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Longitudinal study on the health status of children in a rural Tanzanian community: parasitoses and nutrition following control measures against intestinal parasites

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Summary

Three repeated cross-sectional surveys were undertaken among children (1 month to 15 years) of a rural community in southeastern Tanzania. The study was part of a longitudinal project on the interactions among nutrition, parasitic infections and immunity within a primary health care programme emphasizing village health workers. All children underwent interviews and parasitological, anthropometric, anamnestic and clinical examinations. Out of 550–590 children examined each year, a cohort of 170 children could be followed for three consecutive years.

Malaria was holo- to hyperendemic in the community, *P. falciparum* accounting for >90% of the infections. The parasite and spleen rates were 88% and 67%, respectively, and the average enlarged spleen index was 2.0 among children from 2–9 years in 1982. Transmission of malaria was high and stable as indicated by a parasite rate of 80% among infants between 1 month and 1 year during the whole period of study. *G. lamblia,* hookworm *(N. americanus), Strongyloides* spp. and *Schistosoma haematobium* were highly prevalent and annual incidence rates were high, while *Entamoeba histolytica, Ascaris* and *Trichuris* were of minor importance. Prevalence and incidence of parasitic infections did not differ by sex. Multiparasitism was very frequent and <11% of all children were parasite-free in each year. Not a single child remained parasite-free for three consecutive years.

An anthropometric assessment showed a high degree of stunting (35–71%) and a substantial proportion of wasting (3–20%). The growth potential was

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normal in girls and boys during the whole period of study. There were indications that malaria was the main contributory factor to growth retardation among young children. Hookworm infection did not significantly affect the packed-cell volume of the children, probably owing to the low intensity of infection. Due to the multiparasitism and the lack of parasite-free individuals, single-parasite and single-nutrient effects were difficult to unravel.

A latrine campaign followed by a single mass treatment against hookworm (single oral dose of albendazole, 400 mg) and/or *G. lamblia* (single oral dose of ornidazole, 40 mg/kg) only temporarily affected the prevalence and incidence of *G. lamblia,* and only resulted in a decrease in the intensity of hookworm infections up to six months after the interventions.

As the effects of the latrine campaign and a single mass treatment on the parasite load were only transient, no sustained impact on nutritional variables was observed. The overall anthropometric assessment of the children (weight-height, height-age, weight-age, weight and height gain) did not show any substantial improvement among treated children when compared to untreated within the first 18 months after the interventions. The importance of these findings for the planning of disease control measures is discussed.

Key words: Tanzania; child health, nutritional status; parasitic infections; control measures.

Introduction

The intimate relationship between nutritional status, infection load and immune status of an individual has been recognized by various authors working with experimental models or under field conditions (reviews by Scrimshaw et al., 1968; Chandra and Newberne, 1977; Buck et al., 1978c; Isliker and Schürch, 1981; Beisel, 1982; Watson and Petro, 1982; Chandra, 1984). Most studies have investigated two elements of the triangle of the complex interrelationship, i.e. interactions between immunocompetence and nutrition (Chandra, 1981; Beisel, 1982) or between infections and nutrition or immunity (summarized in: Lee and Aboko-Cole, 1981; Crouch, 1982; Isliker and Schürch, 1981; Hall, 1985; Keusch and Solomons, 1985). A few well established single nutrient effects on parasitic infections and the immune system have been reported (summarized in: Beisel, 1982; Chandra, 1984; McMurray, 1984; Stürchler, 1984). On the other hand there are numerous studies describing how single infections boost or impair the immune system and the nutritional status (Chandra and Newberne, 1977; Cohen and Warren, 1982; Stürchler, 1984).

Interactions between nutrition, infection and immunity should also be assessed against the background of a particular environment. Multiparasitism and/or different concurrent viral and bacterial infections are the rule rather

than the exception in endemic areas (Buck et al., 1978a, b, c; Keusch and Migasena, 1982). The pattern of multiple and concurrent infestations which an individual host has experienced in the past and is facing at a particular moment, along with his/her nutritional status, trigger and determine the reaction of his immune system and may have a significant impact on his growth, development and well-being. The complexity (mixed infections, varying degree of nutritional deficiencies, migration of population) of an appropriate study design for unravelling parasite and nutrient effects explains the scarcity of studies of this kind in endemic areas. However, such studies are needed in order to pin-point parasite-associated nutritional effects important for public health and to define appropriate control strategies (Cole and Parkin, 1977; Latham, 1982, 1984; Keusch, 1982; Pines, 1982; Keusch and Scrimshaw, 1986). Some investigations have been described, for example Murray et al. (1978, 1980) provided evidence that iron repletion can aggravate clinical symptoms of malaria and amebiasis. Gupta (1980) and Gupta and Urrutia (1982) demonstrated an increased height-gain among Guatemalan pre-school children treated against *G. lamblia.* A better catch-up growth was observed in Australian children treated against *G. lamblia* (Kay et al., 1977). With regard to intestinal helminths, some community-based studies advocate periodic deworming in order to overcome the nutritional impact associated with *Ascaris* (Gupta et al., 1977; Willett et al., 1979; Stephenson et al., 1980; Stephenson, 1984), hookworm infections (Variyam and Banwell, 1982; Haller and Lauber, 1980) or schistosomiasis (Stephenson et al., 1985a, b). Although these studies indicate important interrelations, the relevance of such findings for community-based interventions is conflicting (Schultz, 1982; Latham, 1982; Stevens, 1985).

Tanzania faces a plethora of parasitic diseases which affect both rural and urban populations. Malaria is hyper- and holoendemic in large parts of the country (Clyde, 1967) and, in addition to diarrhoeal diseases and acute respiratory tract infections, substantially contributes to morbidity and mortality among young children (UNICEF, 1985). Intestinal protozoa and helminths are also highly prevalent. It has been suggested that these infestations are causal factors of nutritional disorders in children (Nhonoli et al., 1974; Willett et al., 1979; Meakins et al., 1981; Carswell et al., 1981; UNICEF, 1985). A comprehensive review of the anthropometric data on children in the various regions of Tanzania reveals a substantial overall point prevalence of acute malnutrition (prevalence of wasting 8%) and growth retardation (prevalence of stunting 47%; Kimati and Scrimshaw, 1985). The data show widespread dietary inadequacies. Their coexistence with various diseases results in mutually reinforcing interactions (Chandra and Newberne, 1977) which are the immediate causes of the death of young children (UNICEF, 1985; Kimati, 1986). The underlying causes represent a complex interplay between agricultural and dietary practices, health care delivery and the political, social and economic situation in each particular setting (Jonsson, 1983; Latham, 1984; UNICEF, 1985; Vuylsteke et al., 1986; Kimati, 1986).

Considering these problems, and in view of the scarcity of longitudinal community-based studies, we attempted to investigate aspects of the interaction between disease and nutrition within a rural community of lowland Tanzania. The community is representative of Tanzanian health problems (UNICEF, 1985; Tanner et al., 1982, 1987) and the health care system based on national primary health care (PHC) guidelines (Ministry of Health, 1983) is being strengthened.

The present study describes part of the results of the triennial surveys undertaken to monitor the health status of children within a large rural community, Kikwawila village (Kilombero District) in southeastern Tanzania. These surveys are a cornerstone of the applied research project within a PHC programme in Kikwawila which aims to:

1. investigate how interactions between nutrition, parasitic infection, immunity and environmental factors determine the health status of children, and
2. evaluate the effect of various health interventions such as PHC implementation, selective population chemotherapy, health education, sanitation and schistosomiasis transmission control on the health status of children.

The selection criteria and the description of the study area as well as the outline of the study design including the PHC component have been described in detail in a previous paper in this volume (Tanner et al., 1987). The present paper summarizes the baseline data on the prevalence and incidence of parasitic infections and the relation to nutritional variables such as haematological and anthropometric values. In addition, the 6 and 18 months assessment of the impact of a combined latrine campaign and mass treatment against *G. lamblia* and hookworm are discussed.

Study area, Material and Methods

Study area

The study was undertaken in the Kapolo and Kikwawila sectors of Kikwawila village, which is situated in the Kilombero District, southeastern Tanzania. The study area is described in detail by Tanner et al. (1987, Figs. 1, 2, this volume).

Study population

The study focused on children between 1 month and 15 years of age living in the nucleated settlements of the Kapolo and Kikwawila sectors. All children underwent comprehensive anthropometric, parasitological, clinical, biochemical and serological examinations in October of the years 1982, 1983 and 1984 (Table 1). As outlined above (Tanner et al., 1987) between 550 and 590 children were seen each year. One third (170/565) could be examined during three consecutive years. Table 2 shows the age/sex structure of the sample of children examined in 1982, 1983 and 1984 as well as of the cohort followed for three consecutive surveys. There were no significant differences with regard to age and sex between the three cross-sectional samples and the cohort of children followed for three years. The results presented in this paper focus on the cohort of 170 children.

Table 1. Data collected from children (1 month to 15 years) during the annual health status surveys in October 1982, 1983 and 1984

Personal data	age, sex, house
History	MCH card, health problems
Anthropometry	weight, height, mid-upper arm circumference
Blood (thin and thick smear, serum, plasma)	Hc, parasitology, serology, SPE, ZN and retinol determination
Stool	MIF, Kato, culture
Urine	filtration, dip sticks[a]
Clinics	general, hair, eyes, skin, liver, spleen
Sonography[b]	kidney, bladder

MCH = mother child health
Hc = haematocrit
SPE = serum protein electrophoresis
ZN = zinc
MIF = merthiolate-iodine-formalin fixation
[a] mainly for the presence of blood, protein and leucocytes
[b] for in-depth study on schistosome morbidity (cf. Degrémont et al., 1985; Burki et al., 1986)

Table 2. Age/sex structure of the children examined in three surveys and of the cohort followed for three consecutive years

Age groups (years)	Number of children (%)							
	Cross-sectional surveys						Cohort	
	1982		1983		1984		1982 – 83 – 84[a]	
	male	female	male	female	male	female	male	female
<2	46 (17)	42 (14)	47 (18)	38 (13)	38 (14)	47 (15)	12 (15)	7 (8)
2–5	92 (34)	92 (32)	72 (28)	88 (30)	71 (26)	82 (26)	26 (33)	24 (27)
6–10	97 (35)	110 (38)	79 (31)	93 (32)	92 (33)	98 (32)	27 (34)	36 (41)
11–15	38 (14)	46 (16)	59 (23)	73 (25)	75 (27)	82 (27)	14 (18)	21 (24)
no info	1	1	1	2	2	1	2	1
All	274	291	258	294	278	310	81	89
Ratio male/female	0.94		0.88		0.90		0.91	

[a] age groups according to age 1982

Study design and health interventions

The complete design of our longitudinal project on the health status of children in Kikwawila and Kapolo is outlined in the preceding paper (Tanner et al., 1987, this volume). The elements relevant for this paper are:

Health status surveys were always undertaken in October, i.e. at the end of the dry season.

During this time of the year, most inhabitants actually stayed in the village and were not much engaged in agricultural work, travelling or additional income generating activities such as fishing. The population was informed by their leaders about the study and the children were brought to the primary school of Kikwawila or Kapolo on a specified date. The examinations (Table 1) took place in the premises of the school. They were done by virtually the same team, each time; only auxiliary staff rotated from year to year.

Health interventions

After each comprehensive health survey, the children and their parents were informed about the outcome of the examinations by their village health worker. Both infected and symptomatic subjects received a form indicating their problem(s). These individuals were subsequently advised to seek medical care at the village health posts, the dispensary at Kibaoni (2–8 km west of the village) or at the District hospital in Ifakara (for map cf. Tanner et al., 1987).

Based on the results of the surveys, feedback was given to the villagers and control measures were launched if the need for these was also perceived by the population (Tanner et al., 1986 a). After a sanitation campaign by the District health office and the village government from December to April 1982/83 (cf. Tanner et al., 1987, this volume, Fig. 5), mass treatment was offered to all adults and children of the community in April/May 1983. The people of the Kapolo sector were treated with a single oral dose of 40 mg/kg ornidazole (Tiberal, kindly provided by Hoffmann-La Roche), while the population of Kikwawila received ornidazole and a single oral dose of 400 mg albendazole (Zentel, kindly provided by Smith Kline & French Labs.). Pregnant women and children under 6 months (ornidazole) or 24 months (albendazole) were excluded from treatment. The effect of the combined intervention (sanitation campaign and mass treatment) was evaluated 6 and 18 months after the mass treatment, during the health status surveys of October 1983 and 1984.

Demographic and anamnestic data

Each child was identified by his (her) name, sex, age, household and ten-cell leader (balozi). The age was taken from the mother and child health (MCH) card if available; if not the child or a relative (or caretaker) was asked. Demographic and anamnestic data were recorded by a Medical Assistant student or a local staff member of the project team who were always assisted by a village health worker (VHW). VHWs knew most of the children and could assess their age. A brief history was taken from the MCH card (spells of fever/malaria and diarrhoea, vaccination status) and by interview. In latter, questions included the following: haematuria, dysuria, number of stools the day before the survey, and the major health problem as perceived and experienced by the child. Mothers/caretakers answered for under-fives.

Anthropometry

Weight of children >2 years was recorded by a beam balance to the nearest 100 g. Children <2 were weighed hanging in a "bag" using a spring balance with increments of 100 g.

A height rod was used for children >2 years and a length board for those younger. Height/length were recorded to the nearest 0.5 cm. Mid-upper arm circumference (left) was also measured to the nearest 0.1 cm. The data were compared to the National Centre for Health Statistics (NCHS) standards as summarized in WHO (1983). In addition, the classification of wasting and stunting (Waterlow, 1972; Waterlow et al., 1977) was applied using Tanzania adapted Harvard standards (Kilimanjaro Christian Medical Centre, KCMC, 1978).

Urine examinations

Urine was collected between 9 and 12 a.m. in plastic bags and brought to the laboratory. Macrohaematuria was assessed and the presence of blood, protein, leucocytes and nitrite was semi-quantitatively measured by dip sticks (Combur, Boehringer Mannheim, FRG). Subsequently, the urine was filtered through a 12 µm Nucleopore polycarbonate membrane ($\varnothing = 23$ mm, Nucleopore Corp., Pleasanton, CA, USA) and the filter was examined under the microscope for the presence of

schistosome eggs. Intensity of *S. haematobium* infection was expressed as number of eggs/10 ml urine (cf. also Zumstein, 1983).

Blood / Serum

Blood was collected from finger pricks only. A thick smear and two thin smears were made from each child. In addition, duplicate haematocrit was made (heparinized tubes, No. 1020, Clay Adams, USA) and a blood sample was collected in a small plastic tube (300 µl, Semadeni, Switzerland) or Microtainers (No. 5960, Becton-Dickinson) for serum. Haematocrits and serum were kept at 4–8 °C during the transport from the field to the laboratory.

Thick smears were stained with Giemsa and examined for malaria parasites and microfilariae. The malaria parasite density was semiquantitatively assessed by counting the number of parasites/field (at least 50 fields examined at 6×50 magnification): ((+)) = 1 parasite/field or <400/µl, (+) = 2–3 or 400–1200, + = 4–10 or 1600–4000, ++ = 11–30 or 4400–12,000, +++ = >30 or >12,000. Species determination was always confirmed by thin smears. Thin smears were stained with May-Gruenwald-Giemsa which allowed the differentiation of white blood cells and the determination of malaria parasite species.

After the haematocrit tubes were centrifuged and read, the capillary tubes were broken at the interface between packed cells and plasma. The portion containing plasma was sealed with plasticine and kept frozen at –20 °C until shipment to Switzerland on dry ice.

The blood sample for serum collection was allowed to clot and was subsequently centrifuged. The serum was kept frozen at –20 °C until serum protein electrophoresis was performed or a sample was shipped to Switzerland on dry ice. In Switzerland, sera and plasma were stored at –70 °C.

Stool

Village health workers (VHW) informed the households and distributed stool containers to all children to be surveyed the evening before the survey. The children or their parents/attendants were asked to bring a fresh stool sample on the day of the survey. Each stool sample was processed in the laboratory using three different methods:

- fixation in MIF (merthiolate-iodine-formalin) for the parasties, mainly protozoa (WHO, 1980);
- Kato/Katz method (Katz et al., 1972) using the OVO-FEC-kit of Boehringer Mannheim (Rio de Janeiro, Brazil) for quantifying helminth eggs;
- cultivation on agar for 10 days. The bottom of petridishes (diameter = 10 cm) was coated with a 1–2 mm layer of 2% agar (Gibco M 0010). A small sample (about 1 cm in diameter) of fresh stool was placed on the agar and 10 ml of sterile distilled water was added. The cultures were left at ambient temperature (26–30 °C) for ten days. After 10 days the culture liquid was recovered and centrifuged, and the pellet was examined for hookworm and *Strongyloides* larvae. This method allowed the distinction of filariform larvae of *Strongyloides* spp., *Ancylostoma* and *Necator.*

Clinical examinations

Each child underwent a brief clinical examination. The general health status was assessed and the presence/absence of clinical signs related to the nutritional status with special regard to eyes, mouth, skin and hair was recorded. The liver was palpated and splenomegaly was classified according to Hackett (Bruce-Chwatt, 1980).

Statistical analysis

All data were coded and analyzed on a DEC PDP-11 system with the SPSS-II programmes (Morrison, 1982). Some data were analyzed on an IBM MVS/OS with the SAS programme, release 5 (SAS, 1985).

The present longitudinal investigation was designed as a community intervention study (cf. Tanner et al., 1987). As randomization was not the basis of the allocation of children to the different cohorts (e.g. treated, untreated) followed for three consecutive years, and since the PHC activities occurred in the village parallel to our applied research, the study had the character of a "quasi-ex-

perimental multiple group comparison" (cf. Campbell and Stanley, 1963; Kleinbaum et al., 1982). This design implied that the statistical analysis was done as an exploratory attempt to show major trends. The results of significance tests are therefore reported not in a confirmatory sense but as means of description. The calculated error probabilities p provide a measure of the weight to be attached to the corresponding findings, but do not represent significance probabilities in the strict sense.

Results

Prevalence of parasitic infections

The present study summarizes the results of the comprehensive health status survey among 170 children of the Kikwawila and Kapolo sectors from 1 month to 15 years of age (cf. Table 2 for age-sex structure) followed for three consecutive years. The number of blood, urine and stool samples analyzed does not always total 170 in the tables and graphs below, as some samples could not be obtained in one of the surveys or, not all of the tests could be made because only a small volume of blood or stool was obtained. Statements on age or age-groups refer to the initial age in the first survey year, 1982. Thus, age groups shown for three consecutive years represent cohorts of children.

Fig. 1 shows the overall prevalence of the most frequent parasitic infections found among children ≤5 years and of 6–15 years in the Kikwawila and Kapolo sectors. This figure does not distinguish between children treated in 1983 and untreated ones. The effect of the mass treatment in May 1983 (see Material and Methods) will be analyzed and described later. The high prevalence of *G. lamblia* (overall 1982: 24%) and *Strongyloides* (assessed by coproculture overall 1982: 17%) established from a single stool examination per child and year was striking. The prevalence data for *E. histolytica* and *G. lamblia* are based on cysts found in the stool samples fixed in MIF.

Hookworm and urinary schistosomiasis were highly prevalent. Significantly more *S. haematobium* infections were found among children living in the Kikwawila sector (1982: 74% vs 26% from Kapolo). The same was true for hookworm (89% vs 11%). The predominance of hookworm and schistosomiasis in the Kikwawila sector was found in all survey years. While the prevalence of *Strongyloides* and *E. histolytica* remained stable for three years, there was a decrease in the prevalence of hookworm and *G. lamblia* from 1982 to 1983 followed by a reversal to the initial prevalences in 1984. The influence of the health interventions on prevalence will be shown below. The prevalence of *S. haematobium* increased with age reaching 69% among children of 6–15 years. Tables 3 A, B show the geometric mean intensity of hookworm and *S. haematobium* infections in three age-groups and for three consecutive years. The proportions of children with heavy infections (*S. haematobium* ≥50 eggs/10 ml, hookworm ≥1000 eggs/g stool) are indicated. There was no distinct intensity pattern for a particular age group. *S. haematobium* showed an increase of

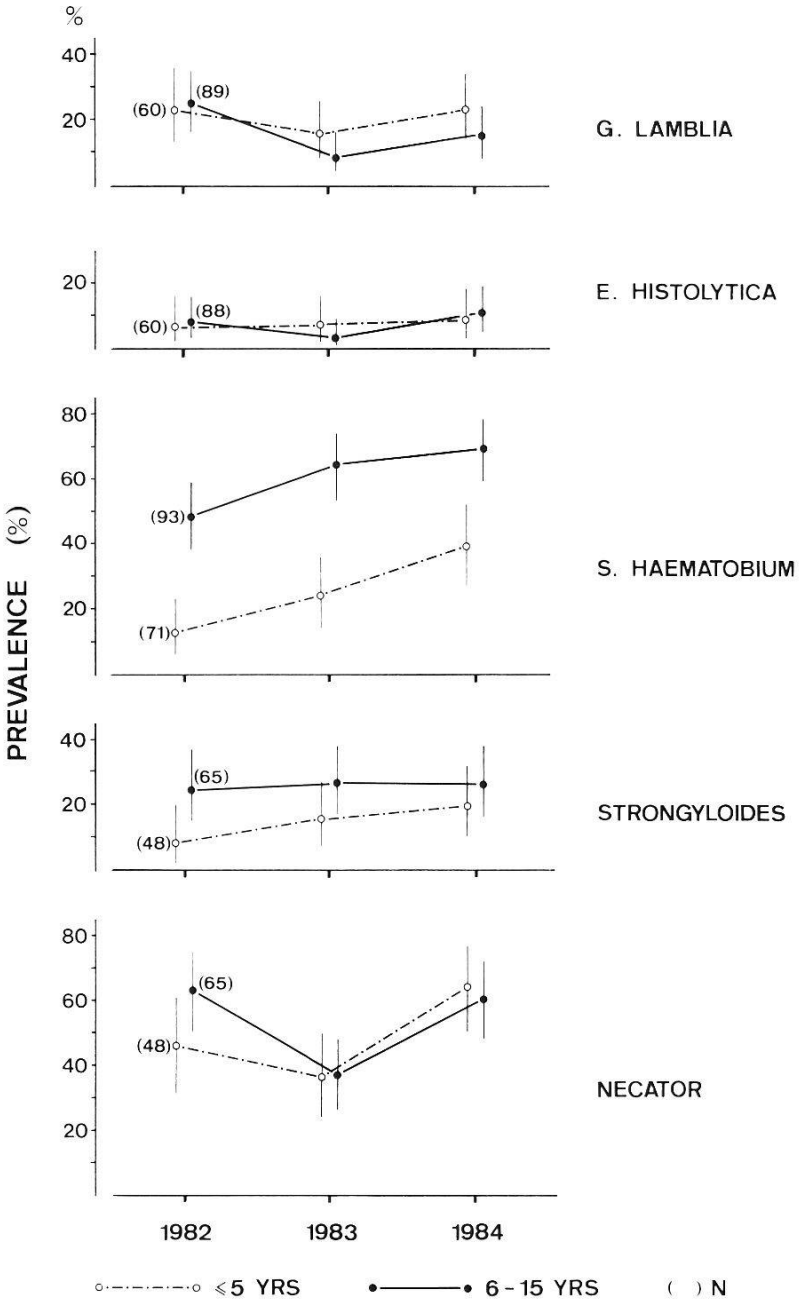

Fig. 1. Prevalence of parasitic infections among a cohort of 170 children of Kikwawila village followed for three consecutive years. Proportion of infected children with 95% confidence intervals of binominal proportion are represented for two age groups (0–5 years and 6–15 years).

intensity with age while the trends and levels were similar in all age groups for hookworm (cf. Tables 3 A and B).

All heavy infections with *S. haematobium* (1982: 15/54, Table 3 A) and three-quarters of the heavy hookworm infections (1982: 6/8 Table 3 B) were recorded from children living in the Kikwawila sector. There was no obvious sex-related difference among infected subjects. The intensity of *S. haematobium* infections increased from 1982 to 1983 and then decreased slightly (proportion of heavy infections stable in 1983, 1984), despite the increasing prevalence from 1982–83 to 1984 (Fig. 1). The intensity of hookworm infections

Table 3A. Intensity of *S. haematobium* infection (eggs/10 ml urine) among children of the Kikwawila and Kapolo sectors; geometric mean (gx) and proportion of heavy infection (≥50 eggs/10 ml)

Population	Year of survey	Age group (years)									All		
		0–5			6–10			11–15					
		N	gx	%≥50	N	gx	%≥50	N	gx	%≥50	N	gx	%≥50
Infected children	1982	9	20	44	30	17	27	15	15	20	54	16	28
	1983	16	13	25	43	31	40	16	26	44	75	25	37
	1984	26	9	23	44	13	23	21	7	14	91	10	21
All children[a] in cohort	1982	71	2	6	62	4	13	31	4	10	164	3	9
	1983	67	2	6	61	12	29	31	6	23	159	5	18
	1984	67	3	9	61	7	16	33	4	9	161	4	12

[a] includes uninfected children, egg counts calculated by (x+1) transformation

Table 3B. Intensity of hookworm infection (*N. americanus*) among children of the Kikwawila and Kapolo sectors: geometric mean (gx) eggs/g stool and proportion of heavy infections (≥1000 eggs/g)

Population	Year of survey	Age group (years)									All		
		0–5			6–10			11–15					
		N	gx	%≥1000	N	gx	%≥1000	N	gx	%≥1000	N	gx	%≥1000
All infected children	1982	21	199	19	24	173	13	8	190	13	53	185	15
	1983	10	125	10	7	53	0	7	62	0	24	79	4
	1984	21	238	5	16	206	6	12	163	0	49	206	4
All children[a] in cohort	1982	48	67	8	46	77	7	19	65	5	113	70	7
	1983	46	36	2	44	29	0	21	38	0	111	34	1
	1984	37	94	3	35	70	3	19	91	0	91	84	2

[a] includes uninfected children, egg counts calculated by (x+1) transformation

followed the pattern of hookworm prevalence and incidence (Figs. 1, 2 and Table 6). The intensity of hookworm infections decreased from 1982 to 1983 (Table 3 B). This decrease was statistically significant (Wilcoxon, $p < 0.001$). Consequently, the increase from 1983 to 1984 reaching the level of 1982 again was also highly significant (Wilcoxon, $p < 0.00001$). However, there were hardly any heavy hookworm infections in 1983 and 1984 (Table 3 B). The morphological assessment of the filariform larvae recovered from coprocultures showed that *N. americanus* was the highly predominant (>98%) species.

Table 4 summarizes malaria parasite rates, spleen rates and the average enlarged spleen (AES, Hackett Classification) of the children in the Kikwawila and Kapolo sectors. In this area, malaria is hyperendemic according to WHO

Table 4. Malariometric parameters of children of the Kikwawila and Kapolo sectors followed for three consecutive years (age groups according to age 1982)

	Age groups								
	<2 years			2–9 years			10–15 years		
	1982	1983	1984	1982	1983	1984	1982	1983	1984
Parasite rate %	89	84	74	83	73	49	75	40	54
Spleen rate %	84	79	58	67	66	46	52	58	21
AES $\bar{x}$	2.6	2.2	1.8	2.0	2.2	1.6	1.7	1.9	1.4
Number of children ..		17			85			36	

AES = average enlarged spleen (Hackett classification)

classification (Bruce-Chwatt, 1980). The development of semi-immunity with age among these children was indicated by decreasing spleen rates and AES. The high parasite rate (nearly 90%) among children <2 years reflects a high transmission dynamic. The same picture was found in the cross-sectional survey of children 1 month to 1 year. Their parasite rate was always over 80%, 1982 49/57 (86%), 1983 34/39 (87%), 1984 28/34 (82%), reflecting high and stable transmission. All four human malaria species were observed, but *P. falciparum* was the most important species (>90%) in Kikwawila village. Mixed infections were frequent and ranged from 8–15% among the children followed for three years.

Ascaris, Trichuris and *S. mansoni* infections were only occasionally recorded (<1%) among the children of the Kikwawila and Kapolo sectors. In 1982, provocation with diethylcarbamazine (DEC-citrate 50 mg for children, 100 mg for adults 45–60 min before making a thick smear, WHO, 1984) was employed to detect microfilariae of *W. bancrofti*. Only 1% of all children examined (N = 565, cf. Table 2) and 17% of a sample of adults (N = 115) were found to be positive for microfilariae. The provocative day-test was not repeated in 1983 and 1984.

There were no sex-related differences in the prevalences and incidences of any of the parasitic infections investigated.

Multiparasitism

Multiple parasitic infections were frequent among the children of Kikwawila village. Table 5 summarizes the degree of multiparasitism with the six most common parasites found in the Kikwawila and Kapolo sectors: Only ≤11% of the children had no parasites in any one survey. One third had one and another third harboured two parasite species. One fifth even harboured three species. There was no clustering of multiparasitism and parasite-free individuals in a

Table 5. Multiple parasitic infections among 170 children (1 month to 15 years) of the Kikwawila and Kapolo sectors followed for three consecutive years

No. of species[a]	% infected			cumulative %		
	1982	1983	1984	1982	1983	1984
None	7	11	11	7	11	11
1	37	36	31	44	47	42
2	32	32	25	76	79	67
3	18	15	25	94	94	92
4	5	5	7	99	99	99
5	1	1	1	100	100	100
6	–	–	–			

[a] *G. lamblia, E. histolytica, Plasmodium* spp., *N. americanus, Strongyloides* spp., *S. haematobium*

particular village part or sector. No child was found to be parasite-free for three consecutive years. The health interventions undertaken in Kikwawila/Kapolo (cf. Tanner et al., 1987a) had no influence on the frequency of multiparasitism (Table 5), as reflected by the stable frequencies throughout the three years studied.

Risk of parasitic infections, incidences

Fig. 2 depicts the cumulative incidence rates (Kleinbaum et al., 1982) for *G. lamblia, E. histolytica, N. americanus, Strongyloides* spp. and *S. haematobium.* The incidences among young children (0–5 years) were higher for *G. lamblia,* and *Necator* compared to older children (6–15 years). The opposite was found for *Strongyloides* spp. and *S. haematobium.* The 95% confidence intervals give an indication of the precision of the incidence. The high incidence of *N. americanus, Strongyloides* spp. and *S. haematobium,* and also of *G. lamblia* 1983–1984, reflects established transmission of the parasites in the village.

Based on the incidences represented in Fig. 2, the risk ratios (RR_i, = relative risk) were calculated for children ≤5 years vs older (6–15 years) children as well as for the periods 1982–1983 vs 1983–1984 (Table 6). The RR_i of acquiring *G. lamblia* infection was more than twice in the period 1983–1984 when compared to the preceding period (1982–1983) in both age groups. It was even three times higher for *E. histolytica* among older children. On the other hand, the risk for hookworm, *Strongyloides* and schistosome infections was similar during both periods. The incidences for all parasitic infections were not different among stunted or wasted children when compared to children with normal anthropometric values (data not shown).

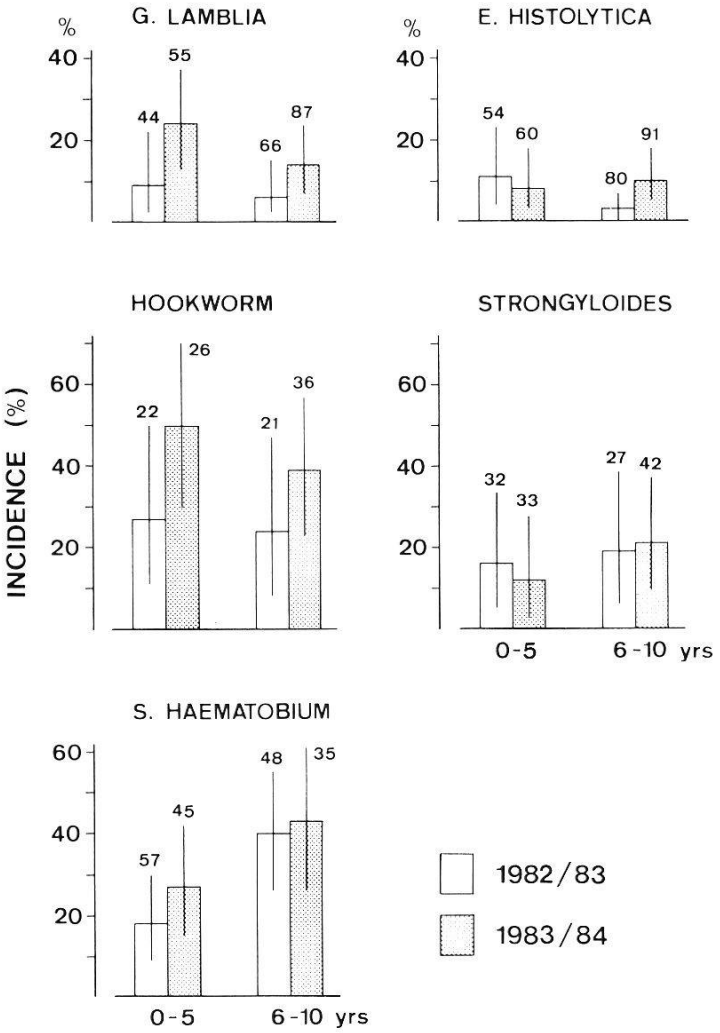

Fig. 2. Incidences of major parasitic diseases among a cohort of 170 children followed for three consecutive years. Incidences for the period 1982–1983 and 1983–1984 (and 95% confidence intervals) for two age groups (0–5 years and 6–15 years) are shown. Number (N) of children on top of the bars.

Table 6. Risk ratios (RR_i) of parasitic infections among children of Kikwawila village (based on Fig. 2)

	Age groups 0–5 years vs 6–15 years		Survey years 1982/83 vs 1983/84	
	RR_i (82–83)	RR_i (83–84)	RR_i 0–5 years	RR_i 6–15 years
Giardia lamblia	0.67	0.58	2.67	2.33
Entamoeba histolytica ...	0.27	1.25	0.73	3.33
Necator americanus	0.89	0.78	1.85	1.63
Strongyloides spp.	1.19	1.75	0.75	1.11
Schistosoma haematobium	2.22	1.59	1.50	1.08

Nutritional status

Weight, height and age of the children (≤10 years) were compared to the NCHS standards (WHO, 1983). Fig. 3 shows the frequency distribution (S. D. score distribution around median) for weight for height (W–H), weight for age (W–A) and height for age (H–A) as measured in 1982. W–H showed a near normal distribution (as by definition does the reference population). W–A and H–A showed a clear shift to the left with the peak at –2 standard deviations (H–A) or –1 standard deviation (W–A), but the near normal distribution pattern was retained. This shift to the left was probably influenced by an overestimation of age. Table 7 summarizes the frequency of wasting and stunting (Tanzania adapted Harvard standards, KCMC, 1978) among children ≤5 years and children from 6–10 years followed for the three consecutive years. The frequency of wasting (W–H), reflecting current malnutrition (Waterlow, 1972), was high in 1982 (≤5 years: 9%, 6–10 years: 20%) and decreased to lower levels (3–9% in both age groups in 1983/84, Table 7). Stunting (H–A) reflecting retardation, i. e. past malnutrition, was very prevalent among under-fives (71%) and 6–10-year-old children (70%) in 1982. An improvement was seen in 1983 (drop to 44% and 35%, resp.) which was partly a treatment effect (Fig. 7, see below). This improved level could only be maintained among older children (6–10 years) in 1984, whereas the children ≤5 years reverted to the levels of 1982 (64%, cf. Table 7). There was a clear trend towards a higher proportion of growth retardation (stunting and wasting) among children ≤5 years (Table 7). The trend of an improved nutritional status in 1983 compared to 1982 and the reversal in 1984 was also observed at the clinical level (Degrémont et al., 1987).

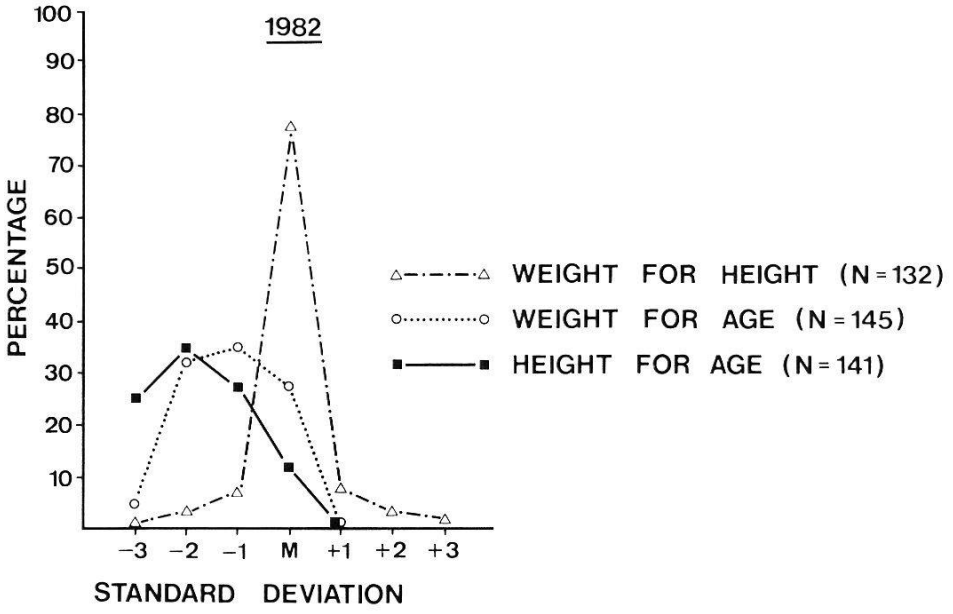

Fig. 3. Frequency distribution of SD scores around the median of NCHS reference population (WHO 1983) for weight for height, weight for age and height for age of the children ≤10 years of Kikwawila village in 1982 (initial survey).

Table 7. Stunting and wasting among the children of Kikwawila village; cohorts 1 month to 5 years and 6 to 10 years followed for three consecutive years

Classification of stunting and wasting[b]	1982			1983			1984		
	N	%	(95% CI)[c]	N	%	(95% CI)	N	%	(95% CI)
Age[a] 0–5 years									
normal	17	25.8	(16–38)	33	54.1	(41–67)	22	34.4	(23–47)
wasting	2	3.0	(0–11)	1	2.0	(0– 9)	1	1.6	(0– 8)
stunting	43	65.2	(52–77)	26	41.9	(30–56)	40	62.4	(50–74)
wasting and stunting	4	6.0	(2–15)	1	2.0	(0– 9)	1	1.6	(0– 8)
Total	66			61			64		
Age[a] 6–10 years									
normal	14	23.7	(14–37)	33	61.1	(47–74)	23	52.3	(37–68)
wasting	4	6.8	(2–16)	2	3.7	(1–13)	1	2.3	(0–12)
stunting	33	55.9	(42–69)	17	31.5	(20–46)	17	38.6	(24–55)
wasting and stunting	8	13.6	(6–25)	2	3.7	(1–13)	3	6.8	(1–19)
Total	59			54			44		

[a] Age groups according to age 1982
[b] Tanzania adapted Harvard standards (KCMC, 1978; Waterlow, 1972)
stunting = stages 2 and 3, wasting = stages 2 and 3
[c] CI = 95% Confidence interval of binomial proportion

The growth potential of male and female children was assessed by testing the relationship of log(weight) to height (Davies et al., 1974; Ehrenberg, 1968), Table 8 shows that all groups of children (female, male, treated 1983, untreated) had a normal comparable growth potential, i.e. the relationship log(weight) to height was highly significant.

Wasting (stages 2 and 3; Waterlow, 1972; KCMC, 1978) and stunting (stages 2 and 3) were not found to be associated with multiparasitism or the presence or absence of malaria parasitemia, *G. lamblia,* hookworms, *Strongyloides* or *S. haematobium* infections among the children (1 month to 10 years) in any of the three surveys.

Fig. 4 shows the mean haematocrit (packed cell volume, PCV) values (±SD) of each age group followed for three years. The mean PCV levels are within the normal ranges and increase with age among the younger children (0–5 years). No significant increase or decrease was observed during the study period. The PCV values were similar among treated and untreated children (data not shown). There was no correlation between malaria parasitemia at the day of the survey and the PCV.

Furthermore, there was no relation between hookworm infection and the PCV (data not shown).

Further nutritional parameters such as mid-upper arm circumference,

Table 8. Growth potential[a] of children from Kikwawila village before (1982) and after (1983, 1984) treatment with ornidazole and albendazole

Year	Sex	Group	N	r	Slope	P value for H_0: slope = 0
1982	female	not treated	41	0.93	0.008	0.0001
		treated	40	0.96	0.008	0.0001
	male	not treated	38	0.97	0.008	0.0001
		treated	33	0.94	0.008	0.0001
1983	female	not treated	44	0.98	0.009	0.0001
		treated	45	0.97	0.008	0.0001
	male	not treated	37	0.99	0.008	0.0001
		treated	36	0.93	0.007	0.0001
1984	female	not treated	44	0.92	0.007	0.0001
		treated	39	0.98	0.008	0.0001
	male	not treated	38	0.98	0.008	0.0001
		treated	38	0.97	0.008	0.0001

[a] Log 10 (weight) = intercept + slope × height (Ehrenberg, 1968; Davies et al., 1974)
H_0 = null hypothesis

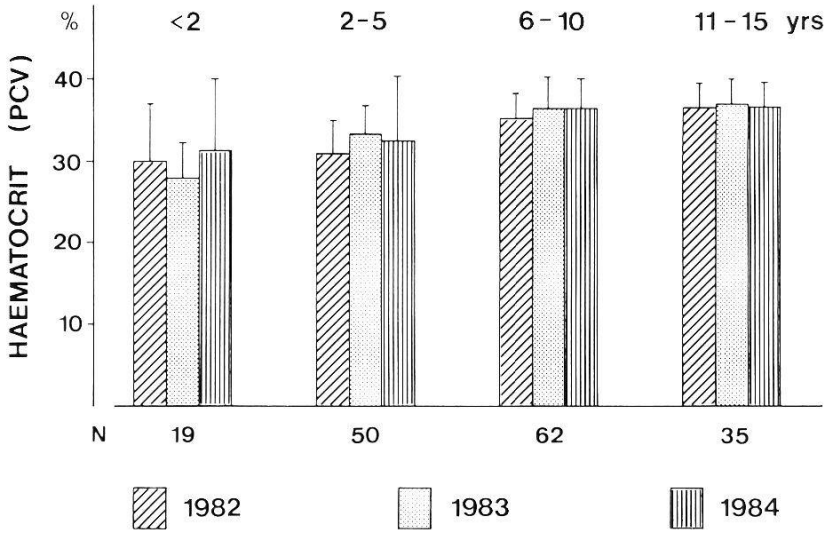

Fig. 4. Mean haematocrit values (PCV, packed-cell-volume) of four cohorts of children followed for three consecutive years. Bars indicate SD.

serum protein levels, retinol and zinc, were measured and related to the presence of parasitic infections and/or the health interventions. The comprehensive results and discussion of these nutritional variables are published elsewhere in this volume (Betschart et al., 1987; Stürchler et al., 1987).

Mass treatment and latrine campaign

The first community health survey in 1982 identified giardiasis, hookworm, strongyloidosis and urinary schistosomiasis to be the major parasitic diseases besides malaria (cf. Figs. 1, 2). Nevertheless, the population mainly complained about intestinal problems (Tanner et al., 1986a; Degrémont et al., 1987), and hardly perceived schistosomiasis as a disease (Lwihula, 1985; Tanner et al., 1986a), despite its high prevalence and incidence (Figs. 1, 2) and the substantial morbidity observed (Degrémont et al., 1985; Burki et al., 1986). Consideration of these facts and findings gave the basis for the first health interventions in the Kapolo and Kikwawila sectors in 1983; i.e. interventions in addition to the activities of VHW initiated in 1982 (STIFL/DHO, 1985; Tanner et al., 1987). After meetings of the District Health Office with the villagers, a latrine campaign was initiated by December 1982. The target was that 80% of the households should have a simple pit latrine built with local material (cf. Tanner et al., 1987) before mass treatments with ornidazole or ornidazole and albendazole could be launched. In April 1983, 82% of the households had a pit latrine – although some were still under repair – and treatment was offered to the population of Kapolo (ornidazole, 400 mg/kg single oral dose) and Kikwawila (ornidazole and albendazole, 400 mg single oral dose). As shown earlier (Tanner et al., 1987), the latrine situation deteriorated again after the rainy season, i.e. only 50% of the households maintained good latrines in 1984. A total of 380 children (≤15 years) and adults were treated in Kapolo with ornidazole (compared to census 1982: compliance rate = 49%) and 392 inhabitants of Kikwawila were treated with albendazole and ornidazole (compliance rate = 62%).

The effects of the mass treatments which followed the latrine campaign were evaluated 6 and 18 months later, i.e. on the following annual surveys in October 1983 and 1984.

The following data on the treatment effect again only focus on the cohort of children followed for the three consecutive years. Three groups were compared:
A. children not treated in 1983, N = 84;
B. children treated with ornidazole only (Kapolo), N = 34;
C. children treated with ornidazole and albendazole (Kikwawila), N = 49.

Ornidazole treatment

Fig. 5 shows the prevalence and the cumulative incidence of *G. lamblia* among treated (groups B+C) and untreated children (group A) followed for three years. The prevalence of *G. lamblia* decreased after treatment mainly among the treated children. The decrease of prevalence among treated children was statistically significant (chi-square, $P < 0.0025$) as well as the difference to the untreated individuals in 1983 ($P < 0.0025$). The treatment effect was not maintained in 1984 when the prevalence reached pretreatment levels. The changes in prevalence between treated and untreated children are best reflected

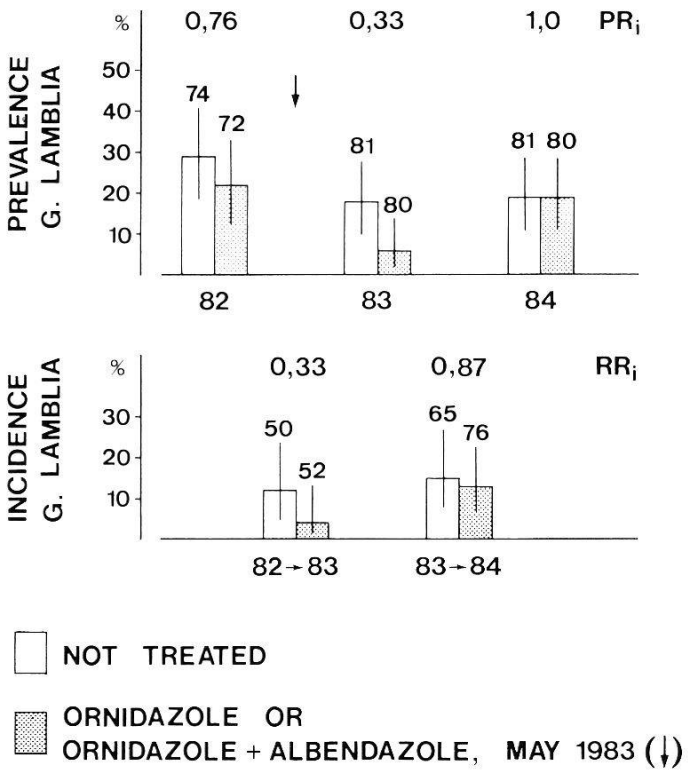

Fig. 5. The effect of ornidazole treatment (May 1983, following a latrine campaign) on the prevalence and incidence of *G. lamblia* among children of Kikwawila village six and 18 months after treatment. Proportions show data of treated and untreated children, the 95% confidence intervals are added for comparison (number = N of children on top of bars). The PR_i (prevalence ratio) and RR_i (risk ratio = relative risk) are given for each year.

by the prevalence ratio (PR_i, Prevalence of treated/Prevalence of untreated) shown in Fig. 5. Substantial differences were seen in 1983 (PR_i = 0.33). The incidence was lower among treated children 6 months after treatment. This difference was no longer observed in 1984. The risk ratio (RR_i) was 0.33 in 1983, indicating a threefold lower risk of *G. lamblia* infection among treated children, but it reverted to 0.87 in 1984.

Albendazole + ornidazole treatment

Fig. 6 shows the prevalence and incidence of hookworm infections among treated (group C, see above) and untreated children. Six months after treatment no substantial reduction of prevalence was observed when compared to the pre-treatment levels (PR_i 1.02 vs 0.89). The prevalence of hookworm infections increased from 1983 to 1984 among the untreated children (30/84 vs 46/74, chi-square, $p < 0.001$). This trend was not observed among the treated children. The incidence of hookworm infection parallelled the prevalence trends. No difference was observed in the first period (1982–1983), when treatment took place. Only incidences of the second period (1983–1984) showed an important difference: the risk of hookworm infection was nearly twice as high among untreated children compared to those treated (RR_i = 0.53). The intensity of

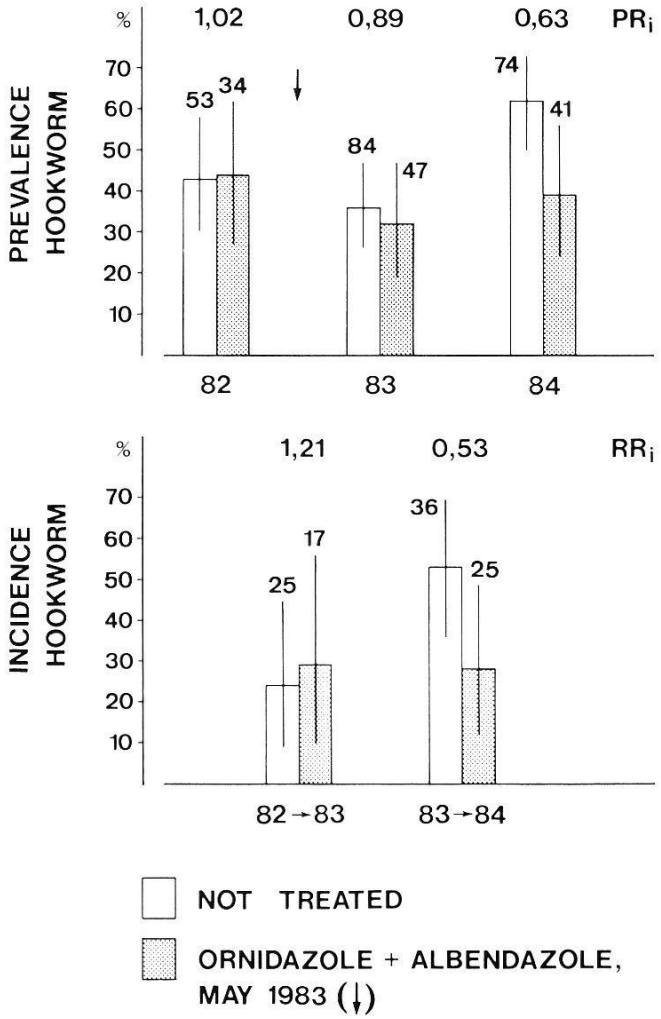

Fig. 6. The effect of albendazole treatment (May 1983, following a latrine campaign) on the prevalence and incidence of hookworm infections (*N. americanus*) among children of Kikwawila village six and 18 months after treatment. Proportions show data of treated and untreated children, the 95% confidence intervals are added for comparison (number = N of children on top of bars); PR_i = prevalence ratio, RR_i = risk ratio (s. Fig. 5).

hookworm infections significantly decreased among treated children from 1982 to 1983 (g mean (x+1) 336 eggs/g to 48, Wilcoxon, $P < 0.005$) while the decrease among untreated children was not significant (g mean (x+1) 120 eggs/g to 72, $P > 0.05$). The decrease of hookworm intensity after treatment between 1982 and 1983 was followed by a significant overall increase from 1983 to 1984. The increase was seen among treated and untreated individuals and reached the pretreatment levels of 1982 (treated: 48 eggs/g to 216, $P < 0.005$, untreated: 72 eggs/g to 240, $P < 0.02$). However, the proportion of heavy infections among the whole community was reduced to virtually nil after the latrine and treatment campaign and this was sustained until 1984 (cf. Table 3 A, B). Again as for ornidazole and *G. lamblia* the latrine campaign/treatment overall effect was mainly transient.

The single dose treatment with albendazole affected neither the prevalence

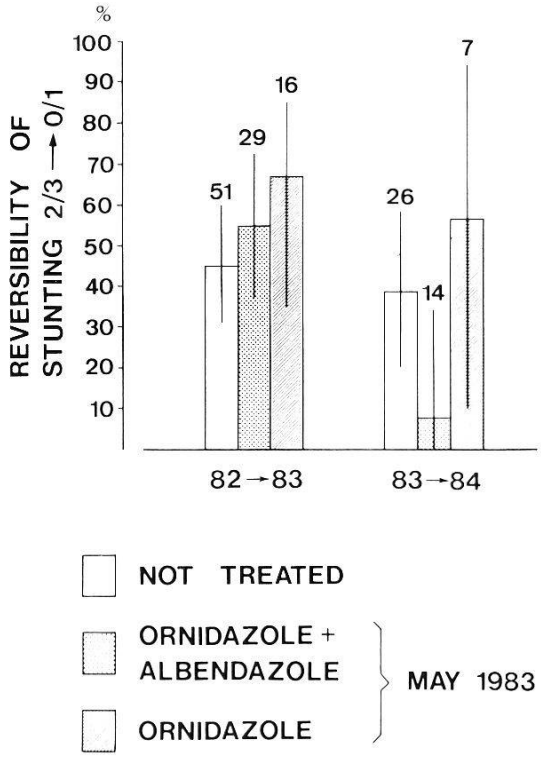

Fig. 7. Frequency of recovery from stunting stage 2 and 3 to stage 0 and 1 among treated and untreated children of Kikwawila village six and 18 months after latrine campaign/treatments; 95% confidence intervals are added for comparisons.

of *Strongyloides* in the community nor its incidence. Both remained stable during the study period (cf. Figs. 1, 2). There was also no difference between treated or untreated subjects with regard to the prevalence and incidence of *S. haematobium.*

Fig. 7 depicts a trend in the influence of treatment on the recovery rates of stunting stage 2 and 3 (retardation, past malnutrition) to stunting stage 0 and 1 following treatment. Six months after ornidazole or ornidazole + albendazole treatment a higher proportion of children improved from stunting stage 2 and 3 to 0 and 1 compared to untreated children (16/29 and 10/16 vs 23/51). This trend was no longer seen 18 months after treatment, and was not observed for wasting (reflecting current malnutrition, W stage 2 and 3 to W stage 0 and 1 1982/83: 5/6 treated vs 12/12 untreated, 1983/1984: 0 vs 2/2).

No sustained effect of the latrine and single mass treatment campaign was observed with regard to prevalence and incidence of *G. lamblia* and hookworm, nor was there compensation of past malnutrition (cf. Figs. 5, 6). Similarly, no significant impact of the latrine and single mass treatment campaign on the overall nutritional parameters of the community could be found within the first 18 months following the interventions. Figs. 8–10 summarize these findings for weight for age (Fig. 8), height for age (Fig. 9) and weight for height (Fig. 10) for all three groups (treated and untreated) and the three consecutive years. There was no significant change of the SD score distribution pattern and no shift of the

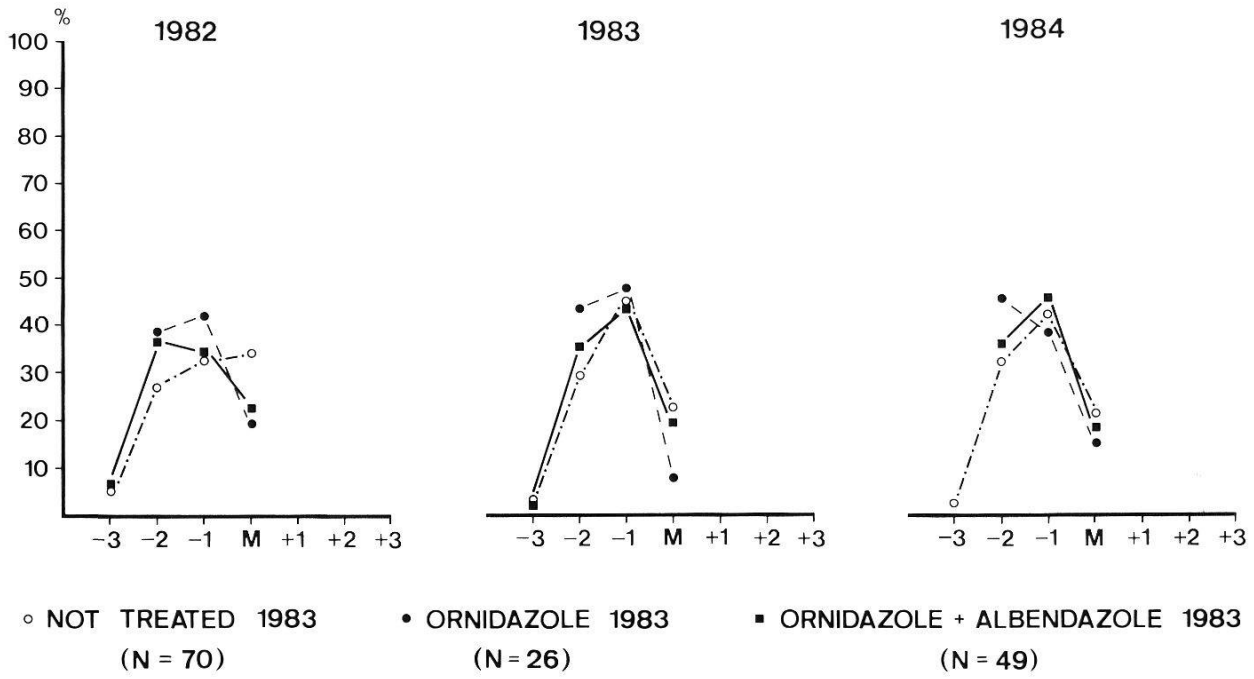

Fig. 8. Frequency distribution of SD scores around the median of the NCHS reference population for weight for age among treated and untreated children (treatment May 1983) followed for three consecutive years.

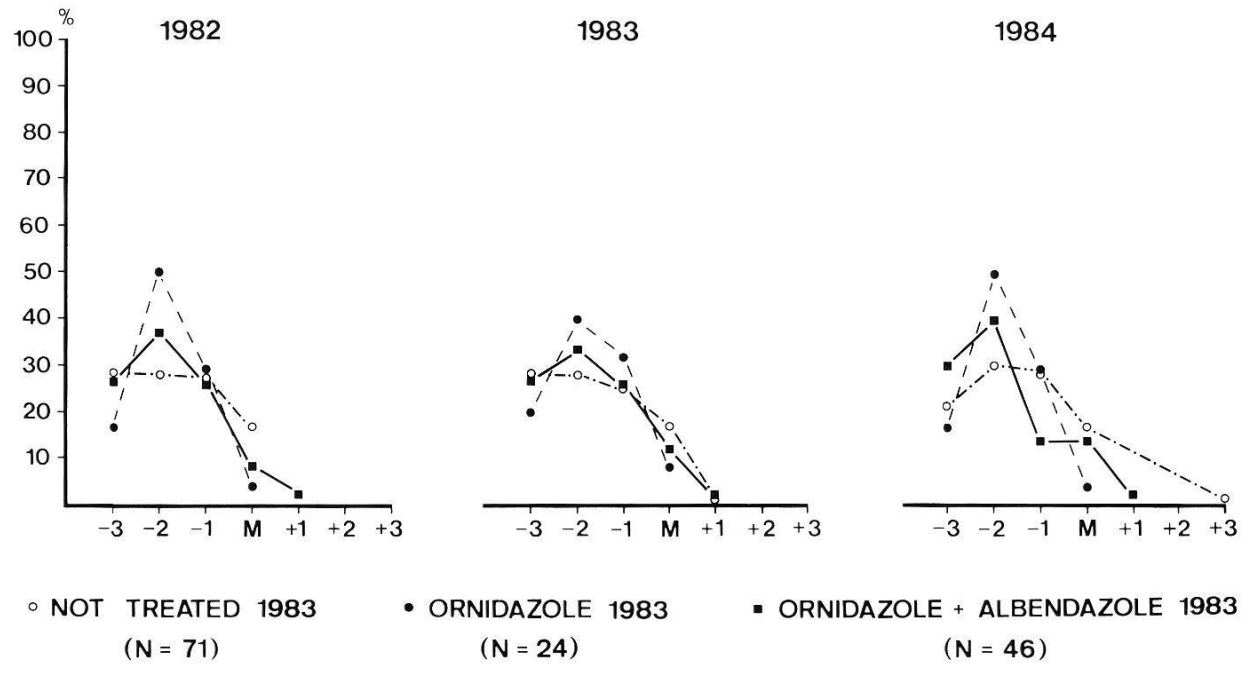

Fig. 9. Frequency distribution of SD scores around the median of the NCHS reference population for height for age among treated and untreated children (treatment May 1983) followed for three consecutive years.

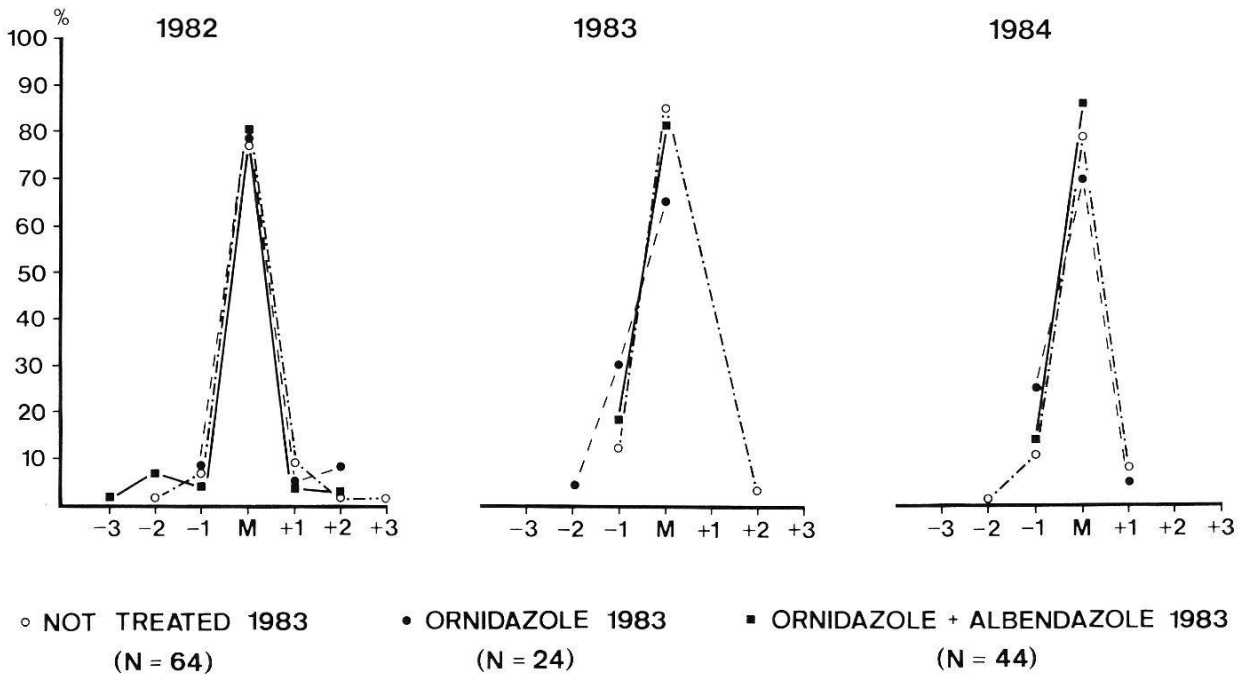

Fig. 10. Frequency distribution of SD scores around the median of the NCHS reference population for weight for height among treated and untreated children (treatment May 1983) followed for three consecutive years.

distribution curve to the right or left either from 1982 to 1984 or among treated and untreated individuals.

The analysis of body weight gain (kg per year) and height gain (cm per year) showed no significant differences (t-test) between treated and untreated children among three age groups (0–5 years, 6–10, 11–15, see Figs. 11, 12). The growth potential of all children was also not found to be affected by the latrine and treatment campaign (Table 8).

An attempt was made to analyze the multiparasite situation and the intervention effects among the cohort of children followed for three consecutive years by analysis of covariance. The models were designed with gain in mid-upper arm circumference and in body weight and height as dependent variables, and the models covered the period from 1982 to 1984. Table 9 shows the inventory with the description of the variables introduced into the models of analysis of covariance. The mid-upper arm circumference was only analyzed for children ≤ 5 years. Since weight gain and height gain showed a nonlinear relationship at all ages, age was broken down into two age groups (0–5 years and 6–15 years). Within each of the groups the linear relationship was retained when the intersection of the two regression lines was set between 5 and 6 years. Tables 10 A, B, C show the data for each of the three representative covariance models. The models do not account for any linear dependence between the introduced

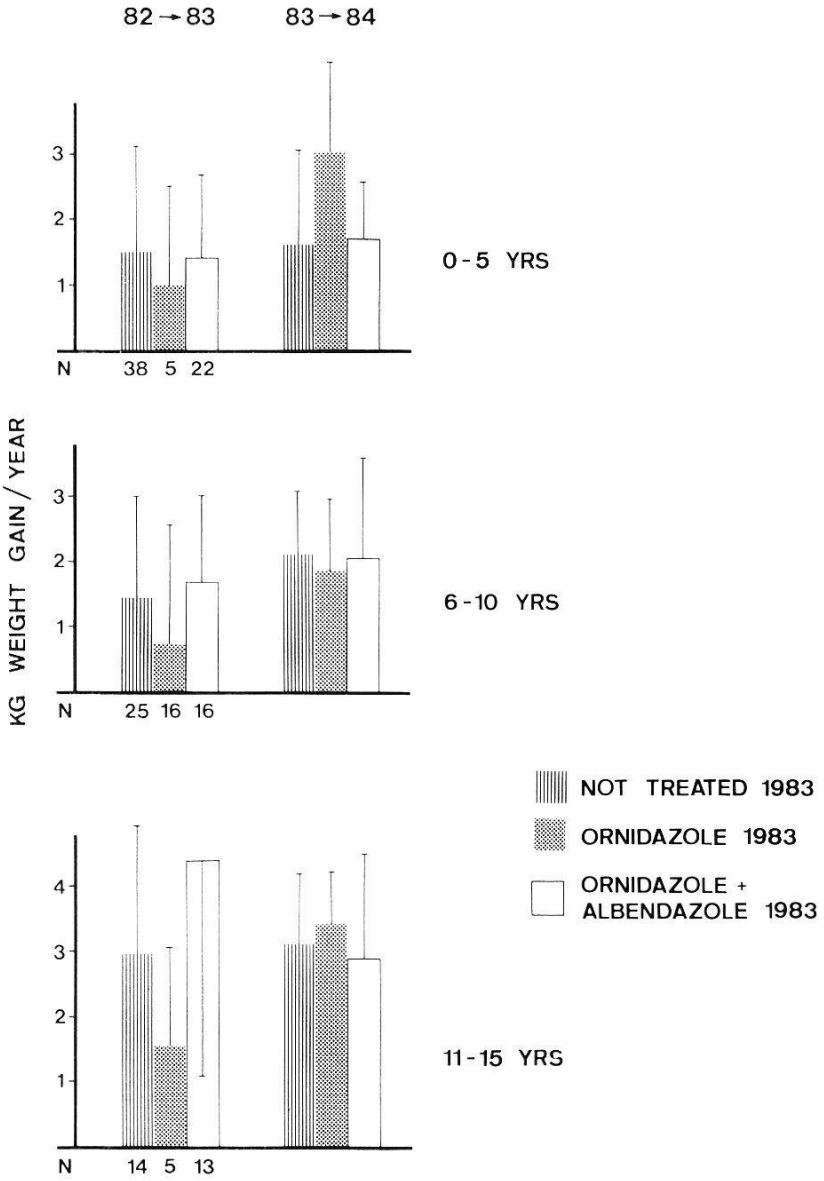

Fig. 11. Weight gain (kg/year ±SD) among treated and untreated children (treatment May 1983) of three age groups from 1982 to 1983 and from 1983 to 1984.

independent variables. With this restriction, the data show that only age explained the designed models for gain in mid-upper arm circumference (Table 10 A), body weight (Table 10 B), and height (Table 10 C). No single parasite effect emerged and no effect of the treatment campaign was revealed in the models shown here, covering the period 1982 to 1984. Similar results were obtained with the same covariance models for the periods 1982 to 1983 and 1983 to 1984 (data not shown). Furthermore, various approaches using stepwise multiple regression models (data not shown) did not reveal parasitic predictors which could be applied for the anthropometric parameters (mid-upper arm circumference gain, weight and height gain) of the cohort of children studied.

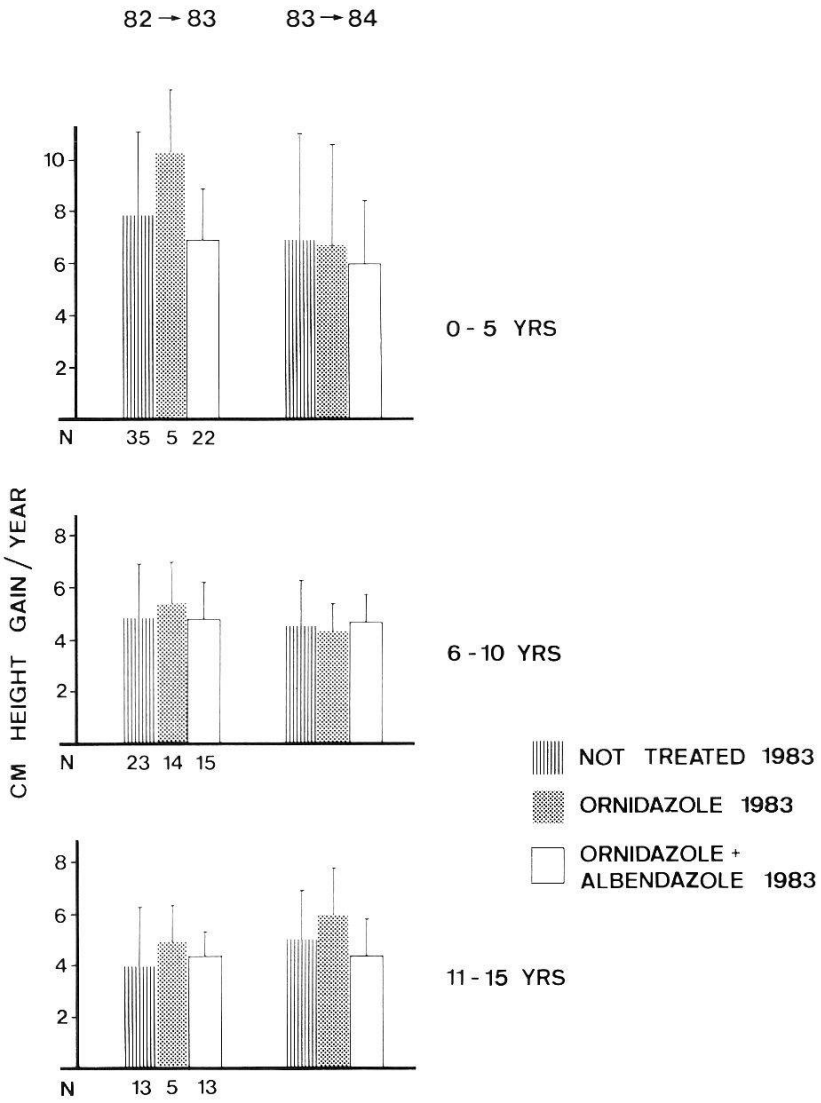

Fig. 12. Height gain (cm/year ± SD) among treated and untreated children (treatment May 1983) for three age groups from 1982 to 1983 and from 1983 to 1984.

Discussion

As mentioned earlier (see Material and Methods), the present investigation was not designed as a randomized intervention study. PHC emphasizing village health workers was implemented in the community of Kikwawila from 1982 onwards (STIFL/DHO, 1985; Tanner et al., 1987) and village health workers undertook curative and preventive measures. Parallel to this ongoing health intervention, the applied research project reported in this paper was started to monitor the health status. The latrine campaign and mass treatment were launched on the basis of the results of the first community health survey (cf. Fig. 1) after consideration of the health problems as they were perceived by the community (Tanner et al., 1986a; Degrémont et al., 1987). This led to a quasi-experimental study design (Kleinbaum et al., 1982) with multiple group

Table 9. Inventory of variables for analysis of covariance

Variable	N observations in analysis	Levels	Values	Remarks
A. Dependent variables				
Midarm gain	37		% gain in midupper arm circumference (100% = 1982)	only for children ≤5 years
Height gain	82		% gain in height (100% = height 1982)	
Weight gain	79		% gain in weight (100% = weight 1982)	
B. Model variables				
Factors:				
Malaria parasitemia ...		2	negative vs positive	0 and ((+)) vs ≥(+) cf. Material and Methods
Hookworm		2	negative vs positive	based on stool examinations
Strongyloides		2	negative vs positive	
G. lamblia		2	negative vs positive	
Treatment		2	chemotherapy 1983 yes vs no	
Covariates:				
Age		continuous	age in years, 1982	
Age in age classes		continuous[a]	two classes ≤5 years vs >5 years	
S. haematobium		continuous	eggs per 10 ml urine	

[a] continuous within each age class

Tables 10 A–C. Analysis of covariance among a cohort of children followed for three consecutive years. Models were designed with gain in midupper arm circumference (A), body weight (B) and height (C) as dependent variables and with parasitic infections and mass-chemotherapy as independent variables for the period 1982–1984.

Table 10 A.

I: Model

Dependent variable: midarm gain (%)

	DF	Sum of squares	F-value	P>F
Model	7	1734.86	2.69	0.03
Error	29	2675.36		
Corrected total	36	4410.22		

II: Source

Independent variable

	DF	Type III SS[a]	F-value	P>F
Factors				
Malaria parasitemia	1	0.003	0.00	0.99
Hookworm	1	5.63	0.06	0.81
Strongyloides	1	24.12	0.29	0.59
G. lamblia	1	40.42	0.44	0.51
Treatment 1983	1	37.12	0.40	0.53
Covariates				
Age	1	885.78	9.60	0.004
S. haematobium	1	3.63	0.04	0.84

[a] Type III SS according to SAS manual release 5 (SAS, 1985). Type III SS indicates the amount of sum of squares contributed by a single model variable provided that all the other model variables have already been fitted.

[b] Age broken down into two classes 0–5 years vs 6–15 years

DF = Degree of freedom

For variable description see Table 9.

comparisons. The data analysis consequently could only show trends as randomization was not the basis of group allocation. However, the treatment groups of a particular sector were highly comparable with regard to age and sex structure, initial parasite load (cf. Fig. 1) and nutritional parameters (cf. Tables 7 and 8, Figs. 8–10).

Prevalence of the parasitic infections was based on one stool, urine or blood sample per survey year. This is of particular importance with regard to *G. lamblia,* because it is shed irregularly, and because the diagnosis was made from MIF preserved stool samples. These procedures both tend to lead to an underestimation of the prevalence of *G. lamblia,* as has been shown in many

Table 10 B.

I: Model

Dependent variable: weight gain (%)

	DF	Sum of squares	F-value	P>F
Model	8	14683.94	4.33	0.0003
Error	70	29684.46		
Corrected total	78	44368.40		

II: Source

Independent variable

	DF	Type III SS[a]	F-value	P>F
Factors				
Malaria parasitemia	1	258.95	0.61	0.44
Hookworm	1	115.99	0.27	0.60
Strongyloides	1	112.98	0.27	0.61
G. lamblia	1	310.78	0.73	0.40
Treatment 1983	1	7.18	0.02	0.90
Covariates				
Age in age classes[b]	2	9368.18	11.05	0.0001
S. haematobium	1	309.64	0.73	0.40

Table 10 C.

I: Model

Dependent variable: height gain (%)

	DF	Sum of squares	F-value	P>F
Model	8	2063.44	22.78	0.0001
Error	73	826.57		
Corrected total	81	2890.01		

II: Source

Independent variable

	DF	Type III SS[a]	F-value	P>F
Factors				
Malaria parasitemia	1	1.00	0.09	0.77
Hookworm	1	56.15	4.96	0.03
Strongyloides	1	20.58	1.82	0.18
G. lamblia	1	13.32	1.18	0.28
Treatment 1983	1	7.35	0.65	0.42
Covariates				
Age in age classes[b]	1	1382.36	61.04	0.0001
S. haematobium	1	0.36	0.03	0.86

earlier studies where single and repeated samples were analyzed and/or MIF fixation was used (Burke, 1977; Kamath and Murugasu, 1974; Craft, 1982). Besides this predictive inaccuracy, MIF also mainly preserves cysts. Thus, the method does hardly detect people who are suffering from an active *G. lamblia* infection and are shedding vegetative forms. In view of this, *G. lamblia* is to be considered highly endemic among children of Kikwawila village (cf. Fig. 1).

The overall prevalence of parasitic infections and the parasite species oberved among the children of Kikwawila village is comparable to the patterns commonly found in rural areas of East Africa (Kloos et al., 1981; Chunge et al., 1985) and Tanzania in particular (Burgess et al., 1969; Kihamia, 1981; Prag et al., 1983). The high prevalence (20%) of *Strongyloides* established through one coproculture was striking. *Strongyloides* prevalences over 15% have so far only been reported from northern parts of Tanzania (reviewed by Kihamia, 1981). The study confirmed the opinion of Medical Officers of Ifakara and the results of investigations at the St. Francis District Hospital, Ifakara (Robyn, 1986; STIFL, 1986), that infections with *E. histolytica* are of comparatively low importance in the Ifakara area. This is in contrast to the data from the Kilimanjaro Region (N-Tanzania) where amebiasis is very common (Timmermanns, 1981). Hookworm (>98% confirmed *N. americanus* based on examination of filariform larvae from coproculture) was highly prevalent among all children (1 month to 15 years). The intensity of hookworm infections was very low (cf. Table 3); this may reflect the seasonality of transmission which can be explained by the climate and soil conditions prevailing in Kikwawila (Tanner et al., 1987; Zehnder et al., 1986). The absence of a relationship between hookworm infection and the packed cell volume (PCV, haematocrit) can be explained by the very low average intensity of infection and the parasite species. *Necator americanus* consumes approximately 5 times less blood than *A. duodenale* (Roche and Layrisse, 1966) and hookworm-related anemia is observed mainly in individuals more heavily parasitized than these in our community (Udonsi, 1984; reviewed by Variyam and Banwell, 1982). No difference in the parasite load between males and females was observed in this study, although sex differences had been reported from various endemic areas (Nawalinski et al., 1978; Schad and Anderson, 1985).

As shown in previous studies (Zumstein, 1983), urinary schistosomiasis is highly endemic in the Ifakara area and the morbidity caused is important; i. e. a high frequency of haematuria and proteinuria and a substantial degree of kidney and bladder pathology was observed (Tanner et al., 1983; Degrémont et al., 1985; Burki et al., 1986; Table 3 A). The data on the *S. haematobium* intensities can only be compared with those from other endemic areas with some reservation, since urine was collected from 9–12 a.m. which can be suboptimal for maximum egg recovery from the urine (Jordan, 1982). The considerable prevalence of *S. haematobium* among under-fives and the lack of any sex-specific infection pattern among all age groups suggest that *S. haematobium* transmis-

sion takes place within the village area. Three quarters of all *S. haematobium* infections were found among children living in the Kikwawila sector. These findings on the possible sites of transmission were confirmed by studies on intermediate host population dynamics (Marti et al., 1985), transmission patterns (Suter, 1986) and human water contact activities (Marti and Tanner, 1982; Lwihula, 1985). The transmission was found to be highly seasonal (Suter, 1986; Lwihula, 1985).

S. mansoni was not prevalent among the Kikwawila community and its intermediate hosts were not found in the rivers, ponds and swamps of the Kikwawila village area (Zumstein, 1983; Marti et al., 1985).

Malaria was hyperendemic (WHO classification, Bruce-Chwatt, 1980), and was the most important parasitic infection in Kikwawila (Table 4). This finding is in accordance with earlier studies on malaria in the Kilombero valley (Clyde, 1967; Freyvogel and Kihaule, 1968). Transmission occurs during 8–9 months as revealed by entomological investigations in Kikwawila (Biro, 1987). The population is exposed to a high number of infectious bites during the transmission period (up to 3 infectious bites/man and night, Biro, 1987) which leads to a rapid development of semi-immunity with age (Table 4) and the high frequency of anti-sporozoite antibodies in the community. These antibodies are detected already in young children and were also shown to be related to resistance to infection (Tanner et al., 1986 b; Del Giudice et al., 1987). However, the substantial change of the malariometric parameters among the cohort of children studied (Table 4) despite the high and stable transmission observed in the area cannot exclusively be attributed to the development of semi-immunity. Regular malaria treatment given at village health posts probably also played an important role. From 1982 onwards, two village health posts were operating within the area (STIFL/DHO, 1985). According to our records, the village health posts of Kikwawila and Kapolo distributed an average of 3000 tablets of chloroquine per month (STIFL/DHO, 1985) and there was hardly any drug shortage until the end of 1984. However, the consumption pattern of these drugs could not be evaluated.

Malaria has a potential for influencing the nutritional status (reviewed by McGregor, 1982) and may have, directly or indirectly, probably together with the less frequent episodes of gastroenteritis (Degrémont et al., 1987), contributed to the high proportion of growth retardation observed among the children of this community (Table 7, Fig. 3). It becomes especially important among young children who are going through the critical weaning period and who have not yet established semi-immunity to malaria (cf. Table 4), and thus have higher and more frequent parasitemia. The trend showing a higher proportion of young children (0–5 years) with growth retardation in this community (Table 7) is consistent with such a relationship. Due to the high endemicity of malaria among the whole cohort of children it was not possible to unravel the influence of malaria on the observed haematological variables such as PCV (Fig. 4) or

serum protein concentrations (Betschart et al., 1987). The PCV measured in this study are substantially higher than values reported earlier from other malaria-endemic areas of central and coastal Tanzania (Sturrock, 1966; Burgess et al., 1968, 1969).

The incidences calculated for each of the recorded parasitic infections and for under-fives as well as for older children showed an increasing risk of infection from 1982 to 1984 (Table 6, Fig. 2). This trend was mainly found among older (6–15 years) children where all RR_i were >1, and was most clearly expressed for *G. lamblia* (2.3) and *E. histolytica* (3.3). These data also indicate that multiparasitism was very common in Kikwawila and Kapolo (Table 5) and that no child was found parasite-free for three consecutive years. The variety of risks faced by the community which also reflects changing patterns of transmission for each of the parasitoses (except malaria, see above) make it difficult to trace the effects of a single parasite or even a single nutrient among these children. Even parasite specific treatment effects (see below) are complex and difficult to unravel in such a multiparasitized community, that also faces non-parasite-related gastroenteritis, additional communicable diseases and other health problems (Degrémont et al., 1987) as well as deficiencies in food production and consumption (Zehnder et al., 1986, 1987; Tanner and Lukmanji, 1987).

The assessment of the nutritional status showed a high prevalence of stunting (27–71%), reflecting past malnutrition (Waterlow, 1972), during all three years while wasting was only observed to any great extent (9–20%) in 1982. Our results compare well with the comprehensive data of a cross-sectional anthropometric survey among Tanzanian children (Kimati and Scrimshaw, 1985). These authors reported a mean prevalence of 47% for stunting in all Tanzanian regions (Morogoro: 67%) and 8% for wasting (Morogoro: 4%).

Kimati and Scrimshaw (1985) also stressed the fact that despite the steady improvement of health services during the last 15 years, the nutritional status of Tanzanian children had not significantly improved. The growth potential (Table 8) did not change during the study period and was found to be normal. The growth retardation which seems to occur early in life (s. above, Table 7) did not affect the growth potential. This was also observed among children from an urban area of Dar-es-Salaam (Davies et al., 1974). The trend of the anthropometric data (stunting, wasting), which showed an improvement in 1983 and a reversal to the level of 1982 in 1984 (stunting) is well paralleled by the frequency of nutritional deficiencies shown by the condition of the hair and skin observed during the clinical investigation (Degrémont et al., 1987).

The health interventions undertaken during the present study were of an integrated nature. Firstly, the community was engaged in a latrine campaign led by the District Health Office and the village health workers. Secondly, when in 1983 as a result of the campaign 80% of the households had simple pit latrines (initially 57%, census 1982 cf. Tanner et al., 1987), mass treatment with ornidazole and albendazole was offered to the community (see above). As outlined

earlier, these interventions were based on the results of the first health status survey (Figs. 1, 2), and respected the health problems and felt needs of the community (Tanner et al., 1986a; Lwihula, 1985; Degrémont et al., 1987). The evaluation of the campaign was done by the subsequent health status surveys six and 18 months afterwards. It was not intended to evaluate the efficacy of the drugs. Ornidazole is effective as a single oral dose of 40 mg/kg against *G. lamblia* and anaerobic bacteria without substantial side-effects (Iyngkaran et al., 1978; Lasserre, 1978; McLean et al., 1984). *E. histolytica* infections are only partly affected by a single dose treatment (Degrémont et al., 1981). Albendazole is an anthelmintic of broad spectrum and was shown to be highly effective (cure rate >90%) against hookworm infections when used as a single oral dose of 400 mg (Pène et al., 1982; Rossignol and Maisonneuve, 1983). The drug also acts against pre-intestinal stages of *N. americanus* (Cline et al. 1984) and it exerts an ovicidal activity against hookworm eggs (Maisonneuve and Rossignol, 1985).

When ornidazole (Kapolo, compliance rate 49%) and ornidazole plus albendazole (Kikwawila, compliance rate 62%) were used for mass treatment (children+adults) on a community basis following the latrine campaign, a reduced prevalence and incidence was observed only for *G. lamblia* among the ornidazole treated children six months later (Fig. 5). The treatment effect of ornidazole was not sustained as reflected by the *G. lamblia* prevalence 1984 and the incidence 1983/84 which reached pretreatment levels again (Fig. 5). The incubation period of *G. lamblia* is short (1–2 weeks) and immunity to re-infection does not seem to be fully active (reviewed by Craft, 1982; Stevens, 1985). Consideration of these facts explains why the treatment effect could not be fully assessed after 6 and 18 months.

The prevalence and incidence of hookworm were hardly affected by the treatment with albendazole. However, the intensity of hookworm infection was significantly reduced (6 months) after treatment, and the few heavy infections were virtually eliminated. The prevalence of hookworm did not increase among treated individuals from 1983 to 1984, whereas a substantial increase was observed among untreated children. In addition, the incidence for the period 1983–1984 was significantly lower in treated subjects, i. e. the risk of acquiring a new hookworm infection among treated individuals was only half that for untreated ones ($RR_i = 0.53$, Fig. 6). This phenomenon, which was seen particularly clearly in the Kikwawila sector because of the predominance of hookworm infection there, raises interesting questions about hookworm transmission in the community. It may be suggested that transmission of hookworm mainly occurs at the household level, since no increase of prevalence of those treated was observed despite the high incidence among untreated individuals of the same community who also showed a substantial increase of prevalence. These findings and the recently described indication that the majority of hookworms occur in small fractions of the population, which reflects predisposition to an

infection (Schad and Anderson, 1985), imply that selective treatment would be appropriate. It remains to be established whether targeted treatment at household level, e.g. by village health workers, could be more effective than a mass treatment strategy in a community with epidemiological features like those of Kikwawila.

There were no differences among treated and untreated children with regard to overall weight and height gain for either period 1982 to 1983 or 1983 to 1984. These findings were supported by our models of analysis of covariance where there was no obvious effect of the treatment campaign and no single parasite effect emerged (Tables 10 A–C). However, detection of single parasite effects was rendered difficult as the independent variables are considerably correlated and produced collinearity of the model; e.g. most parasitic infections are correlated with age and therefore their influence can hardly be unravelled from the influence of age. It is important to note that the models of analysis of covariance could not really take into account the changing patterns of multiparasitism. Furthermore, only 18 months of follow-up after the mass treatment were analysed which may be sub-optimal for an impact assessment using anthropometrical parameters.

The pathological changes in the small intestine and the pathogenesis of malabsorption following *G. lamblia* infections have been well established and analyzed (reviewed by Brasitus, 1983; Gillon, 1984). However, the effect of *G. lamblia* on the growth of children under field conditions is not clear and so far only conflicting results are available (see reviews Solomons, 1982; Stevens, 1985). Gupta (1980) and Gupta and Urrutia (1982) reported a significantly higher, although minimal, height gain among Guatemalan preschool children treated with metronidazole. Kay et al. (1977) demonstrated significant catch-up growth among *G. lamblia* infected Australian children following metronidazole treatment. Similar effects have also been described in animal models (Roberts-Thomson et al., 1976). Loewenson et al. (1986) described an association of *G. lamblia* infection and undernutrition among schoolchildren in Zimbabwe. Such single parasite effects of *G. lamblia* could not be demonstrated in our study: Owing to the high degree of multiparasitism in our study community and the lack of adequate controls due to the fact that only 10% of individuals were parasite-free (cf. Table 5), the effect of one parasite, e.g. *G. lamblia,* is difficult to separate from those of other parasites and/or concurrent microbial infections on nutrition. This is a situation commonly found in epidemiological studies in endemic areas (Buck et al., 1978a, b, c). This and the very low hookworm intensity also may explain why the single antihelminthic treatment of our study did not exert a measurable effect either on anthropometric variables or on haematocrit values.

Meakins et al. (1981) could only observe a slight association of parasitic infections with malnutrition among Tanzanian children by combining the prevalence of all helminth infestations. Effects on nutritional status have, however,

been found by other workers, for example Haller and Lauber (1980) reported an improved growth of schoolchildren who had been treated four times per year with mebendazole and levamisole against intestinal helminths. This treatment also led to a significant increase in serum vitamin levels (vitamin C, carotenes), but the improvement of nutritional parameters was not reflected in the haematological profiles of treated children (Haller and Lauber, 1980).

Stephenson et al. (1985b), faced with a multiparasite system in Kenya (*S. haematobium,* hookworm, malaria), performed stepwise multiple regression analysis and showed improved child growth six months after treatment of *S. haematobium.* The significance of such findings for a sustained better growth of children and for control measures against these complex host-parasite interactions still remain to be established.

The data presented in the present study were collected only once per year, i.e. in October during the post-harvest period towards the end of the dry season. Thus, we lack information with regard to the impact of seasonal variations of transmission on nutritional parameters. Malaria (Tanner et al., 1986b; Del Giudice et al., 1987; Biro, 1987) and urinary schistosomiasis (Marti et al., 1985; Suter, 1986) show striking variations between households and a distinct seasonality in the study area. On the other hand, the seasonality of hookworm, intestinal protozoa and of the pattern of multiparasitism has not been investigated during the first years of the study. However, these data might be essential for the evaluation of single/multiple parasite effects and for the interpretation of the significance of the variations seen at the level of nutritional and biochemical parameters (Table 7; Betschart et al., 1987; Stürchler et al., 1987, this volume) as well as of seasonal variations in food consumption patterns and food production (Tanner and Lukmanji, 1987; Zehnder et al., 1987, this volume) in Kikwawila village.

The parasitological and nutritional variables from the first three years of the present community-based study showed that the effects of the latrine campaign followed by a single mass treatment were mainly transient (Figs. 5, 6, 8–10). The continuing cross-sectional surveys will help to evaluate the long-term effects of the interventions; i.e. if and how the trend towards a better nutritional status, which is indicated by anthropometric variables, and which may be partly attributed to the combined interventions (cf. Fig. 7, Table 7, and Degrémont et al., 1987, this volume), can be consolidated for the benefit of child health in Kikwawila village.

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