voorhorst & Van Krieken, 1973a; Pepys, 1974). When selecting allergens for skin testing, it is important to include those found in the individual's immediate environment. The omission of commonly encountered allergens would lead to false conclusions.

(b) Serum Immunoglobulin levels

(i) Total Immunoglobulin E (IgE)

In their initial classification of atopy in 1923, Coca and Cooke mentioned atopic reagin, which could not be transferred passively to normal individuals by blood. In 1925 Coca and Grove found the atopic reagin to be heat labile and present in the blood of subjects suffering from hayfever and asthma. It was also responsible for prolonged sensitisation.

In 1966 Ishizaka, Ishizaka & Hornbrook showed that atopic reagin or reaginic antibodies belonged to a particular immunoglobulin class - gamma E globulin, called immunoglobulin E or IgE (Bull, WHO 1968).

In 1967 Johansson found that IgE (then called IgND) was raised in the serum of patients suffering from "allergic" asthma as compared to patients with "non-allergic" asthma. This raised serum IgE level in individuals

suffering from allergic disorders has been clearly documented (Gleich, Averbeck & Swedlund, 1971; Hogarth-Scott et al, 1971; Havnen et al, 1973; Halpern, 1974; Saha et al, 1975; Yunginger & Gleich, 1975; Kjellman, 1976).

IgE concentration in serum is raised not only in allergic individuals but also in individuals with parasitic infestation. The association of a raised serum IgE level and intestinal helminthic infestation is well established (Reviewed by Ogilvie & Jones, 1973; Orren, 1974). Orren (1974) studied adults in Cape Town ranging in age from 18 to 65 years in three racial groups: White, Coloured and Bantu. Bantu subjects had a marked raised serum IgE level, while the White group had the lowest levels. Helminthic infestation was common in the Bantu subjects, but uncommon in the White group. No difference in values could be shown between Bantu subjects with or without evidence of such infestation. The following table shows the median, upper 75th and upper 95th percentiles of serum IgE concentrations for male and female blood donors of the 3 racial groups in the Western Cape (Orren, 1974; Orren & Dowdle, 1975b):

	White		Coloured		Bantu	
	Male	Female	Male	Female	Male	Female
Number	2159	950	773	398	144	16
Median IgE, u/ml	85	70	226	180	642	289
Upper 75th percentile, u/ml	172	136	564	446	1765	715
Upper 95th percentile, u/ml	648	460	4096	2331	18519	2885

No values for serum IgE levels for South African children are available.

Raised serum IgE levels have also been found in rare disorders such as the Wiskott-Aldrich syndrome (Waldmann, Polman & Balestra, 1972), the Di-George syndrome (Polman, Waldmann & Terry, 1972), the Guillain-Barré syndrome during the acute phase (Huang, 1975) and the acute mucocutaneous lymph node syndrome (Kusakawa & Heiner, 1976).

(ii) Allergen-Specific IgE

An in-vitro test has been developed to measure allergen-specific IgE with the Radioallergosorbent technique (RAST) (Wide, Bennich & Johansson, 1967). This is a sensitive semi-quantitative test (Wide, 1973).

Highly significant correlations have been obtained between measurements by the RAST method of specific IgE to common allergens (such as the house dust mites, Dermatophagoides pteronyssinus and Dermatophagoides farinae, grass pollen, dog epithelium and purified allergen from codfish) and the results of skin tests, provocation tests and clinical history (Stenius et al, 1971; Stenius, Wide & Seymore, 1972; Aas & Lundkvist, 1973; Ahlstedt et al, 1974; Pepys, Roth & Carroll, 1975).

Serum IgE specific to particular allergens can be identified by a prick skin test, which is quick and simple to do, whereas the RAST requires sophisticated laboratory equipment, including a gamma counter. Moreover, the relatively high cost of the RAST makes it impractical in an epidemiological study involving large numbers.

(iii) Immunoglobulins A, G and M (IgA, IgG and IgM)

Serum immunoglobulins have been measured in children with allergic disorders. In most studies serum IgA, IgG and IgM levels were found to be within the same range as in normal controls (Huntley & Lyerly, 1963; Buckley, Dees & O'Fallon, 1968; Palma-Carlos & Palma-Carlos, 1971). An isolated IgA deficiency, however, seems to be more prevalent among allergic subjects. Saha et al (1975) found 11 of 69 cases (15,9%) with asthma to have an isolated IgA deficiency, while Kaufman & Hobbs (1970) found an IgA deficiency in 6,7% of 641 atopic individuals.

(c) Total Eosinophil Count in the Peripheral Blood

An elevation in the total number of eosinophils is a common finding in patients with allergic disorders (Lowell, 1967; Sheldon, Lovell & Mathews, 1967). Numerous non-allergic disorders, however, have also been associated with an increase in the number of eosinophils in the peripheral blood (Lecks & Karvis, 1969). It is commonly found in patients with helminthic infestations (Rees et al, 1974; Warrell et al, 1975).

(d) Radiographs of the Chest

Radiographic examination of the chest should be done in any child suspected of suffering from asthma in order to exclude other chest diseases which might not be detectable by clinical examination as well as other pathological processes which may mimic the clinical picture of

asthma. These disorders include compression of a bronchus eg. by tuberculous glands or aspiration of a foreign body; cystic fibrosis and bronchiectasis.

In uncomplicated cases of asthma, a radiograph of the chest taken between attacks may be completely normal (Sheldon, Lovell & Mathews, 1967; Jones, 1976). Persistent abnormalities such as hyperinflation may be detected between attacks only in the severe cases, but these cases represent a minor group of the asthmatic population (McNicol & Williams, 1973; Sheldon, Lovell & Mathews, 1967).

CHAPTER 4 : EXERCISE-INDUCED ASTHMA

It has been recognised for many years that exercise may affect sufferers from asthma. It is also known that different types of exercise engaged in by asthmatics produce different responses. This received little attention until 1962, when Jones, Buston & Wharton reported the effect of exercise in asthmatic children. They noted that exercise over a short period (1-2 minutes) resulted in broncho-dilatation; while if this was prolonged (8-12 minutes) marked broncho-constriction resulted. This led to a renewed interest in exercise-induced asthma and many studies on the subject have since been published.

Initial results were conflicting as children and adults were often studied in the same groups and the exercise stimulus varied from free running, running up and down stairs, treadmill-running and walking, cycling on an ergometer and swimming (Heimlich, Strick & Busser, 1966; McNeill et al, 1966; Rebuck & Read, 1968; Seaton et al, 1969; Fisher et al, 1970; Poppius et al, 1970; Chan-Yeung, Vyas & Grzybowski, 1971; Fitch & Morton, 1971).

Controlled trials have clarified many of the uncertainties about exercise-induced asthma and the assessment of an asthmatic child by means of exercise-induced bronchospasm has become an important non-invasive diagnostic aid in the armamentarium of the allergologist. However, the mechanisms by which exercise provokes bronchoconstriction remain uncertain.

Response to Exercise in an Asthmatic Child

The response to an appropriate exercise stimulus in an asthmatic child is typical (Anderson, McEvoy & Bianco, 1972; Silverman & Anderson, 1972; Godfrey, Silverman & Anderson, 1973). During the early stage of exercise, bronchodilatation is found. This is after about 4 minutes of running followed by marked bronchoconstriction which reaches its maximum 3 to 5 minutes after stopping the exercise. The bronchoconstriction then reverses spontaneously and this is usually complete 20 to 30 minutes after exercise. The clinical presentation is that of an acute attack of bronchospasm with wheezing and rhonchi on auscultation, dyspnoea and occasionally even respiratory distress and cyanosis.

The degree of bronchoconstriction is most commonly measured with simple lung function apparatus. The most useful measurements indicating changes in the airway calibre are the Peak Expiratory Flow Rate (PEFR) (Silverman & Anderson ,1972; Godfrey, Silverman & Anderson, 1973; Burr, Eldridge & Borysiewicz, 1974) and the Forced Expiratory Volume in 1 second (FEV_1) (Jones, Buston & Wharton, 1962; Jones, Wharton & Buston, 1963; Bierman, Kawabori & Pierson, 1975). PEFR is usually measured on a Wright Peak Flow Meter and the FEV_1 on a Vitalograph, both of which are simple to operate.

The sensitivity of the various parameters in detecting exercise-induced bronchospasm was evaluated by Cropp (1975). Specific airway conduction (SGAW) was the most sensitive parameter, while PEFR and maximum mid-expiratory flow (MMEF) were approximately equal but less sensitive than SGAW. Reductions in FEV_1 were less marked and less frequent than that

for PEFR and MMEF. A reduction in forced vital capacity (FVC) was the least severe and the least often observed. A high non-linear correlation was found for SGAW to PEFR ($r = 0,84$), to MMEF ($r = 0,86$) and to FEV_1 ($r = 0,93$). Reductions of 20% to 75% from resting values were more or less equally frequent for SGAW, PEFR and MMEF and less frequent for FEV_1; FVC was the least sensitive and changes occurred only when the exercise-induced asthma was severe. The author concluded that the use of peak flow and spirometric measurements make these tests ideal for detecting exercise-induced asthma. These 2 parameters will yield as many positive results as sophisticated plethysmographic tests (Weng et al, 1969; Anderson, McEvoy & Bianco, 1972; Benatar & König, 1974, Cropp, 1975).

In a review of exercise-induced asthma, Godfrey (1974) stated that it mattered little which index of airway calibre was used. The PEFR is very simple to measure with a Wright Peak Flow Meter and gives reproducible results. The FEV_1 is rather more reliable and can be measured with relatively simple equipment.

The degree of bronchoconstriction developing as a result of exercise challenge is expressed as the percentage fall in FEV_1 or PEFR from pre-exercise resting values. The formula used is that of Silverman & Anderson (1972):

$$\text{Percentage fall} = \frac{\text{Pre-exercise} - \text{Post-exercise } FEV_1 \text{ or PEFR}}{\text{Pre-exercise } FEV_1 \text{ or PEFR}} \times 100$$

Effect of the Type, Severity and Duration of Exercise

The response to exercise in the asthmatic child is closely related to its form. The effect of the type, severity and duration of exercise in causing bronchoconstriction have been clarified by using different standardised types of exercise (Fitch & Morton, 1971; Silverman & Anderson, 1972; Vassallo, Gee & Domm, 1972; Godfrey, Silverman & Anderson, 1973; Eggelston & Guerrant, 1976).

The most potent stimulus causing exercise-induced asthma has been found to be free running, with swimming and walking the least (Godfrey, Silverman & Anderson, 1973; Godfrey, 1975; Fitch, 1975).

In one trial, children were exercised at a constant workload on a treadmill (Silverman & Anderson, 1972). The effect of the duration of exercise was assessed by determining the percentage fall in PEFR after 1, 2, 4, 8, 12 and 16 minutes. When exercise time was short (1-2 minutes) broncho-dilatation resulted, but when it was longer the PEFR fell, the greatest percentage fall being after 6 - 8 minutes of exercise. Strangely enough, further prolongation of exercise up to 16 minutes, often resulted in lesser degrees of bronchoconstriction than in the intermediate 6 - 8 minute period, some patients showing no bronchoconstriction whatever after running for 16 minutes.

By changing the severity, ie. the workload, a difference in response to exercise is also found. The maximum bronchoconstriction results from running at a speed representing about 70% of the maximum aerobic power of the subject which should raise the heart rate in children to about 170 to 180 beats per minute (Silverman & Anderson, 1972; Anderson et al, 1975).

Diurnal variation in response to exercise has not been found to be prominent (Silverman & Anderson, 1972; Eggelston & Guerrant, 1976; Haynes, Ingram & McFadden, 1976).

Conflicting findings on the effect of airborne pollens on exercise-induced asthma have been reported. Pierson & Bierman (1975) found the lowest rates of exercise-induced asthma occurred during the summer and the highest rates during autumn and winter, representing an inverse correlation between exercise-induced asthma and airborne pollens. Eggelston (1975) found, on the other hand, that 11 of 16 extrinsic asthmatic patients had a greater fall in FEV_1 during the pollen season than before or after it. The fall in FEV_1 was still significant, even during the non-pollen season.

What is Exercise-Induced Asthma ?

The criteria used in defining exercise-induced asthma varied considerably and were, in many cases, purely arbitrary, ranging from wheezing only after exercise (Engström et al, 1960) to a drop from resting values of 25% or more in PEFR or FEV_1 (Poppius et al, 1970; Cropp & Schmultzler, 1975). A drop of 10% in PEFR from resting values has been suggested as the upper limit of normal (mean $\pm$ 2 SD) (Silverman & Anderson, 1972). In an extensive survey of 12-year-old school children, Burr, Eldridge and Borysiewicz (1974) showed that 92,3% of entirely healthy children had a fall less than 10% and 97,8% had a fall less than 15%. Many workers, however, define exercise-induced asthma as a fall of 15% or greater in in PEFR or FEV_1 from resting values (Chan-Yeung, Vyas & Grzybowski, 1971; Silverman & Andrea, 1972; Fitch, 1975; Kiviloog, 1975; Pierson & Bierman, 1975; Tinkelman, Cavanaugh & Cooper, 1976).

The asthmatic child responds to an appropriate exercise stimulus with a marked and often precipitous drop in PEFR or FEV_1, the values being between 20% and 50% of the pre-exercise levels (Silverman & Anderson, 1972; Burr, Eldridge & Borysiewicz, 1974; Eggelston, 1975).

The form of exercise most likely to bring on an attack of asthma is free range running at a steady, moderate pace, reflecting a submaximal effort lasting 6 to 8 minutes, which raised the pulse rate to 170 or more beats a minute. The maximum bronchoconstriction occurs 3 to 5 minutes after the exercise is completed, measured as a drop of 15% or more in FEV_1 or PEFR.

Bronchoconstriction provoked by exercise is unique and occurs only in the asthmatic child. It is so constant that failure to demonstrate it should lead to reconsideration either of the technique of the test used, or of the diagnosis of asthma itself (Jones, Wharton & Buston, 1963). This view is supported by Godfrey, who calls it "the hallmark of the asthmatic child" (Godfrey, 1974).

Response to Exercise in the Normal Child

In contrast to the asthmatic, the normal child shows very little change in bronchial calibre as a result of exercise (Heimlich, Strick & Busser, 1966; Jones & Jones, 1966; Anderson, Connolly & Godfrey, 1971; Fitch & Morton, 1971; Lefcoe, Carter & Ahmad, 1971; Silverman & Anderson, 1972; Bierman, Kawabori & Pierson, 1975). The mean percentage drop in PEFR for 548 normal 12 year old school children after 6 minutes free running was 2,5% with the results distributed symmetrically around the

mean (Burr, Eldridge & Borysiewicz, 1974). The upper limit for the percentage fall in PEFR (mean + 2 S.D.) was defined as 10% in 19 normal children (Silverman & Anderson, 1972).

Effect of Exercise in Children with other Pulmonary Disorders

The response to exercise in patients suffering from other lung conditions has also been studied. These include children with wheezy bronchitis, "ex-wheezers", "ex-asthmatics", their relatives, patients suffering from cystic fibrosis and tuberculosis.

An increased bronchial lability has been found in children who suffered from "wheezy bronchitis" in infancy or were currently suffering from it (König, Godfrey & Abrahamov, 1972; Burr, Eldridge & Borysiewicz, 1974). This increased bronchial lability was also demonstrated in adults who had had asthma as children, but who had been symptom-free for a long time (Jones & Jones, 1966). These interesting findings were further demonstrated in studies involving the relatives of children who suffered from wheezy bronchitis and asthma. Thirty-eight relatives of 16 infants with wheezy bronchitis were investigated and an increased bronchial lability was found in 29% of healthy relatives following exercise. Only 5% of relatives of a control group had increased bronchial lability (König & Godfrey, 1973b).

In a further study, 65 relatives of 12 asthmatic children were investigated. Of these, abnormal bronchial lability was found in 33% of relatives with a past history of asthma, in 40% of those suffering from hayfever and in 32% of clinically healthy individuals. Some suffered

from active asthma and had a tendency to bronchoconstriction, while the others had a tendency to bronchodilatation (König & Godfrey, 1973a). In all the groups studied, bronchial lability consisted more of bronchodilatation than bronchoconstriction. In no case was the percentage fall from resting values more than 10%. A marked post-exercise fall is found only in the asthmatic subject.

In a study of children suffering from cystic fibrosis, they were shown to have increased bronchial lability post-exercise (Day & Mearns, 1973; Counahan & Mearns, 1975). Bronchodilatation was the main form of the bronchial lability in these children and bronchoconstriction was mild. In another study, groups of patients suffering from cystic fibrosis, tuberculosis as well as normal controls were indistinguishable from one another in the drop of FEV_1 after exercise (Heimlich, Strick & Busser, 1966).

Although abnormal bronchial lability has been seen in other conditions, the characteristic response of mild bronchial dilatation during exercise and severe bronchoconstriction after exercise has been found only in asthmatic subjects.

Incidence of Exercise-Induced Asthma

It is intimately related to the type, duration and severity of the exercise. By subjecting asthmatic children to appropriate exercise, however, nearly all should develop exercise-induced asthma. Failure to provoke it seriously questions the diagnosis of asthma (Jones, Wharton & Buston, 1963; Godfrey, 1974).

TABLE II.7

INCIDENCE OF EXERCISE-INDUCED ASTHMA AFTER RUNNING

AUTHOR	EXERCISE STIMULUS AND DURATION	INCIDENCE
Jones et al (1962)	Free running - 8-12 min	90%
Jones et al (1963)	Free running - 5-10 min	94%
Fitch & Morton (1971)	Treadmill running - 8 min	72,5%
Silverman & Anderson (1972)	Treadmill running - 6 min	73%
Godfrey (1974)	Free running	97% if test repeated
Eggelston & Guerrant (1976)	Treadmill running - 5 min	71%

The incidence of exercise-induced asthma after a single stimulus varies little in controlled trials using standardised appropriate exercise. (Table II.7) The mean percentage of asthmatic subjects who developed exercise-induced asthma following a single exercise stimulus (running) over 5 studies was 80,1% with a range of 71% to 94%. The study of Godfrey, 1974 was excluded from the calculations because it represented repeated testing, while the others only one single stimulus.

The Mechanism by which Exercise provokes Bronchoconstriction

The bronchoconstriction which occurs in exercise-induced asthma has been shown to be in the larger airways, small airways obstruction being usually mild (Mildon et al, 1974; Cropp & Schmultzler, 1975). The efficacy of sympathomimetic drugs in relieving the bronchial obstruction (Godfrey & König, 1975; Anderson et al, 1976) indicates that there is bronchial smooth muscle contraction. The protective effect of sodium cromoglycate (Silverman & Andrea, 1972; Godfrey & König, 1975) suggests that there may be release of chemical mediator from mast cells during, or shortly after stopping the exercise, resulting in exercise-induced bronchoconstriction. Various factors have been put forward as the triggering mechanism of the exercise-induced bronchoconstriction, including hyperventilation, hypoxaemia, hypocapnia and acidosis (Herxheimer, 1946; McNeill et al, 1966; Crompton, 1968; Rebuck & Read, 1968; Seaton et al,,1969; Fisher et al, 1970; Chan-Yeung, Vyas & Grzybowski, 1971). The evidence in support of these hypotheses has often been based on poorly controlled and isolated unusual cases of exercise-induced asthma. In properly controlled studies, however, where oxygen consumption, minute ventilation, heart rate and duration of work

was kept constant, no single factor could be identified and the triggering mechanism responsible for exercise-induced bronchoconstriction is still unknown (Beaudry, Wise & Seely, 1967; Anderson, Silverman & Walker, 1972; Vassallo, Gee & Domm, 1972).

The severity of the exercise-induced asthma as indicated by a percentage fall from pre-exercise resting levels in FEV_1 and PEFR is largely independent of the absolute level of the resting lung function (Poppius et al, 1970; Sly, 1970; Silverman & Anderson, 1972). Treatment of an asthmatic child may improve his lung function as reflected by an increase in PEFR values pre-exercise, but the magnitude of the percentage fall post-exercise is not affected (Silverman et al, 1972).

No correlation could be shown between the drop in FEV_1 post-exercise and serum IgE levels (Morton, Turner & Fitch, 1973). A good correlation has been found between histamine provocation and exercise-induced asthma in the identification of asthmatic children (Mellis et al, 1977). Histamine provocation as a means of identifying asthmatic children is, however, an impractical procedure for use in a field survey involving large numbers of children.

Conclusions

1. Exercise-induced asthma (E.I.A.) is an almost universal finding in the asthmatic child. Asthmatic children may deny the presence of E.I.A., but this only reflects their voluntary restriction of those exercises which will induce bronchospasm.

2. E.I.A. is unique for the asthmatic child and is a non-invasive method of distinguishing the asthmatic from the non-asthmatic child. Failure to provoke bronchoconstriction by appropriate exercise should raise serious doubts of the diagnosis of asthma. The Vitalograph for measuring FEV_1 and the Wright Peak Flow Meter for measuring PEFR are simple and easy to use. Both measurements are sensitive and reliable in detecting exercise-induced asthma.

3. Some types of exercise provoke E.I.A. more readily than others. Free running for 6 - 8 minutes at a steady pace is the most asthmagenic. The duration and pace of the running should be such to raise the heart rate to 170 to 180 beats per minute. Maximum bronchoconstriction occurs 3 - 5 minutes after stopping the exercise.

4. Approximately 80% of asthmatic children would be expected to develop exercise-induced asthma after a single test of free running for 6 to 8 minutes.

5. If resting FEV_1 and PEFR values are well below the predicted normal for height, a child should not be made to do exercise which might provoke bronchospasm, since a further drop could cause acute respiratory embarrassment.

6. It is not influenced by pulmonary disorders other than asthma.

7. The time of day or year has no significant influence.

SECTION III

SUBJECTS AND METHODS

CHAPTER 1 : SUBJECTS

Selection of Children

Two samples of Xhosa children were studied, one living in an urban and the other in a rural environment. The object was to study asthma in Black children not only in terms of the broad division between urban and rural conditions, but also in terms of the home environment and lifestyle which might influence the development of asthma. To eliminate racial differences, only children from the Xhosa nation were selected.

No prevalence rate for asthma exists for Black children. Based on a hypothetical prevalence rate of 5 per 1000, it was estimated that the minimal acceptable sample size was 500 children.

The following were the requirements for the study:

1. The children's ages had to be between 6 and 9 years, inclusive
2. They should have lived for the last 4 or more years in the designated area
3. The children's parents had to be Xhosas
4. Subjects had to be capable of running on flat ground for 6 minutes and of performing pulmonary function tests. All children with physical handicaps, eg. cerebral palsy or moderate to severe mental retardation, who were unable to perform these, were excluded. No number of the latter group was kept.

Consent

The study was explained to all parents in whose family an eligible child was found. Informed consent was obtained in each case from a parent or legal guardian of the child. The procedures were also explained to each child individually and any child who was unco-operative or refused to participate was not accepted for the study. Of the Guguletu sample 7 children and of the Tsolo District sample 12 children who were unco-operative had to be excluded from the study.

CHAPTER 2 : THE COMMUNITIES AND THEIR SETTING

THE URBAN COMMUNITY : GUGULETU

Guguletu, a black township forming part of the greater metropolitan area of Cape Town, was selected for the purpose of the study.

(a) Geographic situation

Guguletu is situated 22km from the centre of Cape Town (latitude 33°55'S and longitude 18°28'E) on a flat plain 30m above sealevel - the so-called Cape Flats. Cape Town is a coastal city with a winter rainfall.

(b) Size

Surface area	Approximately 627 hectares
Number of houses	130 x 2 roomed
	7226 x 4 roomed
	20 x 5 roomed
	7376 Total
Number of inhabitants	28 671 males
	27 033 females
	55 704 Total

Roads: All main and subsidiary roads, totalling approximately 7 235 km, are cement-surfaced.

Schools:	17 primary and secondary schools
Hospital and Welfare Organisations:	1 Day Hospital
	1 Polyclinic
	1 Tuberculosis clinic
	4 Créches
	1 Day Centre for palsied children
	3 Community Centres for multifarious activities of a social welfare and like nature
Churches:	34 for various religious denominations are scattered throughout the area
Miscellaneous amenities:	114 shops and businesses serving the community, including banks and a post office, representing all the facilities of a modern industrialised western society
	Recreational facilities exist for a wide variety of activities including, amongst others, 3 stadiums, swimming pools, tennis courts, a library, a civic centre and open spaces as recreational sports fields.

(c) Housing

All houses are permanent dwellings built with bricks and mortar. In terms of the relevant legislation (Government Notice R1036)[1] it is permissible for the tenant to improve the property upon request, at his own expense and initiative. Electricity is not routinely installed,

[1]Government Gazette Extra ordinary R1036, 14 June, 1968

but installation and connection is permitted upon application, the full cost being borne by the applicant. Some 580 houses have been wired for electricity in this way. Each house has water laid on and all sewage is waterborne.

The allocation of houses to particular families is governed by regulation[1] and is not made on social or economic distinctions.

(d) Selection of Subjects

Thirteen hundred houses were randomly selected from the addresses of all the houses supplied by the Director of the Bantu Affairs Administration Board, Peninsula Area. A field worker visited each sampled house and recorded the names of all eligible children. Certain units had to be excluded for various reasons, such as being unable to find the house, receiving no co-operation or there being no eligible children.

All eligible children from each of the selected houses were included in the final sample. Children were examined more or less in the sequence in which their names appeared in the randomly drawn list. There was no substitution of one child's name for another or one house for another. If a house could not be included in the sample, the next on the random list was used.

After the field workers had visited the first hundred or so houses, the selected children were medically examined while the field workers continued with their visits. These investigators who had visited the houses of the respective groups of children called for medical examination were present to assist in identification and interpretation.

[1]Government Gazette No. 4971, 30 January, 1976

The final sample consisted of 694 children from 416 families.

THE RURAL COMMUNITY : ST. CUTHBERT'S MISSION AREA

For the purpose of the study all locations in a radius of 9 km from the St. Cuthbert's Mission, Tsolo District, Republic of Transkei, were selected.

(a) Geographical setting

The Mission is situated in rural Transkei (latitude 31°19'S and longitude 28°39'E) amongst rolling hills of grasslands. Umtata, capital of the Republic of Transkei, is 51 km away by road from the Mission. Of this distance, 37 km is tarred, and the remaining 14 km is gravel road.

The Mission falls in the Tsolo District and is 14 km from the village of Tsolo, over poor gravel roads. Attached to the Mission is a school and a hospital, the St. Lucy's Hospital. Apart from providing for these needs of the community, it has not changed the lifestyle of the people of the district, who still live according to traditional and often tribal customs. Nurses in training live at the hospital, but all other people live in the locations. The district is 1020m above sealevel and has a summer rainfall.

(b) Locations

The people of Tsolo district live in so-called locations. These locations consist of families each living in their own homestead and forming small communities of variable sizes. The locations are from 2 to 5 km apart

and 8 locations fall within a radius of 9 km from St. Cuthbert's Mission. The sizes in terms of number of families and distance from the Mission for the 8 locations are as follows:

Qudu & Nkwanca	88 families	0,5 km
Bantubabi	57	2,0
Jojweni	35	3,0
Zincuka	323	4,5
Nohmala	113	5,0
Belesi	203	6,0
Elujecweni	77	9,0
Total	896 families	

Only 3 locations have a shop supplying only the basic needs of the community. All other business must be done in Tsolo or Umtata. Each location has its own school.

(c) Homes (Kraals)

The home or homestead of a family, also known as a kraal, differs from the urban concept of a house. It consists of from one to seven separate rondavels or rectangular huts built closely together. Each of these rondavels or huts is used for different purposes, eg. a sleep hut, a cooking hut, and a living hut.

The rondavels are built of mudbricks with a floor of cow dung and a thatched roof. Only in a minority of rondavels were cement bricks and zinc roofs used. The kraals or homesteads in the location are not laid out according to any plan and no roads except footpaths exist between kraals. None of the kraals have electricity. Water for the household is collected from the nearest stream and carried to the kraals by the women. No facilities exist for the disposal of sewage. Each kraal or

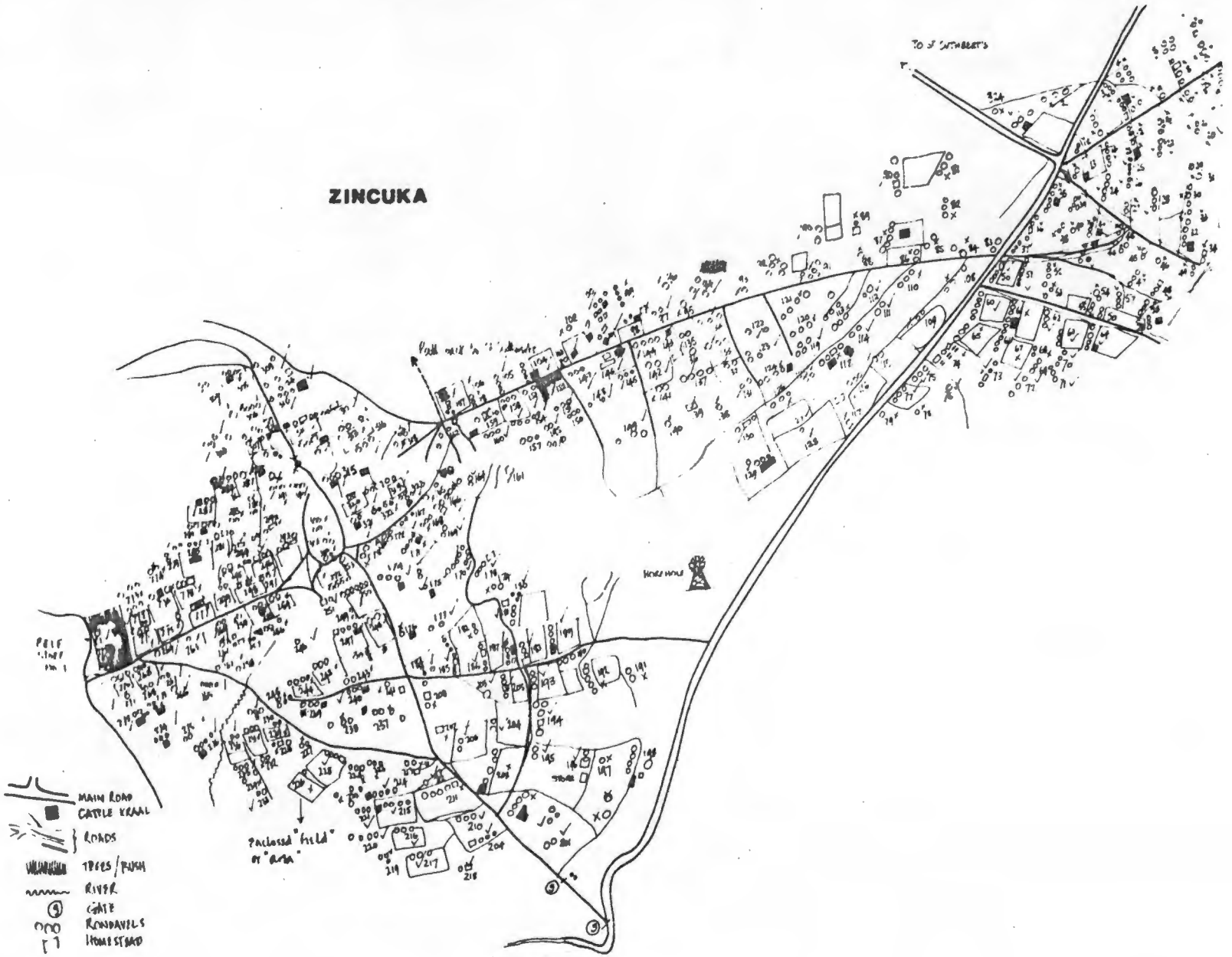

Fig. 1 : MAP OF ZINCUKA LOCATION

homestead provides for its own needs in the form of either a dugout pit toilet, or indiscriminate use of the surrounding fields.

(d) Selection of Subjects

None of the kraals or separate rondavels in any of the locations are numbered and no numbers exist for them. From aerial photographs of the area (ref.no. TAAK 733, Str.4: 0249, 0251, 0255, 0253, 0257; Str.5: 0019, 0017, 0015, 0013, 0011 and Str.6: 9909, 9907, 9905, 9903 and 9901) obtained from the Director-General of Surveys, Pretoria, each location was redrawn by hand onto a large sheet of paper. In this way each small location with its kraal as seen on an aerial photograph could be magnified many times. Separate kraals could then be identified.

Kraal-to-kraal visits were done on foot and the handdrawn map of each location was checked, amended and altered as and where necessary. Every kraal or homestead was numbered on the handdrawn maps of the different locations. Fig. 1 is an example of such a corrected, numbered map of the location, Zincuka.

It had initially been decided to select a random sample from the numbered huts, but after a preliminary kraal-to-kraal survey by field workers, it soon became apparent that the locations were sparsely populated. As this precluded random selection all eligible children from the 8 locations were studied. The final sample consisted of 671 children from 459 families.

CHAPTER 3 : METHODS

In order to determine the prevalence of asthma and explore the factors responsible for its development in the two samples, it was necessary to identify the asthmatic subjects and obtain a detailed description of the home environment and lifestyle of each family. Only in this way could meaningful results and conclusions be reached.

1. IDENTIFICATION OF THE ASTHMATIC CHILD

All children who participated in the study were subjected to an exercise stimulus and the children who demonstrated exercise-induced asthma were identified as being asthmatic.

The single diagnostic criterion for defining asthma was :

> a drop of 15% or more post-exercise in both FEV_1 and PEFR from pre-exercise values. The exercise stimulus was free running on a flat area in the open air for 6 minutes at a pace which raised the heart rate to 170 or more beats per minute.

Apparatus

Both the FEV_1 and the PEFR were measured in every case. The FEV_1 was measured on a dry spirometer, Vitalograph, using specific commercially supplied Vitalogram charts (Vitalograph Ltd, Moreton House, Buckingham, England) and the PEFR on a Wright Peak Flow Meter (Airmed Ltd, Harlow, England).

The same two instruments were used for all the recordings throughout the study. Both the instruments were calibrated twice - at the start of the urban study and at the start of the rural study.

Method of Eliciting Exercise-Induced Asthma

The children were examined in groups of 5. The pre- and post-exercise FEV_1 and PEFR recordings were done individually on each child in succession. They ran in a group of 5.

1. The Vitalograph, Wright Peak Flow Meter and the test procedure were demonstrated to all 5 children simultaneously.
2. They were shown how to inhale maximally, to place the lips around the mouthpiece and to exhale forcefully and maximally into either apparatus.
3. Disposable cardboard mouthpieces were used and a few practice blows were done by the groups.
4. Each child was then tested individually, first on the Vitalograph and immediately afterwards on the Wright Peak Flow Meter.
5. With the child standing comfortably erect and the mouthpiece adjusted to the same height as the child's mouth, a maximal breath was inhaled. The lips then were placed around the mouthpiece and a maximal forced expiration was performed.
6. During the whole procedure the child was urged to inhale as deeply as possible, and to exhale as hard, quickly and forcefully as possible.
7. The FEV_1 was recorded onto the Vitalogram chart, special care being taken to ensure that the writing stylus was at 0 for each recording.

8. The PEFR was read directly from the measuring dial of the Wright Peak Flow Meter.

9. A number of trials were done for each procedure until the child understood the technique. The best of 3 recordings was taken as the actual value. Care was taken to avoid recording values during practice fatigue.

10. When all 5 children in a group had completed the pre-exercise FEV_1 and PEFR recordings, they ran for 6 minutes on a flat area in the open air. The Guguletu groups ran around 2 tennis courts, while the Transkei groups ran round a football field. They were continuously encouraged to run as fast as possible.

11. The pulse rate was recorded immediately after 6 minutes of running in the first 200 cases and found to be over 175 beats per minute in every case. Thereafter the pulse rate was recorded on about every 50th to 75th child. It was always found to be above 175 beats per minute.

12. The FEV_1 and PEFR were then recorded again, beginning with the first child of the group at 4 minutes after stopping the exercise, so that the last child of the group completed the recordings at from 5 to 6 minutes after stopping the exercise.

13. If any child experienced a tightness of the chest with rhonchi on auscultation and a significant drop in post-exercise pulmonary test values, two puffs of fenoterol inhalant were administered and the FEV_1 and PEFR were repeated 5 to 10 minutes later.

14. The pulmonary function tests on the children from Guguletu were done under the supervision of the author by a white state registered nurse, C. Swanepoel, who spoke the Xhosa language fluently. In the Tsolo district these tests were performed by Professor H. deV. Heese and a Xhosa state registered nurse, H. Vumindaba.

Calculations

$$\frac{\text{Pre-exercise } FEV_1 - \text{Post-exercise } FEV_1}{\text{Pre-exercise } FEV_1} \times 100 \quad \text{and}$$

expressed as a percentage.

The change in PEFR was calculated in the same way.

Interpretation

A child was defined as an asthmatic if both the post-exercise FEV_1 and PEFR values dropped 15% or more from the resting pre-exercise levels. A drop of 15% or more in only one of the two measurements was not considered to indicate exercise-induced asthma.

2. ASSESSMENT OF THE CHILD AND HIS FAMILY

A socio-economic assessment of every family and history, clinical examination and special diagnostic procedures of each child included in the study was recorded.

Each home or kraal was visited individually and detailed information obtained from the mother or guardian of the eligible children. The information, obtained by the field worker, was entered onto specially structured schedules suitable for computer analysis.

(A) Family Socio-Economic Assessment

The following were obtained for each household:

(i) Average Income Ratio (A.I.R.)

In order to compare the financial status of the families, the Average Income Ratio (Batson, 1942) was adapted. The A.I.R. is the total available income of the individual members of a household calculated as a ratio of the primary household subsistence level (Potgieter, 1976) plus allowances for household fuel, lighting, washing and cleansing materials, and an allowance for rent and transport costs. This ratio is expressed as a percentage.

Interpretation of Ratios

A score of 100% designates the bare minimum basic requirements, less than 100% indicates that the household is subsisting below the bare minimum level. One hundred and fifty percent is the effective minimum level (Batson, 1942).

The A.I.R. was not calculated for Tsolo District because a considerable number of men from the selected families worked as migrant labourers and their income could not be assessed.

(ii) Household Density Ratio (H.D.R.)

This is the actual number of persons in a household calculated as a ratio of the number of persons which the Survey Occupancy Standard, Cape Town (Batson, 1944) permits for the number of rooms occupied by

the household for sleeping. The ratio is expressed as a percentage.

The Survey Occupancy Standard lays down that every person aged 10 or more years counts as an adult and the younger children as half-adults. A household of not more than $2\frac{1}{2}$ adults requires 1 room; 3 to $3\frac{1}{2}$ adults require 2 rooms; 4 to 5 adults require 3 rooms, and so on.

Interpretation of Ratios

Homes with a H.D.R. of 100% or above were termed crowded and those below 100% were termed uncrowded.

(iii) Education

The level of education of the parents and also that of paternal and maternal grandparents were recorded in 7 categories ie. post-matric, form 4-5, form 1-3, standard 3-5, sub A to standard 2, nil and unknown.

(iv) Head of the Household

One of three was established: father, mother or grandparent.

(v) Occupation of the Head of the Household

The occupation of the head of the household was recorded in 8 different categories ie. unskilled labourer, semi-skilled labourer, craftsman, clerical worker, business or professional, pensioner and unemployed.

(vi) Assessment of the Degree of Urbanisation

The degree of urbanisation was classified under three groups: the first group consisted of those families who had a home in town and

and regarded the town as their home; the second group was the country people who regarded the country as their home. The third group was made up of people who had accepted a home in town, but still had strong ties with the country.

Use of medical services were used as an added indication of the extent to which a family had adopted a western urban style of living. Three classes were used:

1. western remedies totally, excluding traditional beliefs
2. traditional remedies and beliefs only
3. a combination of the above 1 and 2

B. Personal History

When the home visit was made, information on the personal history of each child was also obtained by the field worker and entered on the specially structured schedules. The object was to obtain information on each individual child relating to allergenic contact and the way of life that might influence the development of allergic disorders. The personal history was not used in any way to identify the asthmatic child.

Factors that were investigated were sleeping habits, animal contact, exposure to maize food and pollen, dietary history during infancy ie. duration of breastfeeding, age of introduction of cow's milk and solids and current dietary intake.

C. Clinical Examination

Each child was subjected to a clinical examination and special diagnostic procedures. The findings of the clinical examination were recorded on

forms specially designed for computer analysis, under (a) allergy orientated and (b) additional findings. The clinical examination or special diagnostic procedures were in no way used to identify the child with asthma.

The children living in Guguletu were seen at the Institute of Child Health at the Red Cross War Memorial Children's Hospital, Rondebosch, Cape Town, in groups of from 6 to 20. The children of the Tsolo district were seen at St. Cuthbert's Mission in groups of between 50 and 55.

(i) Allergy Orientated Clinical Signs

1. Chest Shapes were defined according to Pappworth (1971) as:
   (a) Funnel chest: a depression of the lower end of the sternum, also called pectus excavatum
   (b) Barrel deformity: characterised by an increase in the antero-posterior diameter with a dorsal kyphosis; the ribs and clavicle are more horizontal than normal, the supra-clavicular fossae are filled in and the subcostal angle is greater than the normal 90°
   (c) Pigeon chest or pectus carinatum: marked prominence of the upper part of the sternum and adjacent costal cartilages; the lower sternum is usually also prominent but occasionally is depressed. The ribs are usually unduly sloped and gross kyphoscoliosis is common.

(ii) Auscultatory findings

Auscultatory findings were classified as normal or adventitious; adventitious sounds were defined (Pappworth, 1971) as:

(a) Rhonchi: continuous sounds which diminish on cough and are audible during the greater part of inspiration and expiration. They are produced in the bronchi and indicate partial bronchial obstruction.

(b) Wheezing: is heard without the aid of a stethoscope in patients with bronchospasm or any other obstruction in a main bronchus.

(c) Crepitations: interrupted sounds heard mainly at the height of inspiration and the beginning of expiration

(iii) Nasal Obstruction due to Mucosal Oedema

This is classified from 0 to 9. In the categories 0 - 3 , the turbinates did not touch the nasal septum. At 4, in the absence of obvious septal deviation, a turbinate just touched the septum. Categories 4 to 6 designated mild to moderate swelling of the turbinates, while 7 to 9 represented severe swelling to completely occluded nasal passages. The turbinates were judged to be swollen if one or both of them touched the nasal septum in the absence of obvious septal deviation.

(iv) Nasal Secretions

The appearance of the nasal secretions were classified as nil, clear or purulent. The amount of secretion was also graded from 0 to 9, with from 4 - 9 being significant. Nasal secretions were judged to be present if several strands of mucus adhered to the turbinates or if there was a pool of fluid on the floor of the nasal cavity (grade 4 to 9).

(v) Eczema

The skin shows manifestations of erythema, papules or lichenification, involving mainly the flexural surfaces (Sheldon, Lovell & Mathews, 1967).

D. Additional

Anthropometric Measurements

a. Standing Height was measured with the heels together and the head adjusted so that the lower rim of the orbit and the auditory canal were in a horizontal plane. The child stood with his back to a calibrated vertical steel rod and a sliding horizontal flap at right angles to the rod was lowered to just touch the top of the head. Height was measured from the floor to the level of the flap to the nearest 0,1 cm.

b. Weight was measured on a Detecto-Medic Beam Scale (Detecto Scales Inc., Brooklyn, N.Y., United States of America), which was certified accurate by the Department of Commerce, Division of Weights and Measures, Cape Town (Certificate Ref. CT9/9/C/45/57). Readings were expressed to the nearest 100g.

c. Subcutaneous fat thickness was measured with a Harpenden Skinfold Caliper (British Indicators Ltd., Acrewood Way, Hatfield Road, St. Albans, Herts, England) and recorded to the nearest 0,1mm. The skinfold thickness was measured over the left triceps muscle halfway down the upper arm between the tip of the acromion and the top of the radius. A layer of skin and subcutaneous tissue was pinched

between the finger and thumb, pulled away from the underlying muscle and then measured (Tanner & Whitehouse, 1975).

The children were dressed only in their underpants during the clinical examination and the taking of the measurements as above.

CHAPTER 4 : SPECIAL DIAGNOSTIC PROCEDURES AND LABORATORY TECHNIQUES

1. COLLECTION OF SPECIMENS

(a) Blood

From each child examined, 13 ml blood was drawn from a cubital vein, using either a standard 20 ml plastic disposable syringe, Steripath, (Monoplast (Pty) Ltd.) and a No. 19 S.W.G. needle, or the B-D Vacutainer system (Van Becton-Dickinson, Research Surgical (Pty) Ltd, 32 de Ville Street, Langlaagte). After detaching the needle, 2,5ml blood was placed in a tube containing potassium EDTA (Teklab 2,5 ml; Laboratory and Scientific Equipment Co., P.O. Box 2110, Cape Town) as coagulant and mixed thoroughly. Another 3 ml blood was placed in a 5 ml plastic tube containing 80 u heparin as coagulant and mixed gently. This latter tube was placed upright in a container with ice. The plasma was separated in a centrifuge spun at 1 000 rpm at 4°C. The plasma was then transferred to 2 ml plastic tubes and stored at -20°C.

The remaining 7,5 ml blood was placed in a 10 ml plain glass tube and left upright until clotted. The clotted specimens were then spun at 2 500 rpm for 15 minutes to separate the serum. Alloquats of 0,4 ml serum were transferred to 2 ml plastic tubes and stored at -20°C until immediately prior to the laboratory investigation procedures, when they were thawed. The time from the taking of the blood sample until the separated serum was placed in storage at -20°C never exceeded 8 hours.

Because eosinophils in peripheral blood show a diurnal variation, being lowest between 08h00 and 09h30 and returning to baseline levels between 11h00 and 12h00 (Blumberg & Buckley, 1975), all blood samples were taken after 11h00.

(b) Stools

Stool samples were collected in plastic jars with clip-on plastic tops, which were delivered to each home in Guguletu two days before the child's visit to the Institute of Child Health for the clinical examination. Stool samples from the children in the Tsolo district were collected on the day of the clinical examination.

(c) Nasal Secretions

Nasal secretions were collected from each child during the clinical examination. These were obtained by having the child blow directly onto a glass slide and spreading the film evenly on the slide. Occasionally a glass rod applicator was used to obtain specimens from the anterior part of the nasal cavity, but this was found to cause discomfort to the children.

All tubes, glass slides and plastic jars were prelabelled with the code number (cf.p.95) of each child.

The total eosinophil count in peripheral blood, haemoglobin, total leucocyte count, nasal smear and stool examination were done on the day the specimens were collected.

2. TECHNIQUES

(a) Prick Skin Test

Prick skin tests against 13 allergens were performed on each child. The allergens selected were those commonly found in the environment of a child living either in the urban or the rural area. Commercially prepared allergenic solutions were used (Bencard Prick Scratch Testing Solution) (Beecham Research Laboratories, P.O. Box 2127, Johannesburg). The specific allergens chosen were:

1. Maize pollen (Zea maize) 2,5% (4921) D1783 Bencards
2. Maize grain 10% (5104) E1681 Bencards
3. Feathers mixed 150% (3202) D0852 Bencards
4. Housedust 150% (3201) C2342 Bencards
5. Dermatophagoides pteronyssinus 1,2% (2801) E1061 Bencards
6. Dermatophagoides farinae (Culinae) 1,2% (2800) E1414 Bencards
7. Epicoccum purpurascens 10% (1702) D0446 Bencards
8. Group M3 (Moulds) Cladosporium herbarum 10% (1300) D0434 Bencards
9. Group M1 (Moulds) Alternaria tenius 10% (1100) E1100 Bencards
10. Cat fur 150% (3204) D0848 Bencards
11. Dog hair 150% (3205) E1673 Bencards
12. Group B52 (Pollens) South African Grass 2,5% D1886 Bencards
13. Milk (Cow) 10% (5202) E2478 Bencards

A positive control, histamine 1:1000 and a negative control, Control E1306 (Bencards) was used in each case.

Method

1. An area on the flexor surface of one of the child's forearms, free from any sign of infection, scars or blemishes, was cleaned with a cotton wool swab moistened with water and allowed to dry.
2. Fifteen sites, in two rows of 8, were marked on the skin with a felt tipped pen where each individual allergenic solution and the 2 controls were to be placed.
3. These sites were at least 3 cm apart.
4. Care was taken to avoid the cubital fossa, the wrist and skin over tendons.
5. A drop of the allergenic testing solution was placed on the skin beside the appropriate mark. The sequence of allergenic testing solutions was kept constant, care being taken to separate related allergens so as to avoid cross-reaction eg. between D.pteronyssinus and D.farinae.
6. The order of the solutions, starting proximally, was as follows:

maize pollen	Histamine 1:1000
feather	Group M3 moulds
maize food	Group B52 South African grass
D.pteronyssinus	Dog fur
E.purpurascens	Cat fur
housedust	milk
D.farinae	Group M1 moulds
	negative control

7. The skin was pricked through each drop of test solution, using a blunt needle to avoid piercing the skin and drawing blood. The needle was cleaned on a dry cotton swab between pricks to avoid transferring allergens from one site to another.

8. After 8 minutes the test solutions were removed from the skin, each spot being wiped individually and care being taken not to smear the fluid over the skin to another site.
9. The reaction on the skin was read 18 minutes after the skin was pricked.

Interpretation

The reaction was recorded as positive or negative.

A positive reaction was defined as a wheal of diameter 2mm greater than the negative control.

(b) Serum Immunoglobulins

These estimations were performed on the serum stored at -20°C in alloquats of 0,4 ml/tube.

(i) Measurement of IgE

The technique of radioactive immunodiffusion described by Rowe (1969) with modification (Orren & Dowdle, 1975a) was used. All IgE measurements were done in the Department of Clinical Science, Medical School, University of Cape Town.

Method

1. Agarose gel slabs measuring 200 x 200 x 1mm containing 100 wells 2,0 mm in diameter and approximately 1,5 cm apart were prepared.
2. Each slab contained sheep anti-human IgE obtained from Pharmacia, Uppsala, Sweden.

3. Each assay slab was standardised with 6 working standards (1604 u/ml, 793 u/ml, 417 u/ml, 203 u/ml, 101 u/ml and 50,5 u/ml IgE) that had been calibrated against the World Health Organisation Standard 68/341 (supplied by Rowe, W.H.O. International Reference Centre for Immunoglobulin, Lausanne, Switzerland).
4. Unknown serum samples were assayed in duplicate on each slab using 5 µl of the standard or serum per well.
5. The slab was left for 72 hours for diffusion to take place, after which the unreacted antibody was removed by washing with phosphate buffered saline.
6. The slab was then flooded with rabbit anti-sheep IgG (Orren & Dowdle, 1975a) for 18 hours.
7. Unreacted antibody was removed by washing with phosphate buffered saline and the slab was dried. It was then placed in direct contact with a radiographic film and exposed.
8. The film was developed and the diameters (D) of the rings measured.
9. A standard curve was drawn for every plate using the results obtained from the international standards. A plot of squared diameters (D^2) as a function of units of IgE introduced into the standard wells should be linear over the range of 50 to 1500 u/ml.
10. From this curve the IgE concentrations of the unknown sera in u/ml were obtained.
11. If a serum IgE concentration was found to be greater than 1600 u/ml, the serum was diluted twice with saline to 1:5 and 1:10 dilutions and the estimations repeated. The results obtained

were corrected with factors of 5 and 10 respectively.

12. The lower unit of sensitivity of the method was 50 u/ml.

(ii) Immunoglobulins A, G and M (IgA, IgG and IgM)

The method used was that based on the Mancini technique (Mancini, Carbonara & Heremans, 1965) using commercially available immunodiffusion plates, Tri-Partigen (Behring Institute, West Germany through Hoechst Pharmaceuticals (Pty) Ltd, P.O. Box 8692, Johannesburg 2000), for the quantitative determination of these immunoglobulins. The plates were as follows:

IgG (Cat.No. OTDS 03), IgA (Cat.No.OTDT 03) and IgM (Cat.No. OTDU 03).

Commercially accurate control serum or plasma was used in every plate. The controls were OTCM 03 control serum (human) for Tri-partigen and OTCO 03 control plasma (human) for Partigen.

An International reference serum, International Immunoglobulin Reference preparation 67/97, supplied by W.H.O. Reference Centre, Institut de Biochemie, 21 Rue de Bugnon, 1011 Lausanne, Switzerland, was used as a quality control. (Table III.1, p.104.)

Method

1. The Manufacturers' instructions were followed.
2. Undiluted serum was used to determine IgA and IgM while the serum for IgG and the accuracy control serum were diluted 1:10 with isotonic saline.

3. Wells 1, 2 and 3 were filled with 5 µl of the respective standard immunoglobulin dilution (IgG 1, 11, 111, Cat.No. OTRA 07; IgA 1, 11, 111, Cat.No. OTRB 07 and IgM 1, 11, 111, Cat.No. OTRC 07) and well 4 with the international reference serum.
4. Wells 5 to 12 were each filled with 5 µl of the subject's serum.
5. The plate was closed tightly and left to stand at room temperature for a diffusion time of 80 hours.
6. The diameter (D) of the precipitin rings was then measured to an accuracy of 0,1 mm using a Measuring Viewer for Immunoanalysis with a digital counter (Behringwerke AG, West Germany).

Calculation

The squared diameters (D^2) of the precipitin rings obtained from the standard dilutions were plotted against their respective concentrations in linear graph paper. A straight line intercepting the ordinate between 8,5 and 13,5 mm^2 was obtained. The values for the subject's serum was determined by reference to this calibration curve.

When IgG was determined, the value found was multiplied by the dilution factor of 10.

If the protein concentrations of the serum sample differed significantly from the normal values and the resulting precipitin ring diameters fell outside the range of the reference curve, the examination was then repeated using higher or lower dilutions of the serum sample.

Accuracy and Quality Control

The values found with the accuracy control serum or plasma were within the acceptable range for each of the immunoglobulins for every plate. The values for the International reference standards were determined on 17 occasions (Table III.1). The International Reference Preparation 67/97 contained 96,2 I.U./ml IgG, 95,3 I.U./ml IgA and 96,2 I.U./ml IgM.

(iii) Allergen-Specific Serum IgE

Allergen-specific serum IgE antibodies were determined against 10 allergens based on the Radioallergosorbent Technique (RAST) principle (Wide, Bennich & Johansson, 1967). Because of the high cost of the test, allergen-specific serum IgE was measured only in the sera of selected cases.

The selected cases were:

(a) all children identified as asthmatics
(b) 48 and 47 children each from Guguletu and Tsolo district whose serum IgE levels were the highest for each sample respectively

Commercially supplied test kits, Phadebas RAST were used (Pharmacia Diagnostics AB. Uppsala, Sweden).

The RAST quantitates the amount of circulating allergen-specific IgE in serum. Specific serum IgE was measured against the following allergens:

1. D.pteronyssinus (d1)
2. D.farinae (d2)
3. Housedust (h2)

4.	Bermuda grass	(g2)
5.	Meadow fescue	(g4)
6.	Cat epithelium	(e1)
7.	Dog epithelium	(e2)
8.	Milk	(f2)
9.	C.herbarum	(m2)
10.	A.tenius	(m6)

Method

The manufacturers' instructions were followed.

1. One reference disc was placed in each of the test tubes numbered 3 - 10.
2. One of the test discs was placed in each of the tubes numbered 11 - 300.
3. 50 µl of reference sera A - D was pipetted in duplicate onto the discs in the tubes numbered 3 - 10.
4. 50 µl of the serum from the subjects was pipetted onto the discs in the tubes numbered 11 - 300.
5. The test tubes were then covered with foil and left to stand at room temperature for 3 hours.
6. The liquid from each test tube was then removed and
7. the discs were rinsed with 2,5 ml 0,9% saline.
8. 50 µl of Anti-IgE-^{125}I solution was then pipetted into the bottom of all the tubes. Tubes 1 and 2 contained only Anti-IgE-^{125}I solution and were used to determine the total activity.
9. The tubes were again covered with foil and left overnight at room temperature.

10. The liquid was then removed and the discs washed with 0,9% saline.
11. The bound radioactivity in all tubes was measured with a Packard Auto-Gamma Scintillation Spectrometer Model 5120 (Packard Instruments (Pty) Ltd, P.O. Box 401, Bellville). Counting time of 2 minutes per tube was allowed.
12. Duplicate readings were done for the reference tubes and single readings for the subjects' sera.

Calculation of Results

(a) The mean values for the duplicate count-rates for the reference series were calculated.

(b) The allergy response for each subject's serum was classified by comparing the count-rate with the upper and lower limits obtained from the reference sera for each class of response.

(c) Evaluation of classes:

1. Count-rates above that for reference A were classified as 4 and regarded as highly positive responses.
2. Count-rates between those for references A and B were classified as 3 and regarded as strong positive responses.
3. Count-rates between those for references B and C were classified as 2 and regarded as clear positive responses.
4. Count-rates between those for references C and D were classified as 1 and regarded as borderline.
5. Count-rates below that for reference D were classified as 0 and regarded as negative responses.

(c) Total Eosinophil Count in Peripheral Blood (T.E.C.)

This was determined as follows: (Dacie & Lewis, 1975)

1. The blood (2,5 ml) in the tube containing the potassium E.D.T.A. was mixed for approximately 5 minutes on a rotator.
2. Diluting fluid 0,95 ml was added to 0,05 ml of blood from the tube, making a 1 in 20 dilution which was again mixed for about 30 seconds on the rotator.
3. The diluting fluid was made up of 5 ml 2% eosin aqueous (200 g/l), 5 ml acetone and 90 ml distilled water stored at 4°C and filtered before use.
4. A haemacytometer with improved Neubauer ruling counting chamber was filled with the mixture using a stout glass capillary tube.
5. This was allowed to settle for 5 minutes in a moist chamber consisting of a petri dish with a cover and a swab of damp cotton wool.
6. The eosinophil granules stained brightly and distinctly while lysis of the red cells and other leucocytes occurred.
7. The whole area of the counting chamber was counted microscopically.
8. The number of eosinophils was calculated as:

X = number of cells counted

1/20 = dilution

0,1 mm = depth of counting chamber

9 mm^2 = area counted

$$T.E.C. = \frac{1}{9} \times \frac{10}{1} \times \frac{20}{1} \times \frac{X}{1}$$

$$= 22 \times X/\mu l$$

Normal range = 40 - 440/µl

All eosinophil counts were performed within 4 hours of collection.

(d) Examination of Nasal Secretions for Presence of Eosinophils

Hansel's staining method (Sheldon, Lovell & Mathews, 1967) was modified to facilitate the satisfactory staining of large numbers of slides daily.

1. The slides were air dried.
2. They were then flooded with a 1:200 eosin stain (0,30 g eosin in 60 ml methyl alcohol) for 1 minute.
3. The eosin stain was washed off with water.
4. The slides were again flooded with 1:100 methylene blue stain (0,60 g methylene blue in 60 ml methyl alcohol) for 10 seconds
5. The slides were again washed off with tap water and air dried.
6. Each slide was examined microscopically.
7. At least 12 fields were examined on each slide and a mean number of eosinophils per high power field was taken.

Nasal mucus appeared blue and homogenous. Nasal epithelial cells had unlobulated blue nuclei and abundant pale cytoplasm. Neutrophils had blue-staining lobed nuclei and pale pink cytoplasm.

Eosinophils had blue-staining lobed nuclei and intensely eosinophilic cytoplasmic granules.

Results were recorded as:

negative = 0 - 9 cells per high power field
10% = 10 - 19 cells per high power field
20% = 20 - 29 cells per high power field
30% = 30 - 39 cells per high power field

Ten or more eosinophils per high power field were considered abnormal.

(e) Stools

These were examined for the presence of helminthic ova. The Zinc-sulphate ($ZnSO_4$) flotation method for concentration of ova, adapted from the direct centrifugal flotation method of Chandler & Read (1961) was used.

A $ZnSO_4$ solution with a specific gravity (S.G.) of 1,180 was used. The S.G. was checked frequently and adjusted when necessary and maintained at 1,180 (Frankel, Rietman & Sonnenwirth, 1970).

The method of $ZnSO_4$ flotation is as follows:

1. Approximately 1g of stool is thoroughly mixed with water in a 15 ml conical centrifuge tube.
2. The tube is spun for about 2 minutes at 1 000 rpm.
3. The supernatent is poured off and 10 ml $ZnSO_4$ solution added to the deposit.
4. The mixture is spun for another minute at 1 000 rpm.
5. More $ZnSO_4$ solution is added to fill the tube completely.
6. A 22 mm x 22 mm coverglass was placed over the tube which was allowed to stand vertically for 20 - 30 minutes.
7. The coverglass was removed, placed on a slide and examined microscopically.

Direct microscopic examination was used for unformed stools and stools with blood and mucus, when a specimen was spread directly onto a slide.

Helminthic ova were identified according to Jeffrey & Leach's Atlas of Medical Helminthology and Protozoology (1968).

(f) Estimation of Haemoglobin (Hb)

(i) Tsolo district specimens

The cyanmethaemoglobin method was used (Drabkin & Austin, 1932, as described by Dacie & Lewis, 1975).

Standardisation

1. A HiCN reference solution (Acuglobin, Ethnor (Pty) Ltd, New Road, Halfway House, Transvaal) with a known Hb value was used. A series of dilutions of this was made in Drabkin's modified solution (Cyanide-ferricyanide reagent).
2. The absorbance of these dilutions was measured on a Beckman Spectrophotometer, Model 25 (Beckman Instruments (Pty) Ltd, P.O. Box 963, Cape Town) with automatic flow cell accessory at a wavelength of 540 nm, using the cyanide-ferricyanide reagent as a blank.
3. The readings were plotted on linear graph paper using arithmetical scales, with absorbance as ordinate and Hb (g/dl) as abscissa. The graph should be a straight line through the origin.
4. When a dilution of 20 µl of 4C NORMAL (Coulter Diagnostic Inc., Hialeah, Florida, U.S.A.) in 4 ml of Drabkin's solution was made and the absorbance measured, the Hb values should be within the expected range.
5. In order to simplify the readings for the large number of specimens which were handled daily, a chart was drawn up from the graph, giving the Hb values from 2,0 g/dl to 19,0 g/dl with their corresponding absorbances and the Hb values were read from this in g/dl.

Method

1. Blood from the children was collected into 2,5 ml potassium E.D.T.A. tubes and rotated for 5 minutes.
2. A dilution of 20 µl potassium E.D.T.A. blood into 4 ml Drabkin's modified solution was made and left to stand for 10 minutes.
3. The absorbance of the solution was then measured on the spectrophotometer.
4. The Hb value corresponding to the absorbance was read from the chart.
5. The absorbance values of a blank of cyanide-ferricyanide reagent and a HiCN reference solution were measured at the beginning, the middle and the end of each batch of specimens tested.
6. Drabkin's solution was prepared from Aculute Diluent Pellets (ORTHO Diagnostics Division of ETHNOR, Ethnor (Pty) Ltd, New Road, Halfway House, Transvaal).

(ii) Guguletu Specimens

The specimens from the Guguletu children were all done in the routine haematology laboratory of the Department of Pathology at the Red Cross War Memorial Children's Hospital and the Institute of Child Health. The specimens were done simultaneously with routine hospital specimens.

The Hb and total leucocyte count was measured on whole blood with a Coulter Counter, Model S Sr. (Coulter Electronics Inc., 590 West Twentieth Street, Hialeah, Florida, United States of America).

Calibration was done with 4C NORMAL. The author was supplied with only the results on a printout card.

Normal range (mean $\pm$ 2 S.D.) = 13,0 $\pm$ 1,5 g/dl (Dacie & Lewis, 1975).

(g) Total Leucocyte Counts in Peripheral Blood for Tsolo District Specimens

Method (Dacie & Lewis, 1975)

1. Diluting fluid was made up of 2% (20 ml/l) acetic acid coloured pale violet with gentian violet.
2. A 1:20 dilution of the potassium E.D.T.A. blood was made up by adding 20 µl blood to 0,38 ml diluting fluid.
3. The mixture was rotated for about 2 minutes.
4. A haemocytometer with improved Neubauer ruling counting chamber was filled with the mixture using a microhaematocrit tube.
5. The diluting solution lysed the red blood cells and the leucocytes remained intact with their nuclei stained deep violet-black.
6. The leucocytes in 5 of the 1 mm^2 areas were counted. If less than 100 cells were counted, more areas were counted.

Calculation

N = number of cells counted in 5 mm^2
depth of chamber = 1/10 mm
N cells counted in (5/10) 4 mm^2 of diluted blood
Dilution = 1 : 20
Leucocyte per µl = N x 40

(h) Total Serum Protein, Serum Albumin and Serum Cholesterol

In view of the large number of samples, these estimations were done in the routine Chemistry laboratory of the Department of Pathology at the Red Cross War Memorial Children's Hospital and Institute of Child Health. Plasma from whole blood collected into the heparin-containing tubes was used for these determinations.

Estimations for the total protein and albumin in the serum were all performed on a Technicon Auto-Analyzer II (Technicon Instruments Corp., Tarrytown, New York, 10591). The estimations were done according to the instruction manual (Technicon Auto-Analyzer II) and the reagents used were commercially available (Albustrate, General Diagnostics, Division of Warner-Lambert Company, Morris Plains, New Jersey 07950; and from Technicon Instruments Corp.).

For cholesterol a commercial test "A-Gent Cholesterol Test" from Abbott Diagnostic Division (Abbott Laboratories, 820 Mission Street, So.Pasadena, Ca 91030) was used and measurements were done on a Varian Techtron UV-VIS Spectrophotometer Model 635 set at 500 nm (Baird & Tatlock, S.A. (Pty) Ltd, P.O. Box 378, Paarden Eiland).

The estimations were all done simultaneously with the routine specimens from the Red Cross War Memorial Children's Hospital by staff of the particular laboratory. The author was provided with the results.

CHAPTER 5 : PROCESSING OF INFORMATION

All the information obtained on every child and his family was entered on forms constructed for computer analysis. This information was then transferred to punch cards.

Analysis of the information was done on a UNIVAC 1100 Computer at the University of Cape Town computer centre.

Extensive use was made of the Biomedical Computer Programs Series P Program Package.[1]

A maximum likelihood chisquare test to test for differences in discreet data was used unless otherwise stated.

Logarithmic transformation was done where necessary to approximate normal distribution.

A three-way analysis of variance with age, sex and place as the factors was used to test for differences in continuous data.

A p value less than 0,001 was considered as significant, except for the three-way analysis of variance where a p value of less than 0,05 was considered significant.

The punching of the cards was done by the Institute of Biostatistics of the South African Medical Research Council.

The programming and operating of the computer were done by Dr D.J. van Schalkwyk (Ph.D) and Mr T.J. Hastie (B.Sc.Hons) of the Institute for Biostatistics of the South African Medical Research Council.

[1] Dixon, W.J. (ed) B.M.D.P. University of California Press, Berkeley, 1975

CHAPTER 6 : PLANNING AND ORGANISATION

1. SEASON

Cape Town has a winter rainfall, while Tsolo District has a summer rainfall. All house-to-house visits had to be done on foot and roads in Transkei frequently become impassable during the rainy season. All children had to run in the open as part of the assessment. The major part of the study therefore was conducted in the autumn in Guguletu and during the autumn and winter in Tsolo district.

House visits began in Guguletu on 14th February, 1976 and concluded at the end of May, 1976. In Tsolo district, the home visits started on 1st March, 1976 and were completed on 10th June, 1976. The clinical examination of the children from Guguletu started on 1st April, 1976 and the last child was seen on 14th July, 1976. All the children from Tsolo district were seen between 4th and 20th August, 1976, with an average of 52 children per day.

2. IDENTIFICATION OF SUBJECTS

Guguletu was designated Area 1 and Tsolo district Area 2.

Each house, in which a child eligible for the study lived, was given a code number from 001 to 999 for each area. These numbers were allocated sequentially at the time of the home visit.

Each eligible child in a particular home was also allotted a code number in sequence from oldest to youngest, starting from 01 for the eldest.

The final code number given to a child therefore consisted of 5 digits, eg. 57203. The first 3 digits, 572, referred to the family living in a particular house and the last 2, 03, referred to the child, who had two older siblings also fulfilling the criteria for inclusion in the study and respectively coded as 57201 and 57202. The same code number for a child was used throughout the study on all schedules or forms and specimen tubes and jars. The area of the home (Guguletu = 1, Tsolo district = 2) was also recorded on every schedule and form for each child.

During the clinical examination the child was identified according to the code number allotted to him or her. The children were transported from home either to the Institute of Child Health at the Red Cross War Memorial Children's Hospital or to St. Cuthbert's Mission for the clinical examination. On arrival, the field worker identified the child according to name and code number. The code number was then written onto a plastic identity tag which was fastened to the child's wrist.

The planning and organisation differed to some extent in the two areas and they will therefore be discussed separately.

3. GUGULETU

Permits to enter Guguletu and the list of numbered houses (street name and house number) as used by the Rates Department were supplied by the Office of the Director, Bantu Affairs Administration Board, Peninsula Area.

(a) Home Visits

The house-to-house visits were done by three state registered Xhosa nurses resident in Guguletu. All the information on the family structure and the personal history of each individual child was obtained at the home visit from the parents or legal guardian. Every completed schedule was screened before the child was accepted into the study.

The field worker advised the parents 3 to 4 days before the date the child was to be collected for the clinical examination. At the same time she left with the parents a plastic jar with clip-on top labelled with the child's code number, in which to collect a stool specimen from the child and which was to be brought by the child to the clinical examination.

The field workers were chosen specially because they were Xhosa, spoke the language of the subjects to be studied, lived in Guguletu and were known and respected in the community.

A social worker was intimately involved with the construction of socio-economic schedules and supervised collection of information by the field workers. She also screened and assessed the completed schedules.

Altogether 1 300 homes were visited. Out of these, 41 families did not wish to participate in the study, the commonest reason given being that the child was well and the parents saw no reason to participate. In all there were 416 houses included in the study in which a child fulfilled the criteria for the study, ie. one out of every three houses.

(b) Clinical Examination and Special Procedures

A taxi was hired on a contractual basis to transport the children between the Institute of Child Health and their homes. A pre-selected group of children, whose parents were advised in advance, were collected in the morning and returned home in the afternoon. A midday meal was provided, since the children spent most of the day at the Institute of Child Health.

(c) Equipment

The Institute of Child Health and the Red Cross War Memorial Children's Hospital possess all the equipment required for the study and this was made available for use by the author.

(d) Procedures

The prick skin test, pulmonary function tests, venipuncture, clinical examination and anthropometric measurements were all done in the same laboratory and the equipment used was set aside for the exclusive use of the study. The same Vitalograph and Wright Peak Flow Meter were used throughout. The chest radiographs were done by the staff of the Department of Radiology at the Red Cross War Memorial Children's Hospital.

The prick skin testing and pulmonary function tests were done by the same person viz. a white state registered nurse.

(e) Team

The team consisted of :

Social Worker

Three Xhosa field workers

State registered nurse

Author

Two paediatricians

Medical technologist from the Institute of Child Health

4. TSOLO DISTRICT

This aspect of the study posed a number of problems not encountered in the Guguletu section. The chosen community lived in rural Transkei where contact with towns was at a bare minimum over poor gravel roads. Transkei had its own government at the time, although it was not yet independent. The author lived in Cape Town, which is more than 1 300 km from Tsolo district. St. Cuthbert's Mission and St. Lucy's Hospital could not provide any facilities other than accommodation. The study had to be self-supporting in all respects - from mobile laboratories to generating electricity. In the planning and organisation, these factors had to be borne in mind.

(a) Locality

The area selected had to :

1. be rural in all respects
2. be able to provide accommodation for a team of workers from Cape Town

Dr Guy Daynes, then Medical Superintendent of St. Lucy's Hospital, kindly suggested St. Cuthbert's Mission, which fulfilled both criteria.

Accordingly the Tsolo district in which St. Cuthbert's Mission is situated was selected as the area from which the rural sample was drawn.

(b) Permits

The study was discussed with the Secretary of Health for the Transkei, Dr D.R. Arbuckle, who gave it his full support. Permits to enter a Bantu Area, BA1183, were obtained from the Secretary of Bantu Administration and Development, Pretoria, for each member of the team.

The Magistrate of Tsolo district was informed and supplied with the names of the team members who would be visiting St. Cuthbert's Mission.

Visits were paid to the headman of each location to explain the nature of the study and obtain permission and goodwill to conduct the study in his location.

(c) Kraal Visits

These visits were done on foot over rough footpaths. The information on the family structure and personal history was collected by 5 field workers. These field workers were women from the different kraals recommended by the headmen and the Hospital Matron as being wellknown, beloved and respected members of the community. If the information collected was to be meaningful, it was essential that the field workers be known and trusted by the population under study, particularly since that population was rural and many of them still lived according to tribal customs. The field workers were assisted by a medical student doing an elective period at St. Lucy's Hospital.

The author visited the Tsolo district during March and May 1976 to supervise and solve problems arising from the field work. Here too, all completed schedules were checked before a child was included in the study.

(d) Facilities

Accommodation at St. Cuthbert's Mission was available only for the period 1st to 21st August, 1976. All equipment, including radiographic facilities, had to be brought to the Mission from Cape Town.

The following is a list of equipment taken to St. Cuthbert's Mission:

1. Mobile laboratory fitted onto a 5 ton truck from the Institute of Child Health
2. Mobile laboratory with radiographic unit and developing facilities from the South African Medical Research Council, as well as all radiographic film and developing chemicals
3. Generators (4) to supply electricity
4. Centrifuges
5. Microscopes
6. Spectrophotometer
7. All test tubes, plastic jars, reagents and slides
8. Two gas refrigerators and one gas deep freeze unit
9. All syringes and needles
10. Scale, skin caliper, steel tape measure
11. Fuel, gas and paraffin, and 4 paraffin heaters
12. Vitalograph and Vitalogram charts
13. Wright Peak Flow Meter

A light pick-up van was hired to transport the children, who were collected at the fringes of the locations in the mornings, brought to the Mission and returned to the locations in the afternoons. The roads between the Mission and the locations were gravel and in poor condition. No roads existed in the locations, only footpaths. Each child was given a meal at midday, and packets of tea and sugar were given to each child as a gift for the parents.

(e) Procedures

All procedures were done when the child was brought to the Mission for the clinical examination. The skin testing, pulmonary function tests and anthropometric measurements were each performed by the same person throughout.

Specimens of frozen serum were packed in styrofoam containers with 'dry ice', transported to East London from St. Cuthbert's Mission and thence by airfreight to Cape Town. Serum IgA, IgG, IgM, total serum protein, albumin and cholesterol were determined in the various laboratories in Cape Town.

(f) Team

Apart from the 5 field workers and 2 nurse aids who acted as interpreters, all the team members came from Cape Town.

The team consisted of :

3 Paediatricians

the author

2 state registered nurses

4 medical technologists

2 radiographers

Mrs L. Shore

1 secretary

The 5 field workers and 2 nurse aids who lived locally assisted with the assembling and collection of the children each day, identified the children and acted as interpreters.

T A B L E III.1

VALUES FOR THE INTERNATIONAL REFERENCE STANDARD 67/97

AS DETERMINED IN THIS STUDY

IgG		IgA		IgM	
mg %	I.U./ml	mg %	I.U./ml	mg %	I.U./ml
980	112.7	175	104.1	83	95.4
980	112.7	175	104.1	83	95.4
960	110.4	162	96.4	84	96.6
870	100.0	161	95.8	84	96.6
900	103.5	161	95.8	-	-
920	105.8	177	105.3	78	89.7
870	100.0	177	105.3	78	89.7
960	110.4	177	105.3	78	89.7
920	105.8	163	96.9	84	96.6
940	108.1	191	113.6	75	86.2
900	103.5	184	109.4	75	86.2
860	98.9	176	104.7	73	83.9
900	103.5	168	99.9	93	106.9
900	103.5	176	104.7	85	97.7
860	98.9	168	99.9	85	97.7
860	98.9	168	99.9	79	90.8
980	112.7	168	99.9	79	90.8
mean	105,2	mean	102,4	mean	87,6
S.D.	± 5,10	S.D.	± 4,96	S.D.	± 5,80
S.E.M.	± 1,24	S.E.M.	± 1,20	S.E.M.	± 1,45

SECTION IV

RESULTS

The results will be presented in the following way:

Results of the children defined as asthmatics;

Results of all the children in 3 main categories ie. Asthma Guguletu, non-asthma Guguletu and Tsolo District. The Tsolo District group will include the single asthmatic child from that area.

Where a discrepancy between the total number of children in a sample and the number given in either a table or a figure exists, this reflects information that was not obtainable or available for that particular factor.

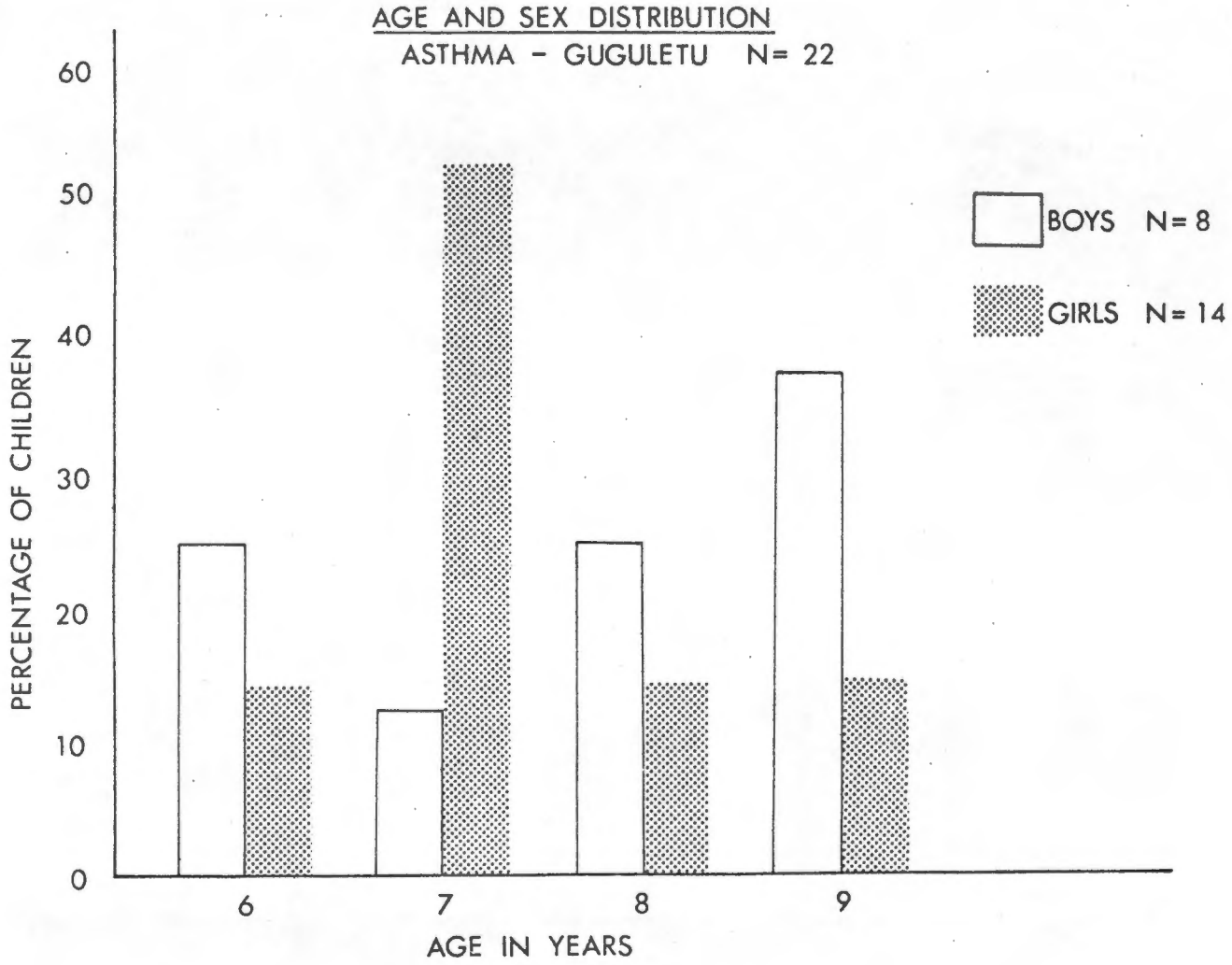

Fig. 2 : DISTRIBUTION OF CHILDREN ACCORDING TO AGE AND SEX

ASTHMA - GUGULETU

TABLE IV.1

PERCENTAGE DROP IN POST-EXERCISE FEV_1 and PEFR VALUES

FOR ASTHMATIC CHILDREN - INITIAL TESTING

Code No.	FEV_1 % DROP	PEFR % DROP
	GUGULETU	
56102	15,8	17,2
57701	51,0	50,0
42402	68,8	53,1
43701	23,5	33,3
07701	61,1	48,5
09802	28,6	19,2
21002	16,6	22,9
10303	28,6	21,9
31701	18,5	15,9
33402	38,5	50,0
51401	16,7	15,4
54702	37,5	30,0
63301	35,0	20,0
14002	20,8	29,5
33202	26,1	25,6
71303	32,1	57,7
38402	44,0	36,8
35001	25,0	18,8
17201	66,1	66,7
60401	20,3	22,2
62001	25,8	50,0
66201	35,0	20,0
	TSOLO	
00101	17,0	15,4

Guguletu children only: $\bar{x}$ = mean

FEV_1	N = 22	$\bar{x}$ = 33,4	SD = $\pm$ 15,9	SEM = $\pm$ 3,4
PEFR	N = 22	$\bar{x}$ = 32,9	SD = $\pm$ 15,8	SEM = $\pm$ 3,4

Guguletu and Transkei children:

FEV_1	N = 23	$\bar{x}$ = 32,7	SD = $\pm$ 15,9	SEM = $\pm$ 3,3
PEFR	N = 23	$\bar{x}$ = 32,2	SD = $\pm$ 15,9	SEM = $\pm$ 3,3

TABLE IV.2

PERCENTAGE DROP IN POST-EXERCISE FEV_1 AND PEFR VALUES FOR ASTHMATIC CHILDREN - REPEAT TESTING

Guguletu children only

Code No.	FEV_1 % Drop	PEFR % Drop
57701	49,3	53,8
43701	44,0	52,3
10303	42,0	53,3
54702	44,0	50,0
21002	36,0	25,5
38402	52,6	57,8
71303	18,0	23,9
17201	53,8	21,0
66201	25,7	23,2
60401	25,0	33,0
62001	33,0	32,0
35001	20,6	15,1

FEV_1

N = 12 $\bar{x}$ = 37,0 SD = ± 12,5 SEM = ± 3,6

PEFR

N = 12 $\bar{x}$ = 36,7 SD = ± 15,5 SEM = ± 4,5

$\bar{x}$ = mean

TABLE IV.3

IMPROVEMENT IN FEV_1 AFTER FENOTEROL INHALATION : ASTHMATIC CHILDREN

Code No.	Pre-Exercise FEV_1 (L/Min)	Post-Exercise FEV_1 (L/Min)	Post-Fenoterol FEV_1 (L/Min)	% Drop	% of Original
56102	-	-	-	-	-
57701	1,02	0,50	1,00	51,0	98,0
42402	0,80	0,25	0,65	68,8	81,3
43701	0,85	0,65	0,75	23,5	88,2
07701	0,90	0,35	0,60	61,1	66,7
09802	1,40	1,00	1,05	28,6	75,0
21002	-	-	-	-	-
10303	1,05	0,75	1,00	28,6	95,2
31701	-	-	-	-	-
33402	0,65	0,40	0,70	38,5	107,7
51401	1,20	1,00	1,05	16,7	87,5
54702	1,20	0,75	1,20	37,5	100,0
63301	1,20	0,78	1,14	35,0	95,0
14002	-	-	-	-	-
33202	-	-	-	-	-
71303	1,12	0,76	1,10	32,1	98,2
38402	1,25	0,70	1,10	44,0	88,0
35001	-	-	-	-	-
17201	1,18	0,40	1,23	66,1	109,8
60401	1,23	0,98	1,30	20,3	105,7
62001	1,55	1,15	1,42	25,8	91,6
66201	1,00	0,65	1,00	35,0	100,0
00101	1,47	1,22	1,35	17,0	91,8

Guguletu children Only: $\bar{x}$ = mean

% Drop	N = 16	$\bar{x}$ = 38,3	SD = $\pm$ 16,0	SEM = $\pm$ 4,0
% Original	N = 16	$\bar{x}$ = 93,0	SD = $\pm$ 11,7	SEM = $\pm$ 2,9

Guguletu and Transkei children:

% Drop	N = 17	$\bar{x}$ = 37,0	SD = $\pm$ 16,3	SEM = $\pm$ 4,0
% Original	N = 17	$\bar{x}$ = 92,9	SD = $\pm$ 11,3	SEM = $\pm$ 2,7

TABLE IV.4

IMPROVEMENT IN PEFR AFTER FENOTEROL INHALATION : ASTHMATIC CHILDREN

Code No.	Pre-Exercise PEFR (L/Min)	Post-Exercise PEFR (L/Min)	Post-Fenoterol PEFR (L/Min)	% Drop	% Original
56102	-	-	-	-	-
57701	140	70	140	50,0	100,0
42402	160	75	145	53,1	90,6
43701	210	140	180	33,3	85,7
07701	165	85	125	48,5	75,8
09802	260	210	210	19,2	80,8
21002	-	-	-	-	-
10303	160	125	190	21,9	118,8
31701	-	-	-	-	-
33402	120	60	140	50,0	116,7
51401	260	220	250	15,4	96,2
54702	250	175	240	30,0	96,0
63301	250	200	260	20,0	104,0
14002	-	-	-	-	-
33202	-	-	-	-	-
71303	260	110	250	57,7	96,2
38402	190	120	220	36,8	115,8
35001	-	-	-	-	-
17201	180	60	170	66,7	94,4
60401	180	140	200	22,2	111,1
62001	280	140	270	50,0	96,4
66201	200	160	210	20,0	105,0
00101	260	220	220	15,4	84,6

Guguletu children only:

% Drop N = 16 $\bar{x}$ = 37,2 SD = $\pm$ 16,5 SEM = $\pm$ 4,1

% of Original N = 16 $\bar{x}$ = 99,0 SD = $\pm$ 12,6 SEM = $\pm$ 3,1

Guguletu and Transkei children:

% Drop N = 17 $\bar{x}$ = 35,8 SD = $\pm$ 16,8 SEM = $\pm$ 4,1

% of Original N = 17 $\bar{x}$ = 98,1 SD = $\pm$ 12,6 SEM = $\pm$ 3,1

STATISTICAL ANALYSIS OF DIFFERENCES IN CHANGES IN FEV_1 AND PEFR VALUES

PAIRED t TEST

% of Original FEV_1 vs PEFR

Guguletu only:

t = 2,356 Df = 15 P = 0,033

Transkei and Guguletu:

t = 2,076 Df = 16 P = 0,054

PAIRED t TEST

% Drop FEV_1 vs PEFR - Initial Run

Guguletu only:

t = 0,2 Df = 21 P = 0,843

Transkei and Guguletu:

t = 0,230 Df = 22 P = 0,820

% Drop FEV_1 vs PEFR - Repeat Run

Guguletu only:

t = 0,0742 Df = 11 P = 0,9422

UNPAIRED t TEST

% Drop FEV_1 and PEFR - Initial Run vs Second Run

FEV_1 : t = 0,6717 Df = 32 P = 0,5066

PEFR : t = 0,6730 Df = 32 P = 0,5058

Initial Run N = 22

Repeat Run N = 12

TABLE IV.5

CHARACTERISTICS OF CHILDREN DEFINED AS ASTHMATIC : SOCIO-ECONOMIC

BOYS

Code No.	Age (Years)	Length of stay in area (years)	No. of rooms per home	No. of persons per homestead	Household density ratio (%)	No. of persons per bedroom
56102	6	6	4	7	120	4
57701	6	6	4	5	114	3
21002	7	7	4	7	120	4
33202	8	8	4	17	250	8
38402	8	8	4	14	240	5
35001	9	9	4	8	171	4
60401	9	9	4	8	140	3
62001	9	6	4	11	200	4

GIRLS

42402	6	6	4	6	114	6
43701	6	6	4	10	190	5
07701	7	7	4	10	160	4
09802	7	7	4	3	57	2
10303	7	7	5	11	190	4
31701	7	7	4	12	200	5
33402	7	7	4	12	126	6
51401	7	7	4	6	128	3
54702	7	7	4	13	220	5
63301	7	7	4	7	120	3
14002	8	8	4	11	200	5
71303	8	8	4	10	140	7
17201	9	9	4	7	157	6
66201	9	9	4	9	150	4
00101	8	8	4	2	060	4

Child 00101 is from Tsolo District.

TABLE IV.5 (Continued)

CHARACTERISTICS OF CHILDREN DEFINED AS ASTHMATICS : SOCIO-ECONOMIC

BOYS

Code No.	Average Income Ratio %	Electricity
56102	168	No
57701	283	No
21102	59	No
33202	60	No
38402	70	No
35001	128	Yes
60401	122	No
62001	31	No

GIRLS

42402	117	No
43701	88	No
07701	160	No
09802	84	Yes
10303	165	No
31701	33	No
33402	122	No
51401	159	No
54702	90	No
63301	40	Yes
14002	31	No
71303	170	Yes
17201	11	No
66201	71	No
00101	-	Yes

TABLE IV.6

CHARACTERISTICS OF CHILDREN DEFINED AS ASTHMATICS : SOCIO-ECONOMIC

Code No.	Head of Household & Occupation		Degree of Urbanisation	Use of Medical Services
		BOYS		
56102	Father	Clerical	Urban	Western
57701	Father	Transport	Urban	Western
21002	Father	Unskilled	Urban	Western
33202	Grandparent	Pensioner	Urban	Western
38402	Grandparent	Unskilled	Urban	Western
35001	Father	Semi-skilled	Urban	Western
60401	Grandparent	Pensioner	Urban	Western
62001	Father	Pensioner	Urban	Western
		GIRLS		
42402	Father	Unskilled	Urban	Western
43701	Father	Semi-skilled	Urban	Western
07701	Grandparent	Pensioner	Urban	Western
09802	Mother	Unskilled	Urban	Western
10303	Father	Unskilled	Urban	Western
31701	Father	Unskilled	Country	Western
33402	Father	Unskilled	Country	Traditional
51401	Father	Unskilled	Urban	Western
54702	Father	Unskilled	Urban	Western
63301	Mother	Semi-skilled	Urban	Western
14002	Father	Unskilled	Urban	Western
71303	Father	Semi-skilled	Urban	Western
17201	Grandparent	Pensioner	Urban	Western
66201	Mother	Unskilled	Urban	Western
00101	Mother	Nurse	School	Western

TABLE IV.7

CHARACTERISTICS OF CHILDREN DEFINED AS ASTHMATICS : ALLERGEN CONTACT

Code No.	Sleeping Habits	Animals in the home	Maize Contact: Home Grown and ground	Maize Contact: Home Grown and mill ground	Maize Contact: Bought
		BOYS			
56102	Bed with pillow	None	No	No	Yes
57701	Bed with pillow	None	No	No	Yes
21002	Bed with pillow	Chickens, dog, cat	No	No	Yes
33202	Bed with pillow	Dog	No	No	No
38402	Bed with pillow	Dog & Cat	No	No	Yes
35001	Bed with pillow	Dog	No	No	Yes
60401	Bed without pillow	None	No	No	Yes
62001	Bed with pillow	None	No	No	Yes
		GIRLS			
42402	Bed with pillow	None	No	No	Yes
43701	Bed with pillow	None	No	No	Yes
07701	Bed with pillow	None	No	No	Yes
09802	Bed with pillow	None	No	No	Yes
10303	Bed with pillow	Chickens, dog, cat	No	No	Yes
31701	Bed with pillow	Cat	No	No	Yes
33402	Bed with pillow	None	No	No	Yes
51401	Bed with pillow	Dog	No	No	No
54702	Bed with pillow	Chickens, cat	No	No	Yes
63301	Bed with pillow	None	No	No	Yes
14002	Bed without pillow	Dog	No	No	No
71303	Bed without pillow	Dog	No	No	Yes
17201	Bed with pillow	None	No	No	Yes
66201	Bed with pillow	Cat	No	No	No
00101	Bed with pillow	None	Yes	Yes	Yes

TABLE IV.8

EDUCATIONAL LEVEL OF PARENTS AND GRANDPARENTS : ASTHMATIC CHILDREN

	Father	Mother	Mat.G.Ma	Mat.G.Pa	Pat.G.Ma	Pat.G.Pa
			BOYS			
56102	F 4-5	Std 3-5	Unknown	Unknown	Unknown	Unknown
57701	Unknown	F 4-5	Std 3-5	F 1-3	Unknown	Unknown
21002	Std 3-5	SubA-Std2	Nil	Nil	Unknown	Unknown
33202	Unknown	F 1-3	Std 3-5	Std 3-5	Unknown	Unknown
38402	Unknown	Std 3-5	Std 3-5	Unknown	Unknown	Unknown
35001	Std 3-5	Std 3-5	Unknown	Unknown	Unknown	Unknown
60401	Std 3-5	Std 3-5	SubA-Std2	SubA-Std2	SubA-Std2	SubA-Std2
62001	F 4-5	F 1-3	Unknown	Unknown	Unknown	Unknown
			GIRLS			
42402	Std 3-5	F 1-3	F 1-3	F 1-3	Unknown	Unknown
43701	F 1-3	F 1-3	F 1-3	F 1-3	Unknown	Std 3-5
07701	Unknown	F 1-3	Std 3-5	Nil	Unknown	Unknown
09802	F 4-5	F 1-3	F 1-3	Unknown	Unknown	Unknown
10303	Std 3-5	Nil	Nil	Nil	Nil	Nil
31701	Std 3-5	Std 3-5	Unknown	Unknown	Std 3-5	F 1-3
33402	Std 3-5	Std 3-5	Unknown	Unknown	Unknown	Unknown
51401	Std 3-5	F 1-3	F 1-3	Unknown	Nil	Nil
54702	SubA-Std2	Std 3-5	Nil	F 1-3	Unknown	Unknown
63301	Unknown	F 1-3	Std 3-5	Std 3-5	Unknown	Unknown
14002	Std 3-5	Std 3-5	Unknown	Unknown	Nil	Nil
71303	Std 3-5	Post Matric	Std 3-5	Std 3-5	Std 3-5	Std 3-5
17201	F 1-3	Nil	Unknown	Unknown	Nil	Std 3-5
66201	SubA-Std2	F 1-3	Std 3-5	Std 3-5	Unknown	Unknown
00101	Std 3-5	Std 3-5	Unknown	Unknown	Unknown	Unknown

F = form Std = Standard

TABLE IV.9

FEEDING PATTERNS DURING INFANCY FOR ASTHMATIC CHILDREN

Code No.	Duration of Breast Feeding in months	Age Cow's Milk introduced in months	Age Solids introduced in months
	BOYS		
56102	6	6	5
57701	0	0	4
21002	24	24	3
33202	8	8	6
38402	3	0	4
35001	1	0	1
60401	4	0	3
62001	5	0	3
	GIRLS		
42402	0	0	4
43701	1	0	6
07701	3	3	3
09802	0	0	1
10303	5	5	4
31701	0	0	3
33402	0	0	Not known
51401	12	3	3
54702	12	5	5
63301	1	0	2
14002	4	4	3
71303	6	0	4
17201	4	2	3
66201	14	0	2
00101	12	4	3

CLINICAL EXAMINATION

ASSOCIATED ALLERGIC DISORDERS

Two children (8,7%) had evidence of associated features suggesting allergic rhinitis. The features were:

	Allergic Shiners	Nasal Crease	Nasal Secretions	Nasal Obstructions	Nasal Eosinophilia
35001	Yes	Yes	Severe clear	Severe	Present
14002	Yes	Yes	Nil	Nil	Present

Allergic shiners were seen in 9 children, but only 2 children (Nos. 35001 and 14002) had associated nasal creases.

Nasal secretions were judged severe in only one child (No. 35001) who also had severe obstruction. Mild secretions present in 4 were purulent in 2 children.

Moderate to severe nasal obstruction was present in 4 children. In only one child (No. 35001) judged to have allergic rhinitis, was it associated with increased nasal secretions.

No child had evidence of eczema.

CLINICAL EXAMINATION OF THE CHEST

Two children (8,7%) had barrel-shaped chests, while the other twency had normally shaped chests.

Rhonchi were present in 7 children (30,4%).

Abnormalities on radiographic examination of the chest were present in 4 children (17,4%).

The two children with the barrel-shaped chests (Nos. 57701 and 33402) had rhonchi on auscultation and evidence of bronchitis (No. 57701) and bronchial wall thickening (No. 33402) on chest radiography.

TABLE IV.10

CLINICAL FINDINGS IN THE ASTHMATIC CHILDREN

Code No.	Shiners	Nasal Crease	Nasal Discharge	Nasal Obstruction	Nasal Secretion
			BOYS		
56102	No	No	Nil	Nil	Nil
57701	No	No		Severe	Mild
21002	No	No	Nil	Nil	Nil
33202	Yes	No		Nil	Nil
38402	Yes	No		Mild	Nil
35001	Yes	Yes	Clear	Severe	Severe
60401	No	No	Nil	Nil	Nil
62001	No	No		Moderate	Mild
			GIRLS		
42402	No	No	Nil	Nil	Nil
43701	No	No	Nil	Nil	Nil
07701	Yes	No	Nil	Nil	Nil
09802	Yes	No	Nil	Nil	Nil
10303	Yes	No	Nil	Nil	Nil
31701	No	No	Nil	Nil	Nil
33402	Yes	No	Nil	Nil	Nil
51401	No	No	Nil	Nil	Nil
54702	No	No	Purulent	Mild	Mild
63301	No	No	Nil	Severe	Nil
14002	Yes	Yes	Nil	Nil	Nil
71303	No	No	Nil	Nil	Nil
17201	Yes	No	Nil	Nil	Nil
66201	No	No	Muco-purulent	Mild	Mild
00101	Yes	No	Nil	Nil	Nil

TABLE IV.10 (Continued)

Code No.	Eczema	Chest Shape	Auscultation
		BOYS	
56102	No	Normal	Normal
57701	No	Barrel	Rhonchi
21002	No	Normal	Normal
33202	No	Normal	Normal
38402	No	Normal	Normal
35001	No	Normal	Normal
60401	No	Normal	Rhonchi
62001	No	Normal	Normal
		GIRLS	
42402	No	Normal	Rhonchi
43701	No	Normal	Normal
07701	No	Normal	Rhonchi
09802	No	Normal	Normal
10303	No	Normal	Normal
31701	No	Normal	Normal
33402	No	Barrel	Rhonchi
51401	No	Normal	Rhonchi
54702	No	Normal	Normal
63301	No	Normal	Normal
14002	No	Normal	Normal
71303	No	Normal	Normal
17201	No	Normal	Rhonchi
66201	No	Normal	Normal
00101	No	Normal	Normal

TABLE IV.11

NUTRITIONAL STATUS OF THE ASTHMATIC CHILDREN

Code No.	Weight kg	Height cm	Triceps skin-fold thickness mm	Total Serum Protein g/ℓ	Serum Albumin g/ℓ
		BOYS			
56102	18,6	110,6	9,4	75	40
57701	20,0	113,5	7,2	78	36
21002	Not done	Not done	8,6	78	42
33202	17,3	118,8	6,2	71	35
38402	26,6	116,0	7,2	74	39
35001	28,0	129,0	7,6	77	43
60401	21,0	125,0	5,8	76	41
62001	31,7	133,0	6,4	74	42
		GIRLS			
42402	15,5	103,0	9,4	72	40
43701	17,2	110,5	6,6	77	42
07701	22,0	111,0	9,2	69	43
09802	30,4	127,0	13,4	83	42
10303	20,0	123,5	12,2	82	41
31701	20,3	115,4	8,0	82	38
33402	17,5	110,8	8,8	79	41
51401	34,0	125,0	16,0	79	42
54702	25,0	121,0	10,4	84	41
63301	20,0	115,5	7,4	78	41
14002	20,0	119,0	5,0	73	42
71303	28,6	133,0	13,0	84	40
17201	25,0	128,0	10,4	76	42
66201	25,0	123,0	11,8	80	39
00101	26,4	123,0	8,7	59	37

TABLE IV.12

CHARACTERISTICS OF CHILDREN DEFINED AS ASTHMATICS : LABORATORY INVESTIGATIONS

Code No.	Total Eosinophil Count per µl	Serum IgE µ/ml	Parasitic ova in faeces	Radio-graphy of chest	Nasal Eosino-philia
56102	1088	5393	T.trichiura	Normal	Negative
57701	1000	9275	T.trichiura	Bronchitis	Negative
21002	711	109	T.trichiura	Normal	Negative
33202	1177	5900	A.lumbricoides & T.trichiura	Normal	Negative
38402	444	4727	A.lumbricoides & T.trichiura	Normal	Negative
35001	1288	1451	A.lumbricoides & T.trichiura	Normal	Positive
60401	533	245	No stool	Normal	Negative
62001	Not done	1371	A.lumbricoides & T.trichiura	Normal	Negative
42402	1422	2405	A.lumbricoides & T.trichiura	Normal	Negative
43701	1066	345	A.lumbricoides & T.trichiura	Normal	Negative
07701	333	128	A.lumbricoides & T.trichiura	Normal	Negative
09802	511	731	T.trichiura	Normal	Negative
10303	355	146	T.trichiura	Normal	Negative
31701	488	1377	A.lumbricoides & T.trichiura	Bronchitis	Negative
33402	2266	7439	A.lumbricoides & T.trichiura	Bronchial wall thickening	Negative
51401	866	1409	A.lumbricoides & T.trichiura	Normal	Negative
54702	688	2446	T.trichiura & H.nana	Normal	Negative
63301	488	369	A.lumbricoides & T.trichiura	Normal	Negative
14002	444	245	A.lumbricoides & T.trichiura	Normal	Positive
71303	377	1590	No stool	Normal	Negative
17201	333	2939	A.lumbricoides & T.trichiura	Bronchial wall thickening	Negative
66201	422	3506	A.lumbricoides & T.trichiura	Normal	Negative
00101	66	1547	No stool	Calcified hilar glands	Negative

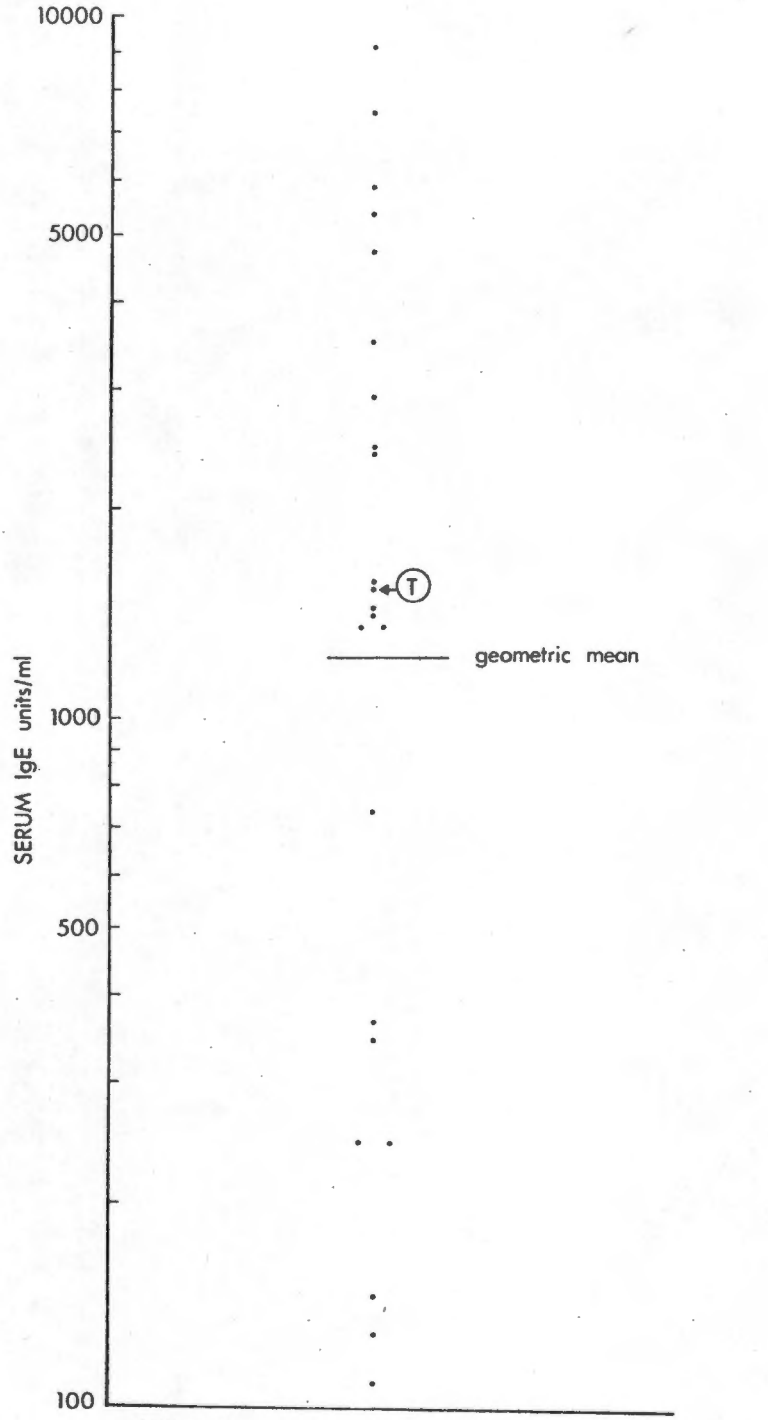

Fig. 3 : SERUM IgE LEVELS IN 23 ASTHMATIC CHILDREN FROM GUGULETU (22) AND TSOLO DISTRICT (1)

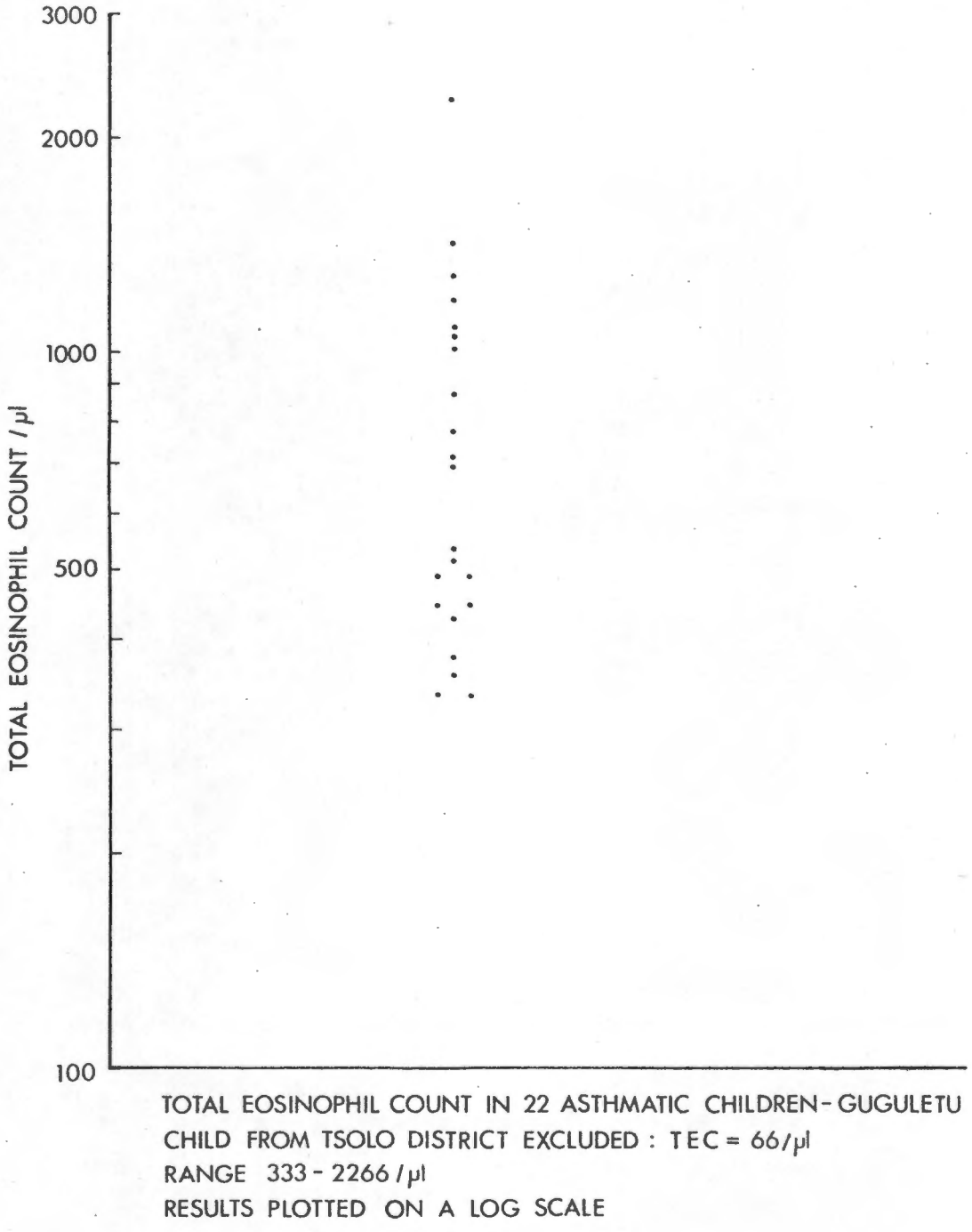

Fig. 4 : TOTAL EOSINOPHIL COUNT IN PERIPHERAL BLOOD OF 22 ASTHMATIC CHILDREN FROM GUGULETU

TABLE IV.13

RESULTS OF PRICK SKIN TESTING IN THE ASTHMATIC CHILDREN

Code No.	ALLERGENS											
	M.P.	M.F.	Grass	Feathers	Cat	Dog	Milk	D.p	D.f.	H.D.	Alt.	Clad.	Epi
56102													
57701													
21002													
33202													
38402													
35001													
60401													
62001									+				
42402													
43701													
07701													
09802													
10303													
31701													
33402													
51401													
54702													
63301													
14002													
71303													
17201													
66201													
00101													+

M.P. = maize pollen, M.F. = maize food, feathers = feathers, D.p = D.pteronyssinus
D.f. = D.farinae, H.D. = House dust, Alt. = A.tenius, Clad = C.herbarum
Epi = E.purpurascens

TABLE IV.14

SERUM IMMUNOGLOBULIN LEVELS OF THE ASTHMATIC CHILDREN

Code No.	IgA (mg/100ml)	IgG (mg/100ml)	IgM (mg/100ml)
56102	168	1720	195
57701	176	2160	168
21002	260	1720	205
33202	156	860	180
38402	160	1720	164
35001	256	1520	136
60401	281	1660	180
62001	219	1620	178
42402	176	980	145
43701	160	1660	258
07701	33	1160	156
09802	305	2160	170
10303	79	2400	142
31701	193	2250	250
33402	125	1280	229
51401	151	1280	109
54702	220	2480	266
63301	233	1420	266
14002	96	1270	210
71303	242	2140	208
17201	136	1540	199
66201	154	1800	227
00101	168	1740	315

TABLE IV.15

RAST SCORE FOR VARIOUS ALLERGENS OF THE ASTHMATIC CHILDREN

Code No.	Allergens measured by RAST									
	Be	Me	D.p	D.f	H.D.	Cat	Dog	Milk	Clad	Alt
BOYS										
56102	2	1	1	1	0	0	0	1	0	0
57701	0	0	0	0	0	0	0	0	0	0
21002	0	0	0	0	0	0	0	0	0	0
33202	0	0	0	0	0	0	0	1	0	0
38402	0	0	0	0	0	0	0	0	1	0
35001	0	2	3	3	1	0	0	0	0	0
60401	0	0	0	0	0	0	0	0	0	0
62001	0	1	3	3	1	0	0	0	0	0
GIRLS										
42402	0	0	0	0	0	0	0	0	0	0
43701	0	0	0	0	0	0	0	0	0	0
07701	0	0	0	0	0	0	0	0	0	0
09802	0	0	0	0	0	0	0	0	0	0
10303	0	0	0	0	0	0	0	0	0	0
31701	0	0	0	0	0	0	0	1	0	0
33402	1	0	0	0	0	0	0	0	0	0
51401	2	0	1	0	0	0	0	0	0	0
54702	0	0	0	0	0	0	0	0	0	0
63301	0	0	0	0	0	0	0	0	0	0
14002	0	0	0	0	0	0	0	0	0	0
71303	0	0	2	0	0	0	0	0	0	0
17201	0	0	0	0	0	0	0	1	0	0
66201	0	0	0	0	0	0	0	0	0	0
00101	0	0	0	0	0	0	0	0	0	0

Be = Bermuda grass D.p = D.pteronyssinus H.D. = House dust

Me = Meadow grass D.f = D.farinae Clad = C.herbarum

Alt = A.tenuis

RAST SCORE: 0 = negative, 1 = doubtfull, 2-4 = positive

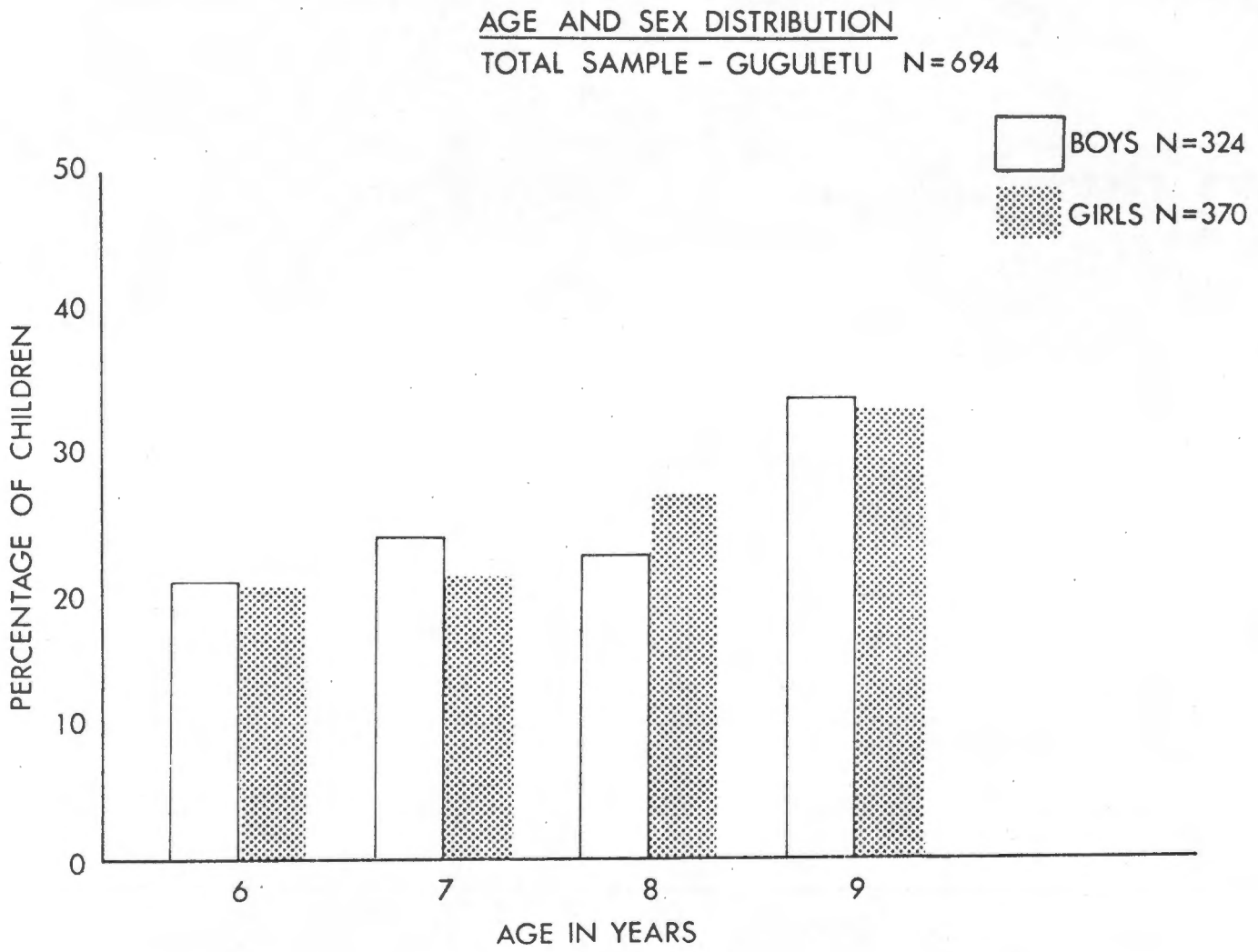

Fig. 5 : DISTRIBUTION OF CHILDREN ACCORDING TO AGE AND SEX - TOTAL SAMPLE GUGULETU

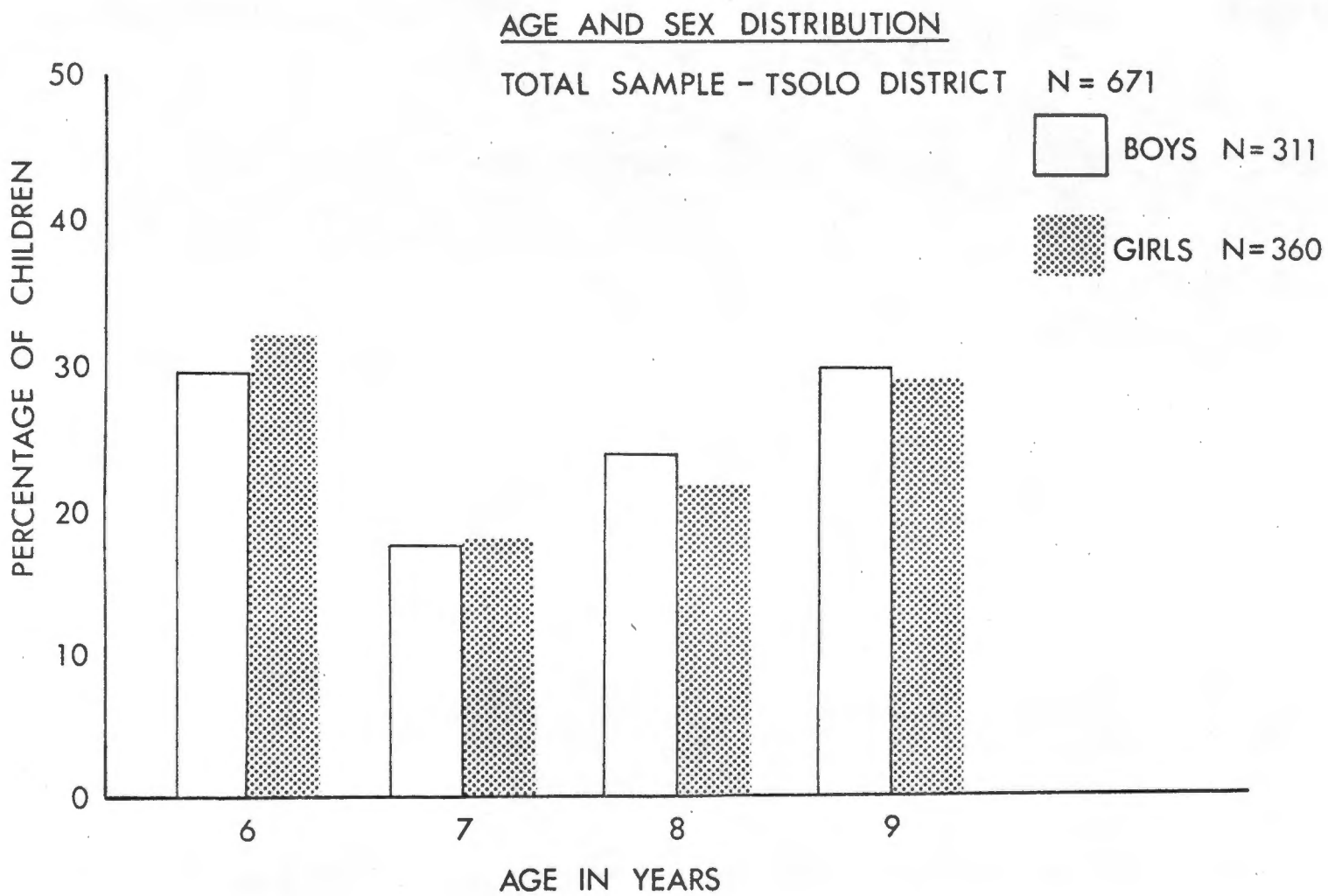

Fig. 6 : DISTRIBUTION OF CHILDREN ACCORDING TO AGE AND SEX - TSOLO DISTRICT

TABLE IV.16

PERCENTAGE DROP OF 15% OR MORE IN POST-EXERCISE FEV_1 OR PEFR FROM PRE-EXERCISE LEVELS FOR INDIVIDUAL NON-ASTHMATIC CHILDREN FROM GUGULETU (N = 45)

Percentage drop in FEV_1	Percentage drop in PEFR
17,4	-1,9
16,7	4,5
26,3	0,0
8,8	15,6
25,0	10,0
20,0	6,0
21,1	10,2
15,4	-2,6
7,4	15,9
25,0	-6,1
26,9	3,7
42,6	14,3
15,8	-4,8
20,0	-13,0
16,7	2,6
18,5	2,4
19,0	9,4
-4,3	25,0
0,0	22,9
21,7	11,4
18,5	6,0
9,1	28,6
38,9	13,5
22,2	4,2
25,0	12,5
23,5	12,5
18,8	-8,3
16,7	0,0
14,8	23,8
21,7	7,7
18,2	-14,3
12,5	15,2

Table IV.16 (Continued)

Percentage drop in FEV_1	Percentage drop in PEFR
29,0	14,3
10,0	39,6
16,0	8,0
16,7	-9,8
7,1	24,2
14,2	20,0
18,5	4,1
14,3	37,3
22,7	11,1
17,6	13,6
15,4	0,0
32,6	10,4
15,0	8,3

TABLE IV.17

PERCENTAGE DROP OF 15% OR MORE IN POST-EXERCISE FEV_1 OR PEFR FROM PRE-EXERCISE LEVELS FOR INDIVIDUAL NON-ASTHMATIC CHILDREN FROM TSOLO DISTRICT (N = 21)

Percentage drop in FEV_1	Percentage drop in PEFR
0,7	17,9
18,4	5,6
15,6	4,8
19,6	9,5
25,0	0,0
11,1	20,0
22,1	2,6
2,9	15,6
4,5	18,2
16,7	-3,6
26,3	-5,0
14,5	27,3
8,3	20,0
16,0	0,0
15,8	2,3
16,7	0,0
16,7	12,5
91,5	-20,0
17,1	7,1
-5,3	15,8
17,3	0,0

TABLE IV.18

PERCENTAGE CHANGE IN POST-EXERCISE FEV_1 AND PEFR VALUES FOR NON-ASTHMATIC CHILDREN

	Guguletu	Tsolo District
	N = 660	N = 663
Mean change in FEV_1 (%)	- 0,80%	0,26%
SEM	± 0,54	± 0,33
	N = 664	N = 664
Mean change in PEFR (%)	0,15%	- 0.17%
SEM	± 0,31	± 0,30

A negative value indicates a rise with bronchodilatation.

A positive value indicates a fall with bronchoconstriction.

Zero indicates no change.

FEV_1	Guguletu vs Tsolo District t test	p = 0,092
PEFR	Guguletu vs Tsolo District t test	p = 0,468

TABLE IV.19

AGE DISTRIBUTION OF BOYS

	6 year	7 year	8 year	9 year
Asthma - Guguletu - Children N = 8	2	1	2	3
Non-Asthma - Guguletu - Children N = 316	63	75	70	108
Tsolo Sample - Children N = 311	91	54	74	92

Asthma vs Non-Asthma Guguletu	$X^2 = 0,65$	Df = 3	p = 0,88514
Non-Asthma Guguletu vs Tsolo District	$X^2 = 9,91$	Df = 3	p = 0,01938

TABLE IV.20

AGE DISTRIBUTION OF GIRLS

	6 year	7 year	8 year	9 year
Asthma - Guguletu - Children N = 14	2	8	2	2
Non-Asthma - Guguletu - Children N = 356	72	69	96	119
Tsolo Sample - Children	115	64	77	104

Asthma vs Non-Asthma Guguletu	$X^2 = 9,49$	Df = 3	p = 0,02347
Non-Asthma Guguletu vs Tsolo District	$X^2 = 13,24$	Df = 3	p = 0,00414

RATIO BOYS TO GIRLS

Guguletu Total Sample	324 : 370 = 1 : 1,14
Tsolo District Sample	311 : 360 = 1 : 1,16
Asthma - Guguletu Sample	8 : 14 = 1 : 1,75
Non-Asthma - Guguletu Sample	316 : 356 = 1 : 1,13

TABLE IV.21

DURATION OF STAY IN STUDY AREA

BOYS

YEARS	4	5	6	7	8	9
Asthma - Guguletu N = 8	0	0	3	1	2	2
Non-Asthma - Guguletu N = 316	2	5	65	71	68	105
Tsolo district N = 311	0	3	94	52	69	93

GIRLS

Asthma - Guguletu N = 14	0	0	2	8	2	2
Non-Asthma - Guguletu N = 356	4	11	71	69	92	109
Tsolo District N = 360	3	5	113	64	74	101

TABLE IV.22

AVERAGE INCOME RATIO OF FAMILIES FROM GUGULETU

Ratio expressed as a percentage	0	20	40	60	80	100	120	140	160	180	200	250	300	350	400
Asthma - Guguletu - Families N = 22	0	1	4	2	2	3	1	3	2	3	0	0	1	0	0
Non-Asthma - Guguletu - Families N = 394	5	8	29	37	59	59	47	48	36	24	12	17	10	2	1

Average income ratio: total range $X^2 = 8,43$ Df = 14 p = 0,86555

Average income ratio: 100, 110-140, 160 and more $X^2 = 0,43$ Df = 2 p = 0,80553

Average income ratic not calculated for Tsolo District

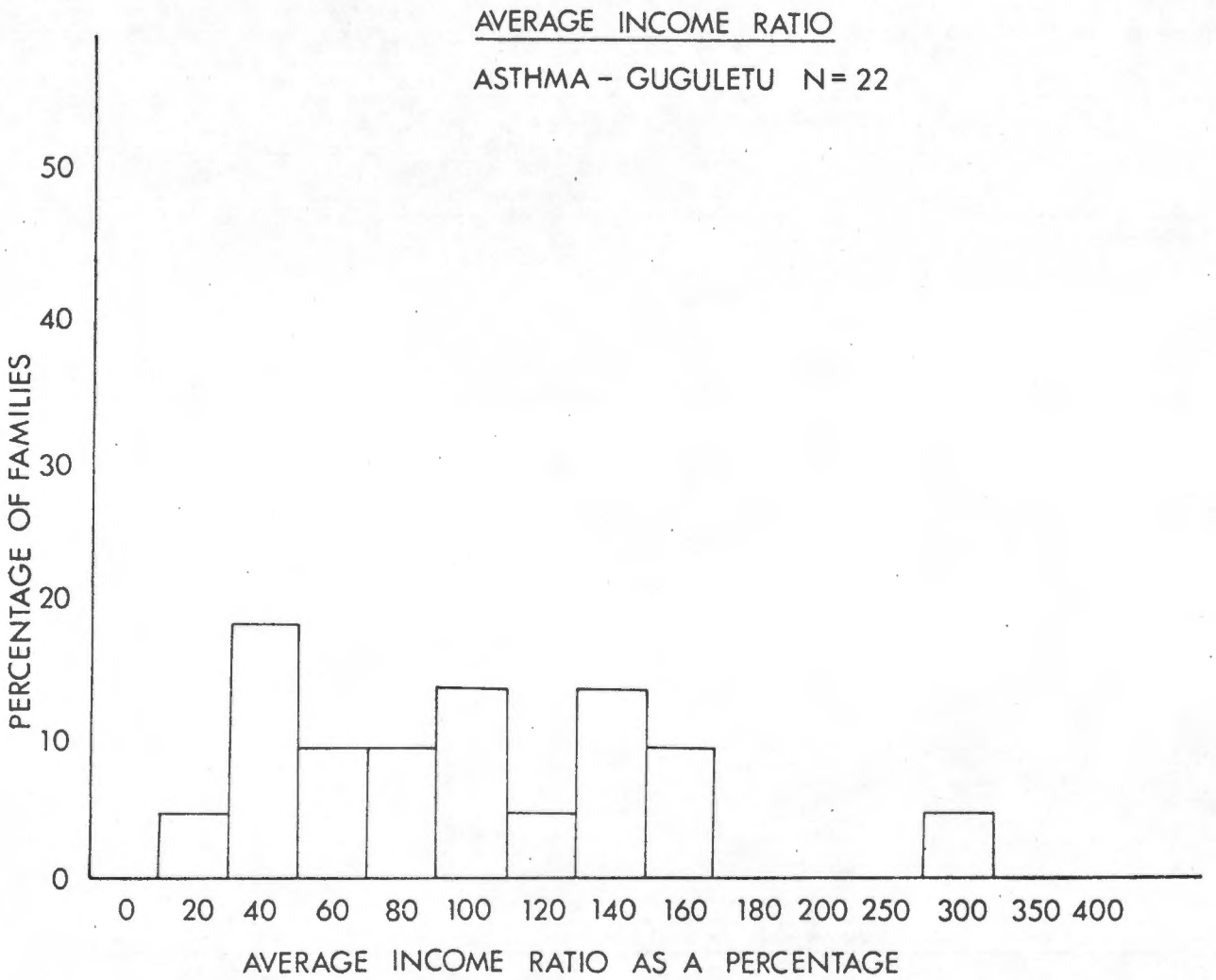

Fig. 7 : AVERAGE INCOME RATIO OF FAMILIES WITH ASTHMATIC CHILDREN FROM GUGULETU

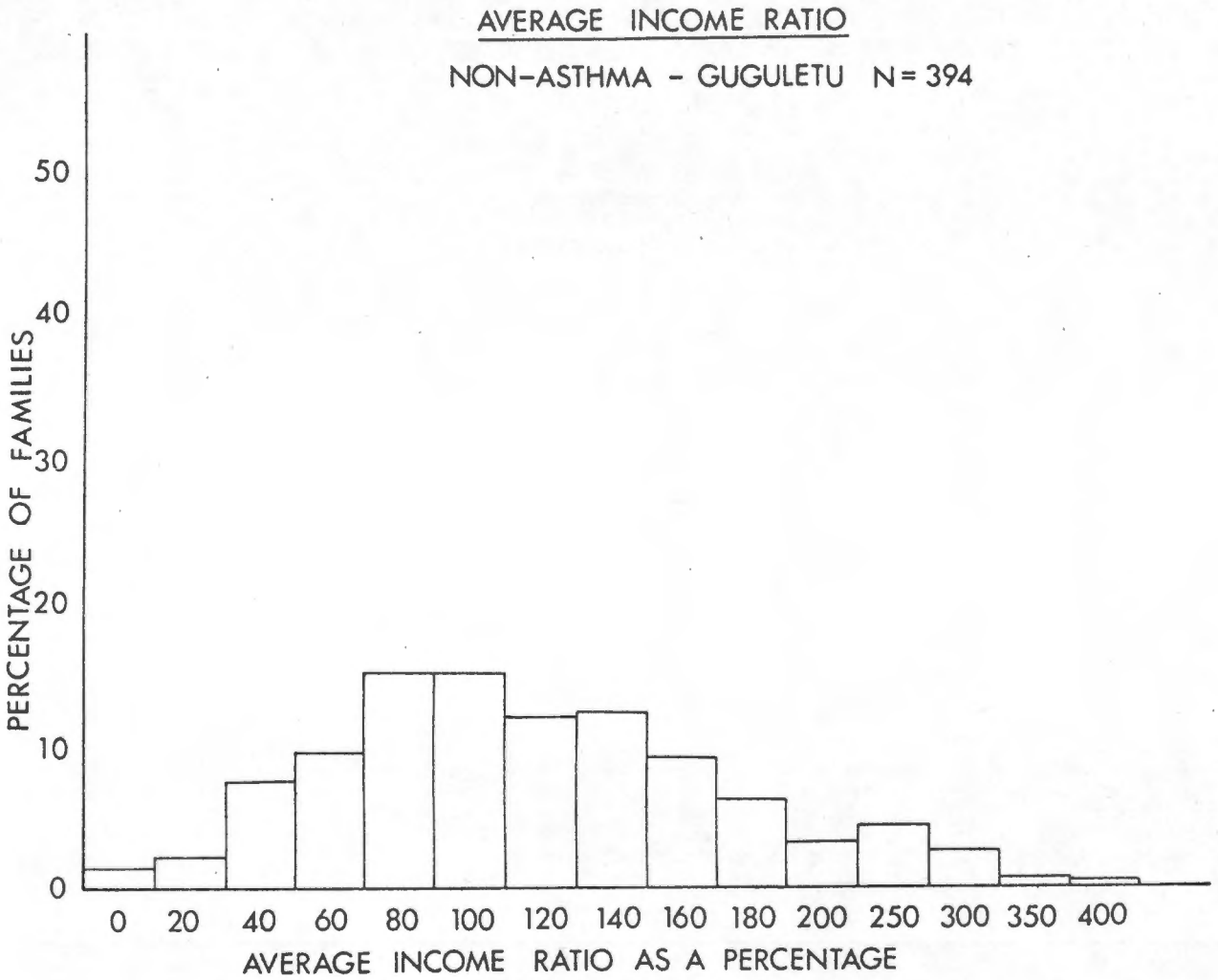

Fig. 8 : AVERAGE INCOME RATIO OF FAMILIES WITH NO ASTHMATIC CHILDREN FROM GUGULETU

TABLE IV.23

HOUSEHOLD DENSITY RATIO OF FAMILIES

Ratio expressed as a percentage	20	40	60	80	100	120	140	160	180	200	220	240	260	300	320
Asthma - Guguletu N = 22	0	0	1	0	0	5	4	3	1	5	1	1	1	0	0
Non-Asthma - Guguletu N = 394	0	0	1	9	41	54	66	75	38	48	20	16	17	8	1
Tsolo District N = 459	1	8	30	28	84	56	61	75	40	38	9	13	7	8	1

Household density ratio less than 100 and 100 or more:

Asthma vs Non-Asthma Guguletu $X^2 = 0,27$ Df = 1 p = 0,60156

Non-Asthma Guguletu vs Tsolo $X^2 = 42,40$ Df = 1 p = 0,00000

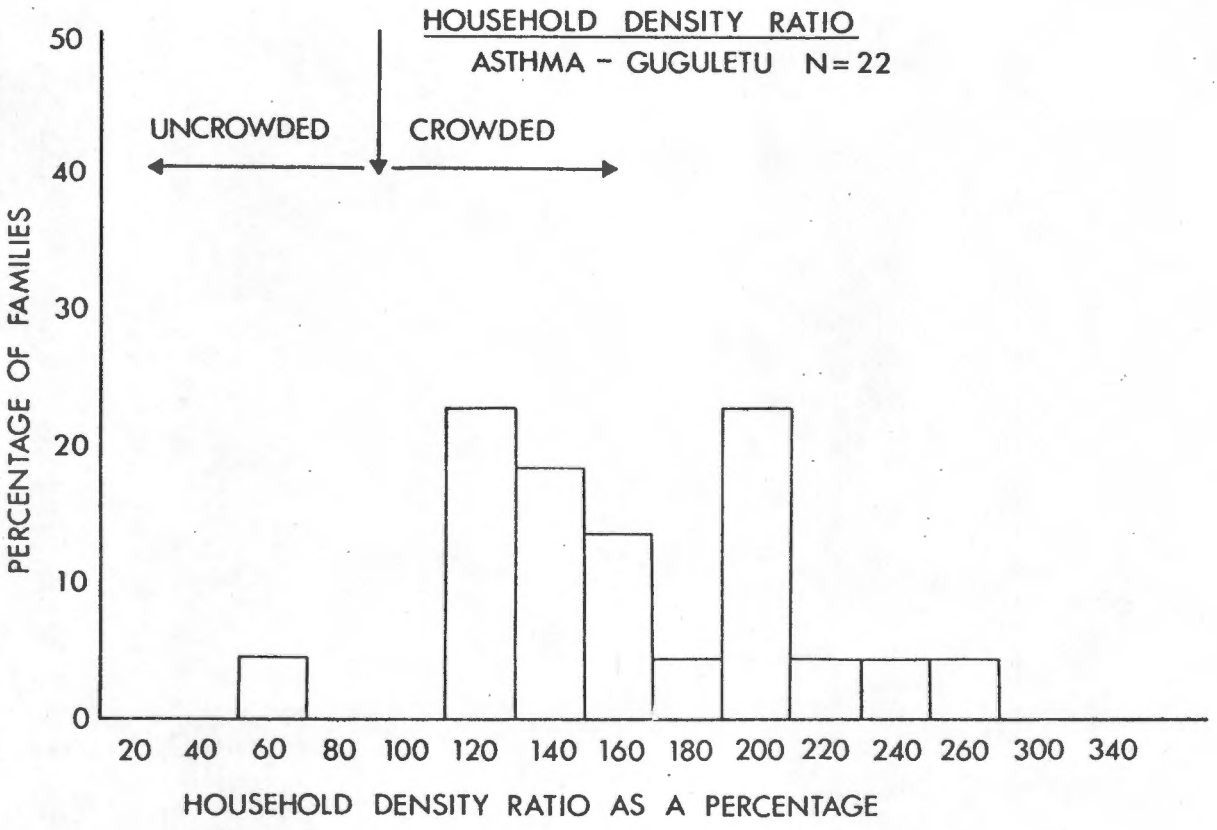

Fig. 9 : HOUSEHOLD DENSITY RATIO OF FAMILIES WITH ASTHMATIC CHILDREN FROM GUGULETU

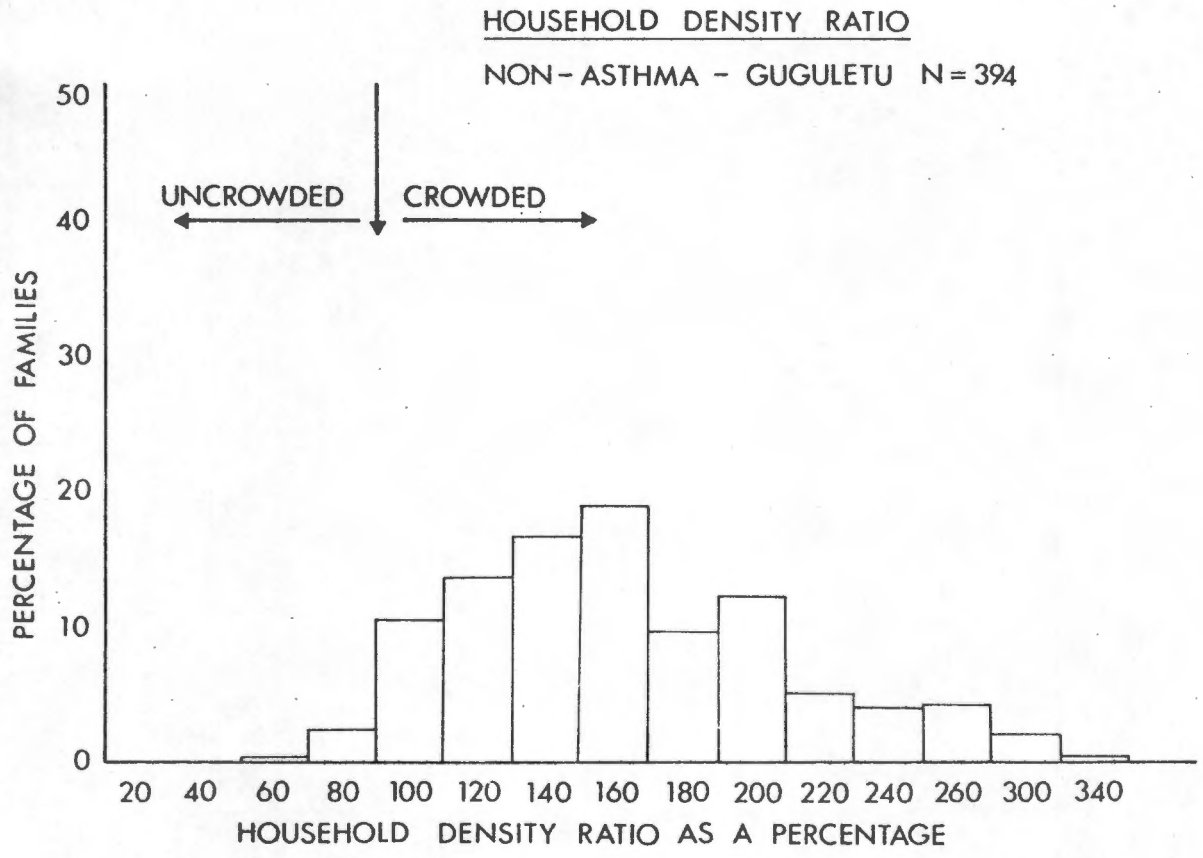

Fig. 10 : HOUSEHOLD DENSITY RATIO OF FAMILIES WITH NO ASTHMATIC CHILDREN FROM GUGULETU

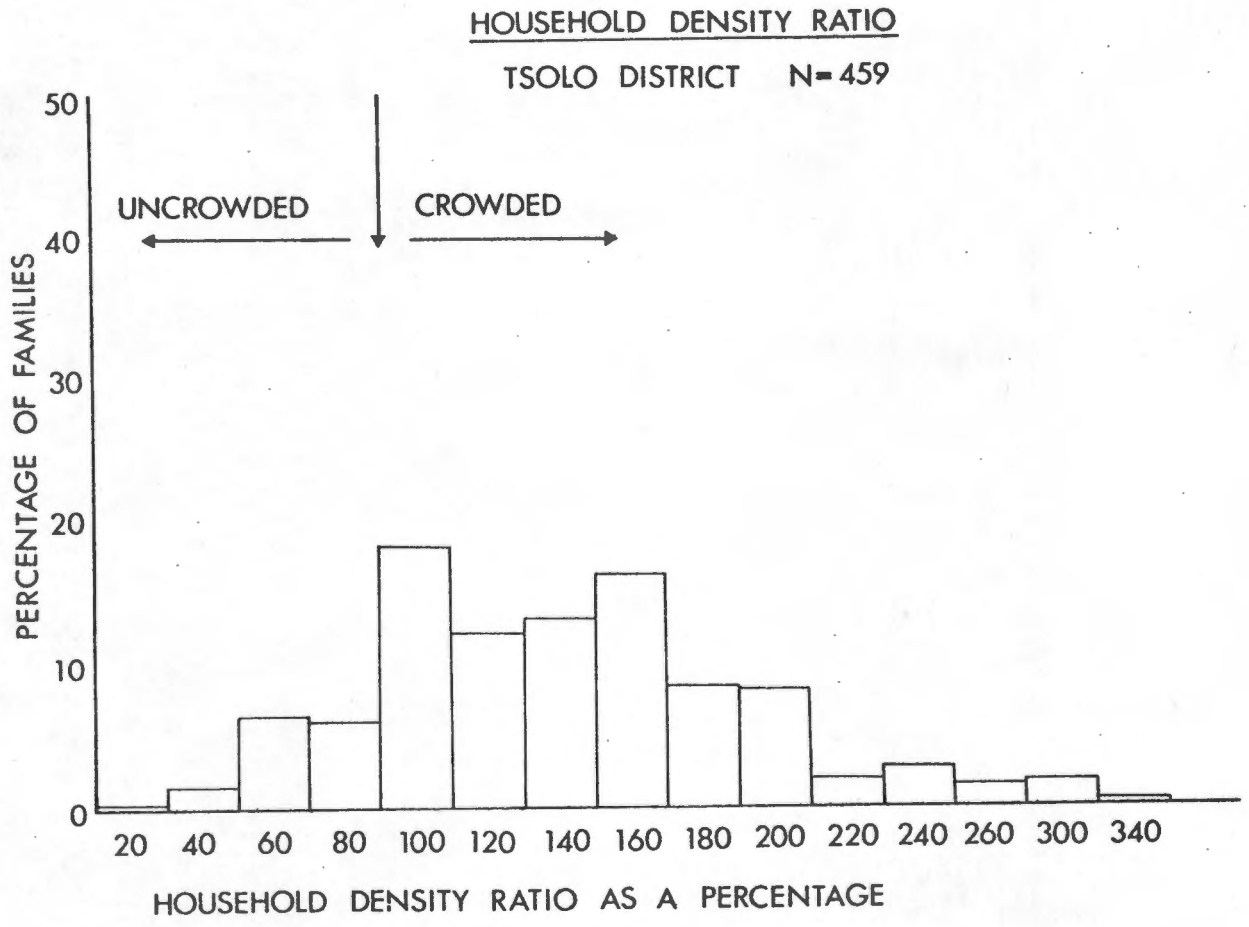

Fig. 11 : HOUSEHOLD DENSITY RATIO OF FAMILIES FROM TSOLO DISTRICT

TABLE IV.24

SIZE OF HOMESTEAD

No. of Rooms/huts	1	2	3	4	5	6	7	8	9	10	11
Asthma - Guguletu N = 22 families	0	0	0	21	1	0	0	0	0	0	0
Non-Asthma - Guguletu N = 394 families	1	7	3	334	40	2	0	7	0	0	0
Tsolo District N = 459 families	48	119	143	74	48	13	5	2	5	0	2

TABLE IV.25

NUMBER OF PERSONS SHARING BEDROOM WITH CHILD

No. of Persons per room	1	2	3	4	5	6	7	8	9	10
Asthma - Guguletu										
N = 22 Children	0	1	4	7	5	3	1	1	0	0
% of total	0	4,5	18,2	31,8	22,7	13,6	4,5	4,5		
Non-Asthma - Guguletu										
N = 667 Children	8	56	121	181	144	70	50	26	9	2
% of total	1,2	8,4	18,1	27,1	21,6	10,5	7,5	3,9	1,3	0,3
Tsolo District										
N = 668 Children	5	27	89	147	156	131	52	36	18	7
% of total	0,7	4,0	13,3	22,0	23,4	19,6	7,8	5,4	2,7	1,0

Asthma vs Non-Asthma Guguletu $X^2 = 1,44$ Df = 9 p = 0,99759

Non-Asthma - Guguletu vs Tsolo District $X^2 = 46,42$ Df = 9 p = 0,00000

TABLE IV.26

EDUCATION OF PARENTS AND GRANDPARENTS OF THE CHILDREN

PARENTS Father	Post Matric	Form 4-5	Form 1-3	Std. 3-5	SubA-Std.2	Nil	Unknown	N
Asthma Guguletu	0	3	2	10	2	0	5	22
%	0	13,6	9,1	45,5	9,1		22,7	
Non-Asthma Guguletu	2	8	76	150	25	24	109	394
%	0,5	2,0	19,3	38,1	6,4	6,1	27,7	
Tsolo District	29	18	19	138	24	210	21	459
%	6,3	3,9	4,1	30,1	5,2	45,8	4,6	
Mother								
Asthma Guguletu	1	1	9	8	1	2	0	22
%	4,6	4,6	40,9	36,3	4,6	9,1	0	
Non-Asthma Guguletu	2	7	123	206	19	18	19	394
%	0,5	1,8	31,2	52,3	4,8	4,6	4,8	
Tsolo District	16	26	32	151	26	195	13	459
%	3,5	5,7	7,0	32,9	5,7	42,5	2,8	
GRANDPARENTS								
Maternal Grandmother								
Asthma Guguletu	0	0	4	7	1	3	7	22
%	0	0	18,2	31,8	4,6	13,6	31,8	
Non-Asthma Guguletu	3	4	28	169	39	79	72	394
%	0,8	1,0	7,1	42,9	9,9	20,1	18,3	
Tsolo District	4	12	9	69	20	199	146	459
%	0,9	2,6	2,0	15,0	4,4	43,4	31,8	

TABLE IV.26 (Continued)

	Post Matric	Form 4-5	Form 1-3	Std. 3-5	SubA-Std.2	Nil	Unknown	N
Maternal Grandfather								
Asthma Guguletu	0	0	4	4	1	3	10	22
%	0	0	18,2	18,2	4,6	13,6	45,5	
Non-Asthma Guguletu	4	3	39	127	42	70	109	394
%	1,0	0,8	9,9	32,2	10,7	17,8	27,7	
Tsolo District	9	11	7	65	17	183	167	459
%	2,0	2,4	1,5	14,2	3,7	39,9	36,4	
Paternal Grandmother								
Asthma Guguletu	0	0	0	2	1	4	15	22
%	0	0	0	9,1	4,6	18,2	68,2	
Non-Asthma Guguletu	1	1	5	59	21	70	237	394
%	0,3	0,3	1,3	15,0	5,3	17,8	60,2	
Tsolo District	2	6	9	40	15	163	224	459
%	0,4	1,3	2,0	8,7	3,3	35,5	48,6	
Paternal Grandfather								
Asthma Guguletu	0	0	1	3	1	3	14	22
%	0	0	4,6	13,6	4,6	13,6	63,6	
Non-Asthma Guguletu	0	2	5	47	25	67	248	394
%	0	0,5	1,3	11,9	6,4	17,0	62,9	
Tsolo District	3	5	7	42	9	162	231	459
%	0,7	1,1	1,5	9,2	2,0	35,3	50,3	

STATISTICAL ANALYSIS OF DIFFERENCES IN EDUCATION

CHISQUARE TEST : EDUCATION OF PARENTS AND GRANDPARENTS

I. Including those with no or unknown education.

1. Paternal vs Maternal

Asthma - Guguletu	$X^2 = 14,198$	Df = 20	p = 0,82030
Non-Asthma - Guguletu	$X^2 = 543,486$	Df = 49	p = 0,00000
Tsolo District	$X^2 = 691,542$	Df = 49	p = 0,00000

2. Maternal Grandmother vs Maternal Grandfather

Asthma - Guguletu	$X^2 = 49,43$	Df = 30	p = 0,00000
Non-Asthma - Guguletu	$X^2 = 1053,557$	Df = 49	p = 0,00000
Tsolo District	$X^2 = 1886,314$	Df = 49	p = 0,00000

3. Paternal Grandmother vs Paternal Grandfather

Asthma - Guguletu	$X^2 = 54,022$	Df = 12	p = 0,00000
Non-Asthma - Guguletu	$X^2 = 1315,865$	Df = 42	p = 0,00000
Tsolo District	$X^2 = 2337,001$	Df = 49	p = 0,00000

II. Excluding those with no or unknown education.

These were analysed only for Non-Asthma - Guguletu vs Tsolo District children.

(a) All Standards ie. combined high and primary school

(b) Only high school

(c) Only primary school.

1. Education of Fathers

(a)	All Standards	$X^2 = 67$	Df = 4	p = 0,00000
(b)	High School	$X^2 = 7,02$	Df = 2	p = 0,00000
(c)	Primary School	$X^2 = 0,02$	Df = 1	p = 0,89053

2. Education of Mothers

(a)	All Standards	$X^2 = 72,08$	Df = 4	p = 0,00000
(b)	High School	$X^2 = 64,5$	Df = 2	p = 0,00000
(c)	Primary School	$X^2 = 3,86$	Df = 1	p = 0,04958

3. Education of Maternal Grandmothers

(a)	All Standards	$X^2 = 16,46$	Df = 4	p = 0,00245
(b)	High School	$X^2 = 12,89$	Df = 2	p = 0,00158
(c)	Primary School	$X^2 = 0,53$	Df = 1	p = 0,46532

4. Education of Maternal Grandfathers

(a)	All Standards	$X^2 = 27,36$	Df = 4	p = 0,00002
(b)	High School	$X^2 = 26,37$	Df = 2	p = 0,00000
(c)	Primary School	$X^2 = 0,53$	Df = 1	p = 0,46687

5. Education of Paternal Grandmothers

(a)	All Standards	$X^2 = 8,72$	Df = 4	p = 0,06855
(b)	High School	$X^2 = 1,16$	Df = 1	p = 0,55859
(c)	Primary School	$X^2 = 0,02$	Df = 1	p = 0,89504

6. Education of Paternal Grandfathers

(a)	All Standards	$X^2 = 12,01$	Df = 4	p = 0,01728
(b)	High School	$X^2 = 2,19$	Df = 2	p = 0,33458
(c)	Primary School	$X^2 = 4,51$	Df = 1	p = 0,03363

TABLE IV.27

HEAD OF THE HOUSEHOLD

Head	Asthma Guguletu	Non-Asthma Guguletu	Tsolo District
Father	14 (63,6%)	222 (56,3%)	319 (69,5%)
Mother	3 (13,6%)	33 (8,4%)	94 (20,5%)
Grandparent	5 (22,8%)	139 (35,3%)	46 (10,0%)
Total Families	22 (100,0%)	394 (100,0%)	459 (100,0%)

Non-Asthma Guguletu vs Tsolo District $X^2 = 92,02$ Df = 2 p = 0,00000

TABLE IV.28

OCCUPATION OF HEAD OF THE HOUSEHOLD

	Unskilled	Semi-skilled	Transport	Craftsman	Clerical	Business	Pensioner	Unemployed
1. FATHER								
Asthma - Guguletu N = 14	8 (57,1)	3 (21,4)	1 (7,1)	0	1 (7,1)	0	1 (7,1)	0
Non-Asthma - Guguletu N = 222	93 (41,9)	58 (26,1)	36 (16,2)	2 (0,9)	7 (3,2)	5 (2,3)	9 (4,1)	12 (5,4)
Tsolo District N = 312	60 (19,2)	112 (35,9)	21 (6,7)	9 (2,9)	8 (2,6)	33 (10,6)	23 (7,4)	46 (14,7)
2. MOTHER								
Asthma - Guguletu N = 3	2 (66,7)	1 (33,3)	0	0	0	0	0	0
Non-Asthma - Guguletu N = 33	11 (33,3)	11 (33,3)	0	0	0	3 (9,1)	2 (6,1)	6 (18,2)
Tsolo District N = 92	6 (6,5)	9 (9,8)	1 (1,1)	0	0	14 (15,2)	34 (37,0)	28 (30,4)
3. GRANDPARENT								
Asthma - Guguletu N = 5	1 (25)	0	0	0	0	0	4 (75)	0
Non-Asthma - Guguletu N = 139	37 (26,6)	26 (18,7)	5 (3,6)	2 (1,4)	1 (0,7)	3 (2,2)	28 (20,1)	37 (26,6)
Tsolo District N = 45	5 (11,1)	4 (8,9)	2 (4,4)	1 (2,2)	0	4 (8,9)	17 (37,8)	12 (26,7)

PERCENTAGES OF TOTALS IN BRACKETS

TABLE IV.29

DEGREE OF URBANISATION

	Urban Home	Country Home	Townsman with Country ties
Asthma - Guguletu	20	2	0
N = 22 families			
Non-Asthma - Guguletu			
N = 394 families	337	55	2
Tsolo District			
N = 458 families	5	452	1

TABLE IV.30

ELECTRICITY CONNECTED TO HOMES

	No	Yes
Asthma - Guguletu N = 22 families	18 (81,8%)	4 (18,2%)
Non-Asthma - Guguletu N = 394 families	359 (91,0%)	35 (9,0%)
Tsolo Sample N = 459 families	449 (97,8%)	10 (2,2%)

Asthma - Guguletu vs Non-Asthma $X^2 = 1,73$ Df = 1 p = 0,18807

TABLE IV.31

USE OF MEDICAL SERVICES

	Western only	Traditional only	Combination
Asthma - Guguletu N = 22 families	20 (90,9%)	1 (4,5%)	1 (4,5%)
Non-Asthma - Guguletu N = 394 families	318 (80,7%)	10 (2,5%)	66 (16,8%)
Tsolo District N = 456 families	284 (62,3%)	61 (13,4%)	111 (24,3%)

Asthma vs Non-Asthma Guguletu	$X^2 = 3,18$	Df = 2	P = 0,20410
Non-Asthma Guguletu vs Tsolo District	$X^2 = 49,67$	Df = 2	P = 0,00000

TABLE IV.32

SLEEPING HABITS OF THE CHILDREN

	Mats	Mattress and pillow	Mattress - no pillow
Asthma - Guguletu - Children	0	20	2
N = 22 - % of total	0	90,9	9,1
Non-Asthma - Guguletu - Children	82	502	83
N = 667 - % of total	12,3	75,3	12,4
Tsolo Sample - Children	354	213	103
N = 670 - % of total	52,8	31,8	15,4

Non-Asthma Guguletu vs Tsolo District $X^2 = 305,25$ Df = 2

p = C,00000

TABLE IV.33

ANIMAL CONTACT IN THE HOME

Type of Animal	0	1	2	3	4	5	6	7	8
Asthma - Guguletu - Children	11	0	5	2	0	1	1	2	0
N = 22 - % of total	50	0	22,7	9,1	0	4,6	4,6	9,1	0
Non-Asthma - Guguletu - Children	284	18	184	77	26	10	64	9	0
N = 672 - % of total	42,3	2,7	27,4	11,5	3,9	1,5	9,5	1,3	0
Tsolo Sample - Children	74	32	89	1	345	14	13	81	22
N = 671 - % of total	11,0	4,8	13,2	0,2	51,4	2,1	1,9	12,1	3,3

0 = None 1 = Chickens 2 = Dog 3 = Cat 4 = Chicken & Dog

5 = Chicken & Cat 6 = Dog & Cat 7 = Chicken, dog & cat 8 = Goats

TABLE IV.34

CONTACT WITH MAIZE (POLLEN & GRAIN) IN THE HOME ENVIRONMENT

Source	Do not use it	Only Bought	Only home grown	Home grown & ground + bought
Asthma - Guguletu - Children	4	18	0	0
N = 22 - % of total	18,2	81,8	0	0
Non-Asthma - Guguletu - Children	201	466	2	3
N = 672 - % of total	29,9	69,4	0,3	0,5
Tsolo Sample - Children	3	12	17	639
N = 671 - % of total	0,4	1,8	2,5	95,2

Non-Asthma Guguletu vs Tsolo District $X^2 = 1660,67$ Df = 3 p = 0

TABLE IV.35

FOODS CURRENTLY INCLUDED IN THE DIETS OF THE CHILDREN

Type of Food		Milk	Bread	Eggs	Choco-late	Oranges	Tomat
Asthma - Guguletu - Children		22	21	22	18	19	19
N = 22	- % of total	100	95,5	100	81,8	86,4	86,4
Non-Asthma - Guguletu - Children		660	664	660	563	649	572
N = 672	- % of total	98,2	98,8	98,2	83,8	96,6	85,1
Tsolo Sample - Children		645	663	556	417	472	375
N = 671	- % of total	96,1	98,8	82,9	62,1	70,3	55,9

STATISTICAL ANALYSIS OF DIFFERENCES IN THE SAMPLES

1. Animal Contact

Chisquare Test:

(a) All types of animals

Asthma vs Non-Asthma Guguletu	$X^2 = 8,15$	Df = 8	p = 0,41915
Non-Asthma Guguletu vs Tsolo District	$X^2 = 725,68$	Df = 8	p = 0,00000

(b) Contact categorised as Yes or No

Asthma vs Non-Asthma Guguletu	$X^2 = 0,00$	Df = 1	p = 0,97447
Non-Asthma Guguletu vs Tsolo District	$X^2 = 233,75$	Df = 1	p = 0,00000

2. Foods currently included in Diet

A. Intake of all foods in combination

Asthma vs Non-Asthma Guguletu	$X^2 = 0,25$	Df = 5	p = 0,99852
Non-Asthma Guguletu vs Tsolo District	$X^2 = 40,78$	Df = 5	p = o,00000

B. Individual foods

1. Milk	$X^2 = 0,31$	Df = 1	p = 0,57676
	$X^2 = 5,45$	Df = 1	p = 0,19533
2. Bread	$X^2 = 1,16$	Df = 1	p = 0,28067
	$X^2 = 0,00$	Df = 1	p = 0,99793
3. Eggs	$X^2 = 0,31$	Df = 1	p = 0,57676
	$X^2 = 105,51$	Df = 1	p = 0,00000
4. Chocolate	$X^2 = 0,06$	Df = 1	p = 0,80903
	$X^2 = 81,4$	Df = 1	p = 0,00000
5. Oranges	$X^2 = 3,83$	Df = 1	p = 0,05026
	$X^2 = 188,00$	Df = 1	p = 0,00000
6. Tomatoes	$X^2 = 0,03$	Df = 1	p = 0,87021
	$X^2 = 142,7$	Df = 1	p = 0,00000

TABLE IV.36

DURATION OF BREAST FEEDING DURING INFANCY

Duration in Months	0	1	2	3	4	5	6	7	8	9	10
Asthma - Guguletu - Children N = 22	5	3	0	2	3	2	1	0	1	0	0
Non-Asthma - Guguletu - Children N = 635	175	34	29	59	23	13	60	7	16	25	3
Tsolo Sample - Children N = 670	19	16	8	15	10	3	29	2	34	17	8

Duration in Months	11	12	13	14	15	16	17	18	19	20	21
Asthma - Guguletu - Children N = 22	1	2	0	1	0	0	0	0	0	0	0
Non-Asthma - Guguletu - Children N = 635	2	89	2	8	3	7	2	21	1	2	1
Tsolo Sample - Children N = 670	4	90	3	4	3	6	3	60	2	7	2

Duration in Months	22	23	24	25	26	27	28	29	30	31	32
Asthma - Guguletu - Children N = 22	0	0	1	0	0	0	0	0	0	0	0
Non-Asthma - Guguletu - Children N = 635	1	0	36	0	0	0	1	0	3	0	0
Tsolo Sample - Children N = 670	2	0	274	1	3	0	4	0	7	0	0

TABLE IV.36 (Continued)

Duration in Months	33	34	35	36	48	52
Asthma - Guguletu - Children N = 22	0	0	0	0	0	0
Non-Asthma - Guguletu - Children N = 635	0	0	0	8	3	1
Tsolo Sample - Children N = 670	0	2	0	32	0	0

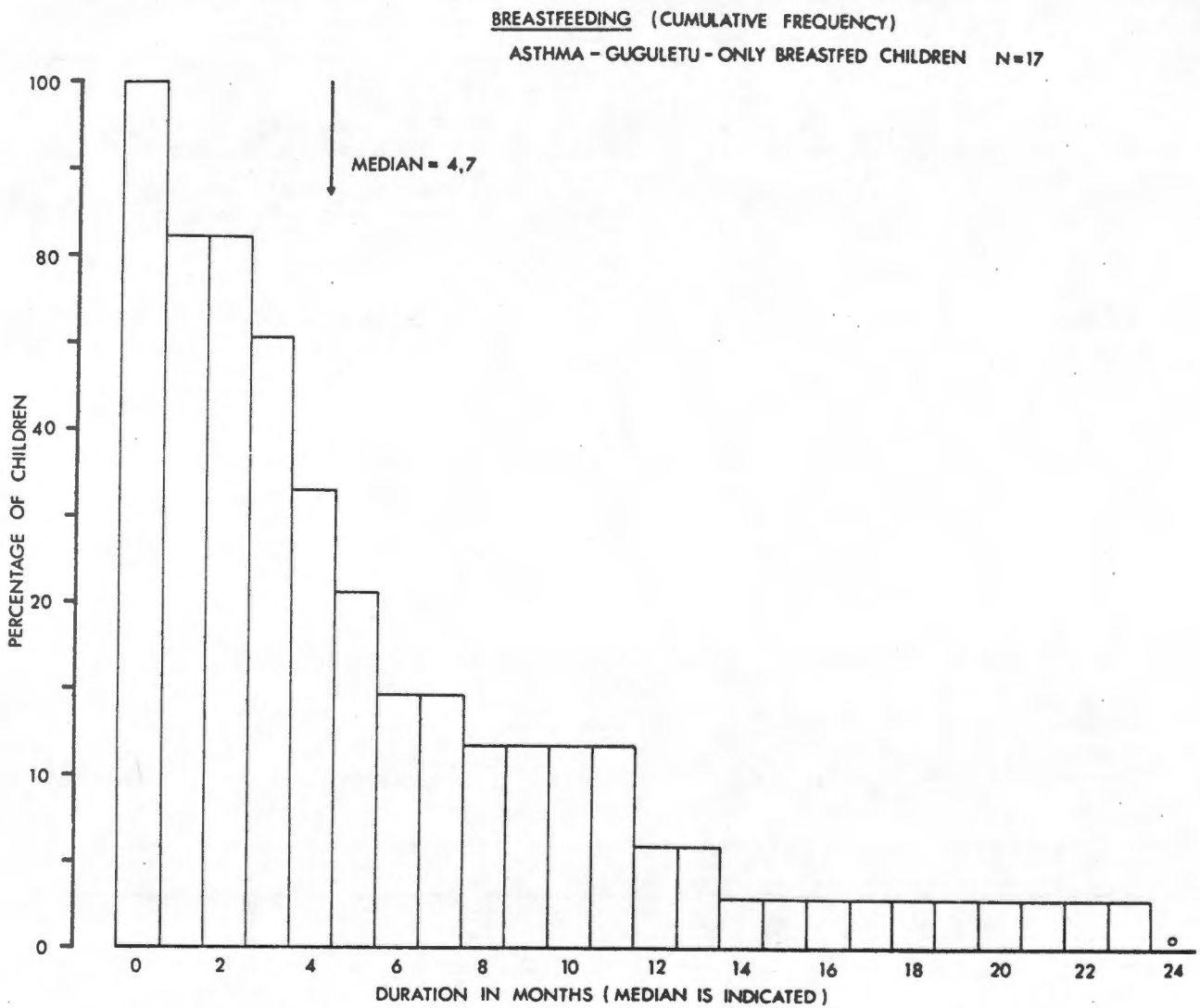

Fig. 12 : CUMULATIVE FREQUENCY OF DURATION OF BREASTFEEDING FOR ASTHMATIC CHILDREN FROM GUGULETU EXCLUDING THOSE NEVER BREASTFED

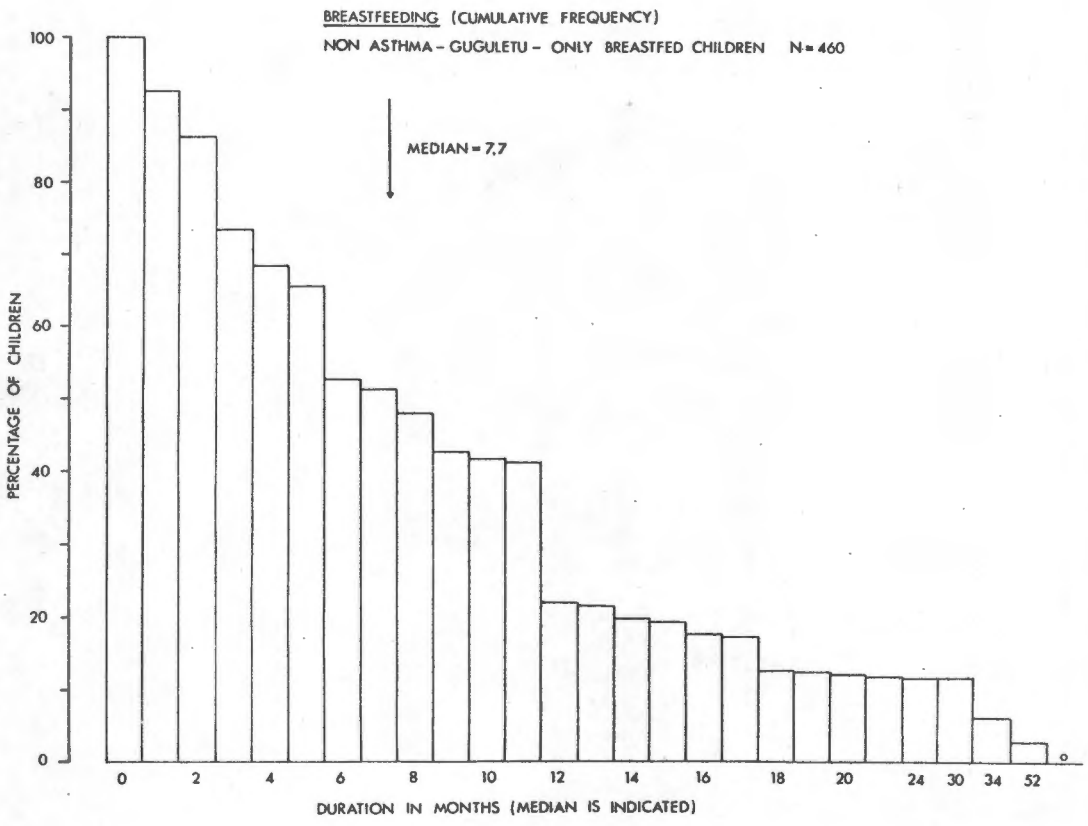

Fig. 13 : CUMULATIVE FREQUENCY OF DURATION OF BREASTFEEDING FOR NON-ASTHMATIC CHILDREN FROM GUGULETU EXCLUDING THOSE NEVER BREASTFED

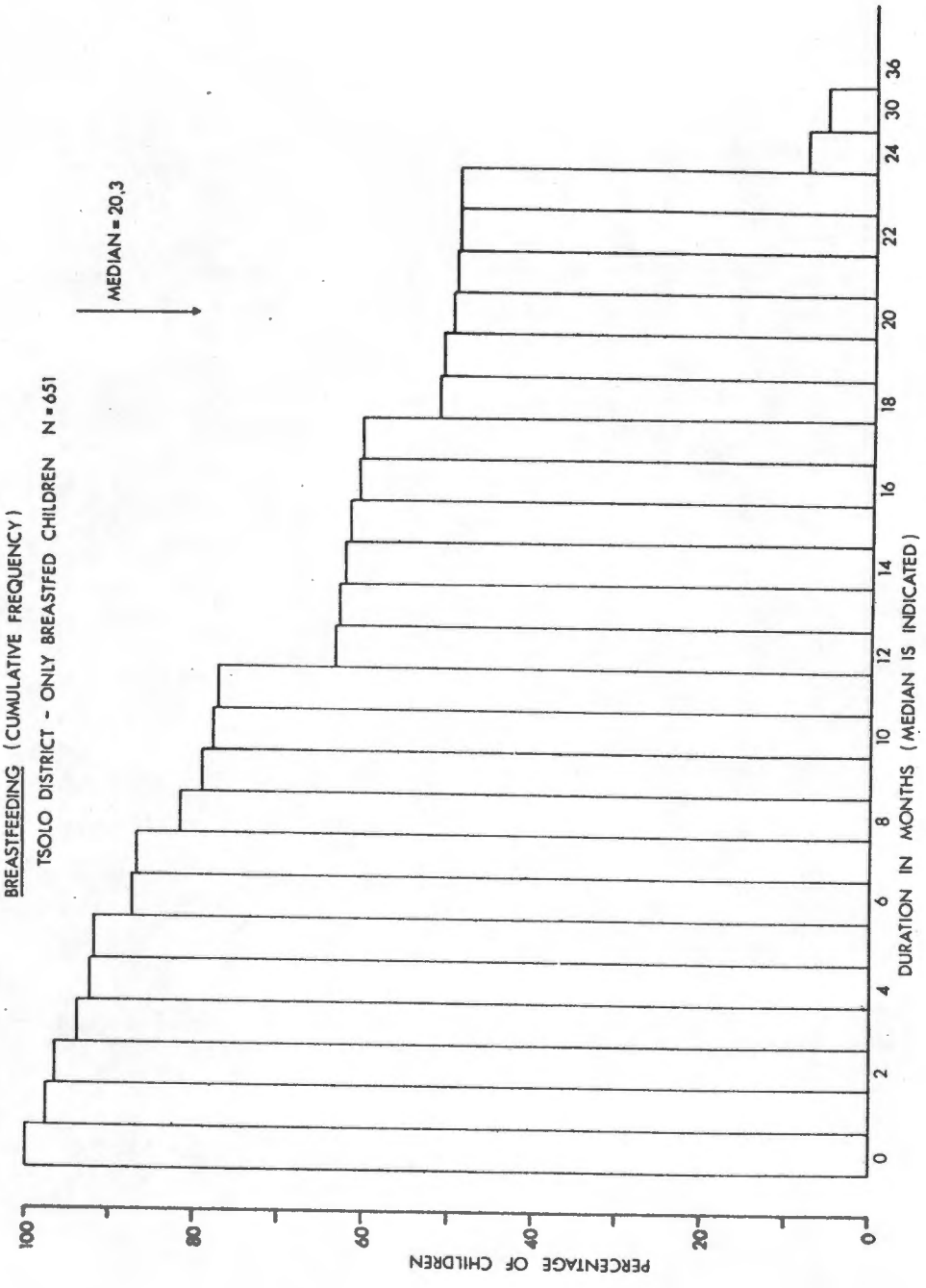

Fig. 14 : CUMULATIVE FREQUENCY OF DURATION OF BREASTFEEDING FOR CHILDREN FROM TSOLO DISTRICT EXCLUDING THOSE NEVER BREASTFED

TABLE IV.37 AGE AT INTRODUCTION OF COW'S MILK

Age in Months	0	1	2	3	4	5	6	7	8	9	10
Asthma - Guguletu - Children N = 22	13	0	1	2	1	2	1	0	1	0	0
Non-Asthma - Guguletu - Children N = 636	339	50	26	49	17	8	34	0	6	16	0
Tsolo Sample - Children N = 670	4	142	59	156	165	28	101	5	4	4	0
Age in Months	11	12	13	14	15	16	17	18	19	20	21
Asthma - Guguletu - Children N = 22	0	0	0	0	0	0	0	0	0	0	0
Non-Asthma - Guguletu - Children N = 636	0	50	3	3	1	2	1	13	0	1	0
Tsolo Sample - Children N = 670	0	2	0	0	0	0	0	0	0	0	0
Age in Months	22	23	24	25	26	27	28	29	30	31	32
Asthma - Guguletu - Children N = 22	0	0	1	0	0	0	0	0	0	0	0
Non-Asthma - Guguletu - Children N = 636	0	0	13	0	0	0	0	0	1	0	0
Tsolo Sample - Children N = 670	0	0	0	0	0	0	0	0	0	0	0

TABLE IV.37 (Continued)

Age in Months	33	34	36	37	38	48
Asthma - Guguletu - Children N = 22	0	0	0	0	0	0
Non-Asthma - Guguletu - Children N = 636	0	0	0	1	0	2
Tsolo Sample - Children N = 670	0	0	0	0	0	0

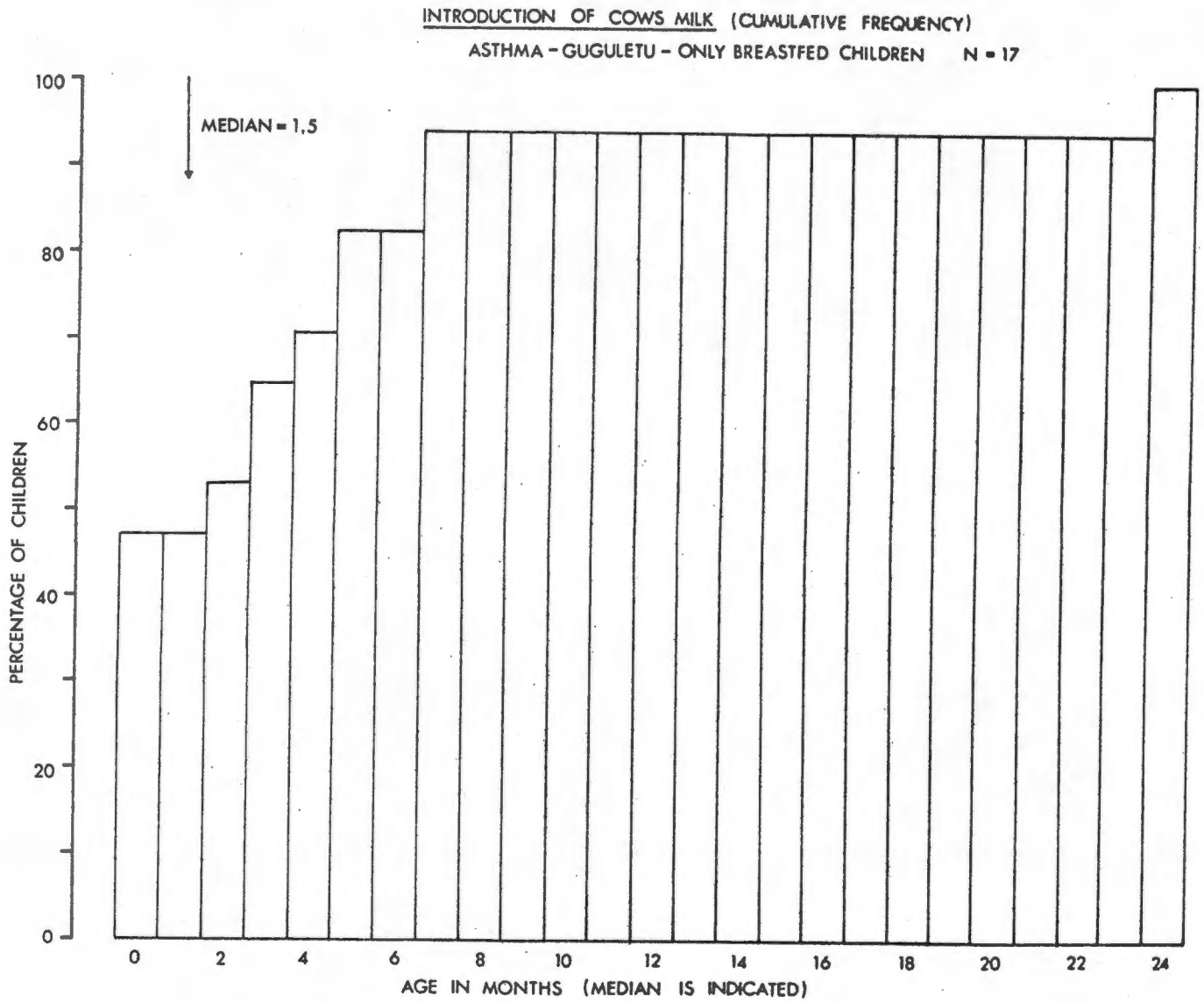

Fig. 15 : CUMULATIVE FREQUENCY OF AGE OF INTRODUCTION OF COW'S MILK INTO THE DIET DURING INFANCY FOR ASTHMATIC CHILDREN FROM GUGULETU EXCLUDING THOSE NEVER BREASTFED

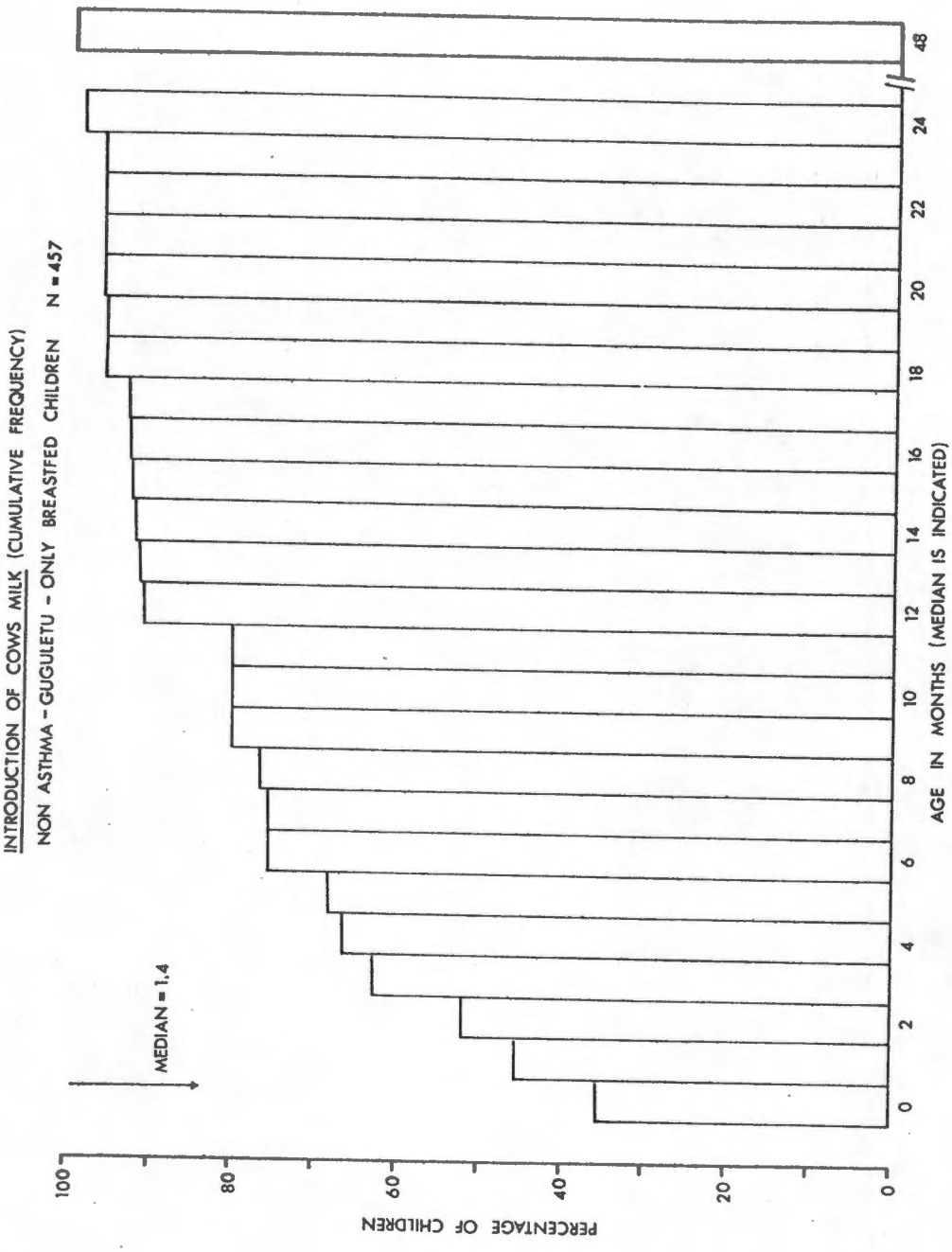

Fig. 16 : CUMULATIVE FREQUENCY OF AGE OF INTRODUCTION OF COW'S MILK INTO THE DIET DURING INFANCY FOR NON-ASTHMATIC CHILDREN FROM GUGULETU EXCLUDING THOSE NEVER BREASTFED

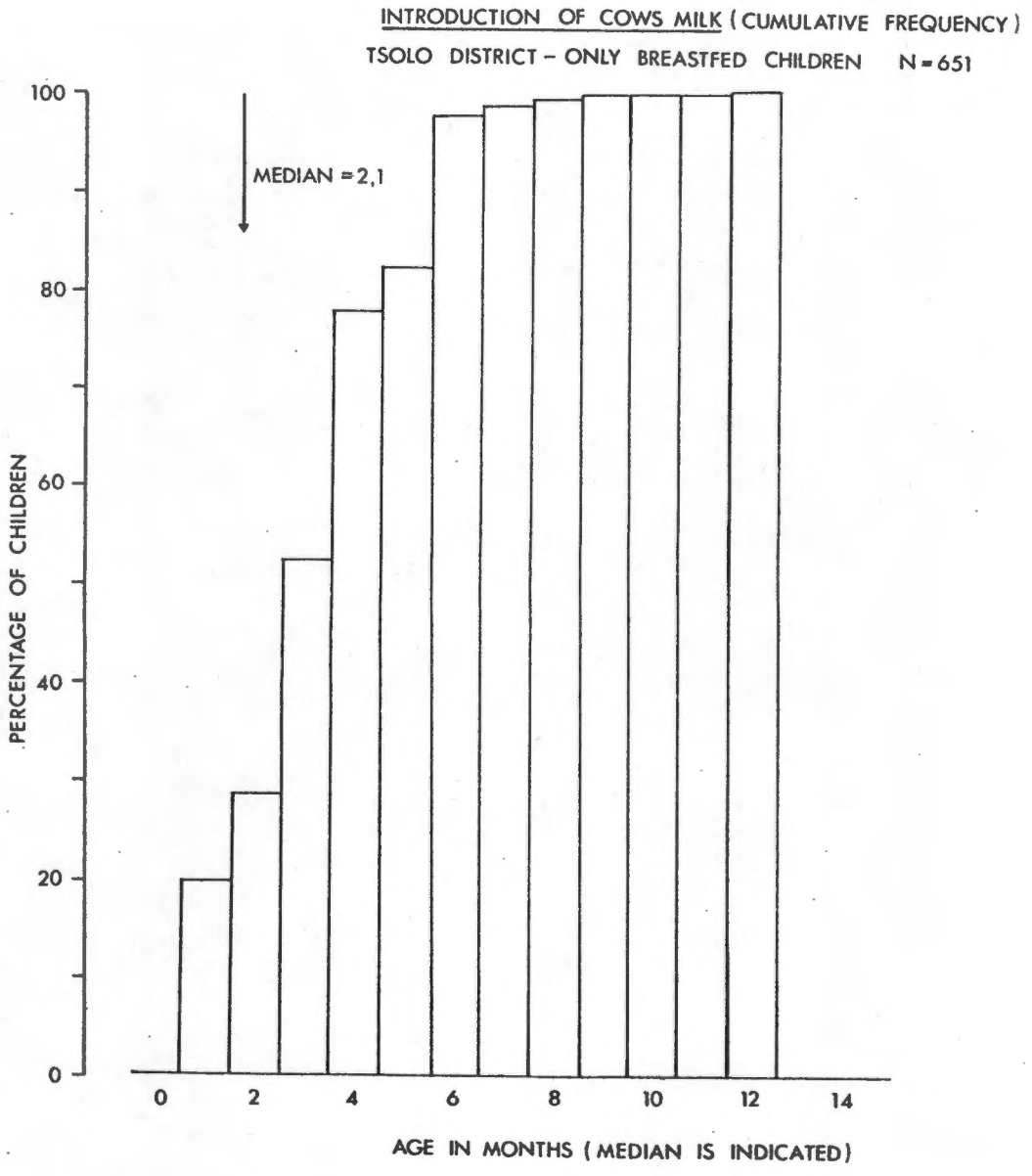

Fig. 17 : CUMULATIVE FREQUENCY OF AGE OF INTRODUCTION OF COW'S MILK INTO THE DIET DURING INFANCY FOR TSOLO DISTRICT CHILDREN EXCLUDING THOSE NEVER BREASTFED

TABLE IV.38

AGE AT INTRODUCTION OF SOLIDS

Age in Months	0	1	2	3	4	5	6	7	8
Asthma - Guguletu N = 21	0	2	2	8	6	1	2	0	0
Non-Asthma - Guguletu N = 628	0	24	59	219	156	51	77	5	9
Tsolo Sample N = 670	1	10	29	29	165	123	234	25	28

Age in Months	9	10	11	12	13	14	15	16	17
Asthma - Guguletu N = 21	0	0	0	0	0	0	0	0	0
Non-Asthma - Guguletu N = 628	12	3	1	5	0	1	0	0	1
Tsolo Sample N = 670	12	4	0	7	1	1	0	0	0

Age in Months	18	19	20	21	22	23	24
Asthma - Guguletu N = 21	0	0	0	0	0	0	0
Non-Asthma - Guguletu N = 628	1	0	0	0	1	0	3
Tsolo Sample N = 670	0	0	0	0	0	0	1

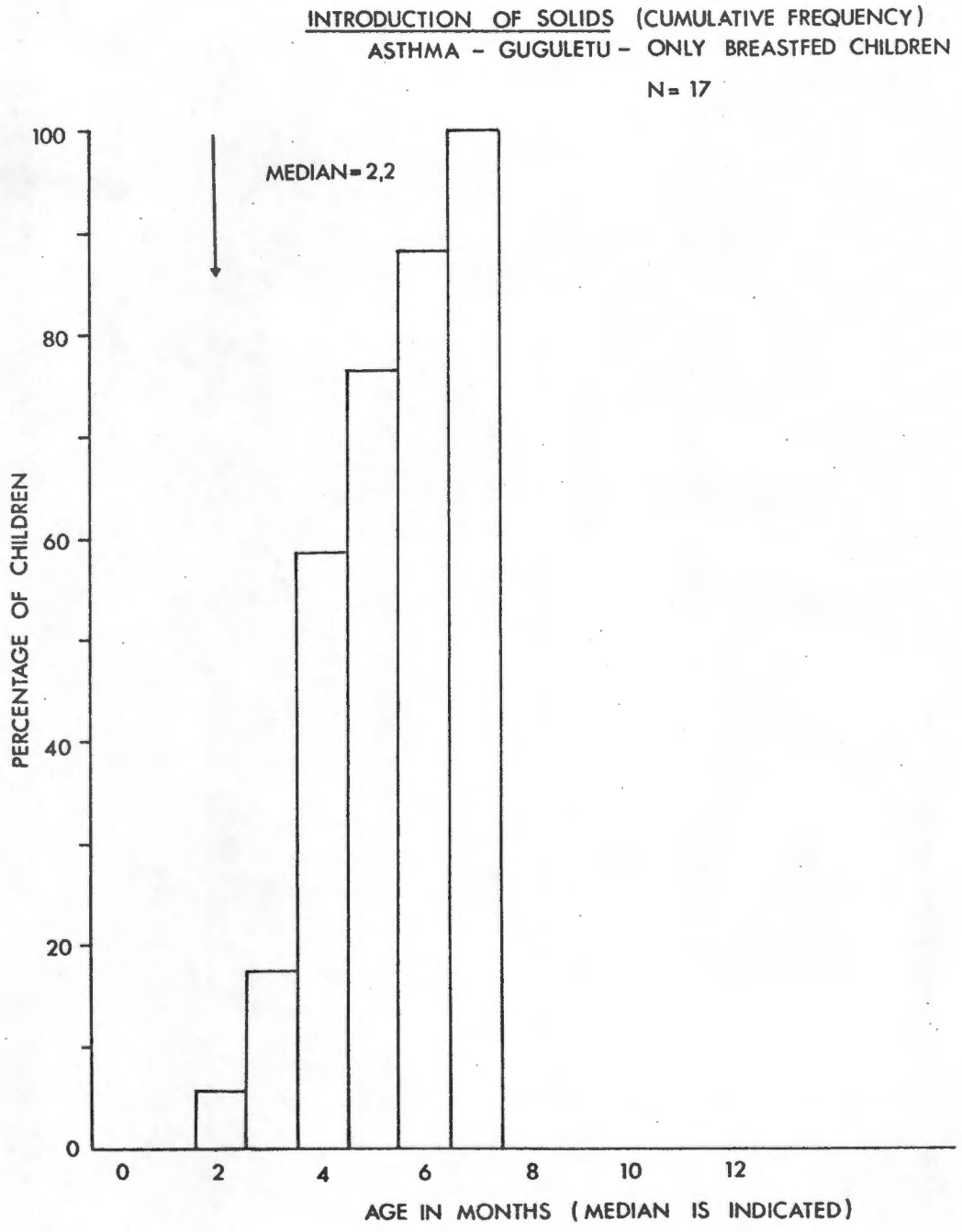

Fig. 18 : CUMULATIVE FREQUENCY OF AGE OF INTRODUCTION OF SOLIDS INTO THE DIET DURING INFANCY FOR ASTHMATIC CHILDREN FROM GUGULETU

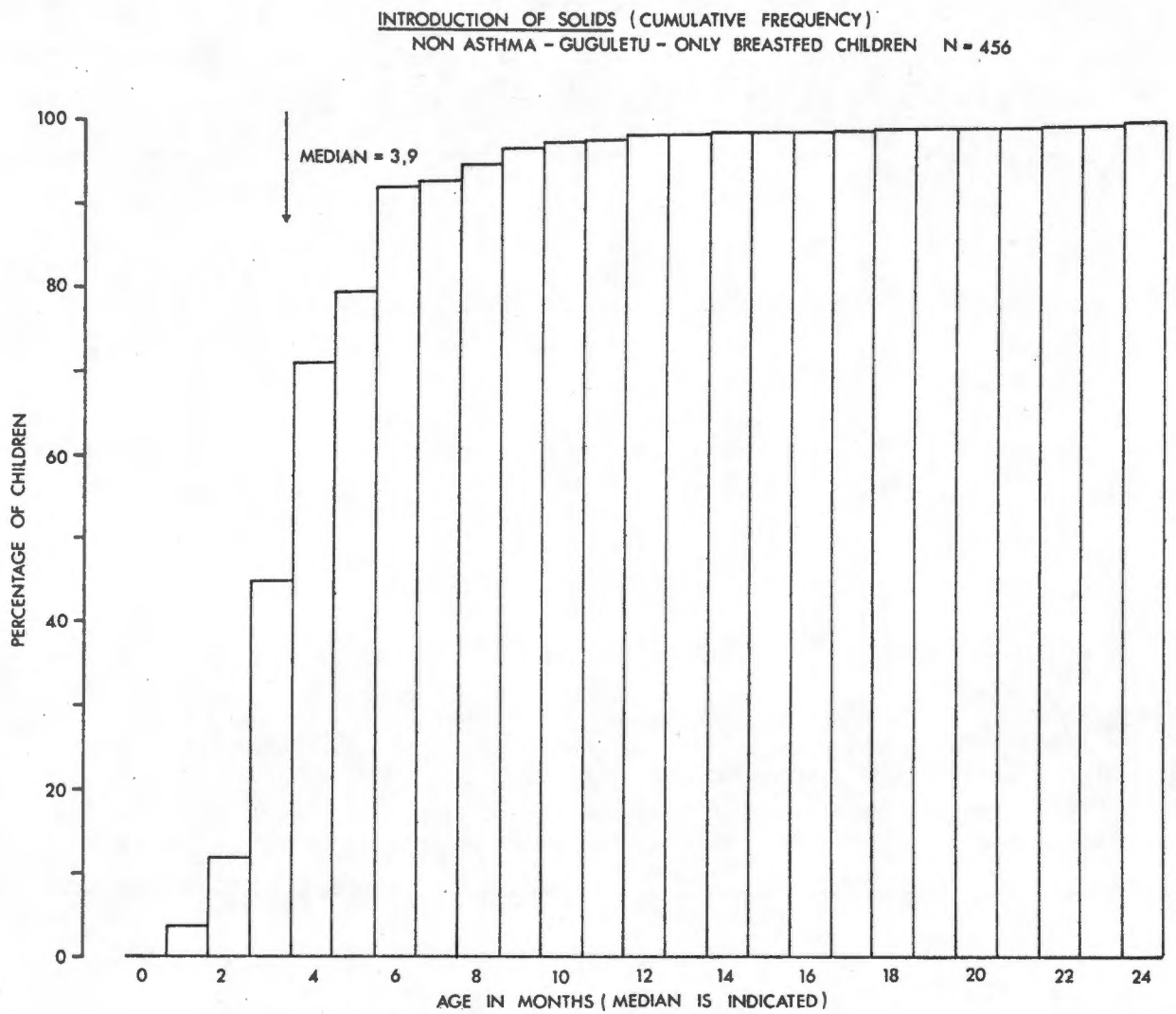

Fig. 19 : CUMULATIVE FREQUENCY OF AGE OF INTRODUCTION OF SOLIDS INTO THE DIET DURING INFANCY FOR NON-ASTHMATIC CHILDREN FROM GUGULETU

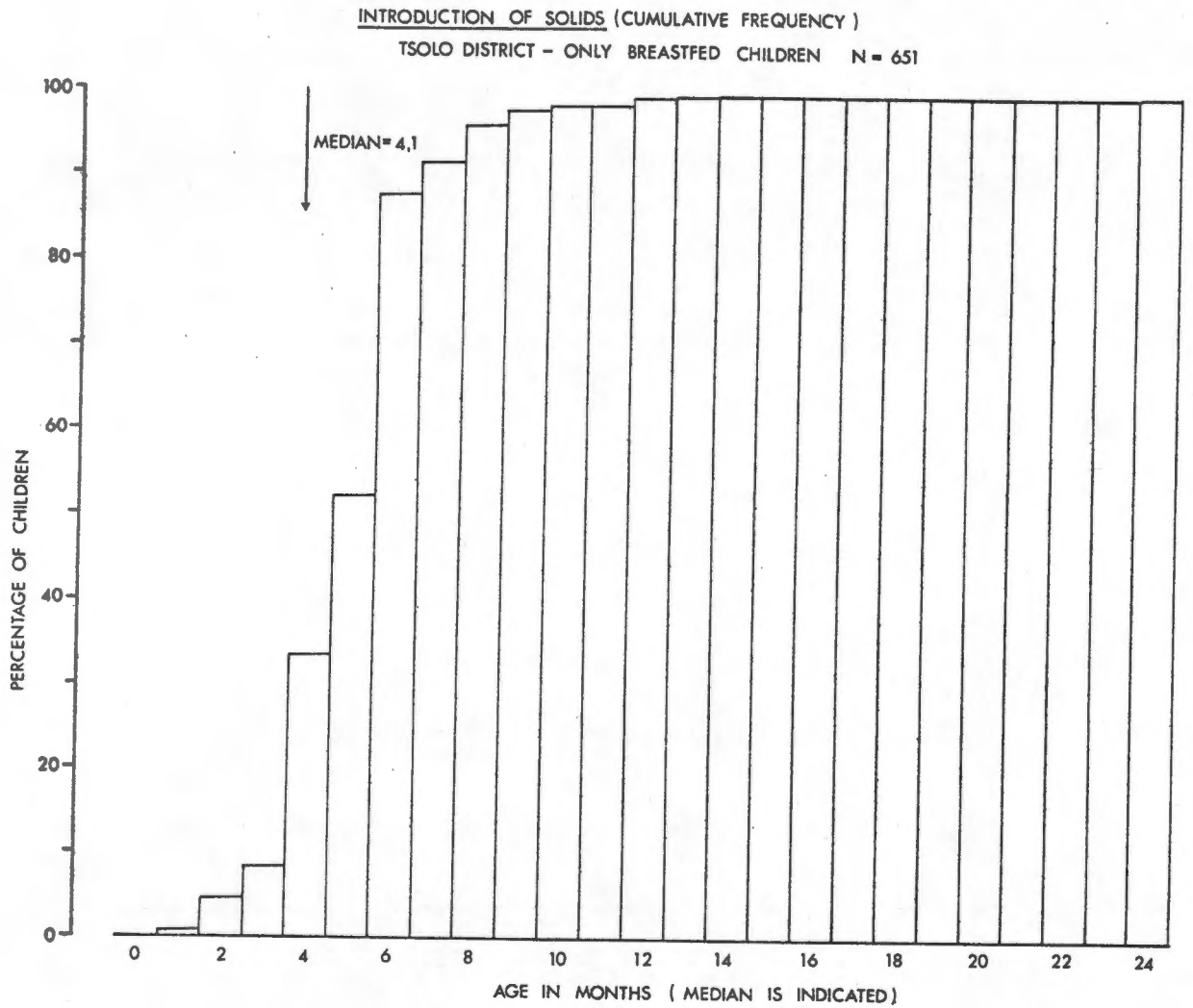

Fig. 20 : CUMULATIVE FREQUENCY OF AGE OF INTRODUCTION OF SOLIDS INTO THE DIET DURING INFANCY FOR CHILDREN FROM TSOLO DISTRICT

TABLE IV.39

DISTRIBUTION OF WEIGHT FOR AGE OF BOYS

ACCORDING TO NCHS PERCENTILES

CHILDREN

Percentiles	5	10	25	50	75	90	95	>95
6 YEARS								
Asthma - Guguletu	0	1	1	0	0	0	0	0
Non-Asthma - Guguletu	18	7	10	18	6	2	1	0
Tsolo District	47	4	15	13	6	0	5	2
7 YEARS								
Asthma - Guguletu	0	0	0	0	0	0	0	0
Non-Asthma - Guguletu	17	19	16	12	6	1	1	1
Tsolo District	14	6	9	13	7	3	2	0
8 YEARS								
Asthma - Guguletu	1	0	0	0	1	0	0	0
Non-Asthma - Guguletu	34	8	18	3	4	2	0	0
Tsolo District	25	9	13	10	10	5	1	0
9 YEARS								
Asthma - Guguletu	1	0	0	1	1	0	0	0
Non-Asthma - Guguletu	32	7	21	31	11	5	0	0
Tsolo District	21	9	20	27	12	3	0	0

PERCENTILES INCLUDE ALL CHILDREN AT OR BELOW A PARTICULAR ONE

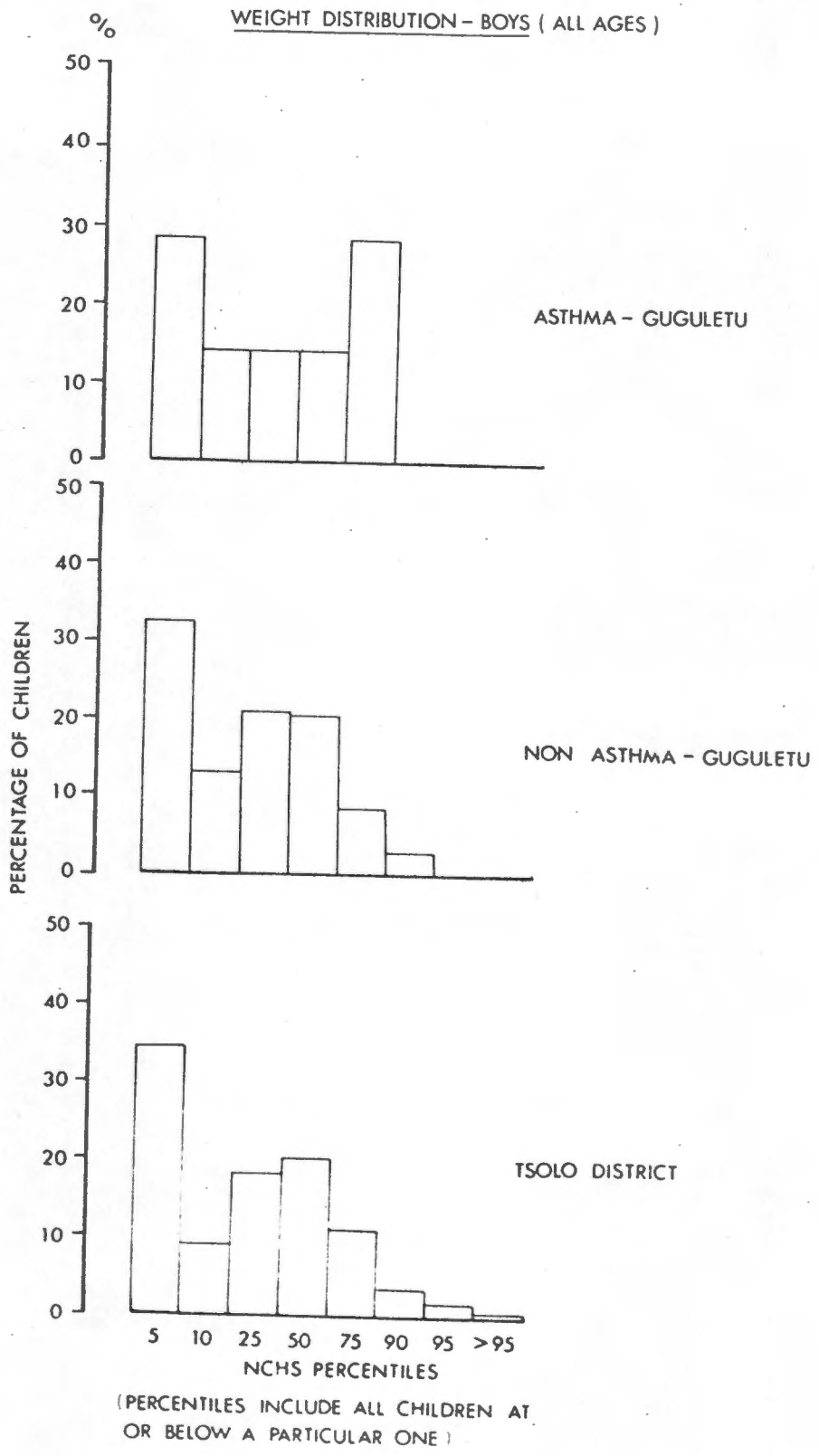

Fig. 21 : DISTRIBUTION OF WEIGHT FOR AGE OF BOYS ACCORDING TO NCHS PERCENTILES. ALL AGES INCLUDED.

TABLE IV.40

DISTRIBUTION OF HEIGHT FOR AGE OF BOYS

ACCORDING TO NCHS PERCENTILES

CHILDREN

Percentiles	5	10	25	50	75	90	95	>95
6 YEARS								
Asthma - Guguletu	0	1	1	0	0	0	0	0
Non-Asthma - Guguletu	36	7	5	8	5	0	0	1
Tsolo District	60	10	8	6	2	1	0	3
7 YEARS								
Asthma - Guguletu	0	0	0	0	0	0	0	0
Non-Asthma - Guguletu	24	16	12	9	6	1	2	2
Tsolo District	22	4	9	7	7	1	3	1
8 YEARS								
Asthma - Guguletu	2	0	0	0	0	0	0	0
Non-Asthma - Guguletu	30	13	19	4	0	3	0	0
Tsolo District	31	12	10	7	6	5	0	2
9 YEARS								
Asthma - Guguletu	0	1	1	1	0	0	0	0
Non-Asthma - Guguletu	38	13	34	11	6	3	2	0
Tsolo District	35	8	19	19	7	2	2	0

PERCENTILES INCLUDE ALL CHILDREN AT OR BELOW A PARTICULAR ONE

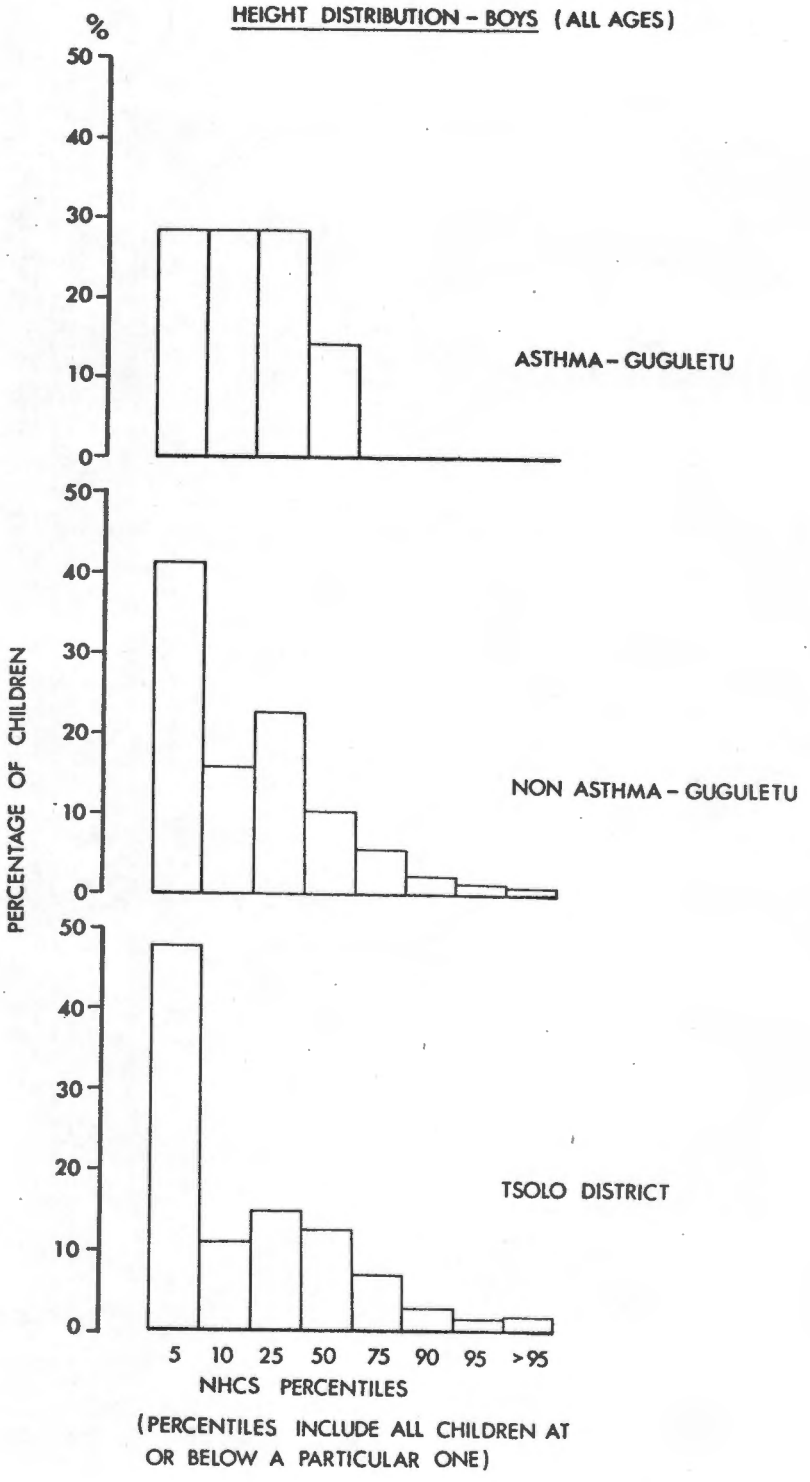

Fig. 22 : DISTRIBUTION OF HEIGHT FOR AGE OF BOYS ACCORDING TO NCHS PERCENTILES. ALL AGES INCLUDED.

TABLE IV.41

DISTRIBUTION OF WEIGHT FOR AGE OF GIRLS

ACCORDING TO NCHS PERCENTILES

CHILDREN

Percentiles	5	10	25	50	75	90	95	>95
6 YEARS								
Asthma - Guguletu	1	1	0	0	0	0	0	0
Non-Asthma - Guguletu	14	4	17	20	10	4	1	0
Tsolo District	40	8	11	29	16	9	3	2
7 YEARS								
Asthma - Guguletu	1	0	3	1	1	0	2	1
Non-Asthma - Guguletu	10	9	20	14	7	3	2	2
Tsolo District	20	11	18	11	3	1	0	0
8 YEARS								
Asthma - Guguletu	2	0	0	0	0	0	0	0
Non-Asthma - Guguletu	25	15	22	16	10	1	2	0
Tsolo District	24	6	15	23	9	0	0	0
9 YEARS								
Asthma - Guguletu	0	0	2	0	0	0	0	0
Non-Asthma - Guguletu	35	16	21	27	10	5	0	0
Tsolo District	34	13	30	19	8	0	0	0

PERCENTILES INCLUDE ALL CHILDREN AT OR BELOW A PARTICULAR ONE

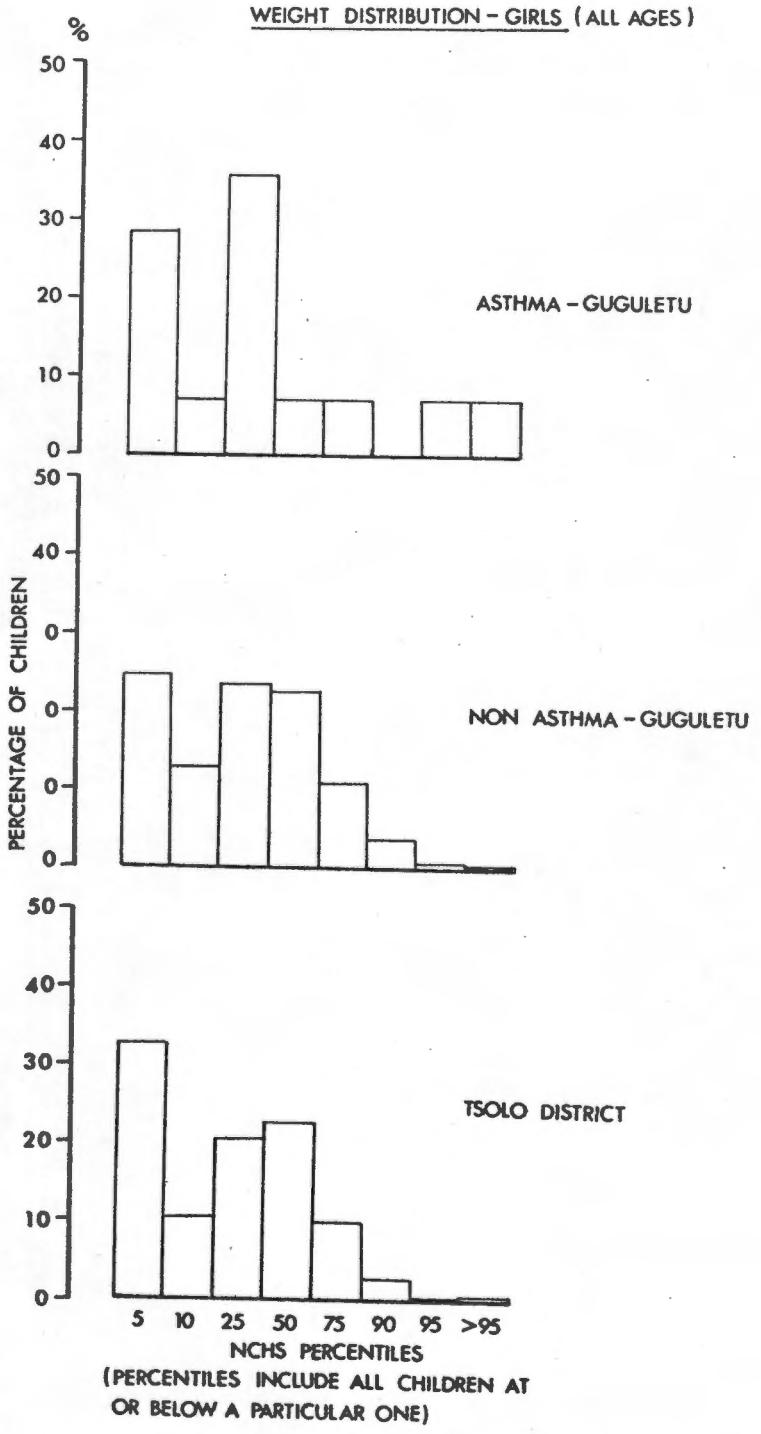

Fig. 23 : DISTRIBUTION OF WEIGHT FOR AGE OF GIRLS ACCORDING TO NCHS PERCENTILES. ALL AGES INCLUDED.

TABLE IV.42

DISTRIBUTION OF HEIGHT FOR AGE OF GIRLS

ACCORDING TO NCHS PERCENTILES

CHILDREN

Percentiles	5	10	25	50	75	90	95	>95
6 YEARS								
Asthma - Guguletu	1	1	0	0	0	0	0	0
Non-Asthma - Guguletu	22	10	17	13	5	2	1	0
Tsolo District	58	12	19	8	10	4	2	3
7 YEARS								
Asthma - Guguletu	2	2	0	1	2	1	0	0
Non-Asthma - Guguletu	21	15	9	12	7	0	0	1
Tsolo District	32	10	13	5	4	0	0	0
8 YEARS								
Asthma - Guguletu	0	1	1	0	0	0	0	0
Non-Asthma - Guguletu	31	8	19	18	11	2	2	0
Tsolo District	39	8	15	11	1	1	1	1
9 YEARS								
Asthma - Guguletu	1	0	1	0	0	0	0	0
Non-Asthma - Guguletu	44	15	20	20	8	3	4	0
Tsolo District	51	8	19	17	6	3	0	0

PERCENTILES INCLUDE ALL CHILDREN AT OR BELOW A PARTICULAR ONE

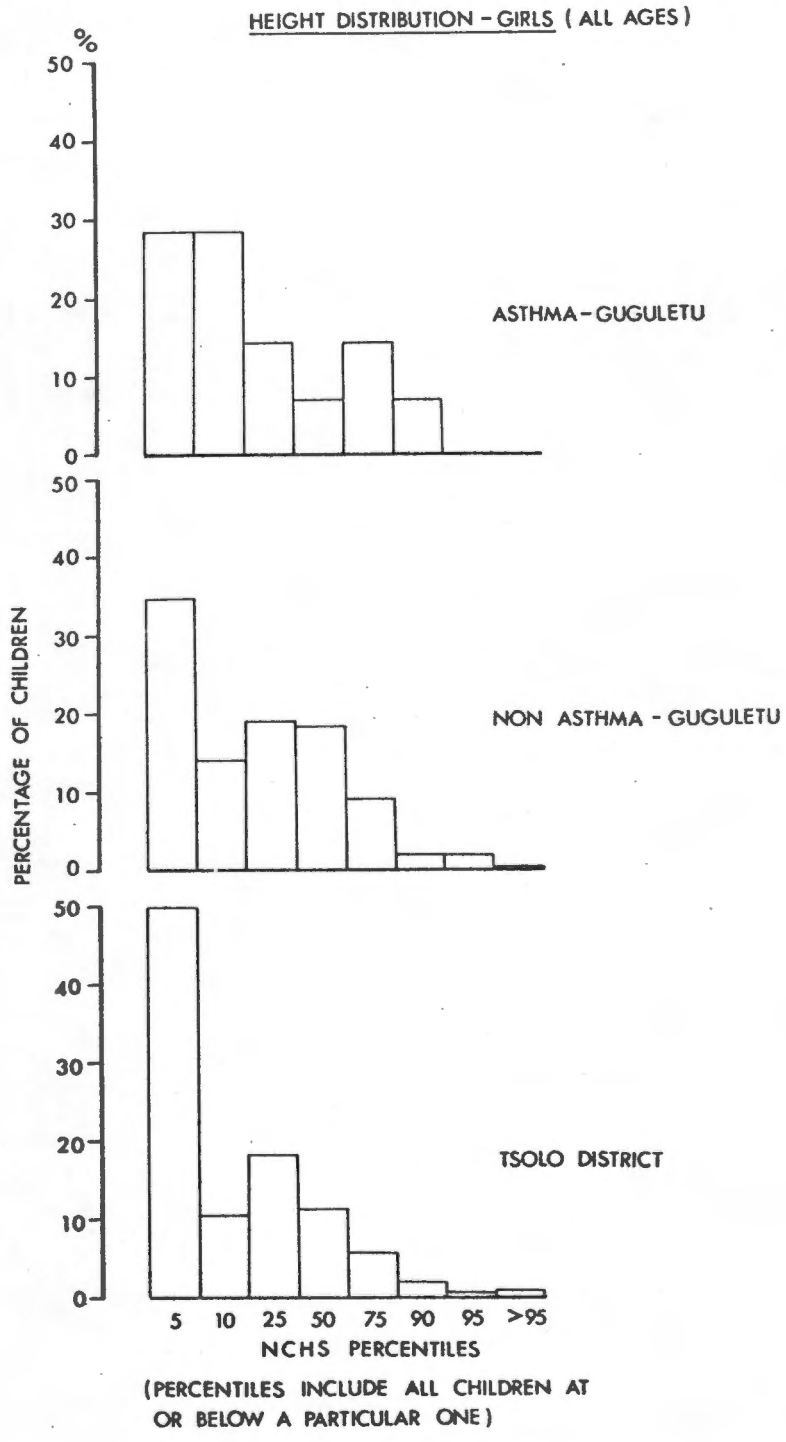

Fig. 24 : DISTRIBUTION OF HEIGHT FOR AGE OF GIRLS ACCORDING TO NCHS PERCENTILES. ALL AGES INCLUDED.

TABLE IV.43

PERCENTAGE OF CHILDREN LESS THAN 3RD PERCENTILE OF WEIGHT FOR AGE : MALNUTRITION

BOYS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	-	-	50,0	33,3	2 (28,6%)
Non-Asthma - Guguletu	26,2	16,7	49,3	25,2	89 (28,8%)
Tsolo District	46,7	20,4	31,5	16,3	91 (29,4%)

Non-Asthma Guguletu - Tsolo District $X^2 = 17,72$ Df = 3 p = 0,00050

GIRLS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	50,0	12,5	50,0	-	3 (21,4%)
Non-Asthma - Guguletu	18,6	12,3	16,5	29,8	70 (20,6%)
Tsolo District	33,6	31,7	23,4	28,8	107 (29,6%)

Non-Asthma Guguletu - Tsolo District - Actual Numbers $X^2 = 11,65$ Df = 3 p = 0,00868

TABLE IV.44

PERCENTAGE OF CHILDREN LESS THAN 3RD PERCENTILE OF HEIGHT FOR AGE : STUNTING

BOYS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	-	-	100	-	2 (28,6%)
Non-Asthma - Guguletu	57,4	33,3	43,5	35,5	127 (41,1%)
Tsolo District	66,7	40,7	42,5	38,0	148 (47,9%)
Non-Asthma - Guguletu vs Tsolo District		χ^2 = 5,28,	Df = 3,	p = 0,15249	

GIRLS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	50,0	25	-	50,0	4 (28,6%)
Non-Asthma - Guguletu	28,6	32,3	33,0	36,0	112 (32,9%)
Tsolo District	48,3	50,0	50,6	48,1	177 (49,0%)
Non-Asthma - Guguletu vs Tsolo District		χ^2 = 7,38,	Df = 3,	p = 0,06075	

TABLE IV.45

PERCENTAGE OF CHILDREN LESS THAN 3RD PERCENTILE OF WEIGHT FOR HEIGHT : WASTING

BOYS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	-	-	-	-	0 (-)
Non-Asthma - Guguletu	1,6	5,6	11,6	11,2	25 (8,1%)
Tsolo District	1,1	-	1,4	1,1	3 (1,0%)
Non-Asthma - Guguletu vs Tsolo District		X^2 = 2,47,	Df = 3,	p = 0,48092	

GIRLS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	-	-	-	-	0 (-)
Non-Asthma - Guguletu	-	3,1	12,1	6,1	20 (5,9%)
Tsolo District	-	1,6	-	1,9	3 (0,8%)
Non-Asthma - Guguletu vs Tsolo District		X^2 = 3,87,	Df = 3,	p = 0,27553	

TABLE IV.46

PERCENTAGE OF CHILDREN WITH SKINFOLD THICKNESS LESS THAN 3RD PERCENTILE

BOYS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	-	-	-	-	
Non-Asthma - Guguletu	1,6	1,4	1,5	1,9	5 (1,6%)
Tsolo District	15,4	7,4	23,8	15,2	45 (14,6%)
Non-Asthma - Guguletu vs Tsolo District		X^2 = 0,92,	Df = 3,	p = 0,82171	

GIRLS

AGE IN YEARS	6	7	8	9	TOTAL ALL AGES
Asthma - Guguletu	-	-	50,0	-	1 (7,1%)
Non-Asthma - Guguletu	7,1	3,1	8,6	2,6	18 (5,2%)
Tsolo District	8,6	21,9	27,3	17,3	63 (17,5%)
Non-Asthma - Guguletu vs Tsolo District		X^2 = 3,27,	Df = 3,	p = 0,35137	

STATISTICAL ANALYSIS OF DIFFERENCE IN HEIGHTS & WEIGHTS

1. CHISQUARE TEST: DISTRIBUTION OF HEIGHT FOR AGE OF BOYS ACCORDING TO NCHS PERCENTILES

For Samples Non-Asthma Guguletu vs Tsolo District

(a)	6 years	$X^2 = 29,16$, Df = 7, p = 0,00014
(b)	7 years	$X^2 = 6,52$, Df = 7, p = 0,48102
(c)	8 years	$X^2 = 14,92$, Df = 7, p = 0,03705
(d)	9 years	$X^2 = 6,94$, Df = 7, p = 0,43554

2. CHISQUARE TEST: DISTRIBUTION OF WEIGHT FOR AGE OF BOYS ACCORDING TO NCHS PERCENTILES

For Samples Non-Asthma Guguletu vs Tsolo District

(a)	6 years	$X^2 = 15,85$, Df = 6, p = 0,01459
(b)	7 years	$X^2 = 8,30$, Df = 6, p = 0,21669
(c)	8 years	$X^2 = 10,96$, Df = 6, p = 0,89741
(d)	9 years	$X^2 = 2,27$, Df = 6, p = 0,89336

3. CHISQUARE TEST: DISTRIBUTION OF HEIGHT FOR AGE OF GIRLS ACCORDING TO NCHS PERCENTILES

For Samples Non-Asthma Guguletu vs Tsolo District

(a)	6 years	X^2 = 2,63, Df = 7, p = 0,93731
(b)	7 years	X^2 = 8,68, Df = 7, p = 0,27608
(c)	8 years	X^2 = 13,20, Df = 7, p = 0,06747
(d)	9 years	X^2 = 7,53, Df = 7, p = 0,37574

4. CHISQUARE TEST: DISTRIBUTION OF WEIGHT FOR AGE OF GIRLS ACCORDING TO NCHS PERCENTILES

For Samples Non-Asthma Guguletu vs Tsolo District

(a)	6 years	X^2 = 10,29, Df = 6, p = 0,11284
(b)	7 years	X^2 = 12,91, Df = 6, p = 0,04443
(c)	8 years	X^2 = 18,75, Df = 6, p = 0,00460
(d)	9 years	X^2 = 25,98, Df = 6, p = 0,00022

TABLE IV.47

RADIOGRAPHS OF THE CHEST

RADIOGRAPHIC APPEARANCE	ASTHMA GUGULETU		NON-ASTHMA GUGULETU		TSOLO DISTRICT	
Normal	18	(81,8)	549	(85,2)	625	(94,0)
Bronchitis	2	(9,1)	15	(2,3)	1	(0,2)
Bronchial wall thickening	2	(9,1)	19	(3,0)	1	(0,2)
Bronchitis with bronchial wall thickening	0		3	(0,5)	2	(0,3)
Hyperinflation	0		2	(0,3)	2	(0,3)
Bronchitis with hyper-inflation	0		0		2	(0,3)
Pneumonia	0		14	(2,2)	8	(1,2)
Pneumonia with atelectasis	0		1	(0,2)	2	(0,3)
Calcified hilar lymph nodes	0		34	(5,3)	16	(2,4)
Active tuberculosis	0		7	(1,1)	6	(0,9)
Number of children	22		644		665	

PERCENTAGE OF TOTALS IN BRACKETS

TABLE IV.48

PRICK SKIN TEST REACTIONS TO 13 ALLERGENS

	Positive	Negative
Asthma Guguletu - Children		
N = 22	1	21
% of total	4,5	95,5
Non-Asthma Guguletu - Children		
N = 669	51	618
% of total	7,6	92,4
Asthma Tsolo District - Children		
N = 1	1	-
% of total	100,0	
Non-Asthma Tsolo District - Children		
N = 670	130	540
% of total	19,4	80,6

TABLE IV.49

POSITIVE PRICK SKIN TEST REACTIONS PER CHILD TO 13 ALLERGENS

No. of Reactions	Asthma Guguletu	Non-Asthma Guguletu	Asthma Tsolo	Non-Asthma Tsolo
1	1	29	1	97
2	-	9	-	19
3	-	10	-	12
4	-	2	-	1
5	-	-	-	1
Total Positive Reactions	1	85	1	182

TABLE IV.50

FREQUENCY OF POSITIVE PRICK SKIN TEST REACTIONS TO 13 ALLERGENS

Allergen	Asthma Guguletu N = 22	Asthma Tsolo N = 1	Non-Asthma Guguletu N = 669	Tsolo District N = 670	p*
Maize pollen	0	0	12	42	0,00004
Maize food	0	0	7	24	0,00152
Feathers	0	0	1	18	0,00000
House dust	0	0	13	13	1,0
D.pteronyssinus	0	0	13	13	0,84892
D.farinae	1	0	21	11	0,04983
E.purpurascens	0	0	1	12	0,00089
A. tenuis	0	0	4	3	0,70279
C.herbarum	0	1	2	16	0,00011
S.African grass	0	0	5	15	0,06812
Dog epithelium	0	0	3	8	0,08571
Cat epithelium	0	0	2	5	0,24871
Milk	0	0	1	3	0,30632
Total Positive Reactions	1	1	85	183	

* p value for Non-Asthma Guguletu vs Tsolo District for individual allergens

All allergens: $X^2 = 47,84$ Df = 12 p = 0,00000

TABLE IV.51

SERUM IMMUNOGLOBULIN E (IgE) LEVEL

(Geometrical mean, 67% and 95% Tolerance Intervals u/ml)

GUGULETU

Age in Years	6	7	8	9
Boys - N	61	71	66	103
IgE u/ml	122-405-1421-4981-16605	131-437-1531-5365-17786	90-299-1048-3675-12250	111-370-1298-4548-15162
Girls - N	68	69	95	118
IgE u/ml	71-235-824-2888-9626	64-214-751-2631-8770	103-344-1205-4226-14086	61-205-718-2518-8394

TSOLO DISTRICT

Boys - N	81	52	64	90
IgE - u/ml	76-252-883-3096-10320	50-165-578-2026-6753	76-254-891-3125-10417	60-201-705-2471-8237
Girls - N	104	60	72	99
IgE - u/ml	63-210-737-2583-8609	88-293-1029-3607-12024	59-198-693-2430-8100	62-207-725-2540-8467

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

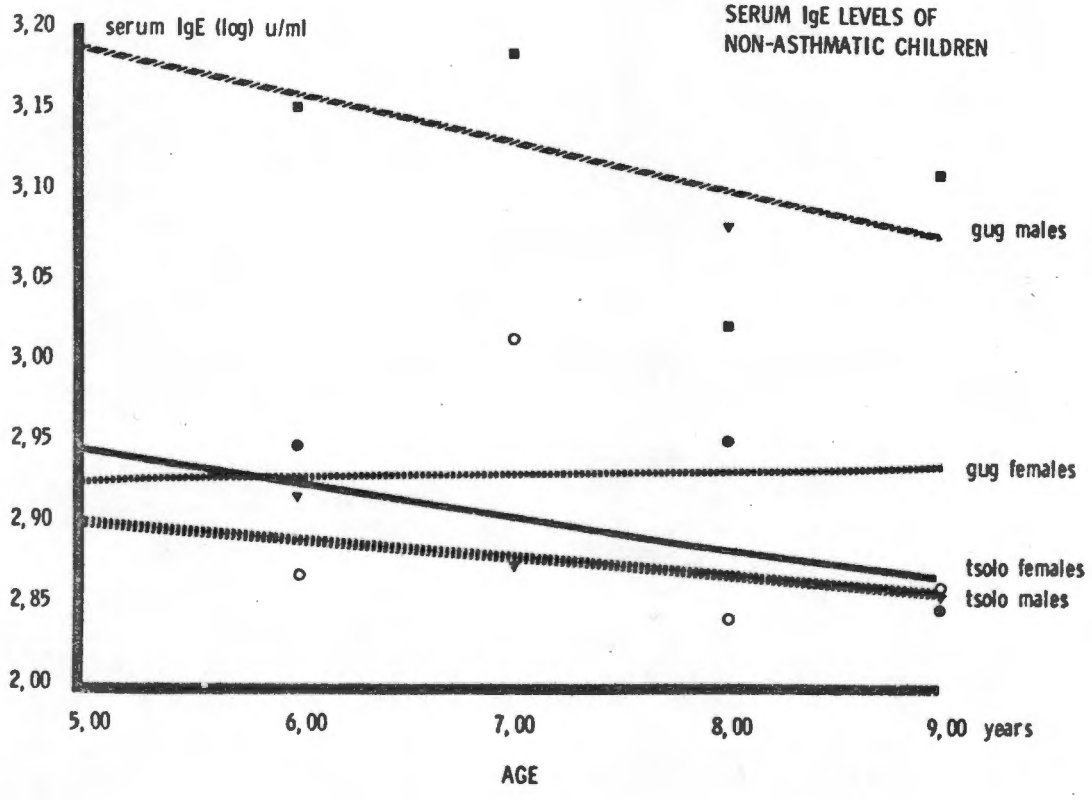

Fig.25 : SERUM IMMUNOGLOBULIN E (IgE) - THREE-WAY ANALYSIS OF VARIANCE

TABLE IV.52

SERUM IMMUNOGLOBULIN G (IgG) LEVEL

(Geometrical mean with 95% tolerance intervals mg per ml)

GUGULETU

Age in Years	6	7	8	9
Boys - IgG mg/ml	1133 - 1740 - 2672	1142 - 1754 - 2694	1125 - 1729 - 2655	1240 - 1904 - 2924
N	62	76	70	107
Girls - IgG mg/ml	1093 - 1679 - 2579	1153 - 1772 - 2721	1185 - 1820 - 2795	1220 - 1875 - 2879
N	71	69	96	118

TSOLO DISTRICT

Age in Years	6	7	8	9
Boys - IgG mg/ml	1049 - 1611 - 2474	1063 - 1633 - 2508	1142 - 1754 - 2695	1129 - 1734 - 2663
N	84	53	71	91
Girls - IgG mg/ml	1079 - 1656 - 2544	1129 - 1734 - 2663	1151 - 1767 - 2714	1175 - 1805 - 2772
N	108	63	77	103

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

TABLE IV.53

SERUM IMMUNOGLOBULIN G (IgG) - THREE-WAY ANALYSIS OF VARIANCE

(mean values mg per 100 ml)

Age in Years	6	7	8	9	PLACE
Non-Asthma - Guguletu IgG mg/ml	1707	1762	1781	1889	1797
Tsolo District IgG mg/ml	1636	1687	1761	1771	1713

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

SIGNIFICANT DIFFERENCES FOR PLACE ($p<0,001$) AND AGE ($p<0,000$)

TABLE IV.54

SERUM IMMUNOGOLBULIN A (IgA) LEVEL

(Geometrical mean with 95% tolerance intervals mg per ml)

GUGULETU

Age in Years	6	7	8	9
Males - IgA mg/ml	76 - 155 - 316	80 - 162 - 331	95 - 194 - 396	94 - 192 - 391
N	62	76	70	107
Females - IgA mg/ml	81 - 165 - 336	82 - 167 - 341	95 - 193 - 393	100 - 203 - 414
N	71	69	96	118

TSOLO DISTRICT

Age in Years	6	7	8	9
Males	85 - 172 - 351	93 - 189 - 385	102 - 209 - 426	108 - 220 - 449
N	84	53	71	91
Females	83 - 168 - 343	92 - 188 - 384	97 - 198 - 404	102 - 208 - 423
N	108	63	77	103

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

TABLE IV.55

SERUM IMMUNOGLOBULIN A (IgA) - THREE-WAY ANALYSIS OF VARIANCE

(mean values mg per 100 ml)

Age in Years	6	7	8	9	PLACE
Non-Asthma - Guguletu	160	165	193	198	182
Tsolo District	170	189	203	213	193

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

SIGNIFICANT DIFFERENCES FOR PLACE ($p<0,000$) AND AGE ($p<0,000$)

TABLE IV.56

SERUM IMMUNOGLOBULIN M (IgM) LEVEL

(Geometrical mean with 95% tolerance intervals mg per ml)

GUGULETU

Age in Years	6	7	8	9
Males - IgM mg/ml	79 - 153 - 296	89 - 173 - 336	83 - 161 - 311	84 - 164 - 317
N	62	76	70	107
Females - IgM mg/ml	90 - 175 - 339	100 - 194 - 376	111 - 215 - 416	112 - 217 - 421
N	71	69	96	118

TSOLO DISTRICT

Age in Years	6	7	8	9
Males - IgM mg/ml	95 - 184 - 357	100 - 195 - 378	104 - 202 - 392	104 - 202 - 392
N	84	53	71	91
Females - IgM mg/ml	113 - 219 - 425	114 - 222 - 430	133 - 257 - 499	121 - 235 - 456
N	108	63	77	103

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

TABLE IV.57

SERUM IMMUNOGLOBULIN M (IgM) - THREE-WAY ANALYSIS OF VARIANCE

(mean values mg per 100 ml)

PLACE	NON-ASTHMA GUGULETU	TSOLO DISTRICT
IgM mg/ml	183	214

SEX	MALE	FEMALE
IgM mg per ml	178	217

AGE	YEARS	6	7	8	9
IgM mg per ml		187	195	210	205

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

SIGNIFICANT DIFFERENCES FOR PLACE ($p<0,000$), SEX ($p<0,000$) AND AGE ($p<0,000$)

TABLE IV.58

ALLERGEN-SPECIFIC IgE MEASURED BY RAST COMPARED TO POSITIVE SKIN REACTIONS AND STOOL FINDINGS FOR 23 ASTHMATIC CHILDREN FROM GUGULETU AND TSOLO DISTRICT

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score									
			Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
		GUGULETU										
109	T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
128	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	0	0	0
146	T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
245	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	0	0	0
245	No stool	Nil	0	0	0	0	0	0	0	0	0	0
345	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	0	0	0
369	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	0	0	0
731	T.trich.	Nil	0	0	0	0	0	0	0	0	0	0
1371	A.lumb., T.trich.	D.farinae	0	1	3	3	1	0	0	0	0	0
1377	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	1	0	0
1409	A.lumb., T.trich.	Nil	2	0	1	0	0	0	0	0	0	0
1451	A.lumb., T.trich.	Nil	0	2	3	3	1	0	0	0	0	0
1590	No stool	Nil	0	0	2	0	0	0	0	0	0	0
2405	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	0	0	0

TABLE IV.58 (Continued)

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
			GUGULETU (Continued)									
2446	A.lumb., H.nana	Nil	0	0	0	0	0	0	0	0	0	0
2939	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	1	0	0
3506	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	0	0	0
4727	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	0	1	0
5395	T.trichiura	Nil	2	1	1	1	0	0	0	1	0	0
5900	A.lumb., T.trich.	Nil	0	0	0	0	0	0	0	1	0	0
7439	A.lumb., T.trich.	Nil	1	0	0	0	0	0	0	0	0	0
9275	T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
			TSOLO DISTRICT									
1547	No stool	C.herbarum	0	0	0	0	0	0	0	0	0	0

Be = Bermuda grass; Me = Meadow grass; Dp = D.pteronyssinus; Df = D.farinae; Hd = Housedust; Clad = C.herbarum; Alt = A.tenius

RAST Score : 0 = Negative; 1 = Doubtful; 2-4 = Positive

A.lumb. = Ascaris lumbricoides; T.trich. = Trichuris trichiura

TABLE IV.59

ALLERGEN-SPECIFIC IgE MEASURED BY RAST COMPARED TO POSITIVE SKIN REACTIONS AND STOOL FINDINGS

FOR 47 CHILDREN FROM TSOLO DISTRICT WITH THE HIGHEST SERUM IgE CONCENTRATIONS

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score									
			Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
4676	No stool	Nil	0	0	0	0	0	1	1	1	0	0
4855	No stool	Nil	0	0	0	0	0	2	2	1	1	0
5150	Nil	Nil	0	0	0	0	0	2	2	1	0	0
5259	Nil	Nil	0	0	0	0	0	2	2	1	0	0
5261	No stool	Nil	0	0	0	0	0	3	3	1	0	0
5272	Nil	Nil	0	0	0	0	0	1	1	1	0	0
5278	No stool	Nil	0	0	0	0	0	1	1	1	1	0
5286	Nil	Maize Food	1	0	0	0	0	3	3	1	0	0
5293	Nil	Nil	0	0	0	0	0	0	0	1	1	0
5348	No stool	Nil	0	0	0	0	0	0	0	0	0	0
5600	No stool	Nil	0	0	0	0	0	1	1	0	0	0
5624	Nil	Housedust	0	0	0	1	0	3	2	0	0	0
5636	Nil	S.A.grass	0	0	0	2	0	2	2	0	0	0
5795	No stool	Nil	0	0	1	0	0	2	1	0	0	0
5802	Nil	Nil	0	1	0	0	0	2	2	0	0	0
5873	No stool	Nil	0	0	0	0	0	1	1	0	0	0
5899	No stool	Nil	0	0	1	0	0	1	1	0	0	0

TABLE IV.59 (Continued)

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score									
			Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
6189	Nil	Nil	3	2	0	0	0	0	0	2	0	0
6334	Nil	SA grass, Maize pollen, Maize food	0	0	0	0	0	1	1	0	0	0
6454	No stool	C.herbarum, Cat, Maize pollen	0	0	0	0	0	0	0	1	0	0
6536	No stool	C.herbarum	0	0	0	0	0	0	0	0	0	0
6564	No stool	Nil	1	1	0	1	0	3	3	1	0	0
6638	Nil	Nil	0	0	0	0	1	1	1	0	0	0
6663	Nil	Nil	0	0	0	1	0	0	0	0	0	0
6714	No stool	Nil	0	0	0	0	0	0	0	1	0	0
6726	Nil	C.herbarum	0	0	0	0	0	2	2	0	0	0
6748	Nil	Nil	0	0	0	0	0	0	0	0	0	0
6823	No stool	Nil	0	0	0	0	0	0	0	0	0	0
6857	Nil	Nil	0	0	0	0	0	1	1	0	0	0
7104	Nil	Housedust	0	0	0	0	0	3	3	0	0	0
7318	No stool	Nil	0	0	0	0	0	0	0	1	0	0
7328	Nil	Nil	0	0	0	0	0	2	2	1	0	0
7476	Nil	Feathers	0	0	0	0	0	0	0	0	0	0
7573	No stool	Nil	0	0	0	0	0	0	0	0	0	0
7818	No stool	Nil	0	1	0	0	0	3	2	1	0	0
8346	No stool	Nil	1	0	0	0	0	3	3	1	0	0

TABLE IV.59 (Continued)

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score									
			Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
8358	No stool	Nil	0	0	0	0	0	0	0	0	0	0
9536	Nil	Nil	0	0	0	2	0	1	1	0	0	0
9687	No stool	Nil	2	2	0	0	0	1	1	0	0	0
9798	T.trichiura	Maize pollen, Maize food, House-dust, C.herbarum	0	0	0	0	0	1	2	0	0	0
9969	Nil	Nil	0	0	0	0	0	0	0	1	0	0
10305	Nil	Nil	0	0	0	0	0	2	2	1	0	0
11223	No stool	Nil	0	0	0	1	0	0	0	0	0	0
11754	Nil	Nil	0	0	0	0	0	0	0	0	0	0
12650	Nil	Nil	0	0	0	0	0	0	0	0	0	0
12886	Nil	Nil	0	0	0	0	0	1	1	0	0	0
18551	No stool	Nil	0	0	0	0	0	2	2	1	0	0

Be = Bermuda grass; Me = Meadow grass; Dp = D.pteronyssinus; Df = D.farinae; Hd = Housedust;

Clad = C.herbarum; Alt = A.tenius

RAST Score: 0 = Negative; 1 = Doubtful; 2-4 = Positive

TABLE IV.60

ALLERGEN-SPECIFIC IgE MEASURED BY RAST COMPARED TO POSITIVE SKIN REACTIONS AND STOOL FINDINGS FOR 48 CHILDREN FROM GUGULETU WITH THE HIGHEST SERUM IgE CONCENTRATIONS

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score									
			Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
6777	No stool	Nil	1	0	0	0	0	0	0	1	0	0
6813	T.trichiura	Nil	0	0	0	0	0	0	0	1	0	0
6964	T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
7251	A.lumbricoides, T.trichiura	Nil	2	0	0	0	0	0	0	1	0	0
7285	T.trichiura	Nil	1	0	0	0	0	0	0	1	0	0
7316	Nil	Nil	0	0	0	0	0	0	0	0	0	0
7357	No stool	Nil	2	1	0	0	0	0	0	1	0	0
7392	A.lumbricoides, T.trichiura	Nil	4	3	0	0	0	0	0	1	0	0
7463	A.lumbricoides, T.trichiura	Nil	1	0	0	1	0	0	0	1	0	0
7521	No stool	Nil	1	0	0	0	0	0	0	0	0	0
7563	A.lumbricoides, T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
7662	A.lumbricoides, T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
7716	A.lumbricoides, T.trichiura	Nil	2	1	0	0	0	0	0	0	0	0
7885	A.lumbricoides, T.trichiura	Nil	1	0	0	0	0	0	0	0	0	0
7955	A.lumbricoides, T.trichiura	Nil	1	0	0	0	0	0	0	0	0	0

TABLE IV.60 (Continued)

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
8335	A.lumbricoides, T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
8429	A.lumbricoides, T.trichiura	Nil	1	0	0	0	0	0	0	0	0	0
8433	A.lumbricoides, T.trichiura	Nil	2	1	3	3	1	0	0	0	0	0
8549	T.trichiura	Nil	2	1	0	0	0	1	0	0	0	0
8651	A.lumbricoides, T.trichiura	Nil	0	0	0	0	0	0	0	1	0	0
8860	A.lumbricoides, T.trichiura	Nil	0	0	0	0	0	0	0	1	0	0
9077	A.lumbricoides	Nil	2	0	0	0	0	0	0	0	0	0
9175	A.lumbricoides, T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
9595	No stool	Nil	1	0	0	0	0	0	0	0	1	0
9597	No stool	Nil	0	0	0	0	0	0	0	0	0	0
9631	A.lumbricoides, T.trichiura, H.nana	Nil	2	1	0	0	0	0	0	1	0	0
9854	No stool	Nil	0	0	0	0	0	0	0	0	0	0
10289	T.trichiura	Nil	1	0	0	0	0	1	0	0	0	0
10538	T.trichiura	Nil	1	1	0	0	1	1	0	1	1	0
10542	A.lumbricoides, T.trichiura	Nil	1	0	0	0	0	0	1	1	1	0
10608	A.lumbricoides, T.trichiura	Nil	3	1	1	0	0	1	1	1	1	0
10837	T.trichiura	Nil	0	1	0	0	0	1	0	0	1	0
11053	No stool	Nil	1	0	0	1	0	1	0	1	1	0

TABLE IV.60 (Continued)

Serum IgE u/ml	Parasitic Ova	Skin Reaction: Allergen	Allergen-Specific IgE : RAST Score Be	Me	Dp	Df	Hd	Cat	Dog	Milk	Clad	Alt
11210	A.lumbricoides, T.trichiura	Nil	1	0	0	0	0	0	1	1	1	0
11382	No stool	Nil	1	0	0	0	1	1	0	1	1	0
11753	T.trichiura	Nil	1	0	0	1	0	1	0	1	1	0
12042	A.lumbricoides, T.trichiura	Nil	1	0	0	0	0	0	0	0	0	0
12430	A.lumbricoides	Nil	1	1	0	0	1	1	1	1	1	1
12610	A.lumbricoides, T.trichiura	Nil	0	0	0	0	0	0	0	0	0	0
13660	No stool	Nil	2	0	3	0	0	0	0	1	0	0
13671	T.trichiura	Nil	0	1	0	0	0	1	0	1	1	1
14088	No stool	Nil	1	0	3	3	0	3	0	0	0	3
14183	A.lumbricoides, T.trichiura	Nil	1	1	0	1	0	1	1	1	1	1
15393	A.lumbricoides, T.trichiura	Nil	1	1	1	1	1	1	0	1	1	0
15622	A.lumbricoides, T.trichiura	Nil	1	1	2	1	1	1	1	1	2	2
17900	A.lumbricoides, T.trichiura	Nil	0	0	0	1	0	1	1	2	1	0
19088	T.trichiura	Nil	2	1	1	1	1	1	1	1	2	0
24860	A.lumbricoides, T.trichiura	Nil	2	1	1	0	1	2	1	2	1	1

Be = Bermuda grass; Me = Meadow grass; Dp = D.pteronyssinus; Df = D.farinae; Hd = Housedust; Clad = C.herbarum; Alt = A.tenius

RAST Score: 0 = negative; 1 = doubtful; 2-4 = positive.

TABLE IV.61

TOTAL EOSINOPHIL COUNT IN PERIPHERAL BLOOD (T.E.C.)

(cells per µl)

Number of Eosinophils	22-440 per µl	441-1000 per µl	more 1000 per µl
Asthma - Guguletu N = 21	5 (23,8%)	9 (42,9%)	7 (33,3%)
Non-Asthma - Guguletu N = 670	241 (36,0%)	301 (44,9%)	128 (19,1%)
Tsolo District N = 648	615 (94,9%)	28 (4,3%)	5 (0,8%)

PERCENTAGE OF TOTALS IN BRACKETS

TABLE IV.62

NUMBER OF STOOLS COLLECTED FROM CHILDREN FROM GUGULETU AND TSOLO DISTRICT

	Guguletu children	Tsolo District children
Sample size	694	671
Stools collected	540 (77,8%)	387 (57,7%)
Stools with parasites	524 (97,0%)	38 (9,8%)
Stools without parasites	16 (3,0%)	349 (90,2%)

TABLE IV.63

TYPES OF HELMINTHIC PARASITIC OVA IN STOOLS OF CHILDREN FROM GUGULETU AND TSOLO DISTRICT

Type of Ova	Guguletu children	Tsolo District children
T.trichiura (total)	485	13
T.trichiura (sole parasite)	143	7
T.trichiura and others		
+ A.lumbricoides	316	2
+ A.lumbricoides and H.nana	17	2
+ H.nana	9	1
+ Taenia	-	1
A.lumbricoides (total)	372	10
A.lumbricoides (sole parasite)	38	5
A.lumbricoides and others		
+ T.trichiura	316	2
+ T.trichiura and H.nana	17	2
+ H.nana	1	1
H.nana (total)	27	23
H.nana (sole parasite)	-	19
H.nana and others		
+ T.trichiura	9	1
+ T.trichiura and A.lumbricoides	17	2
+ A.lumbricoides	1	1
Taenia species	-	1
Hookworm	-	3

TABLE IV.64

HAEMOGLOBIN LEVELS (Hb)

(mean ± 1 S.D., g/dl)

GUGULETU CHILDREN

Age in years	6	7	8	9
Boys - N	61	71	66	103
Hb - g/dl	12,0 ± 1,1	12,5 ± 1,1	12,4 ± 1,0	12,8 ± 0,8
Girls - N	68	69	95	118
Hb - g/dl	12,1 ± 1,1	12,1 ± 1,2	12,6 ± 1,0	12,9 ± 1,1

TSOLO DISTRICT CHILDREN

Boys - N	81	52	64	90
Hb - g/dl	13,5 ± 1,0	13,6 ± 1,0	13,8 ± 0,9	13,9 ± 0,8
Girls - N	104	60	72	99
Hb - g/dl	13,6 ± 1,0	13,6 ± 1,0	14,0 ± 0,8	14,1 ± 1,0

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

TABLE IV.65

HAEMOGLOBIN - THREE-WAY ANALYSIS OF VARIANCE

(mean values g/dl)

Age in Years	6	7	8	9	PLACE
Non-Asthma Guguletu Hb g/dl	12,09	12,29	12,51	12,85	12,49
Tsolo District Hb g/dl	13,53	13,63	13,91	14,03	13,78

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

SIGNIFICANT DIFFERENCES FOR PLACE ($p<0,000$) AND AGE ($p<0,000$) BUT NOT FOR SEX

TABLE IV.66

TOTAL WHITE BLOOD COUNT (W.B.C.)

(mean ± 1 S.D. per µl)

Guguletu Children

Age in Years	6	7	8	9
Boys - N	61	71	66	103
WBC/µl	8718	8990	8844	8365
	±2208	±3130	±3003	±7015
Girls - N	68	69	95	118
WBC/µl	8668	8333	7731	9026
	±2799	±2545	±2755	±2342

Tsolo District Children

Boys - N	81	52	64	90
WBC/µl	9025	8438	8423	8826
	±3284	±2680	±3071	±3248
Girls - N	104	60	72	99
WBC/µl	8694	9715	8858	8465
	±3348	±7111	±3169	±3565

ASTHMATIC CHILDREN FROM GUGULETU EXCLUDED

NO DIFFERENCE FOR PLACE, AGE AND SEX

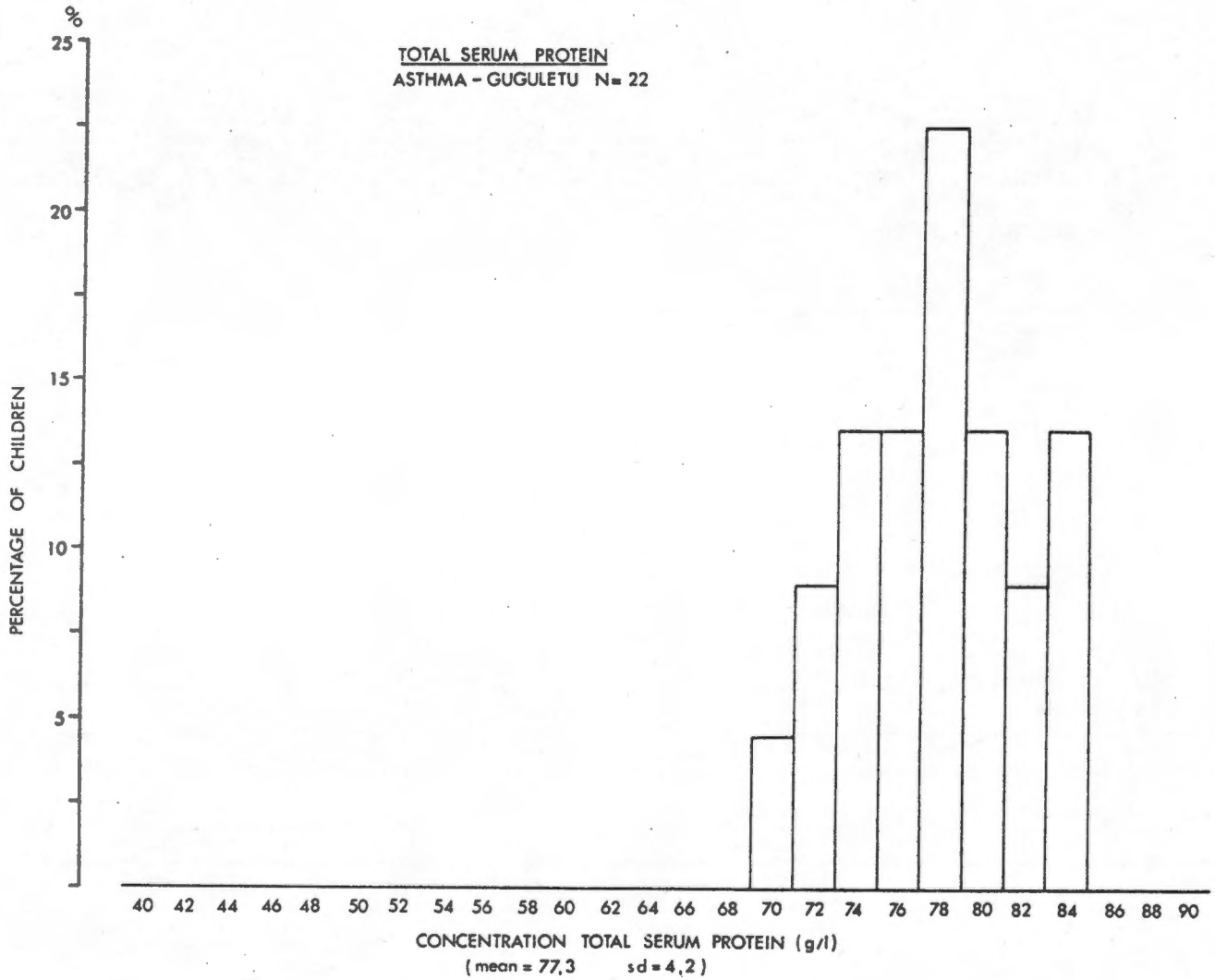

Fig. 26 : TOTAL SERUM PROTEIN LEVELS FOR ASTHMATIC CHILDREN FROM GUGULETU

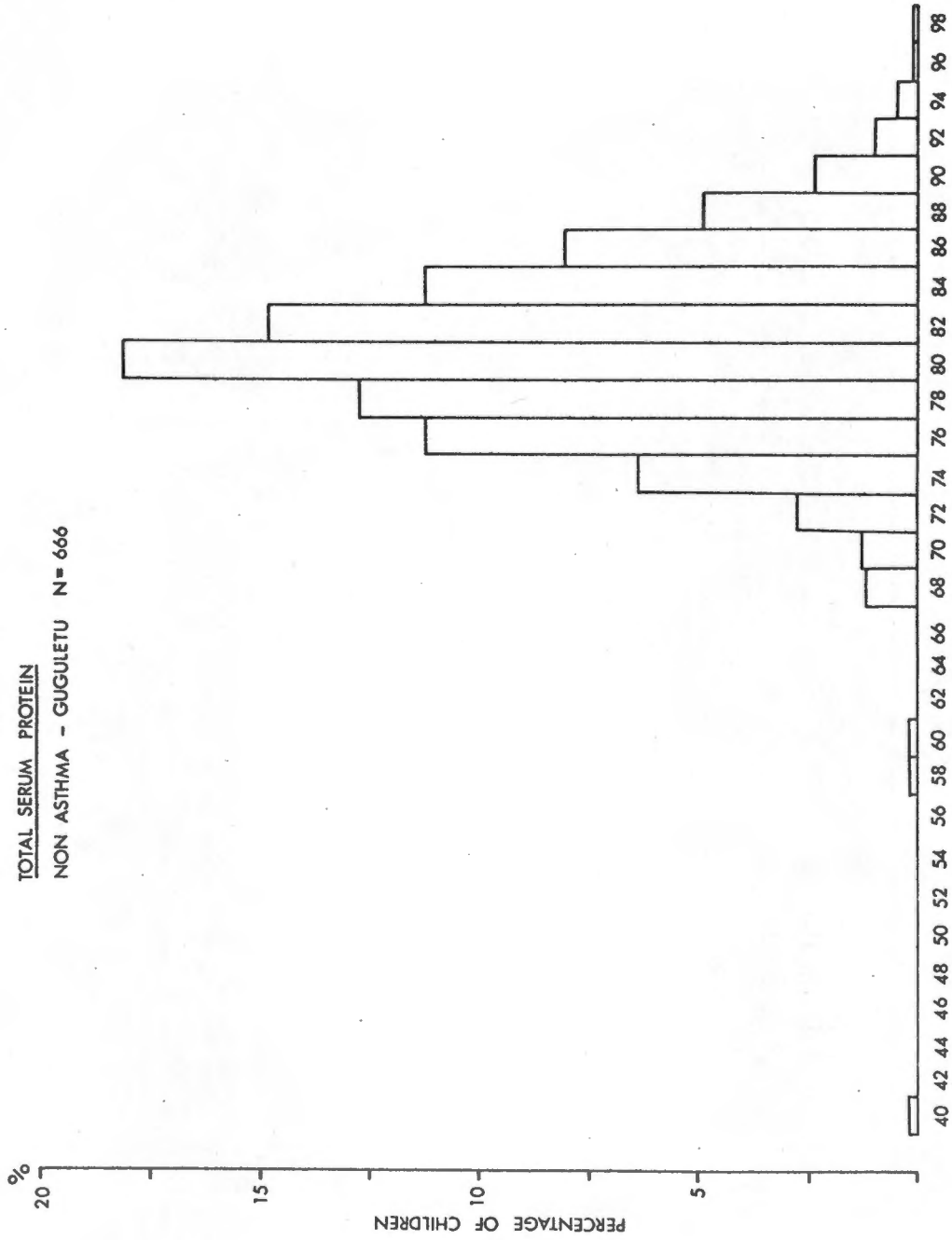

Fig. 27 : TOTAL SERUM PROTEIN LEVELS FOR NON-ASTHMATIC CHILDREN FROM GUGULETU

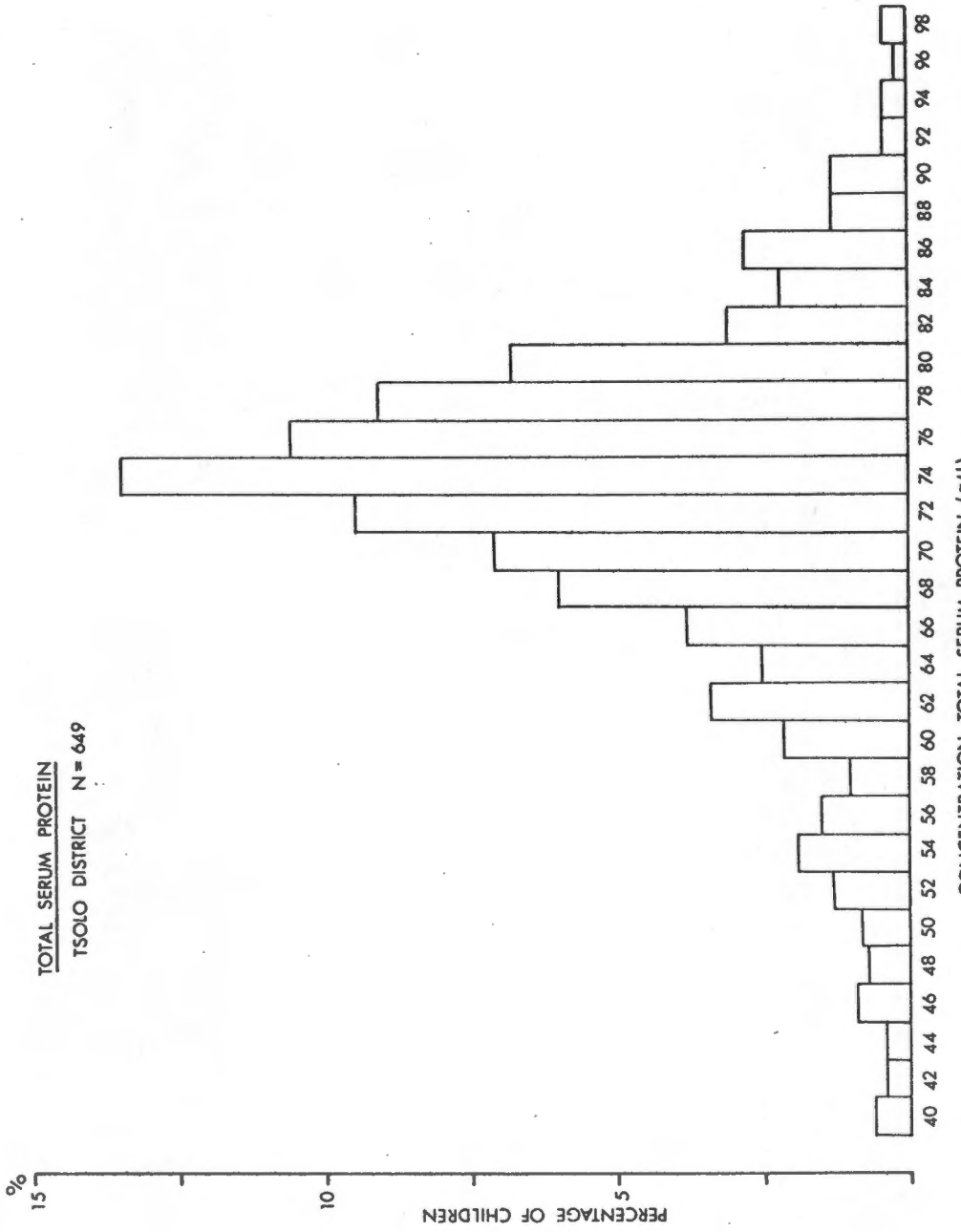

Fig. 28 : TOTAL SERUM PROTEIN LEVELS FOR CHILDREN FROM TSOLO DISTRICT

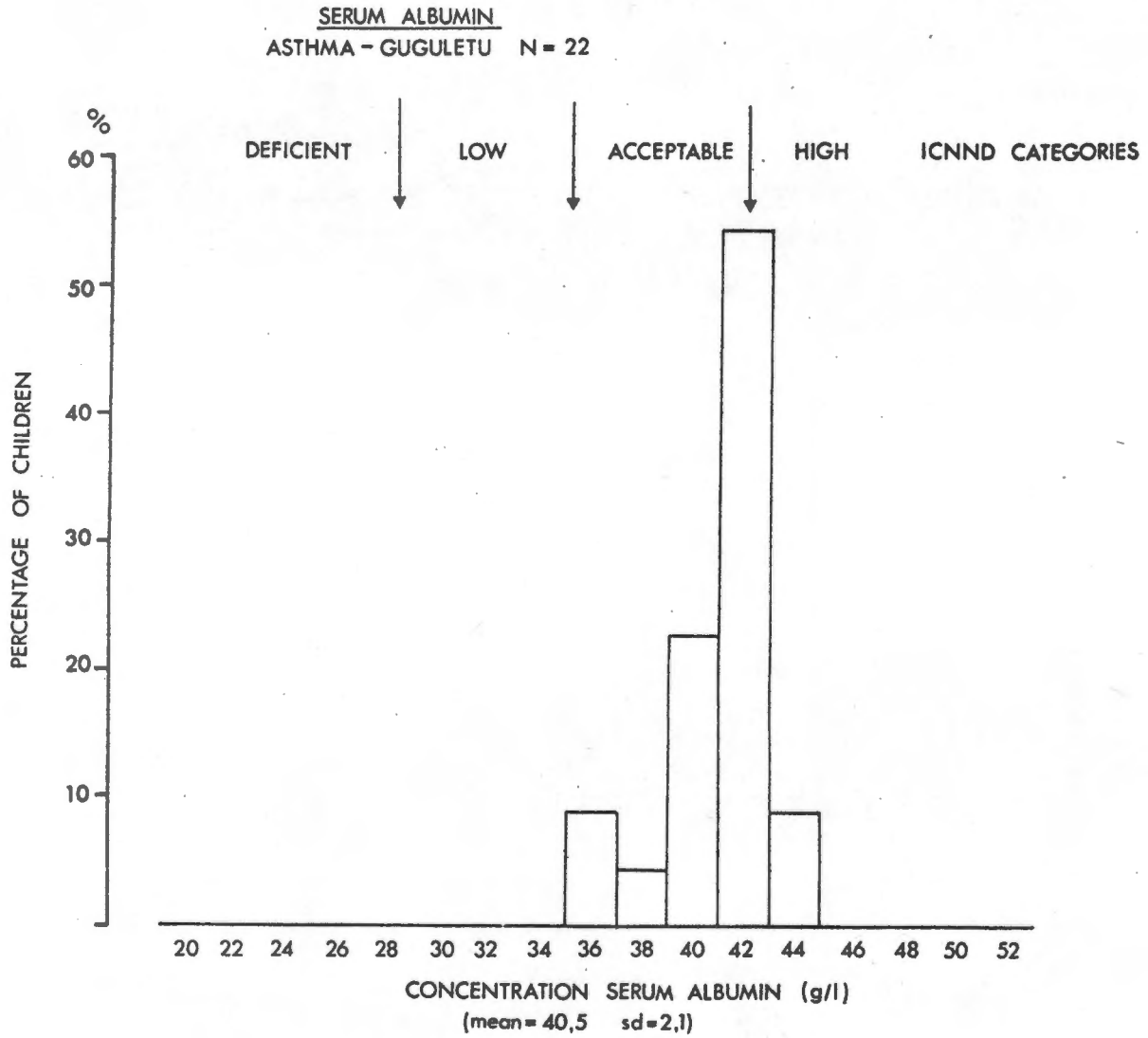

Fig. 29 : SERUM ALBUMIN LEVELS FOR ASTHMATIC CHILDREN FROM GUGULETU

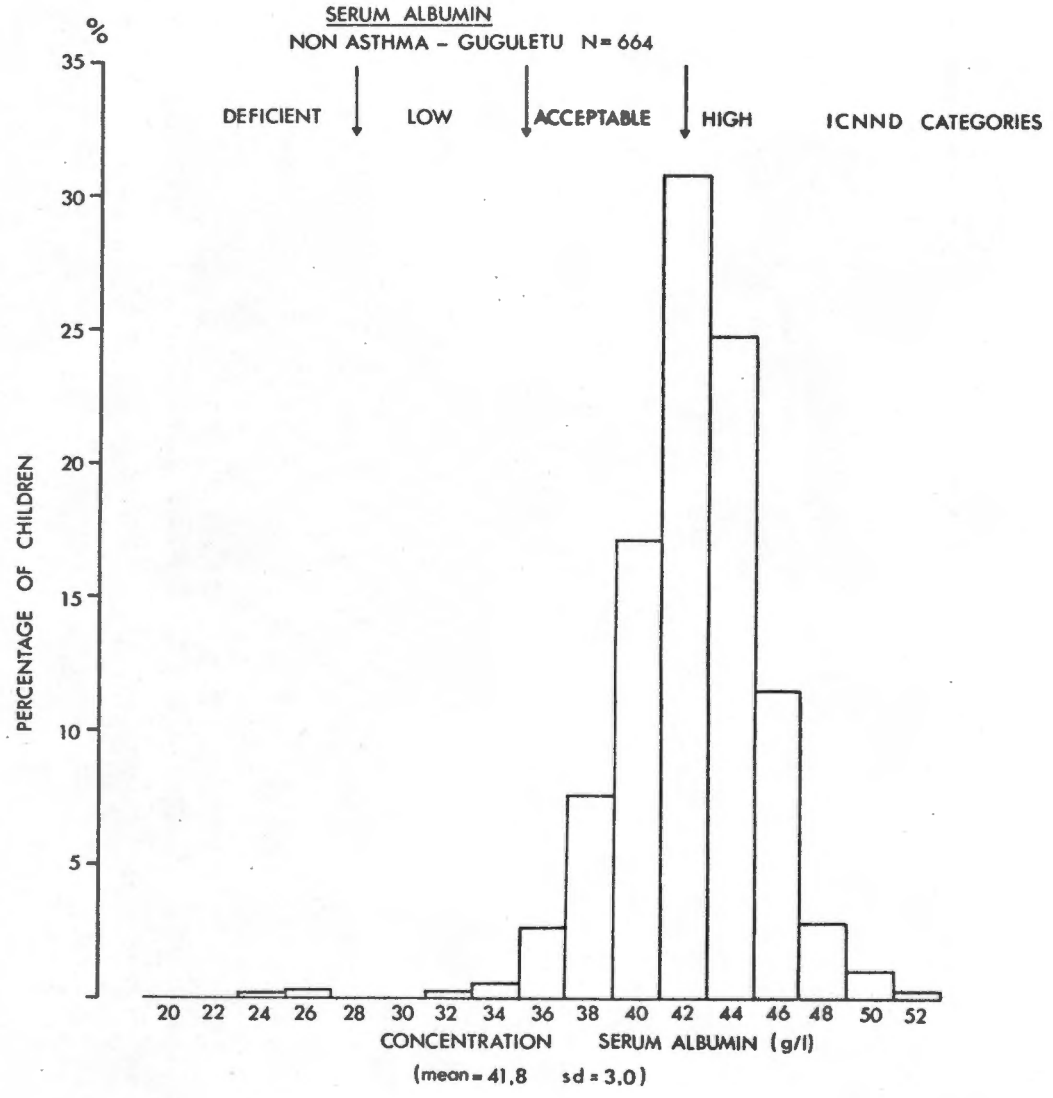

Fig. 30 : SERUM ALBUMIN LEVELS FOR NON-ASTHMATIC CHILDREN FROM GUGULETU

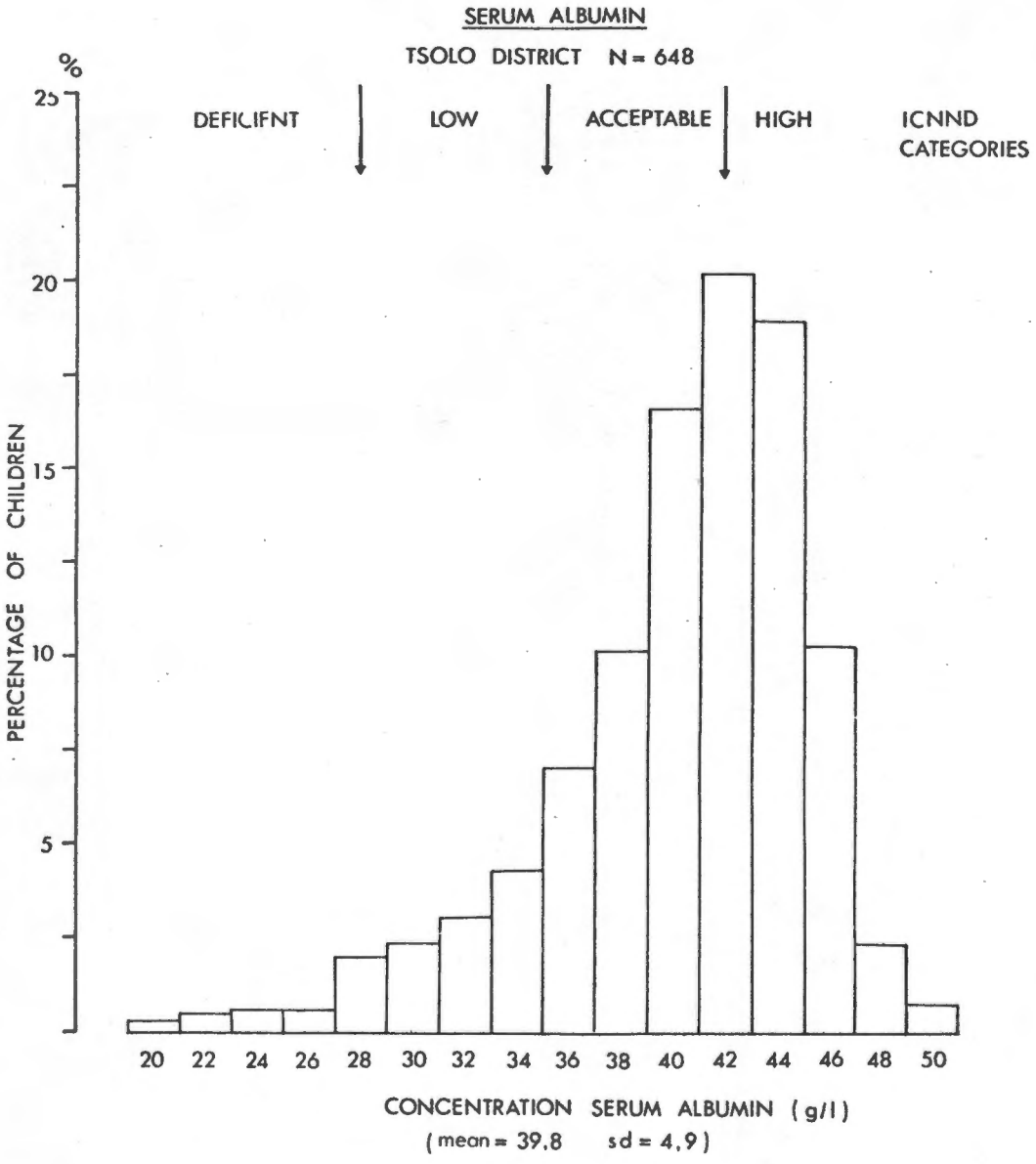

Fig.31 : SERUM ALBUMIN LEVELS FOR CHILDREN FROM TSOLO DISTRICT

SECTION V

DISCUSSION

CHAPTER 1 : INTRODUCTION

Attempts to reach a universally accepted definition of asthma have failed (Ciba Guest Symposium, 1959; Ciba Foundation Study Group 38, 1971). The proposed but not universally accepted definition of asthma as a variable, intermittent, episodic bronchial airways obstruction that changes its severity is only a functional one. It does not in any way relate to the cause. The degrees of variability, intermittency and reversibility of the airways are not stipulated.

This inability to define and lack of agreement as to what constitutes asthma remains an impediment to epidemiological studies of the condition. Such studies are furthermore plagued by many of the methodological problems which characterise investigations of poorly defined chronic conditions with low case-fatality rates. In addition there is the natural history of asthma with its tendency to intermittency and spontaneous remission.

The diagnosis and management of the child with asthma in clinical medical practice is one of continual assessment. This assessment is based on a thorough history and evaluation of the entire clinical picture, relevant investigations and laboratory procedures aimed at the identification of the causative allergens (Sheldon, Lovell & Mathews, 1967; Aas, 1975; Jones, 1976). Of these investigatory procedures, a carefully performed prick skin test is probably the most important.

The accuracy and extent of the history obtained from parents in an epidemiological survey like this one are unreliable. Chai et al (1968) critically evaluated the validity and reliability of a history. They concluded that "any research program that uses only historical data as a measurement in the investigation of asthma will result in incorrect conclusions most of the time".

This related to the fact that reliance on memory was a poor reflection of past events even from very observant mothers. Recall over a 24 hour period was also of a low order of accuracy. Both parents and patients tended to remember only the more serious episodes.

In this survey the populations under study had a different language and culture to that of the author. This necessitated the mandatory use of an interpreter. Misconceptions regarding asthma are not uncommon in rural communities in Africa, where it is thought to be infectious or the cause of infertility (Sofowara, 1970) or where no word for "wheeze" exists in the language (Warrell et al, 1975). Under such circumstances the value of a history is not simply questionable, but useless. There is no reason to believe that the community of rural Tsolo district would differ much in this respect from others in Africa. A considerable number of the families (13,4%) used only traditional Xhosa medicine.

In a study where the point prevalence of asthma is measured, singly performed clinical examinations and pulmonary function tests without challenge, i.e. exercise and others, to identify the asthmatic subjects are of little if any value. The findings of both may vary tremendously in the asthmatic child. No abnormality may be found in between attacks. The absence of clinical signs or abnormal

pulmonary function tests do not rule out ventilatory impairment (Chai et al, 1968). In fact no abnormal signs may be found on examination of the chest even in moderately severe asthma (Jones, 1976; Sheldon, Lovell & Mathews, 1967). McNicol, Williams & Gillam (1970) studied a random sample of 276 asthmatic children with varying grades of asthma. The children were evaluated only between and not during asthmatic attacks. Only 3% of these had evidence of chest deformities. They all fell into the severe grade of asthma.

Of the total sample 12% had rhonchi on auscultation and 7% had evidence of airway obstruction as judged by spirometry. It is, therefore, apparent that clinical signs are uncommon between attacks even in children with proven asthma. The clinical examination and pulmonary function tests relate only to the time when they are undertaken, and in no way to the previous or future status of the bronchial airways.

Investigatory and laboratory procedures are conducted to evaluate the asthmatic child. These include prick skin testing, total serum IgE estimations, total eosinophil count in peripheral blood, radiograph of the chest and allergen-specific IgE in selected cases. There is, however, no single specific or pathognomonic procedure for childhood asthma (Sheldon, Lovell & Mathews, 1967; Aas, 1975).

To measure the prevalence rates of asthma in the 2 samples of children, another way of identifying the asthmatic child had to be used. As the point prevalence was measured, the criteria for identifying the asthmatic subjects had to be specific for asthma.

Exercise-induced asthma was used as the sole diagnostic criterion for identifying the asthmatic children. It is unique for the asthmatic child (Godfrey, 1974) and failure to provoke bronchoconstriction by appropriate exercise raises serious doubts of the diagnosis (Jones, Wharton & Buston, 1963). In view of its high frequency in asthmatic children it was recently suggested as a suitable marker for prevalence studies of asthma (Editorial, Br.Med.J., 1977).

It has been used as such in a limited way in epidemiological studies of asthma, mainly to confirm the diagnosis based on a history. Burr, Eldridge & Borysiewicz (1974) studied PEFR before and after exercise in 812 school children. A questionnaire relating to chest illnesses, amongst others asthma, was completed pre-exercise. The historical findings were then correlated with the drop in PEFR. They found that children with current asthma, as based on the history, had a marked fall in PEFR. In another study induction of bronchospasm by means of exercise was studied in 69 children selected from a group of 2 500 (Nicolaescu et al, 1974). These 69 children were divided into 11 asthmatics diagnosed by general practitioners, 28 "wheezy" children according to parental history and 30 randomly selected normals. Exercise-induced bronchial lability showed significant difference between the 3 groups. The authors suggested that exercise-induced bronchial lability be used as a simple screening test for the separation of normal from abnormal subjects in epidemiological surveys. Exercise-induced asthma was not used in either of these studies as the only diagnostic criterion, but in conjunction with a previously obtained history of asthma.

Carswell, Meakins & Harland (1976), however, used only exercise-induced asthma to identify asthmatic subjects among 128 school children from

Tanzania. Their diagnostic criterion of a drop in PEFR of more than 10% post-exercise is however possibly too small (Van Niekerk et al, 1977).

Exercise-induced asthma was used as the only criterion for identifying the asthmatic children from the selected samples. It was defined as a drop of 15% or more in post-exercise FEV_1 and PEFR from pre-exercise levels. The exercise stimulus used in every child was running on a level in the open for 6 minutes. The pace was such as to raise the heart rate to 170 beats or more per minute. This exercise stimulus has been shown to be the most productive of bronchospasm in an asthmatic (Godfrey, Silverman & Anderson, 1973; Fitch, 1975; Godfrey, 1975).

The children in the Tsolo district sample live in a rural community following traditional Xhosa ways and often tribal customs. The use of a Vitalograph and Wright Peak Flow Meter was very strange to the unsophisticated children, in spite of individual instruction in the use of the apparatus. A drop in both FEV_1 and PEFR was therefore used to eliminate the child where a change might have been due to technical reasons. An equal drop in both these readings would very likely be due to exercise-induced asthma.

Twenty-three of the 1 365 children were identified as asthmatic subjects. All had a drop of 15% or more in both FEV_1 and PEFR post-exercise from pre-exercise values (Table IV.1).

The mean percentage drop in FEV_1 and PEFR was marked (32,7% and 32,2% respectively). The percentage drop in FEV_1 and PEFR were indeed very similar for each individual child. This is to be expected, as both

FEV_1 and PEFR have been shown to correlate closely with sensitive whole body plethysmographic tests (Cropp, 1975). When dealing with a sophisticated sample of children, it probably makes no difference whether the FEV_1 or PEFR test is used for identifying the asthmatic child.

It is unusual for a child under 5 years of age to co-operate sufficiently to be able to perform pulmonary function tests accurately (Woods, 1969). This had to be borne in mind in selecting the sample to be studied. No difficulty was experienced with children for either sample. Aged 6 to 9 years they were all capable of performing both the tests after adequate demonstration.

Children suspected of being asthmatic were given 2 puffs of fenoterol aerosol inhalant after the post-exercise FEV_1 and PEFR were recorded. Each puff contained 200 mcg fenoterol hydrobromide. These recordings were repeated 5 minutes later (Tables IV.3, p. 109; IV.4, p. 110). This procedure was only performed in 17 children. The improvement, judged as the percentage of the original post-exercise FEV_1 and PEFR values after 2 puffs of fenoterol, was marked in all cases. This was evident for both the FEV_1 and PEFR with a mean percentage improvement from the original of 92,9% and 98,1% in FEV_1 and PEFR respectively. In a number of children the percentage improvement was greater than 100%. This suggests that these children may have had bronchospasm before the pulmonary testing procedures. The post-fenoterol FEV_1 and PEFR readings were almost identical to the original pre-exercise values for most of the 17 children. This would indicate a reversal of the bronchoconstriction induced by the exercise stimulus.

The mean percentage drops in post-exercise FEV_1 and PEFR after free running for 6 minutes in these 23 asthmatic children are similar to those found by other authors (Silverman & Anderson, 1972: mean % fall PEFR - 39%; Eggelston, 1975: mean % fall FEV_1 - 27%; Godfrey, Silverman & Anderson, 1973: mean % fall PEFR - 47%; Burr, Eldridge & Borysiewicz, 1974: mean % fall PEFR - 20,5%).

The normal non-asthmatic children in both groups had virtually no change in post-exercise from pre-exercise values (Table IV.18, p. 134). The change in pre-exercise values of FEV_1 to post-exercise levels was -0,80% (S.E.M.± 0,54) for Guguletu children and 0,26% (S.E.M.± 0,33) for Tsolo district children. The PEFR changes were similarly insignificant; 0,15% (S.E.M.± 0,31) and 0,17% (S.E.M.± 0,30) for Guguletu and Tsolo children respectively. A negative value indicates bronchodilatation whereas a positive value indicates bronchoconstriction. These results were similar to those of other workers who reported that normal children show very little change in bronchial calibre as a result of exercise (Heimlich, Strich & Busser, 1966; Jones & Jones, 1966; Anderson, Connolly & Godfrey, 1971; Fitch & Morton, 1971; Lefcoe, Carter & Ahmad, 1971; Burr, Eldridge & Borysiewicz, 1974; Bierman, Kawabori & Pierson, 1975).

Only a small number of children from both samples had a drop of 15% or more in either FEV_1 or PEFR; 45 from Guguletu and 21 from Tsolo district (Tables IV.16, p. 131; IV.17, p.133). Of the 45 children from Guguletu, 34 (75,6%) had a drop of 15% or more in FEV_1 and 11 (24,4%) in PEFR. The findings for children from Tsolo district were similar where 14 (66,7%) had a drop of 15% or more in FEV_1 and 7 (33,3%) in PEFR. These children were not regarded as asthmatic as they did not have a drop of 15% or more in <u>both</u> FEV_1 and PEFR.

The higher number of "false positives" for FEV_1 in a considerable number of children is probably technical. The writing stylus was not at the zero mark in many of these recordings. This would account for the wide discrepancy between FEV_1 and PEFR values found in some of the children. It may also be that the technique of performing the PEFR on the Wright Peak Flow Meter was more readily understood than performing the FEV_1.

There were, however, also children where a considerable drop was measured in both FEV_1 and PEFR (12 from Guguletu and 3 from Tsolo district). It is possible that some of these children were in fact asthmatic and would have been identified as such on repeat testing.

From these results it can be concluded that either FEV_1 or PEFR can be used to determine exercise-induced asthma in a large population survey. Recording only the PEFR on a Wright Peak Flow Meter may be preferable to recording only the FEV_1. This would result in fewer "false positive" recordings.

Subjecting a sample of children to a single exercise stimulus, as in this study, will result in about 80,1% of the asthmatic children developing significant post-exercise bronchoconstriction (cf.p.53). Subjecting the sample to further exercise stimuli should result in identifying almost all of the asthmatic subjects (Godfrey, 1974). Therefore, the 23 children identified as asthmatic subjects in this study represent about 80,1% of the total number in the 2 samples. They are moreover true asthmatics as it is only the child with asthma who will develop significant exercise-induced symptoms. Children suffering from other pulmonary diseases, especially wheezy bronchitis, are excluded from the asthmatic group.

Wheezy bronchitis and other pulmonary disorders may result in increased post-exercise bronchial lability, mainly bronchodilatation. The marked drop in post-exercise FEV_1 and PEFR due to bronchoconstriction is found only in the asthmatic child (cf.pp.50,51). As the normal child shows only a minimal or post-exercise drop (Silverman & Anderson, 1972; Burr, Eldridge & Borysiewicz, 1974) it is highly unlikely that any children were erroneously identified as asthmatic in this study. The ability to reverse the drop in FEV_1 and PEFR (ie. bronchoconstriction) by inhalation of fenoterol, is further supportive evidence that these 23 children are asthmatics.

Exercise-induced asthma is proposed as a reliable method of identifying the asthmatic child in a large epidemiological survey. It has a low false positive response rate as indicated by the finding that 66 of 1 365 (4,8%) children had a drop of 15% or more in either FEV_1 or PEFR. If the change in PEFR alone is measured, 18 of 1 365 (1,3%) children would have erroneously been identified as asthmatic children.

CHAPTER 2 : PREVALENCE RATES OF ASTHMA

Twenty-three of the 1 365 children studied were identified as asthmatic. Of these, 22 were from the urban Guguletu sample and only 1 was from the rural Tsolo district sample. The point prevalence of childhood asthma for children from Guguletu was 3,17% (31,7 per 1 000 children). In contrast a figure of only 0,14% (1,4 per 1 000 children) was found for children from Tsolo district. These prevalence rates refer to children aged 6 to 9 years.

No comparable studies on the prevalence rates of asthma in urban or rural children have been done in this country which represent a childhood population (cf.pp.8,9,10 & 11). The study by Wesley, Clyde & Wallace (1969) only reflected hospital admissions where more stringent criteria were employed for admission of Black children. The prevalence of 7% for asthma found by Shore (1959) only represents the prevalence of asthma among Jewish children and children from policemen's families seen in a specialist referral practice. As the mode of identifying the asthmatic subjects was not indicated by either author, any comparison between their findings and those of this study would be highly speculative and inappropriate.

The prevalence of asthma of 3,17% of the urban Guguletu sample is similar to that reported from the United Kingdom, United States of America, Australia and New Zealand. The "calculated" mean prevalence rates for these countries are 3,49% for the United Kingdom, 4,46% for the United States of America and 4,1% for Australia and New Zealand (cf.p.13). Studies from these countries of industrialised western communities were undertaken. In view of the divergent criteria used to identify asthmatic

subjects in these studies (cf.p.26) the significance of the differences in these reported prevalence rates and that found in this study for Guguletu cannot be assessed. Inter-study comparison is not possible. However, the trends found in these developed western style countries are similar to the 3,17% for the children from Guguletu.

Guguletu forms part of a large westernised metropolitan area. It has the facilities of a western industrialised community. The way of life in Cape Town is like that of cities from the United Kingdom, United States of America, Australia and New Zealand.

One of the criteria for inclusion in the study was that the child had to have lived in the designated area for at least the previous 4 years. The majority of the children studied in Guguletu had lived there most of their lives (Table IV.21, p.137). In fact, it could be assumed that at least 95% of the children studied had probably been born and brought up in Guguletu. The lifestyle to which they are exposed and accustomed is that of a modern western industrialised community.

In striking contrast is the prevalence of 0,14% for asthma in children living in Tsolo district, Republic of Transkei. This prevalence rate compares closely with reports from Africa and other less developed communities: 0,18% for children aged 0 - 10 years from the urban area of Patna, India (Viswanathan et al, 1966); 1,06% for children 5 - 15 years from Barbados (Pearson, 1973) and a calculated prevalence of 0,007% for the Lufa rural area of New Guinea Highlands (Anderson, 1974). Findings on childhood asthma in Africa are based on non-factual statements in studies involving mainly adult subjects. Such reports seem to indicate

that childhood asthma is rare, especially in the rural communities (Johansson, Mellbin & Vahlquist, 1968; Wasunna, 1968; Mitchell, 1970; Godfrey, 1975; Warrell et al, 1975). The only exception to this is a study of Carswell, Meakins and Harland (1976) of 128 Tanzanian school children. The criterion for diagnosing asthma was a drop of more than 10% in post-exercise PEFR value after running on the level for 6 minutes. A prevalence of 3,3% was found. A drop of 15% or more is the more generally accepted cut-off point for the diagnosis of exercise-induced asthma (cf.p.49). A prevalence rate of 3,3% is therefore questionably high.

Prevalence rates of asthma in Scandinavian countries have all been consistently low. These range from 0,25% to 1,37% (cf. Table II.5). The reason for these low rates from these developed and industrialised countries is not clear. It is possible that these studies do not reflect the true prevalence rates. It may well be that they are an under-representation of the prevalence (cf.p.17). It seems, however, unlikely that under-reporting is the cause of the consistently low prevalence rates of asthma in all studies from these countries and other unknown factors may well play an important role. No explanation can be given for the low prevalence rates in the Scandinavian countries which approximate that of rural Tsolo district and other less developed, especially rural, communities in the world.

A male preponderance has been a constant finding in many epidemiologic studies of asthma in children (reviewed by Gordis, 1973). No satisfactory explanation has been given for this, variously reported as 2:1 and 2,7:1 for children up to 15 years (Rackemann & Edwards, 1952; Arbeiter, 1967; Dawson et al, 1969). This preponderance of males is

found until puberty, when a reversal of the sex ratio takes place (Pearson, 1958; Crawford & Beedham, 1976).

In this study a reversed ratio has been found with a female preponderance. The ratio of boys to girls for the group of asthmatic children from Guguletu was 8 boys to 14 girls or 1:1,75. The reason for this is not apparent. The number of asthmatic children is very small and they may not represent the true ratio among all asthmatic children from Guguletu. In the selection of a sample of children to be studied, the male preponderance amongst asthmatic children must be borne in mind. If not, it may influence the findings considerably. Erroneous conclusions may be reached if the population studied comprises a large number of boys. The optimum sample should have a boy to girl ratio as near as 1 to 1 as possible.

In this study the selection of the samples was from the total childhood population of the 2 designated areas. The sample of children from Guguletu was selected randomly from the total number of houses in Guguletu irrespective of schools or area. The sample from Tsolo district included every child aged 6 to 9 years that lived within a radius of 9 km from the St. Cuthbert's Mission. Sample selection based upon school populations may differ significantly from one another in terms of whether the schools are boys', girls' or co-educational, which take children of a particular religion only, or on a socio-economic basis (private schools with high fees compared to government schools with low fees), as well as hostels with children from other areas and different ethnic groups. Added to this is the fact that 35,9% of the children in the Tsolo district were 'Red' people.

The Xhosa nation of the Ciskei and Transkei has been divided, for several generations, into two opposing cultural camps - 'Red' people and 'school' people (Mayer & Mayer, 1974). The 'Red' people are the traditionalist Xhosas, the conservatives who still adhere to the indigenous way of life. The 'school' people are products of the Mission and the school upholding Christianity, literacy, education, etc. as ideals. They are not simply town people or people living under town influence: the division lies in the countryside itself. The 'school' child goes to school, while the 'Red' child goes out herding, helps with household chores or just roams around. Any sample of Transkei children drawn from school populations, therefore, would immediately eliminate a significant number ie. the 'Red' children who do not attend school which would be extremely biased and would not give a true picture of the lifestyle of the rural child.

It was accordingly decided to select the samples from the houses or kraals of the communities under study, in order to obtain an unbiased selection of children representing the broad childhood population of the communities.

The ratio of boys to girls in the two samples was similar. The Guguletu sample comprised 324 boys to 370 girls ie. 1 : 1,14, whereas the Tsolo district sample comprised 311 boys to 360 girls ie. 1 : 1,16.

Even when correction is made for the female preponderance in the sample of Guguletu children, the female preponderance for the asthmatic children is still significant. The 8 asthmatic boys represent 2,47% of the sample of boys and the 14 girls represent 3,78% of the sample of girls. The percentage ratio of boys to girls is still 1 : 1,53.

The age range of the children under study is important not only from the point of view of the age of onset, but also for the upper limit of the ages of the subjects included in a study. Asthma is a dynamic situation and the tendency is for children to outgrow their asthma. Rackemann & Edwards (1952) re-examined 688 children 20 years after they had been diagnosed as asthmatic and found that 30,7% outgrew their symptoms by an average age of 15 years. Another 19,3% were quite well, provided they avoided contact with certain allergens to which they were still sensitive. A further 21,4% or more had little if any asthma, but did have other manifestations of allergy - usually hayfever. Thus 71,4% of the children suffering from asthma were found, 20 years later, to have become free of the disability by about the age of 15 years. Twenty-six percent of the original children still had asthmatic symptoms but this was severe in only 10,9%. Similar results were obtained by Buffum (1963), Freeman & Johnson, (1964) and Buffum & Settipane (1966).

In selecting a population in which the epidemiology of asthma will be studied, the importance of age must be borne in mind. The prevalence rates of asthma in any childhood population should be expressed for the specific age group in order to be meaningful. In the majority of children the onset of asthma will be during the first 5 years of life (cf.p.22) and a significant number of these will outgrow their asthma by 15 years of age. The age range 6 to 9 years is probably the most representative group.

The distribution of boys and girls according to age ie. 6 to 9 years differs significantly between the non-asthmatic Guguletu and the Tsolo district groups (Tables IV.19 and IV.20). It is noteworthy that girls aged 8 and 9 years represented 60,4% of the total sample of girls for the non-asthma

Guguletu group. In Tsolo district they represented 50,3% of the total sample of girls which is the same for boys from both groups of these ages.

The 2 samples were selected in such a way as to represent the larger childhood population. The reason for these differences is not apparent. Neither is the reason for the high number of 7 year old asthmatic girls clear. The 7 year old group of girls from Guguletu represented only 20,8% of the sample of girls.

About 80% of asthmatic children will develop significant exercise-induced asthma if subjected only once to an appropriate exercise stimulus. The 23 asthmatic children (22 from Guguletu and 1 from Tsolo district) represent about 80% of the total number of asthmatics in the 2 samples respectively. Based on this, a 'corrected' prevalence rate representing all asthmatic children can be calculated for both the areas. The 'corrected' prevalence rate is 27,5 of 694 children or 3,96% for urban Guguletu and 1,25 of 671 children or 0,19% for rural Tsolo district. As the 'corrected' prevalence is a calculated assumption, it will not be used. Reference will only be to the prevalence as determined by a single exercise stimulus.

CHAPTER 3 : FACTORS RELATING TO THE PREVALENCE RATES

A significant difference in the prevalence rates for asthma between the two samples of children was found. Is this a real difference and a true reflection of the prevalence of asthma in the children from these 2 communities or does it reflect, as in many previous studies, only a difference in terminology and/or methodology ? The author believes the prevalence rates are real for the 2 samples. The terminology in identifying the asthmatic subject has been constant throughout for each child. The identification depended solely on demonstration of exercise-induced asthma following an appropriate exercise stimulus. The definition of exercise-induced asthma was independent of interpretation by either parents, myself or the people who assisted me. The difference in prevalence rates is also highly unlikely to be due to methodological differences. The same apparatus and techniques were used throughout. The people assisting with the actual recording of the FEV_1 and PEFR in the children were familiar with the techniques. Great care was taken to ensure that the recordings obtained were accurate. The pieces of apparatus, the Vitalograph and Wright Peak Flow Meter, were calibrated before commencing both the Guguletu and the Tsolo district studies. Although actual readings were recorded for each of the indices used, they were used only in the calculation of the percentage drop in post-exercise from pre-exercise values in each case.

Any reading error due to not being calibrated would be a constant one pre- and post-exercise for each child. It would not affect the percentage drop in post-exercise value from pre-exercise levels.

The difference in prevalence of asthma between the 2 samples of children is

real and significant in magnitude. There must be one or more factors responsible for this.

1. Genetic

One of the criteria for "atopic hypersensitiveness, hayfever and asthma" was the inheritance of a dominant gene by the subject (Coca & Cooke, 1923). Many epidemiological studies of asthma have shown a familial tendency towards allergic disorders, supporting the concept that asthma is an inherited disease (Shore, 1959; Maternowski & Mathews, 1962; Freeman & Johnson, 1964; Dawson et al, 1969; Özkarogöz & Çakin, 1969; Anim & Adoo, 1972). The prevalence of asthma among children born of asthmatic parents was higher than that among children born of non-asthmatic parents (Milne, 1969; Turner, Rosman & O'Mahoney, 1974). To establish the mode of inheritance, Edfors-Lubs (1971) studied 7 000 pairs of twins and found low concordance rates in both monozygotic (25,3%) and dizygotic (16,1%) twins. She concluded that a distinction between recessive, dominant and multigenic inheritance could not be made. In a review of the genetic aspects of allergy, Cohen (1974) stressed the difficulties encountered in studies on allergy as a result of sample bias, terminology and methodology. He was unable to provide a clear answer to the mode of inheritance.

Allergic diseases, including asthma, have both an environmental and a hereditary component. The asthmatic individual is genetically selected or predisposed to the development of the clinically recognisable disorder. The environment and consequent exposure to allergens, is essential for the onset of clinically overt allergic disease. The genetically predisposed individual needs to be exposed and sensitised to a specific allergen or

allergens before the clinical disorder will manifest itself. The interplay of hereditary and environmental factors is illustrated in a study of the prevalence of ragweed pollinosis in foreign and native students in the United States of America (Maternowski & Mathews, 1962). The prevalence of ragweed pollinosis in the foreign students from various countries throughout the world was found to be no different from that of students native to America. However, it was notable that 23 of the 38 foreign students who developed ragweed pollinosis had previously had no atopic symptoms in their countries of origin. The onset also occurred more than ten times more frequently among foreign students during their second to fifth ragweed seasons in the United States than among native students of the same age. The explanation for these differences was that latent atopy was present in the families of many of the foreign students but had not resulted in clinically overt atopic disease until some member of the family was exposed to a potent allergen such as ragweed. Strict avoidance of the offending allergens is one of the only two rational means of treating the allergic child (Rapaport, 1972).

Could the difference in the prevalence rate be due to genetic or racial differences ?

The Black people of South Africa are divided into four main cultural-linguistic groups: the Nguni, Sotho-Tswana, Venda and Shangaan-Tsonga. These divisions are based principally on historical, linguistic and cultural differences and can, in turn, be subdivided into nine separate ethnic groups, each with its own language, legal system, lifestyle, values and socio-political identity. The Nguni tribes are concentrated mainly in the eastern and south-eastern areas of South Africa. This major division comprises the Xhosa, Zulu and Swazi nations (Van der Spuy, 1974).

The Xhosa nations live in the Transkei and Ciskei and have always been both a pastoral and an agricultural people.

Over the last two generations a major population trend among the Black people has been their migration to the white urban centres. In 1970 Cape Town had a Black population of 107 877 (Van der Spuy, 1974) of which the overwhelming majority in 1963 were Xhosa (Wilson & Mafeje, 1963). They are not original inhabitants of the Western Cape but have migrated there from Transkei or Ciskei.

Only Xhosa children were admitted into the study from both areas. The difference in prevalence, therefore, is unlikely to be due to either racial or genetic differences.

The Black child has the same if not a greater tendency than White children to develop asthma. Smith (1973) found asthma to be uncommon in Negro immigrant children born outside the United Kingdom. However, it was more prevalent among Negro children born in the United Kingdom than among the indigenous white population.

2. Environment

The environment has a profound effect on the development of allergic disorders, such as asthma. Environment in this instance embraces a very broad concept. It not only relates to the natural environment of the community, but also to the home environment. It is in the home where the child spends a considerable amount of his time. The individual families' home environment reflects their individual way of life. It will differ

from family to family. A family's lifestyle may relate to its own cultural and socio-economic status. This individual way of life may result in a different allergenic exposure or in protective measures for a particular child.

Factors relating to the individual child's way of life and allergenic exposure have been measured. These were urban and rural communities, socio-economic status, exposure to allergens and feeding patterns during infancy. Each of these has been evaluated to try and establish those that may be precipitating or protective factors in the prevalence of asthma in children from Guguletu and Tsolo district.

(a) Urban and Rural Communities

It has not been shown that living in either an urban or rural community influences the prevalence rate of asthma in western industrialised communities. This applies equally to the United Kingdom, United States of America and Australia where the prevalence rate for asthma is similar for urban and rural communities (cf.pp.13, 14). Reports from the Scandinavian countries are more varied. In a study of 7 000 pairs of twins from Sweden, Edfords-Lubs (1971) found no effect of living in an urban or rural community on the prevalence of asthma. Koivikko (1974) found that the area of residence did not have an important influence on the progress of asthma among Finnish children. Peltonen, Kasanen & Peltonen (1954-55) noted the low proportion of allergic conditions among the rural population. Whether the prevalence of asthma was significantly different in urban and rural populations is not apparent from their study.

By contrast, striking differences have been found in the prevalence rates for asthma between urban and rural communities in Africa and most other tropical and underdeveloped areas. The reported findings of virtually no cases of asthma among rural communities from these countries is consistent (cf.Table II.1;p.11). Asthma, however, is more prevalent among those people who have changed from a rural traditional lifestyle to an urban, westernised way of life. Wesley, Clyde & Wallace (1969) from Durban found that 5 cases of asthma among Black children admitted over a 5 year period to the hospital came only from an urban environment.

Godfrey (1975) found no cases of asthma among 1 200 inhabitants of rural Gambian communities, but asthmatic subjects were readily found in the capital town of Banjul. Woolcock (1972) found asthma to be exceedingly rare amongst people living in the rural New Guinea Highlands. Where it did occur it was mainly in people who had been exposed to European communities and the age of onset was delayed. Anderson (1974) found childhood asthma to be exceedingly rare in the rural New Guinea Highlands.

The conclusion can thus be drawn that residence in an urban or rural environment does not significantly influence the prevalence of asthma in the western, industrialised communities. In the tropical and less developed societies, however, asthma is found mainly in urban communities and there appears to be a striking absence of asthmatic cases in rural environments. Is this due to the difference between the urban and rural environments or to a difference in lifestyle between people who live in urban and rural communities ? It would seem to be the way of life rather than the area, which is responsible, as illustrated by reports from westernised, industrialised countries. Herxheimer (1964) found that

bronchial asthma was almost unknown in American Indians before 1931, but since then, a small number of typical cases have been encountered in some tribes in Arizona and New Mexico. The estimated incidence, however, was much less than in the White population. It would, therefore, appear that a change in the lifestyle of these American Indians has taken place and that the difference in prevalence of asthma is not racial. In a review of all hospital admissions since 1946, representing a 17 year period with 5 000 hospital admissions, only 3 cases of asthma were diagnosed for the Canadian Eskimo population in the MacKenzie Delta and the coast east to Booth Peninsula (Herxheimer & Schaefer, 1974). Ford (1969) reviewed 11 551 cases of asthma and found it to be widespread throughout Australia, affecting all ages. Australian Aborigines, however, appeared to be rarely affected.

It would appear as if the American Indians, Canadian Eskimos and Australian Aborigines, although they live in modern industrialised countries, are protected against developing asthma and that this 'protection' is wearing off for the American Indian.

Studies undertaken of Xhosa people in cities have indicated the condition of urbanisation refers not just to the length but to the quality of the life that is lived in town. An individual who has lived for a long time in town will not necessarily show the genuine urbanised quality. He may actually be planning to return to the country; dislike townlife and consider himself an outsider, whereas the urbanised man accepts the values of an industrial society and regards the town as his home. The townsman seeks to achieve a European standard in food, dress and furnishings (Wilson & Mafeje, 1963).

The Xhosa in town can be divided into : (Wilson & Mafeje, 1963; Mayer & Mayer, 1974)

(a) Townrooted people who have a home in town and never want to live in the country

(b) Countryrooted people who after living in town for many years still regard the country as their home

(c) Townrooted people who do not value at all the diacritic institutions of the real townspeople and try to create as far as possible the moral and cultural atmosphere of their old pre-urban homes

The degree of urbanisation was assessed according to these 3 categories as well as the use of medical services, which were divided into modern western medical facilities, or traditional Xhosa medicine or where a combination of both were used (Tables IV.29, IV.31).

As the degree of urbanisation refers more to the urban sample, the division between 'Red' and 'School' people was established for the rural Tsolo District sample.

The use of medical services was also used as an indicator of adapting to a western style of life.

The majority of families from Guguletu (85,5%) regarded themselves as urbanised. They have adopted the city as their home and have no wish to return to living in the country. This finding is not unexpected. Sample selection was such that only children who had lived for the last 4 years or longer in Guguletu were included. Most of the children had lived their

whole lives in Guguletu (Table IV.29, p.154). The use of medical services by families from Guguletu was predominantly that of modern medical facilities. This is in keeping with the fact that most families have adapted largely to the western way of life in the city. It is, however, noteworthy that 16,8% of the families from Guguletu still used a combination of traditional and modern medical methods.

The families from Tsolo District represented a truly rural one and all but one family regarded the country as their home. The use of traditional Xhosa medicine either alone or in combination with available modern medical facilities was favoured by a considerable number of families.

A significant number of families (35,9%) from Tsolo District declared themselves as the 'Red' people. Their way of life would be even less westernised. It would be tribal (Mayer & Mayer, 1974).

The rest regard themselves as being 'school' people. A considerable number of children from the total sample still sleep on mats and most grind their own maize. It can be assumed that although about a third of the families were tribal in their orientation, the rest still lived according to the more traditional rather than the urban way of life.

There has, for a number of years, been a tendency for Black people in South Africa to move to urban areas (Van der Spuy, 1974). This movement entails more than just a move from a home in a rural area to one in a city. It involves a change from a traditional and often tribal to an urban western way of life. Although westernisation may not take place,

certain changes occur in the way of life. It is often a process of acculturation. It results in neither complete substitution of western ways for indigenous ones, but in a new way of life in which continuity with both traditions is recognisable (Pauw, 1973). In spite of the process of acculturation the effect of an urban and western way of life is almost total in certain spheres. The urban Xhosa rely almost exclusively on Western forms of occupation as a means of livelihood. They also follow western patterns in respect to housing, furniture and clothing (Pauw, 1973).

(b) Socio-Economic Factors

The evaluation of information obtained from study in the human sciences is, however, not without limitations. One of the difficulties encountered in obtaining the information is the reliance on memory and personal experience.

The socio-economic status of each family from which an eligible child was selected, was examined. Factors that were looked at were (1) the occupation grade of the head of the household (2) the education of parents and grandparents (3) economic status based on the average income ratio and whether a family had access to electricity and (4) the degree of crowding in the home.

(i) Head of the Household and Occupation Grade (Tables IV.27, IV.28)

The differences in occupation of the head of the household were significantly different for the 2 main populations ie. non-asthma Guguletu and Tsolo district for fathers, mothers and grandparents. The occupation grades of the head of the household from Guguletu

were more in the unskilled and semiskilled grades than from Tsolo district, where more were in the unemployed or pension grades. The percentage in the clerical and business occupations was, however, higher in the Tsolo district.

The father, in all 3 groups of children, was most frequently the head of the household but even more so for families from Tsolo district. A grandparent was more frequently the head for both urban groups ie. asthma and non-asthma families in Guguletu. This was not the case for families from Tsolo district where the mother was more often the head of the household. These differences are statistically significant between non-asthma families from Guguletu and families from Tsolo district.

There is no difference between asthmatic and non-asthmatic families from Guguletu as far as who is head of the household, as well as their occupation, is concerned.

The occupation grade of the head of the household is used as an indicator of social class. In this study it is apparent that the majority of families from Guguletu fell into the lower social classes. In Tsolo District a considerable number were either pensioners or unemployed. It is therefore not possible to categorise them into social classes but likewise the majority of families fell into the lower social classes.

(ii) Formal Education of Parents and Grandparents (Table IV.26)

In the evaluation of the role of education among the parents and grandparents of the children studied, the fact that 35,9% of families from Tsolo district declared themselves as being 'Red' people had to be

borne in mind, This may well account for the high number of people from Tsolo district who had no formal education.

The information on the education levels was analysed as follows: (i) education at all levels ie. post-matric to nil and unknown and (ii) by excluding those individuals who had no education or where it was unknown and were analysed as education for all school standards, only high school and only primary school standards.

In comparing the education of fathers to mothers and grandfathers to grandmothers, there is no difference for the asthmatic children from Guguletu. The differences for the non-asthmatic groups from Guguletu and Tsolo district were highly significant. Parents of the asthmatic children both had some degree of education which was equal. The education of parents and grandparents at each level in the two main population groups also showed differences.

The education of the fathers differed in that more from Tsolo had attended high school compared with Guguletu, whereas the differences at primary school were not significant. Differences similar to those found for fathers also exist for the education of maternal grandmothers, maternal grandfathers and paternal grandfathers, but not for paternal grandmothers.

The education of mothers differed significantly for all standards, for high and for primary school. The differences lie in a variation of the school standard attained by mothers in the 2 groups but does not necessarily indicate a higher level of overall education in a particular group.

These differences are a result of a better educated population in the Tsolo district among those who did attend school ie. excluding the people with no or unknown education. The latter probably represents the 'Red' people who as part of the culture do not attend schools.

The difference in parental education of asthmatic children relates to an overall higher level of education compared with the non-asthmatic children. This is particularly so for the mothers of whom at least 50,1% had been to high school. The majority of parents of asthmatic children had reached standard 3 or higher.

(iii) Economic Status (Tables IV.22, IV.30, Figures 7 and 8)

This was assessed according to the average income ratio and whether or not electricity had been installed in the home. A score of 100% indicates an existence at a bare minimum level and a score of less than 100% indicates that the household is subsisting below the bare minimum level. A score of 150% is the effective minimum level (Batson, 1942). The average income ratio is not a measurement of wealth but of the minimum subsistence level of the household. It should be noted that these levels are an estimate of the income needed by an individual household to maintain a bare minimum level of health and decency. It allows only for the basic requirements of food, clothing, fuel and lighting, cleansing materials, transport and housing. Nothing is allowed for amusement, sport, hobbies, education, medical or dental care, holidays, newspapers, stationery, tobacco, or for comfort or luxuries of any kind nor for replacement of household equipment or furniture or hire purchase, insurance or savings.

The economic status of families with an asthmatic child was not statistically different from those families with non-asthmatic children. More than half of the families for both the asthmatic and non-asthmatic groups from Guguletu had an average income ratio of 100% or less (54,5% and 50,2% families respectively). Only about a quarter (27,3% and 25,7% families respectively) had an average income ratio of 160% or more. It costs about R 400 to have electricity installed into a house in Guguletu. The number of families with asthmatic children (18,2%) who in fact had had electricity installed was slightly less than those who had an average income ratio of 160% or more. Considerably less families from the non-asthmatic group (9,0%) had electricity in their homes. The difference, however, is not statistically significant. The 10 families from Tsolo district who had electricity in their homes were all employed and accommodated at either the Mission or the hospital at St. Cuthbert's.

No attempt was made to correlate the average income ratio to the household density ratio. Most houses in Guguletu are 4 roomed. The allocation of houses is according to a government regulation and not on social or economic grounds. It was not uncommon for the bread-winners in families from Tsolo district to be migrant workers, therefore the average income ratio was not calculated.

The findings on the relationship between social class and the prevalence of asthma are contradictory. The popular clinical impression is that asthma is more frequent in the upper social classes (Editorial, Lancet, 1967). Is this in fact true ?

Graham et al (1967) found that asthma was more prevalent in upper and middle social classes and that the lower social classes were under-represented. They considered the possibility that the difference could be due to greater utilisation of medical services by the higher class mother. Lower class mothers usually worked and tended not to attend school medical examinations, where the data was collected on which this study was based, so that the asthmatic children of these mothers remained unidentified. This possibility seemed to them unlikely. Freeman & Johnson (1964) noted that an allergic history was found three times more frequently among upper social classes than among lower classes, but felt that the availability of medical care was related to the ability to pay and that the apparent difference in prevalence could reflect only differences in the utilisation of services. An increased prevalence for asthma in the upper social classes has also been found in Finland by Peltonen, Kasanen & Peltonen (1954-55). In a survey which included all children aged 0 - 14 years in Finland, Koivikko (1974) found that 10,8% of the cases had educated, non-manual working parents, 58,7% had educated manual working parents and 30,5% had parents who were unskilled manual workers. He concluded that social and living conditions had no marked effect on the severity of asthma.

Dawson et al (1969) screened 2 511 Aberdeen school children and found that children with severe asthma were predominantly from the lower social classes and tended to come from families with 4 or more children. When mild, moderate and severe cases were pooled, no social class differences could be demonstrated. Schmerler & Abramowicz (1974) found that socio-economic status was unimportant in patients admitted to the Bronx Municipal Hospital Centre.

Sofowara (1970), on information obtained from the asthma clinic of the teaching hospital in Ibadan, Nigeria, found that 86% of asthmatic cases came from the lower income groups. However, after the establishment of a special asthma clinic, there was a tendency for an increasing number of patients from higher social classes to attend the clinic. These cases had probably been seen previously by private general practitioners. Anim & Edoo (1972) found the same tendency in Ghana, in that 79% of asthmatic cases came from lower social classes and they commented on the absence of members of the higher socio-economic brackets and suggested that these groups probably consulted private doctors. A somewhat higher prevalence of asthma in the lower income groups was also found by Viswanathan et al (1966) in a study of the population of Patna, India.

The opposite of this has, however, been reported from other parts of the African continent. Rees et al (1974) described the typical asthma patient from Nairobi, Kenya, as a young adult Kikuyu from a reasonable socio-economic background. Wesley, Clyde & Wallace (1969) from Durban found that asthma admissions to the hospital were exceedingly low for Bantu children and the 5 cases seen during a 5 year period all came from comparatively well-to-do families, with at least one parent in the professional class. Godfrey (1975) on the other hand, examined 44 asthmatics who lived in Banjul, capital town of Gambia, and found that 24 were from less privileged homes, whereas the rest (20) came from economically privileged homes. The distinction between economic differences were not defined.

The role of social class in the prevalence of asthma is unclear. It would seem that children with asthma are found in homes of all social classes and no fixed pattern emerges for a relationship between the prevalence of asthma and social class.

(iv) Degree of Crowding (Tables IV.23, IV.24, IV.25, Figures 9 - 11)

The size of the families from Tsolo district tended to be smaller than those of the asthmatic and non-asthmatic children from Guguletu. Only 13 of 459 (2,8%) families from Tsolo had 10 or more inhabitants, whereas 128 of 416 (30,8%) families from Guguletu had 10 or more inhabitants per household. Most of the families from Guguletu lived in 4 roomed houses. The number of people sharing the bedroom with the selected child in each family did not differ significantly for the 3 groups of children. An average of 4,3 people shared the room with the asthmatic child from Guguletu. The household density ratio was significantly different between the non-asthmatic families from Guguletu and those from Tsolo district. The smaller families from Tsolo district were less crowded with 14,5% of them having a household density ratio less than 100%.

All but one of the asthmatic children came from houses which were crowded.

There are many differences between the people from Guguletu and those from Tsolo district regarding social, economic and educational factors, This is not entirely unexpected as the one group lives in a western urban community and the other in a rural community.

The families from Guguletu tended to be larger, more crowded and have a higher number of people sleeping in the same room. Compared to Tsolo district the father is less often the head of the household and a grandparent not uncommonly fulfills this role. The occupation of the head of the household in Guguletu more frequently than in Tsolo district tended to be unskilled or semiskilled. The parents and grandparents of families from Guguletu tended to have a lower level of formal education, reaching only junior school. Of those who attended school in the Tsolo district, more reached high school level. The educational levels for females (ie. mothers and grandmothers) was on the whole higher than that of the males. The only exception to this was for families with an asthmatic child.

The families with an asthmatic child were in no way different from the families with non-asthmatic children as far as crowding, number of people per bedroom or head of the household and his/her occupation are concerned. There was no difference in average income ratio of families in the 2 groups where a considerable number existed below the bare minimum level.

The education of parents of asthmatic children, however, differed from the asthmatic children in that both parents had reached equal levels of education.

As most families in both areas belonged to the lower socio-economic classes and only 22 asthmatic children were identified in Guguletu, the effect of these factors on the prevalence rate of asthma cannot be evaluated.

CHAPTER 4 : EXPOSURE TO ALLERGENS

There is a significant difference in the prevalence of asthma for Xhosa children living in an urban western community and those from a traditional rural community. Seen superficially the difference is a geographical one. The development of the overt clinical picture of allergy is profoundly influenced by exposure to allergens in the environment. Allergic asthma is a manifestation of the immediate local anaphylactic type of allergic tissue reaction (Coombs & Gell, 1975). This reaction is mediated by IgE antibodies which are formed on exposure to allergens. A number of allergens have been shown to be important in the aetiology of asthma. Such allergens are house dust and house dust mites, D.pteronyssinus and D.farinae, plant pollens, animal epithelium, foodstuffs and mould species (Vaughan & Black, 1948; Sheldon, Lovell & Mathews, 1967; Sarsfield et al, 1976).

An attempt has been made to evaluate exposure to allergens in the environment of the 2 groups of children. For the purpose of this study, the environment can be divided into the natural and the home environment.

There is an abundance of flora, both grass and others in the Cape Peninsula where Guguletu is situated. Tsolo district is situated among rolling grass hills with interspaced agricultural lands on which maize and kaffircorn are cultivated. There is no reason to believe that the magnitude of exposure to plant pollens is significantly different in the two areas. Exposure to the grass family pollens may well be even higher in Tsolo district.

The home environment may well differ from family to family eg. the presence or absence of pets. This difference in home environment may well result in a difference in allergenic exposure.

The 4 factors studied from which a difference in allergenic exposure may result were animals in the home, exposure to maize, current dietary intake and sleeping habits.

1. Animals in the Home (Table IV.33)

Exposure to pets can be an important cause of asthma (Sarsfield et al, 1976). It was noticeable that 50% of the asthmatic children from Guguletu had no pets or animals at home. A similar tendency for the non-asthmatic children from Guguletu was noticed in that 42,3% of the children had no pets. Where there were pets in the home, dogs were the commonest. Against this, most children from Tsolo district had some exposure to animals in the home. Only 11% of the children had no animals in the home. Here dogs and chickens were the commonest animals and just over half (51,4%) of the children were exposed to both dogs and chickens in their homes. These chickens wandered in and out of the homes and it was not uncommon to see a hen nesting on eggs in a hut used either for sleeping or living.

2. Maize (Table IV.34)

Maize dust has been shown to be a common cause of respiratory allergy in South Africa (Ordman, 1958b). Maize was used in the diet of a considerable number of children from Guguletu - both asthmatic and non-asthmatic. None of these children, however, were exposed to maize pollen as it was not cultivated in Guguletu. In the Tsolo district, 95,2% of the children were exposed to maize not only as food but also as a cultivated crop which was ground at home.

3. Current dietary intake (Table IV.35)

Certain foodstuffs have been shown to relate to the development of asthma (Vaughan & Black, 1948; Sheldon, Lovell & Mathews, 1967). The ingestion of some of these foods by the children were studied. The intake of these foods was only assessed in terms of whether the child ate the particular food or not. The frequency and quantity ingested was not assessed. The intake of these foods were common in most children irrespective of area. The overall pattern of intake of these foods is, however, statistically significantly different between the non-asthma Guguletu and Tsolo district groups. The differences lie in the intake of milk, eggs, chocolate, oranges and tomatoes. The differences for the intake of milk, although statistically significant (x^2 = 5,45, Df = 1 p = 0,19534) is not really important as only 3,9% (26 of 671) of the children from Tsolo district did not drink milk compared to 1,8% (12 of 672) non-asthmatic children in Guguletu. The difference in the intake of eggs is more definite as 17,1% of children from Tsolo district did not eat eggs compared to 1,8% from Guguletu. Almost all children from Guguletu - both asthmatic and non-asthmatic - had milk, bread and eggs in their diet, while a considerable number ate chocolates, oranges and tomatoes. Milk and bread were eaten by almost all children from Tsolo district. Eggs were eaten by 82,9% of children, compared to 98,2% of children from Guguletu. Chocolate, oranges and tomatoes were eaten less often by children from Tsolo district. The intake of the last 3 items was influenced by the fact that there are only 3 shops in the study area.

It is therefore clear that the children from both urban and rural areas were exposed to a variety of allergens, mainly pollens, in the natural environment. Exposure to allergens in the home environment showed certain

differences in the 2 groups. Animals were more often found as pets in Tsolo district. Exposure to maize allergen - either food pollen or grain was also more common for these children.

The difference in prevalence of asthma for children of the 2 areas is, however, striking. In spite of an abundant exposure to inhalent allergens, ie. pets, grass, maize, as well as dietary allergens, asthma was almost absent among children from Tsolo district.

4. Sleeping Habits (Table IV.32)

The effect on the asthmatic subject of sleeping on a bed has been known for many years. As early as 1684 T. Willis noted "whatsoever therefore makes the blood to boyl, or raises it into an effervescence, as violent motion of the body or mind, excess extern cold or heat, the drinking of Wine, Venery, yea sometimes meer heat of the Bed cloth cause asthmatica assaults to such as predisposed. It is usual that those who are obnoxious to this disease oftentimes dare not enter into a Bed, only sleep in a chair, or on a bed, being covered with garments. The reason wherof is, that the body covered and heated by Bed-cloaths, the blood being a little raised into a more quick motion, and grow hot, requires a more plentiful sucking in of Air .....".

The house dust mites, Dermatophagoides pteronyssinus and D. farinae, have since been shown to be the major allergenic cause of asthma in many parts of the world, including Africa, irrespective of whether the population is urban or rural (El-Hefny, 1966; Mitchell et al, 1969; Buchanan & Jones, 1972; Pearson & Cunnington, 1973; Rees et al, 1974; Sarsfield, 1974; Cookson & Makoni, 1975; Godfrey, 1975; Warrell et al, 1975; Godfrey & Griffiths, 1976; Van Niekerk, Shore & Weinberg, 1977).

The majority of asthmatics from these countries have positive skin reactions to these mites and relate their symptoms to housedust. Mites have been readily isolated from homes in these countries (Buchanan & Jones, 1972 & 1974; Pearson & Cunnington, 1973; Rees et al, 1974; Cookson & Makoni, 1975; Warrell et al, 1975). In a group of 1 100 atopic children, positive skin tests to mites were common in asthmatic children of all races, regardless of country of origin (Smith, 1973).

Mites are ubiquitous in the human environment. It is in the home that the highest concentrations are found where they live off human dander (Blythe, 1976). The highest densities of these mites are found on the surfaces of mattresses. Mite counts from mattresses are many times higher than elsewhere in the home (Maunsell, Wraith & Cunnington, 1968). High densities are also found on soft furnishings (Blythe, 1976). Mattresses are clearly important sources of infestation. Hospital beds enclosed in plastic covers have shown a low density of mites (Blythe et al, 1975). The plastic covers possibly hinder infestation by preventing human dander from reaching the surface of the mattress.

More than half of the children from the Tsolo district slept on mats on the floor. Most children from Guguletu and the 22 children with asthma slept on mattresses. It is noteworthy that the single asthmatic child from Tsolo district also slept on a mattress.

Mats are made of grass stems and/or reeds which are interwoven. It is possible that mats, like plastic covered mattresses, may hinder their infestation by mites. It may be possible that in this way the child living in the rural community is not exposed to the high density of mite populations in their bedding, as is the child from the urban community who sleeps on a mattress.

Mites have been found in homes in Umtata (Ordman, 1971). Tsolo district is 51 km from Umtata by road and 35 km as the crow flies in a north by north-west direction. The 2 areas both have a summer rainfall, while Umtata, at 668M, is 352m lower than Tsolo district in altitude. Although no mite counts have been done from any of the homes from Tsolo district, there is no reason to believe that climatic factors will be unfavourable towards the mite growth there.

Anderson and Cunnington (1974) did mite counts from homes in the Highlands of Papua, New Guinea and found the occurrence of mites similar to that in most European countries. The highest counts, however, were found in homes of "Europeans". It was suggested that high mite counts from "European homes" may reflect the influence of different environmental and social factors, such as the quality and amount of bedding and furnishings and the different habits, practices and styles of living.

In a study of 100 asthmatic Zambians in Ndola, a significantly greater proportion of dust samples from the homes of asthmatics contained the mite, D.pteronyssinus, compared with the homes of the controls (Buchanan & Jones, 1974). The mean mite count from the homes of asthmatic subjects was 176 per gram of dust compared to 72 per gram of dust in the controls. The difference could perhaps be due partly to the more diligent collection of dust by the asthmatics, but it focused attention on the fact that both environmental and constitutional factors are implicated in the causation of asthma.

These exciting observations imply that the home environment of the asthmatic may favour the growth of mites and that this may be reflected in the way of life eg. sleeping on mats instead of mattresses. An individual's

immediate home environment can be profoundly influenced by his social class, income, culture, race, religion and whether he lives in an urban westernised community or a rural undeveloped one. This applies not only to the present adult generation, but may also determine the home environment of their children in the future. By and large, people marry within certain social, racial, ethnic, religious and geographic boundaries (Berger & Berger, 1972), so that a non-fatal, inherited disorder such as asthma may perpetuate itself in a specific group, if present in the parents. The finding that the highest case rate for boys with asthmatic symptoms occurred in social class III to IV of Protestant families in the State of Maryland, United States of America (Nathanson & Rhyne, 1970) serves to illustrate this point, for which no other satisfactory explanation can be given.

Only a few studies have been undertaken to evaluate the home environment and the way of life in epidemiological surveys of asthma. In a study of bronchial asthma in the Nigerian Savanna region, Warrell et al (1975) noted that most of the 106 asthmatic patients lived in mud houses with zinc roofs and cement floors and slept on a bed with mattress and pillow. Only 20% lived in traditional thatched huts and only 14% slept on a mat on the floor. The lifestyle of non-asthmatics was not compared with that of the asthmatics. It was suggested that the healthy controls were protected from atopic disease by their high IgE levels, which were attributed to chronic helminthic infestation. Godfrey (1975) found the rarity of asthma in rural Gambia compatible with the hypothesis that high IgE levels, presumably with specificity for various parasites, helped to prevent the development of atopic illness. He, however, agreed that the findings might have been coincidental and that other factors such as local ethnic differences, which were present but not determined, were important.

He felt their importance was difficult to assess.

The importance of a change in environment on the prevalence of asthma in children was clearly demonstrated by Smith, Harding & Cumming (1971), Smith (1973) and Smith (1976). School children from the city of Birmingham were studied to establish the prevalence of asthma and any changes in the prevalence. During 1968-69 it was noted that Negro children born in England had a higher prevalence rate of asthma than the white indigenous population (5,5% to 2,3%) but Negro children born abroad had a very low prevalence (0,4%) (Smith, Harding & Cumming, 1971). In a study of 1 100 children attending special clinics for atopic diseases in Birmingham, Smith (1973) found that such disorders in immigrant children from tropical countries (ie. born in Asia, Africa and West Indies) were rare. Only 2 out of 1 040 children had asthma, one had eczema and no cases of hayfever were documented. In a follow-up study undertaken during 1974-75 (Smith, 1976) the findings were similar to the previous studies. Asthma was once again more common in Negro children born in England than in either their European or Asian counterparts. It was also much more common in those born in England than in Negro children born abroad. Cases of hayfever were again not found in immigrant children arriving from overseas. The findings of the later study (1974-1975) were, however, less marked than those of the earlier (1968-1969) one. The author suggested the possibilities of changing conditions in the country of origin or that the immigrants to the United Kingdom came from an urban rather than a rural population.

It is obvious that the difference in prevalence of asthma in these groups is not racial. The Negro child arriving in England from abroad has the same potential, if not a greater one, to develop asthma. What was it in

the environment or way of life in his home country that prevented the development of asthma ? Smith (1973) suggested the possibility that the total allergenic bombardment to which children are exposed in England is greater than that in tropical countries.

Can this difference in total allergen bombardment be due to a difference in lifestyle based on social class, cultural background and religion ? Childhood asthma has been found to be exceedingly rare in rural communities from Africa and less industrialised areas. There again, the subjects that did develop asthma all came from an urban community and were exposed to "the European way of life" (Woolcock, 1972).

It would seem as if something protects the child born in a rural community from developing asthma. In spite of exposure to abundant allergens, excluding perhaps mites, asthma was uncommon among children from Tsolo district. The total allergenic background may well relate to factors like sleeping on mats. There must, however, be one or a number of other known or unknown factors associated with the different ways of life in different communities which are responsible for this.

CHAPTER 5 : DIETARY PATTERN DURING INFANCY (Tables IV.36-38, Figs.12-20)

In 1936 Grulee and Sanfords made the observation that the general incidence of infantile eczema was considerably lower in breastfed as compared with artificially fed infants. Since then there has been a continued interest in the interaction between feeding practices, ie. breast or artificial feeding during early infancy and allergic disorders. It has been both claimed and refuted that breastfeeding during early infancy protects against development of allergic disorders.

The practice of prolonged breastfeeding was almost universal among mothers from Tsolo district. Of the 671 children, only 19 had never been breastfed. Of the rest, 63,7% were breastfed for more than 12 months, 50% for 20,3 months and 49,6% till 24 months. There was a sharp decline to 7,5% at 25 months and it would seem that weaning takes place at about this time. In spite of the prolonged breastfeeding, cow's milk was introduced at a median age of 2,1 months. Solids were introduced at a median age of 4,1 months. Although the mothers from Tsolo district breastfed for a considerable period - almost 24 months - cow's milk and solids were introduced at an early age in infancy.

The tendency to prolonged breastfeeding is a characteristic of rural underdeveloped societies. The majority of mothers in a Nigerian village were shown to breastfeed for 23,2 months (Martin, Morley & Woodland, 1964).

The pattern of early infant feeding for the non-asthmatic children from Guguletu was in striking contrast. Cow's milk was given from birth in 175 (27,7%) of the infants, none of whom received any breast milk.

Of those who were initially breastfed, only 21,6% were still on the breast after 12 months. In this group there were 2 periods at which weaning took place, one at 12 months and one at 24 months. Only 36 infants (7,8%) were breastfed up to 24 months. The median period of breastfeeding of 7,7 months was considerably less than that for Tsolo district. Cow's milk was introduced at 1,4 months in 50% of children with solids later at 3,9 months.

In the asthmatic children the median period was even shorter at 4,7 months. The introduction of cow's milk at 1,5 months was similar to the other 2 groups but solids were introduced at an earlier age.

A Kruskal-Wallis one-way analysis of variance test was used to test for differences in the feeding patterns during infancy. The test was performed on (1) the total samples ie. including children that were never breastfed and (11) only children who were breastfed. The differences in the duration of breastfeeding; age of introduction of cow's milk and solids were highly significant ($p = 0,0000$) for all 3 groups ie. asthmatic and non-asthmatic children from Guguletu and children from Tsolo district.

Asthma rarely occurs in children from rural societies in Africa and other undeveloped areas. This is despite exposure to potent allergens in their environment ie. the mite D.pteronyssinus and D.farinae. It seems as if something protects the child born in these countries. Yet if born outside these communities, the Negro child in England, for example, had a greater tendency to develop asthma in response to the same potent allergen found in the rural community. It appears to be not only the environment or exposure to allergens which leads to the development of clinically overt asthma. There may also be an additional factor which protects the child

from developing asthma during childhood but which leaves him unprotected during adult life.

Breast milk has been suggested and indirectly implicated as such a factor. Glaser & Johnstone (1953) studied the prophylaxis of allergic disease in newborns who had a strong family history of allergy and were regarded as potentially allergic. Cow's milk was withheld from 96 of these infants. The majority of these infants were fed on a soybean preparation. Cow's milk was introduced at various ages and these children were assessed at between 7 months and 5 years, and again 10 years later. A retrospective control of siblings and unrelated subjects from allergic families were also assessed. It was found that only 15% of the children from whom cow's milk had been withheld developed allergic diseases, compared with 65% of sibling controls and 52% of unrelated controls who had been fed cow's milk. In a similar but prospective study by Johnstone & Dutton (1966), 235 children chosen from families with strong allergic histories were followed for 10 years. Allergy occurred in 18% of the group fed on a soybean preparation and in 50% of the control group fed on cow's milk. The number of cases who developed asthma before the age of 3 years was significantly less in the group fed on soybean than in the control group. This difference was, however, not significant between 3 years and 10 years of age. After 10 years there was a significantly less number of asthmatic cases in the group fed on soybean preparation compared to the group fed on cow's milk.

These results could not be substantiated by other workers. Brown et al (1969) studied 427 children for 12 to 27 months, in whom soybean preparations and cow's milk were used regardless of family history of allergy. No difference in the number of cases with allergy was found in the two groups. They concluded that feeding with soybean preparations did not

affect the prevalence of allergy.

To observe the development of allergy, Halpern et al (1973) followed 1 751 infants for periods ranging up to 7 years. The infants were fed breast milk, a soybean preparation or cow's milk according to the choice of the mother. No differences were noted in the 3 dietary groups. It was concluded that feeding with soybean or breast milk supplemented with soybean preparations instead of cow's milk during the first 6 months of life did not reduce the incidence of childhood allergy. However, an important observation was that allergic disorders appeared at a significantly later age in the breastfed group compared to the group fed on cow's milk. Asthma was also less frequent in the former group at 36% compared to 48%. This difference, however, was not statistically significant. Perhaps a period of breastfeeding of 6 months is not adequate to prevent or postpone the onset of asthma.

Although the findings of studies on the avoidance of cow's milk in potentially allergic infants are contradictory, the clinical impression is that prolonged breastfeeding may postpone the onset of asthma. This observation was substantiated by Halpern et al (1973) in a prospective study. Özakarogöz and Çakin (1969) studied the prevalence of asthma in children from Anakara, Turkey, where they found an incidence of 10,6%. They attributed the increase in the incidence of atopic disorders to the possibility of a decrease in breastfeeding. Pearson (1973) attributed the low prevalence of eczema in Barbados to a delay in weaning from breast milk or alternatively to climatic influences. In a study of childhood asthma in Finland, Koivikko (1974) found that prolonged breastfeeding had the effect of postponing the onset of respiratory allergy.

The delay in commencing feeding foreign proteins (cow's milk, egg, fish, oranges, cereals and honey) had a similar effect on asthma. He commented on the continuous decline in breastfeeding and felt that it might be an important co-factor in the increasing incidence of childhood asthma. None of his deductions were based on factual evidence.

Glaser & Johnstone (1953) proposed the possibility of a physiological immaturity of the immune system during the early months of life which results in the absorption of unaltered proteins from the intestinal tract of potentially allergic children and causes sensitisation and clinical symptoms.

Taylor et al (1973) studied children of "reaginic parents" and found a transient IgA deficiency at the age of 3 months in those who developed atopy in the first year of life. They speculated that a transient IgA deficiency during early infancy results in excessive entry of allergens leading to excessive IgE stimulation and hence atopic disease. Once the reaginic system has been primed during the deficient period, subsequent administration of only very small amounts of allergen are required to continue the process. Soothill et al (1976) and Matthew et al (1977) found that sensitisation in the newborn period was important in the development of reaginic allergy in infancy and that allergen-avoidance resulted in less eczema at 6 months and 12 months than in a control group. The hypothesis of transient IgA deficiency may underly atopy in early life, but cannot explain the onset of it in later life.

This later onset of disease may be explained in terms of prolonged breast-feeding. This is supported by the observations of Koivikko (1974) and

Özkarogöz and Çakin (1969). In Africa asthma is rare in childhood but common in adults. What is the reason for this ? Prolonged breastfeeding is common in rural communities of Africa. It has been proposed that prolonged breastfeeding results in a low prevalence of childhood asthma and a delay in the onset of asthma and eczema (Koivikko, 1974; Pearson, 1973; Soothill et al, 1976; Matthew et al, 1977; Özkarogöz & Çakin, 1969).

It is well documented that breast milk and especially colostrum is rich in IgA (Ammann & Stiehm, 1966). Not only IgA but also colostral cells have been found in breast milk. These cells are primarily phagocytic macrophages with an abundance of lysosomes and a significant population of lymphocytes which respond to specific allergens (Smith & Goldman, 1968). They also synthesise IgA and β_1C (Murillo & Goldman, 1970). Immunoglobulin A similar to that in breast milk could be readily detected in the stools of breastfed infants. The IgA in breast milk proved to be resistant to the low pH of gastric secretions (Kenny, Boesman & Michaels, 1967).

Breast feeding even for one month can provide protection against respiratory syncytial virus infection (Downham et al, 1976). Inhaled IgA may play a significant role, since infants inhale milk during feeding and regurgitate feed through the nose. The IgA may persist in the respiratory tract for some time, helping not only to prevent infection with respiratory syncytial virus, but also to delay age of onset and decrease severity when it does occur.

The broad implications of these findings may have considerable impact. It has been suggested that breast milk protects the newborn infant against septicaemia (Winberg & Wessner, 1971) and the respiratory syncytial virus

infections (Downham et al, 1976). Is it possible that the IgA secreted in breast milk may protect the potentially allergic infant during the period of transient IgA deficiency ? Prolonged breastfeeding may extend the period of protection with a resultant delay in the onset of allergic diseases. The prolonged breastfeeding, lasting for 18 to 24 months in the rural communities of Africa and other tropical countries, could account for the late onset of asthma in adult life. Hayfever and eczema are almost unknown in these countries (Smith, 1973; Smith, 1976; Warrell et al, 1975). Could prolonged breastfeeding completely abolish the onset of these allergic disorders ?

The possibility of a factor in breast milk capable of postponing or preventing allergic disease in the potentially allergic individual, whether it be IgA or the colostral cells, does not seem to be too far-fetched. The isolation of such a factor could lead to the prevention of allergic disorders in genetically predisposed individuals.

CHAPTER 6 : DIAGNOSTIC AND LABORATORY PROCEDURES

In addition to the clinical examination, a number of diagnostic and laboratory procedures are available for the assessment of the asthmatic child. No single procedure can provide a diagnosis of allergy (Aas, 1975; Sheldon, Lovell & Mathews, 1967). Each has its own limitations and should be evaluated in such a light.

The information of the diagnostic and laboratory procedures in this study will be used to assess:

1. the children identified as asthmatics
2. the asthmatic children in the light of the information obtained from the total samples. The Guguletu sample will represent all non-asthmatic children. The Tsolo district sample includes the one asthmatic child.

The findings of these procedures can be grouped under 2 headings:

(1) Allergy-orientated and (11) General investigations

1. Allergy-orientated Investigations

These procedures are aimed at establishing the response of the allergic child to allergens in the environment.

(a) Prick Skin Testing (Tables IV.48 - 50)

The prick skin test is the most sensitive of the cutaneous tests (Indrajana, Spieksma & Voorhorst, 1971) and is widely used (Vaughan & Black, 1948;

Sheldon, Lovell & Mathews, 1967). It provides a high degree of immunological specificity, diagnostic reliability and safety if done correctly. It remains the single most sensitive test for specific allergic homocytotrophic antibody, IgE, in the skin (Aas, 1975; Yunginger & Gleich, 1975).

Good correlation exists between history, positive skin test reactions to specific inhalent allergens and inhalation challenge tests (Aas, 1975). Care must be exercised in the selection of allergens used for testing. It is important to include those allergens found in the individuals' immediate environment. Erroneous conclusion may be reached if the common occurring allergens are omitted from the skin testing procedure. In a review of skin test results, Voorhorst & Van Krieken (1973b) stated that, because of the variability of the results, skin test reactions might not reflect the real degree of sensitivity of an individual. Likewise, a negative skin test to a particular allergen does not rule out possible sensitivity to that allergen. Hale (1969) presented 3 cases showing dissociation between skin testing and clinical sensitivity and proposed that skin-sensitising antibodies can attach selectively to skin or mucous membranes or both. The plasma cells which form IgE were not found in skin biopsies, which might explain why the intensity of the skin reactions might not reflect the sensitivity (Tada & Ishizaka, 1970).

A skin test merely indicates whether or not the reagin tested for is present in the skin. It does not necessarily distinguish which allergen is of importance for the respiratory tissues (Aas, 1975). Skin tests are a bioassay valid for specific purposes. They cannot replace a thorough history and physical examination, nor can their results supplant physician judgement (Shapiro et al, 1977).

The 1 362 children in whom skin testing was done represented a random sample from the normal childhood population of the 2 areas. These included children with other allergic disorders or with a family history of allergic disorders. Only asthmatic children were excluded. The percentage of children having a positive skin reaction (19,4%) from Tsolo district, was similar to that found by other workers, but the 7,6% for children from Guguletu is lower. In a group of 303 randomly selected school children aged 8 to 14 years, 20,2% of the asymptomatic children had positive skin tests (Godfrey & Griffiths, 1976) while out of 3 101 subjects studied as part of a total community survey, 34% had positive skin reactions (Barbee et al, 1976). Positive skin reactions were found in a considerable number of people with only a family history of allergy (Curran & Goldman, 1961). Whitcomb (1971) found that amongst 50 randomly selected medical students, a history of urticaria, a family history of allergy and a past history of allergy but without clinical symptoms for more than a year were all associated with positive skin tests. He concluded that a positive skin test alone did not necessarily reflect active allergy. If cases are selected on the basis of being non-allergic, and having no family history of allergy, however, only 5% will have a positive scratch test (Curran & Goldman, 1961).

There are certain noteworthy differences in the findings of the skin testing in the 2 samples. A greater number of children from Tsolo district had positive reactions; 130 (19,4%), compared with 51 (7,6%) from Guguletu. Although different observers read the reactions in the 2 samples, the same technique for performing and reading was employed. The differences are, therefore, true for the 2 groups and statistically significant.

The skin reactions were difficult to interpret in the children studied. Flares could not be detected and the wheal sizes were difficult to assess accurately in the black skins of the children. This possibly accounts for the very low number of positive skin reactions among the asthmatic children. There is, however, another possible explanation for the finding of only 1 positive skin reaction among the 22 asthmatics and only 85 positive reactions among the remaining 669 children from Guguletu.

Intestinal parasitic infestation was exceedingly common among children from Guguletu (Tables IV.62 and 63). Parasitic eggs were found in the stool of every asthmatic child from whom a stool was collected and eggs of A.lumbricoides were present in the stools of 19 of the 22 asthmatic children.

Parasites are potent stimulators of serum IgE production (Ogilvie & Jones, 1973). Johansson, Mellbin & Vahlquist (1968) found levels of serum IgE 16 to 20 times higher in Ethiopian children than Swedish children, which was attributed to parasitic infestation.

An important element of the definition of type I allergic tissue reaction ie. hayfever and allergic asthma, is the release of vasoactive amines from mast cells (Coombs & Gell, 1975). This reaction is IgE dependent. Each mast cell has 30,000 - 100,000 IgE receptor sites on its surface capable of binding IgE molecules (Ishizaka, Soto & Ishizaka, 1973). It has been suggested that the development of atopic disease may be prevented by certain parasitic infestations (Preston, 1970; Editorial, Lancet, 1976). This hypothesis is based on the fact that the IgE receptor sites of mast cells become saturated by parasitic induced IgE, which would then prevent

allergen-specific IgE molecule attachment to the mast cell. The subsequent release of vasoactive amines as triggered by the interaction of allergen and its specific homocytrophic IgE would not then take place.

Evidence in support of the hypothesis of mast cell IgE receptor site saturation is based on in-vitro and in-vivo studies. Godfrey & Gradidge (1976) found that if human lung fragments were incubated with IgE rich serum from West African subjects, further passive sensitisation of the mast cells was blocked. If the lung fragments were first passively sensitised, the IgE rich serum failed to block histamine release following allergenic challenge. It would seem that the time exposure to the IgE rich serum is critical.

In-vivo studies, based on passive cutaneous anaphylaxis (P.C.A.), (Prausnitz-Küstner phenomenon) have shown that passive sensitisation cannot be induced in patients with IgE myeloma (Ogawa et al, 1971) and that when myeloma IgE is injected into the skin of normal subjects, P.C.A. to specific reaginic IgE is blocked (Stanworth et al, 1967). Bazaral, Orgel & Hamburger (1973) found that in individuals with parasitic infestation and high levels of circulating IgE, P.C.A. reactions were difficult to elicit.

Saturation by IgE of mast cells in the skin produced by exposure to helminthic infestation in the asthmatic children from Guguletu may have taken place. This may have prevented allergen-specific IgE from attaching to the mast cells and thus resulting in a negative response to prick skin testing. The most likely explanation for the difference in prick skin test results in the 2 areas probably relates to the helminthic infestation.

Helminthic infestation, especially A.lumbricoides, which is a potent stimulator of IgE production,was much more common among the children from Guguletu than among the country children. Mast cell saturation with IgE directed against the helminths may have caused the higher number of negative skin reactions. In the selection of the allergens used in the skin testing, care was taken to include those allergens which are associated with asthmatic symptoms. It is unlikely that the few positive reactions in Guguletu children- asthmatic as well as non-asthmatic - were a result of too few or inappropriately selected allergens.

The one asthmatic child from Guguletu (No. 62001) who had a positive reaction to D.farinae, also had A.lumbricoides and T.trichiura eggs in his stool. His serum IgE level was elevated to 1 371 u/ml. The positive reaction in this child may reflect a high portion of his total serum IgE to be specific for D.farinae.

The pattern of sensitivity judged by skin reactions, also differed in the 2 samples. By and large, the types of positive reactions tended to follow the allergenic exposure experienced by the children from the different areas. Exposure to maize, pollen or food, was of a high order in Tsolo district but less so for Guguletu and it is therefore not surprising that of the positive reactions, 36,1% were against one of these allergens for children from Tsolo district compared to only 22,3% from Guguletu. It was also the most common positive reaction for children from Tsolo district.

Sensitivity to house dust mite related group of allergens was unexpected. No difference in the number of positive reactions to house dust and

D.pteronyssinus were found but significantly more children from Guguletu had a positive reaction to D.farinae. A positive skin reaction to D.farinae was almost twice as common as D.pteronyssinus. The reason for this is not known. Ordman (1971) found D.pteronyssinus to be the predominant mite in South Africa. It is also the commonest one found in homes in the United Kingdom with 94% of housedust sensitive patients having a positive reaction on skin testing (Maunsell, Wraith & Cunnington, 1968). It is, however, of importance to note that positive reactions to the house dust mite related allergens accounted for more than half of the positive skin reactions (40,0%) for Guguletu and only 13,1% of children from Tsolo District (Table V.1, p.279).

Positive skin reactions to the combined mould groups of allergens (16,9%) followed closely on that of the housedust related allergens for children from Tsolo district. The mould species E.purpurascens and C.herbarum resulted in the most frequent positive reactions. The comparatively high number of children from Tsolo district with a positive reaction to moulds may relate to the floors and roofs of the houses. The floors are of cowdung and the roofs of thatch. This may favour growth of moulds. It is noteworthy that the single asthmatic child from Tsolo district only had a positive skin reaction to C.herbarum.

A positive reaction to feathers was more common among Tsolo district children. Chickens often come into the houses and even nest there. This would inevitably result in a high exposure to feathers compared to Guguletu where only 9,4% of children had chickens at home.

Positive reactions to grass were almost equal in the 2 samples. This was expected as grass is common in both areas.

Comparison between differences and their significance in terms of biological importance is obscured by the overwhelming helminthic intestinal infestation among the children from Guguletu. The findings from Tsolo district are probably more representative of the actual skin sensitivity. If the results are expressed as a percentage of the number of positive reactions, a more representative picture is perhaps seen.

T A B L E V.1

POSITIVE REACTIONS OF PRICK SKIN TESTING TO INDIVIDUAL ALLERGENS EXPRESSED AS A PERCENTAGE OF THE TOTAL NUMBER OF POSITIVE REACTIONS

ALLERGEN	NON-ASTHMA GUGULETU	TSOLO DISTRICT
Maize pollen	14,9 %	23,0 %
Maize food	8,2	13,1
Feathers	1,2	9,8
Housedust	15,3	7,1
D.pteronyssinus	15,3	7,1
D.farinae	24,7	6,0
E.purpurascens	1,2	6,6
A.tenius	4,7	1,6
C.herbarum	2,4	8,7
Grass	5,9	8,2
Dog Epithelium	3,5	4,4
Cat Epithelium	2,4	2,7
Milk	1,2	1,6

The actual number of positive reactions to mites do not support the hypothesis that the environment is less favourable to their growth in rural Tsolo district. If the positive reactions are, however, expressed

as a percentage of the total positive reactions, the difference between the 2 areas becomesmore apparent with a smaller percentage of positive reaction resulting from these allergens in children from Tsolo district.

(b) Total Serum Immunoglobulin E (IgE)

Since the discovery of serum IgE (Ishizaka, Ishizaka & Hornbrook, 1966) the association of an elevated serum IgE with allergic disease has been well documented (Johansson, 1967; Gleich, Averbeck & Swedlung, 1971; Hogarth-Scott et al, 1971; Havnen et al, 1973; Halpern, 1974; Saha et al, 1975; Yunginger & Gleich, 1975; Kjellman, 1976).

Initially it was thought that the serum IgE level could be used to distinguish the allergic from the non-allergic individual. In a study of serum IgE concentrations in children suffering from allergic asthma, Havnen et al (1973) concluded that serum IgE was a valuable diagnostic tool which could distinguish between allergic and non-allergic asthma in selected cases when used as a supplement to a thorough investigation of allergy. They found, however, a raised serum IgE in 1 out of 10 children without any evidence of sensitivity.

It is not generally accepted that serum IgE is a useful means of distinguishing between allergic and non-allergic disorders. In a study of the sera of asthmatic children, Hogarth-Scott et al (1971) found that the mean serum IgE level corresponded with the severity of the disease. A significant increase in the mean serum IgE level with the more severe grades of asthma was found in childhood. On the other hand, they found severe asthma cases with normal IgE levels, as well as normal controls

and patients with mild asthma who had very high IgE levels in their sera. In an attempt to correlate serum IgE levels with skin test sensitivity, Loeffler, Cawley & Moeder (1973) found a 'grey' zone for IgE values which included both allergic and non-allergic individuals. The mean serum IgE in skin test negative patients was 461 u/ml. Levels below 400 u/ml were strongly against, whereas levels above 800 u/ml were strongly suggestive of allergy. Levels between 400 u/ml and 800 u/ml represented an indecisive 'grey' zone. It was concluded that serum IgE did not provide a sharp dividing line between allergy and non-allergy. Correlation between total IgE levels and age in allergic patients, with age of onset, duration, severity or frequency of allergic symptoms could not be found (Stenius, Wide & Seymore, 1972).

The serum IgE level can only be interpreted in the light of a history and skin testing. On its own, it cannot be used to distinguish an allergic from a non-allergic individual.

The interpretation of the serum IgE levels, from the children studied in this study, is further complicated. Intestinal helminthic parasitic infestation was present in almost every child from Guguletu from whom a stool specimen was examined (Table IV.62). Only 3,0% had no evidence of parasitic eggs in their stools. A.lumbricoides eggs were found in 71,0% of the stools with parasites. Ethiopian children with proven A.lumbricoides infestation had serum IgE levels 28 times higher than those of Swedish children (Johansson, Mellbin & Vahlquist, 1968). In contrast only 38 (9,8%) of the stools examined from the children in Tsolo district had evidence of helminthic intestinal infestation (Table IV.62, p.211). The type of ova found in the stools of these

children also differed from those from Guguletu. Hymenolepis nana was the commonest helminth found, while A.lumbricoides was present in only 26,3% of stools examined, whereas T.trichiura was the commonest helminth in the stools of children from Guguletu.

The serum IgE levels of the 3 samples of children follow the same trend reported by other workers where studies were done among subjects living in an endemic parasitic area (Table IV.51, p.194, Figure 25, p.195). Values ranged from medians of 2 160 u/ml, 3 240 u/ml and 3 730 u/ml (Turner, Baldo & Anderson, 1975) to means of 1 613 u/ml (Merrett, Merrett & Cookson, 1976) and 1 365 u/ml (Warrell et al, 1975). Studies on Black adults or African children have all shown that the serum IgE levels are invariably raised (Johansson, Mellbin & Vahlquist, 1968, Ethiopia; Orren, 1974 & Orren & Dowdle, 1975b, Cape Town, Republic of South Africa; Godfrey, 1975, The Gambia; Warrell et al, 1975, Nigeria Savannah Region; Merrett, Merrett & Cookson, 1976, Rhodesia).

A striking observation in this study is the very wide range of the serum IgE. The 95% tolerance interval of 131 to 17 786 for boys aged 7 years from Guguletu is especially remarkable. These findings support the view that serum IgE levels have the widest range of all immunoglobulins (Kjellman, 1976). This wide range was evident for both the asthmatic as well as the non-asthmatic children from both areas with and without evidence of parasitic infestation. The serum IgE levels of the individual asthmatic children ranged from 109 to 9 275 u/ml (Figure 3, p.125). The level was greater than 1 000 u/ml in 14 (63,6%) children. In spite of proven parasitic helminthic infestation, 6 (27,3%) asthmatic children had serum IgE levels less than 400 u/ml.

Of the total sample of children from Guguletu (asthma and non-asthma) 372 had evidence of A.lumbricoides. Of these, only 34,5% (128) had a serum IgE level greater than 1 944 u/ml. It is noteworthy that 25,1% (93) had a serum IgE level less than 389 u/ml and even levels below 50 u/ml.

A three-way analysis of variance of the serum IgE levels showed significant differences between the serum IgE levels for sex, age and place for the 2 main population groups ie. non-asthmatics from Guguletu and Tsolo district (Fig. 25, p.195). Males from Guguletu had the highest overall values that decreased slightly with age. Males and females from Tsolo district had very similar mean IgE levels following the same decrease in levels with age, but much lower than for males from Guguletu. The females from Guguletu had a much lower mean level than the males, but did not show this decrease in levels with age. Other workers found no sex difference in serum IgE concentrations in non-allergic subjects (Orren & Dowdle, 1975b; Orren, Walls & Dowdle, 1975; Gleich, Averbeck & Swedlund, 1971).

The difference in serum IgE levels for males and females from Guguletu can perhaps best be explained on the basis of helminthic infestation. Boys often tend to play with toy motor cars in sand, which could result in a heavier helminthic infestation. Girls tend to play with dolls and not so much in the sand. The much lower levels for males and females from Tsolo district probably relates to the fact that helminthic infestation was less prevalent among them.

Serum IgE levels increase with age to reach adult levels at 7 years (Kjellman, 1976). The overwhelming presence of helminthic infestation in the children in this study, however, makes the interpretation of the effect

of age on the serum IgE concentrations impossible.

The limitations of a single serum IgE estimation are obvious. It cannot be used to classify an individual as allergic or non-allergic because of an indecisive 'grey' zone and because of the finding of low IgE levels in allergic subjects and vice versa. Its diagnostic value in tropical countries, where levels in healthy individuals are already abnormally high, has been questioned (Tay et al, 1975). The potent stimulative effect of parasitic infestation on serum IgE production makes it virtually worthless in communities with a high prevelance of helminthic intestinal infestation. Halpern's suggestion (1974) that a serum IgE value of 1000 u/ml is suggestive and one over 2 000 u/ml is almost definitely indicative of parasitic infestation is useful. It is, however, important to remember that a considerable number of children with proven parasitic infestation may have values less than 1 000 u/ml. Of the children from Guguletu 22,4% had serum IgE levels between 389 and 870 u/ml into which the serum IgE value of many allergic patients would fall.

The conclusion reached from the determinations of serum IgE in these 3 groups is limited. These values cannot be analysed in any way to establish normal references for the populations. Although only 9,8% of the children from Tsolo district from whom stools were examined had evidence of intestinal helminthic infestation, it cannot be assumed that the rest were normal. A stool found to be normal by a single examination does not rule out infestation. Also no prediction can be made as to which child was parasite-free prior to the study. In such children, the serum IgE level may be considerably elevated in spite of being free of parasites at the time the study was conducted. There is evidence that the serum IgE level does not return to normal for some time after avoidance

of allergens (Lund & Dowdle, 1977).

(c) Allergen-Specific Serum IgE

A technique has been developed by which allergen-specific IgE in serum can be measured viz. the radioallergosorbent test (RAST) (Wide, Bennich & Johansson, 1967). Significantly high correlations between allergen-specific IgE measured by RAST, history, prick skin testing and provocation tests were found (Stenius et al, 1971; Stenius, Wide & Seymore, 1972; Aas & Lundkvist, 1973; Wide, 1973; Ahlstedt et al, 1974; Pepys, Roth & Carroll, 1975).

Foucard (1973) found that children with 'asthmoid bronchitis' who had strong positive prick skin reactions also had a positive RAST in 86% of cases. This perhaps indicates a higher sensitivity of the skin test or possibly that IgE fixes primarily to tissue mast cells. A certain tissue saturation is necessary before antibodies appear in the serum (Foucard, 1973).

The high correlation between RAST and skin test results (RAST: prick test 98%, positive prick test : RAST 89%, positive history : RAST 91% and positive history : prick test 92%) (Pepys, Roth & Carroll, 1975) and the fact that prick skin tests identify almost all subjects with specific IgE (Stenius et al, 1971) makes the RAST complementary to skin testing.

In view of the difficulty of interpreting results of the prick skin tests in the children studied, it was decided to measure allergen-specific IgE by RAST in selected children (Tables IV.58 to 60). It was also done to

try to establish the specificity of the raised serum IgE found in the non-allergic children which was presumed to be in response to the helminthic infestations.

Allergen-specific IgE was measured against 10 of the allergens for which skin testing was done. The RAST was done on sera of the 23 asthmatic children. It was also done on 48 and 47 non-asthmatic children from Guguletu and Tsolo district respectively. These children were selected as having the highest serum IgE levels of the 2 samples.

A number of differences are noted in the results among the 3 groups. The median serum IgE for the asthmatic group is much lower than for both the other groups. The median IgE of 9 595 u/ml for the Guguletu children is considerably higher than the 6 663 u/ml for the Tsolo district children. This probably relates to the difference in helminthic infestation in the 2 groups.

In only 23,3% (5 of 23) of the asthmatic children was it possible to detect allergen-specific IgE. In these, 62,5% of the positive results were against the mites, D.pteronyssinus and D.farinae. The asthmatic child, 62001, who had a positive prick skin test reaction to D.farinae also had a positive RAST to the same allergen. The reason for the low positive RAST among the asthmatic children is not clear.

Interesting findings arise from the results of the RAST done in the other 2 groups ie. those with the highest serum IgE levels. Several differences are prominent. More children from Tsolo district than Guguletu had one or more positive reaction (21 to 15 children respectively). The number of positive reactions in total were also higher for the Tsolo district children

(41 to 27 Tsolo district to Guguletu respectively.) More than one positive reaction per child was found in 46,7% of the children from Guguletu, whereas 85,7% of the children from Tsolo district had more than one positive reaction per child.

Allergen-specific IgE against grass and mites was common for Guguletu children. Of the positive reactions 13 (48,1%) and 7 (25,9%) were against grass and mites respectively. Only 2 children (4,3%) from Tsolo district had positive reactions to mites. More than 80% of the positive reactions were to either cat or dog epithelium. This would lend further support to the suggestion that mites are probably more common in the home of the child from a western urban society.

There was no correlation between the RAST findings and that of the skin test reactions. This is in direct contrast to the findings in allergic patients where a high correlation is found between findings of the skin test and RAST (Wide, 1973; Pepys, Roth & Carroll, 1975). It is probable that the total serum IgE in allergic patients is of a high allergen specificity to a few allergens. In fact, not a single child from the Guguletu group had a positive reaction on skin testing. This is in keeping with the overall findings of the skin test reactions which may relate to the helminthic infestation.

Orr, Riley & Doe (1971) showed that the reagin response to egg albumin and Bordetella pertussis vaccine in rats was potentiated by infection with the nematode Nippostrongylus brasiliensis. Crude worm extract did not show this potentiated response - it had to be an infection. Jarrett & Stewart (1972) induced reaginic antibodies to various allergens in rats. Subsequent infection with N.brasiliensis greatly potentiated the circu-

lating antibodies against these allergens, amongst others, house dust extract. The parasitic infestation did not cause de novo synthesis of reagin to unrelated antigen but was capable only of potentiating an already existing reagin response. They concluded that a helminthic factor could stimulate the antibody producing cells of any specificity previously programmed for IgE production. Selective potentiation of a reagin response (ie. IgE) could occur.

The suggested clinical implication was that in addition to an anti-helminthic IgE response, an elevation of circulating levels of one or more existing allergen-specific IgE took place in persons with a helminthic infestation.

This would seem to be the case for the children with the high serum IgE from the 2 areas. The RAST is a wualitative and only semi-quantitative measure of allergen-specific IgE. A considerable part of the total serum IgE in these children with helminthic infestation seems to be allergen-specific. Normal people probably can and do at times produce small amounts of IgE (Coombs & Gell, 1975). It is likely that these cells previously programmed for IgE production have been stimulated by the helminthic infestation that is measured as allergen-specific IgE in the 2 groups.

(d) Serum Immunoglobulins G, A and M (Tables IV.52 to 57)

Blacks tend to have higher serum immunoglobulins than Caucasians. Results from both child and adult populations showed an invariable elevation of serum IgG concentration, while the concentrations of IgA and IgM varied

from one African population to another, including South Africa (Turner & Voller, 1966; Johansson, Mellbin & Vahlquist, 1968; Rowe et al, 1968; Rosen, Geefhuysen & Ipp, 1971; Orren, 1974; Shulman & Gilich, 1976). The mean serum IgG levels for Ethiopian children was significantly higher than the mean 778 mg per 100 ml for Swedish children. The serum IgA and IgM levels were also higher, but not significantly more so than for Swedish children. The values for Ethiopian children for IgA, however, peaked between 44,8 and 89,6 mgm per 100 ml. The distribution for Swedish children was much flatter (Johansson, Mellbin & Vahlquist, 1968). Shulman & Gilich (1976) measured serum immunoglobulins A, G and M in 936 Black children seen at the Far East Rand Black Hospital, Transvaal. Measurements were done for 13 age ranges. The ranges for all three immunoglobulins in all age ranges were higher in the Black children compared to available standards for Caucasian children. Rosen, Geefhuysen & Ipp (1971) found similar results in Black children seen at Baragwanath Hospital, Transvaal. The levels of all 3 serum immunoglobulin classes (A, G and M) for the 2 samples in this study followed this trend and were considerably higher than those for Caucasian children (Stiehm & Fudenburg, 1966; Allansmith et al, 1968).

One child from Guguletu had a serum IgA which was undetectable while another 5 children had decreased levels of serum IgA (17, 24, 24, 33, 33 mg/ml). The incidence of total serum IgA deficiency for the Guguletu children is 1 in 693 or 0,14%. Decreased levels of serum IgM or IgG were not found apart from one child who had a serum IgM of 50 mg/ml. A decreased or absent serum IgA level was not found among the children from Tsolo district. The lowest serum IgM level was 84 mg per 100 ml. The incidence of an isolated serum IgA deficiency in the Guguletu childhood population is similar to that reported for Canadian school children (0,28%) (Collins-

Williams et al, 1972) and amongst hospital patients (0,2%) (Hobbs, 1968).

The serum immunoglobulins A, G and M of the asthmatic children did not differ from those of the 2 non-asthmatic populations. This is in keeping with findings from other studies (Huntley & Lyerly, 1963; Buckley, Dees & O'Fallon, 1968; Palma-Carlos & Palma-Carlos, 1971). Although an isolated serum IgA deficiency has been found to be more prevalent among allergic subjects (Kaufman & Hobbs, 1970; Saha et al, 1975), this was not the case for the 23 asthmatic children. This number is, however, too small to make comparison.

A three-way analysis of variance for age, sex and place showed significant differences for serum IgM but only for age and place for serum IgA and IgG in the 2 main groups (non-asthma Guguletu and Tsolo district)(Tables IV.53, IV.55 and IV.57).

There was an increase in levels of all 3 immunoglobulins with age. Serum IgA and IgM levels were higher for the Tsolo district than for Guguletu while the reverse was found for serum IgG. Girls had a higher serum IgM level than boys. The sex difference for serum IgM and the increase in levels of serum immunoglobulins with age have been previously reported (Allansmith et al, 1968; Stiehm & Fudenburg, 1966). The reason for the differences between the 2 populations is not apparent. Overcrowding and bigger families were more common in Guguletu. Intestinal helminthic parasitic infestation was far more common among children from Guguletu than those from Tsolo district. The higher serum immunoglobulin levels in Blacks have speculatively been ascribed to the intensity of antigenic stimulation by various bacterial, viral, protozoal and helminthic diseases. In spite of these being more prevalent in Guguletu children, their levels

of serum IgA and M were statistically significantly lower than for children from Tsolo district. The biological importance of these differences is not clear as the children from Guguletu had even higher mean levels of all three immunoglobulins than Black children reported by Shulman & Gilich (1976) from Johannesburg.

No attempt was made to exclude children with signs of infection from these 2 samples which represented the broad childhood populations in both areas. It is inconceivable that a large number of children were infected at the particular time the blood specimens were obtained. The question arises whether the differences in levels of serum immunoglobulin A, G and M between Blacks and Caucasians are not possibly racial or genetic in origin.

(e) Total Eosinophil Count (TEC) in Peripheral Blood (Table IV.61)

A raised total eosinophil count is often found in patients with allergic disorders (Lowell, 1967; Sheldon, Lovell & Mathews, 1967). Other non-allergic disorders such as polyarthritis nodosa and systemic lupus erythematosus are also associated with an elevated TEC (Lecks & Kravis, 1969). A single estimation of the TEC in the peripheral blood may on its own, therefore, point to a number of disorders.

The interpretation of the TEC in the children studied is also hampered by the fact that parasitic infestation gives rise to an elevation of the total eosinophils in peripheral blood. Blood eosinophilia is notoriously difficult to interpret in parasitic endemic areas (Warrell et al, 1975; Rees et al, 1974). Finding a TEC greater than 440 per µl in 64,0% of the children from Guguletu was not unexpected. It is, however, impossible

to judge the significance of the TEC in the 22 asthmatic children from Guguletu, as all of them from whom stools were examined (20 from 22) had evidence of parasitic infestation.

In contrast a much lower TEC was found for children from Tsolo district. Only 5,1% had an abnormally raised level. This would indicate that current infestation with parasites was probably at a low order as suggested by the stool examination.

The role of the eosinophil and the elevation of the TEC in the IgE-mediated allergic reactions is not clearly understood. Its function in parasitic infestations is believed to be one of a parasite killer cell (Gleich, 1977). However, when taken in conjunction with a history of allergy, a positive prick skin test reaction and a raised serum IgE without evidence of helminthic infestation, it is an added pointer to an allergic disorder. As indicated, its value in this study was limited.

2. General Investigations

These parameters do not relate to allergic disorders but are a means of assessing other disorders. They will therefore only be briefly discussed.

(a) Haemoglobin concentration (Hb) (Table IV.64)

The majority of children had a haemoglobin value within the normal range for age.

Seventy-two children from Guguletu had levels less than the lower limit of normal for age ie. 11,5 g/dl, and only 13 (1,9%) had a value of less than 10 g/dl. Infestation with T.trichiura was exceedingly common in

the children from Guguletu. It is feasible that the abnormally low haemoglobin levels were related to this particular infestation. T.trichiura was present in the vast majority of the stools of the 72 children whose Hb was less than 11,5 g/dl.

Only 5 children from Tsolo district had a haemoglobin value less than 11,5 g/dl. T.trichiura was found in the stools of one of these children. A three-way analysis of variance of haemoglobin values showed that statistically significant differences existed for age and place but not for sex. There was an increase in the haemoglobin level with age from 6 to 9 years. The children from Tsolo district also had a higher level than those from Guguletu.

The differences between Tsolo district and Guguletu can be explained by the higher altitude of Tsolo district and the greater degree of helminthic intestinal infestation, especially with T.trichiura in children from Guguletu. Guguletu is almost at sea level (30 m) whereas Tsolo district is at 1 020 m. The haemoglobin concentration rises 1 g/dl per 2 000 m ascent in altitudes (Dacie & Lewis, 1975).

(b) Nutritional Status (Tables IV.39 - 42; Figs.21 - 24 and 26 - 31)

Malnutrition has been shown to affect the immune system especially the cellular immunity (Smythe et al, 1971). The humoral immunity ie. immunoglobulin A, G and M levels, however, has been shown to be within normal ranges or even elevated in children with kwashiorkor (Keet & Thom, 1969; Rosen, Geefhuysen & Ipp, 1971).

The National Centre for Health and Statistics percentile charts (NCHS growth charts, 1976) for height for age, weight for age and weight for height were used to evaluate these measurements in the children studied. As the NCHS percentile charts have no 3rd percentile, it was calculated for $6\frac{1}{2}$, $7\frac{1}{2}$, $8\frac{1}{2}$ and $9\frac{1}{2}$ years from the actual values obtained from the NCHS tables.

Several differences existed between the 2 main groups regarding their nutritional status (Tables IV.39 to 46; Figures 21 to 24 and 26 to 31). Children from Tsolo district were shorter and lighter than their counterparts in age and sex. The girls aged 7, 8 and 9 years were significantly lighter than those from Guguletu, but there was no difference for height at any age. Boys from Tsolo district aged 6 years were lighter and those aged 6 and 8 years were shorter than those from Guguletu.

Their nutritional status was further assessed for malnutrition ie. weight for age less than 3rd percentile; stunting ie. height for age less than 3rd percentile; and wasting ie. weight for height less than the 3rd percentile.

The incidence of malnutrition at the various ages was statistically significantly different for boys and girls from Guguletu compared to those from Tsolo district. More children from the latter were malnourished. The differences for boys of all ages combined, however, were indeed very small (28,8%) for Guguletu and 29,4% for Tsolo district. The differences for girls of all ages were, however, considerably higher for Tsolo district.

Stunting was common for both groups of all ages and sexes. Girls from Guguletu were the least stunted (32,9%) and girls from Tsolo district (49,0%) the most. The differences in stunting for the 2 main samples

were not statistically different.

Wasting (low weight for height) was far less common than substandard weight and height for all ages. The majority of children, although malnourished and stunted for age, had normal body proportions. The interesting finding is the higher number of children, boys as well as girls, from Guguletu who were wasted. Only 6 of the total sample of children from Tsolo district were wasted. This indicates that the current nutritional status of the children is satisfactory in that their weights are proportionate for height. Having heights and weights below the 3rd percentile for ages indicate the stigmata of previous malnourishment currently not present.

The findings of serum albumin levels and skinfold thickness measurements to a lesser extent support this view. A serum albumin level less than 28 g/l is classified as deficient while a level of 35,2 g/l or less as low (I.C.N.N.D., 1960). Skinfold thickness reflects total body fat stores (Smith, 1977) whereas serum albumin levels reflect current protein status (Hansen, 1965).

Only 3,5% of children from Tsolo district and 0,5% of children from Guguletu had serum albumin levels less than 28 g/l. More children from Tsolo district (16,2%) had serum albumin levels less than 35,2 g/l compared to Guguletu children (2,1%).

Only 3,4% of the children from Guguletu (1,6% of the boys and 5,2% of the girls) had a triceps skinfold thickness less than the 3rd percentile for age (Tanner & Whitehouse, 1975) compared to 16,1% of the children from Tsolo district (14,6% of the boys and 17,5% of the girls). It should

be borne in mind that the standard of measurement used was that for British children. This is not necessarily the optimum standard.

It can be concluded that a considerable number of children from both areas had stigmata of malnutrition in infancy but their current state of nutrition is satisfactory. Although more children from Tsolo district had a serum albumin level classified as deficient, it cannot be regarded as an important factor for the different in prevalence rates of asthma. Judged by serum immunoglobulin levels, all children had a normal humoral immune response.

(c) Radiographs of the Chest and Clinical Examination (Table IV.47)

As these were in no way used to identify the asthmatic child, they will not be discussed. The singly performed clinical examination has severe limitations and without a supporting reliable history, no valid conclusions can be made in this study from these observations regarding the prevalence rate of asthma.

SECTION VI

SUMMARY AND CONCLUSIONS

A review of the world literature shows conflicting prevalence rates of asthma for various childhood populations. Reported prevalence ranges from 0,25% to 7,14%. The prevalence of childhood asthma in Black children in South Africa is not known. To establish the prevalence of childhood asthma in Black children, two samples of Xhosa children, aged 6 to 9 years, were studied. One group lived in an urban western society, ie. Guguletu, Cape Town. The other lived in a rural, traditional Xhosa society, ie. Tsolo district, Republic of Transkei. The samples were selected to represent the broad childhood population of the two areas.

The point prevalence of asthma for the two samples is strikingly different. The children from Guguletu had a prevalence rate of 3,17% (22 of 694) which is comparable with the prevalence rate found in the United Kingdom, Australia, New Zealand and United States of America. The prevalence rate of 0,14% (1 or 671) for children from Tsolo district is similar to that found in other rural areas of Africa, India, New Guinea and Barbados.

These findings are a significant contribution to the epidemiological study of asthma in this country. The need for establishing factual prevalence rates of asthma in children of all racial and cultural groups from other areas in this country is obvious. It is also equally important to establish the rates for the adult populations.

Only by having accurate prevalence rates for children and adults from rural and urban communities, can factors in the pathogenesis of the disorder be elucidated.

There is no universally accepted definition for asthma. This inability of definition has plagued epidemiological studies on the disorder. In many epidemiological studies the diagnosis of asthma has been based upon history obtained from the parents. Obtaining such history has its limitations, especially when dealing with a population where a language difference exists. The word "asthma" is foreign, and in rural communities, as in the Tsolo district, people live according to traditional ways. Exercise-induced asthma was used as the only criterion for identifying the asthmatic children from the selected samples. It was defined as a drop of 15% or more in post-exercise FEV_1 and PEFR from pre-exercise levels. The exercise stimulus used in every child was running on a level in the open for 6 minutes.

In this study, exercise-induced asthma was found to be a satisfactory way of identifying the asthmatic child. Any child, whether sophisticated or not, aged 6 years or more, should be able to perform these pulmonary function tests with ease. The children in the Tsolo district sample are unsophisticated, live in a rural community following traditional Xhosa ways and often tribal customs. A drop in both FEV_1 and PEFR was therefore used to eliminate the child where a change may have been due to technical reasons. The percentage drop in FEV_1 and PEFR were very similar for each individual asthmatic child. The mean percentage drop in FEV_1 and PEFR for the asthmatic children was 32,7% and 32,2% respectively. The normal non-asthmatic children in both groups showed virtually no change in post-exercise from pre-exercise values.

Exercise-induced asthma is proposed as an easily performed and a reliable method of identifying the asthmatic child in a large epidemiological survey. The FEV_1 and PEFR are easy to measure and do not require sophisticated equipment in field work. The results in this study showed that either FEV_1 or PEFR can be used to determine exercise-induced asthma in a large population survey. Recording only the PEFR may be preferable to recording only the FEV_1 as this would result in fewer false positive recordings. When dealing with an unsophisticated sample of children, however, the use of both FEV_1 and PEFR is suggested until future studies confirm the most reliable measurement.

In addition to identifying the asthmatic children, factors which may contribute to, or influence the prevalence rate of asthma, have been evaluated. These were the socio-economic status of each family, exposure to environmental allergens in each individual child, feeding patterns, both current and during infancy, and the sleeping habits.

Both samples came mainly from the lower socio-economic classes, where overcrowding was common. The levels of formal education and occupations of the head of the household were associated with those of the lower social classes. The families from Guguletu with an asthmatic child were in no way different from the families with non-asthmatic children from Guguletu as far as crowding, number of children per bedroom or head of the household and his/her occupation are concerned. There was no difference in the average income rate of families in the two groups, where a considerable number existed below the bare minimum level. Most families from Guguletu regarded themselves as being urbanised and most of the children from this sample were born and had grown up there. All but one family from Tsolo district group regarded the rural

environment as their home and 35,9% declared themselves as "Red" people with tribal customs.

Exposure to allergens was similar in magnitude in both areas although the type of allergens differed. Exposure to pets was more common for children from Tsolo district, where only 11% of the children had no animals in the home and more than half of the children were exposed to both dogs and chickens in their homes. In contrast 42,3% of the non-asthmatic children from Guguletu and 50% of the asthmatic children had no pets or animals in the home. Most of the children in Tsolo district were exposed to both maize and pollen as well as maize food. Almost all families in Tsolo district cultivated maize, whereas it was uncommon in Guguletu. Exposure to grass was common in both areas.

The diet of children in both areas included foodstuff which may relate to the development of asthma and no real important differences exist between the two samples.

There was a striking difference in the patterns of breast feeding during infancy of the two samples. Only 19 or 2,3% of the children from Tsolo district had never been breast fed and the median duration of breast feeding was 20,3 months. In the non-asthmatic group of children from Guguletu 175 or 27,6% had never been breastfed and the median duration of breast feeding for this group was 7,7 months. The median duration of breast feeding for the 22 asthmatic children from Guguletu was 4,7 months and 5 of the asthmatic children were never breastfed.

All asthmatic and most other children from Guguletu slept on mattresses. More than half of the children of Tsolo district slept on mats made from grass stems or reeds.

It is clear that children from both urban and rural areas were exposed to a variety of allergens in their environment. The difference in prevalence of asthma for children of the two areas is, however, striking. In spite of an abundant exposure to inhalant allergens, as well as dietary allergens, asthma was almost absent among children from Tsolo district.

Speculatively, two possibilities may be put forward to explain the striking difference in prevalence of childhood asthma in the two samples.

The first possibility is that the rural child is protected against developing asthma during childhood by prolonged breast feeding which can last up to 24 months. Immunoglobulin A secreted in breast milk protects the infant against septicaemia and infection of the respiratory syncytial virus and also delays the age of onset and the decrease of severity when infection does occur. It is proposed that during prolonged breast feeding the IgA in breast milk, which is also inhaled, prevents absorption of allergens from the respiratory and intestinal mucosa. This prevents priming of IgE forming plasma cells during infancy and thus protects the potentially allergic infant from developing childhood asthma. It does, however, leave the child unprotected in later life with subsequent onset of asthma during the second and third decades.

The second possibility may be a difference in exposure to the housedust mite as a result of the different sleeping habits in the two samples. Most children from Guguletu, including the 22 with asthma, slept on mattresses, whereas most of the children from Tsolo district slept on mats. The single asthmatic child from Tsolo district also slept on a mattress. The density of mites in the home has been shown to be highest on mattresses. When the mattress is covered with plastic, counts drop considerably. Mats may, as a result of the material used, be unfavourable for colonisation by mites. It is speculated that sleeping on mats may reduce the rural child's exposure to highly potent allergens. This exposure may result in a lower prevalence of asthma. Prick skin test reactions to the housedust mites were responsible for 40,0% of all positive reactions in children from Guguletu, compared to 13,1% for those from Tsolo district. These findings lend support to the speculation that children from Tsolo district may be less exposed to these particular allergens. As no mite counts were done, either in Guguletu or Tsolo district homes, this may only be speculated upon, but indicates a very important need for future studies.

A number of diagnostic and laboratory procedures were performed on each child and these procedures were aimed at establishing the response of the allergic child to allergens in the environment. These procedures were prick skin testing, serum IgE, allergen-specific serum IgE, serum immunoglobulins G, A and M and total eosinophil count in the peripheral blood. Interpretation of the diagnostic and laboratory procedures is obscured by the overwhelming presence of helminthic intestinal infestation in most children from Guguletu and 9,8% of children from Tsolo district.

The prick skin test reactions were difficult to interpret accurately in the black skins of the children. A greater number of children in Tsolo district had positive reactions: 130 (19,4%) compared to 51 (7,6%) from Guguletu. The positive skin reactions tended to reflect the allergens encountered in the children's environment. The most common positive reaction for children from Tsolo district was against maize, pollen or food. A positive reaction to feathers was also more common amongst Tsolo district children. Positive reactions to grass were almost equal in the 2 samples. This was expected as grass was common in both areas. The difference in the number of positive prick skin test reactions between the samples, with only 86 positive reactions among the children of Guguletu, probably relates to the overwhelming presence of helminthis intestinal infestation in most children from Guguletu. Saturation of mast cells with serum IgE provoked by helminthic infestation may prevent mast cell degranulation when challenged as in prick skin testing.

Raised serum IgE values were common for children of all ages and sexes from both samples, with boys from Guguletu having the highest values. Parasitic infestation has been shown to be a very potent stimulator, not only of serum IgE, but also of the total eosinophil soung in peripheral blood. No conclusions could be reached from either of these determinations and the routine determination of serum IgE and total eosinophil counts in these populations is not indicated.

Measurements of allergen-specific serum IgE by the RAST in selected children indicated that in many cases the total serum IgE was specific against various allergens. There was, however, no correlation between

the RAST findings and that of the skin test reactions. Allergen-specific IgE against grass and mites were common for Guguletu children and 25,9% of the positive reactions were against mites. Only 4,3% of children from Tsolo district had positive reactions to mites. This would lend further support to the speculation that mites are probably more common in the home of the child from a western urban society. A considerable part of the serum IgE level in the children with helminthic infestation seemed to be allergen specific. Helminthic parasitic infestation probably potentiated serum IgE production already present.

The serum immunoglobulin G, A and M levels followed the trend reported in Black children elsewhere and were considerably higher than those for Caucasian children. The serum immunoglobulins G, A and M of the asthmatic children did not differ from those of the two non-asthmatic populations, as was expected.

No definite conclusions can be reached on many of the findings in epidemiological studies. The prevalence of asthma for the two childhood populations is, however, a definite finding. This study, however, is also of importance in that it indicates trends that need further investigation. Although the way of life differed vastly in the two areas, no single factor could be incriminated as the reason for the difference in prevalence. As only Xhosa children were studied, racial differences are excluded. The importance of prolonged breast feeding and the effect of sleeping on mats can only be a matter of speculation. It may be possible that the way of life of the urban child exposes him or her more to the development of asthma. Conversely the way of life of the rural child results in less exposure to allergens. Less allergenic exposure in the rural sample is not evident from this study.

What will happen to the children from Tsolo district ? Will they be free from asthma during childhood, only to develop this disorder during adult life ? If so, what are the factors precipitating or regulating the age of onset ? The answers to these questions are as yet unknown.

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