Also would you ask the children in your school if they know of any other children of school age who are lame or cannot walk and *do not attend school* and would you also list these children on the accompanying form.

I or my assistant will come by shortly to visit you, to pick up the list, and to examine these lame children. Please hold this list until our arrival.

Thank you for your co-operation.

Sincerely,
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Department of Community Health,
Ghana Medical School,
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Accra.

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Is poliomyelitis a serious problem in developing countries? - lameness in Ghanaian schools

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Summary

A postal survey of lameness in schools throughout Ghana showed an estimated prevalence of lameness attributable to poliomyelitis of 5·8 per 1000 school-aged children and an estimated mean annual incidence of paralytic poliomyelitis of 23 per 100 000 population. Official reported incidence rates range from 0·1 to 2·1 per 100 000 population, indicating that at least 90% of cases are not reported. No evidence of epidemics was found to account for these high rates. These suggest that mean annual incidence rates in tropical endemic countries have always been as great, if not greater, than those experienced by temperate countries during epidemic periods in the twentieth century and that the total number of cases of paralytic poliomyelitis occurring in the world each year has been reduced by only 25% since the advent of polio vaccine. Immunisation against poliomyelitis must have a high priority in Ghana and other tropical countries where the disease is endemic.

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Introduction

Serological surveys have shown that poliomyelitis is endemic in Ghana.[1 2] Low annual incidence rates of 0·1 to 2·1 (mean 1·0) per 100 000 population are reported for paralytic poliomyelitis.[3] No epidemics have ever been recorded. Yet Nicholas and co-workers[4] found a high prevalence of lameness attributable to poliomyelitis (seven per 1000 children aged 6 to 15) in the Danfa Project district of rural Ghana, implying a mean annual incidence of at least 28 per 100 000 population. This supported a suspicion first raised by Paul[5] from studies in Egypt that the mean annual incidence rates in endemic countries might be as high as those in countries experiencing epidemics.

Lameness carries a high social cost. If the prevalence rates throughout Ghana were similar to those in the Danfa area it would mean that immunisation against poliomyelitis should be given a higher priority. During the Danfa study[4] it was found that headteachers were reliable in reporting cases of lameness, and that the prevalence of lameness attributable to poliomyelitis could be estimated from the prevalence of reported cases of lameness due to any cause. We report here the results of a postal survey of lameness in a sample of schools throughout Ghana using a teacher questionnaire that was tested and validated during the Danfa study.

Population and methods

After permission was obtained from the Ministry of Education a questionnaire and a stamped addressed envelope was sent to the headteacher of each school in the sample.

Questionnaire—This requested the school's enrolment and the name, current age, age at onset of lameness, residence at onset, and present class in school of each lame child.

Sampling frame—This was the official 1971 list of all primary and middle schools in Ghana. Schools exist in all districts. Nevertheless, to avoid a bias towards the urban areas, whose population has greater access to schools, a random sample of schools was drawn that was stratified and self-weighted according to the school-age population by region and urban/rural classification of town or village using information from the 1970 national census. Settlements with less than 5000 population were classed as rural. Of children attending primary or middle schools 99% are aged from 6 to 19. Based on confidence intervals for the range of prevalence that might be expected, we calculated a target sample size of 510 schools with an expected enrolment of 60 000. This was a 5% sample of all schools in Ghana.

Results

In the first instance 377 (73·9%) schools responded. After reminders were sent, another 100 schools responded improving the response rate to 93·5%. The total enrolment of schools returning questionnaires was 74 609.

The prevalence of lameness, standardised for school-aged population by strata, was 7·8 per 1000 school children (table I). There were no significant regional differences. The difference in urban and rural prevalence rates was statistically significant ($P < 0.01$). This difference, however, was due primarily to the low urban primary school rate.

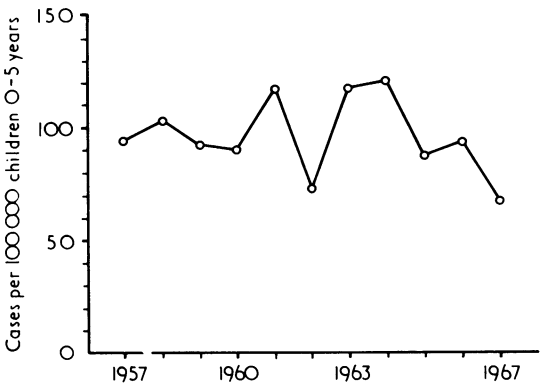
TABLE I—*Prevalence of lameness in Ghanaian schools*

	School enrolment	Cases	Prevalence ± SE* per 1000	Standardised† Prevalence ± SE‡
Urban primary ..	19030	84	4·41 ± 0·43	4·40 ± 0·41
Urban middle ..	4981	39	7·83 ± 1·32	8·16 ± 1·80
Total ..	24011	123	5·12 ± 0·46	5·62 ± 0·69
Rural primary ..	36559	281	7·69 ± 0·64	8·06 ± 0·70
Rural middle ..	14039	136	9·69 ± 1·17	9·85 ± 1·27
Total ..	50598	417	8·24 ± 0·57	8·73 ± 0·63
Grand total ..	74609	540	7·24 ± 0·42	7·83 ± 0·49

*SE (standard error) of a proportion for a random cluster sample with unequal cluster size.[11]
†Standardised for school age population by strata (region, primary/middle school age, urban/rural classification of town or village).
‡SE (standard error) of a proportion for a stratified cluster sample with unequal cluster size.[11]

The median age of onset for cases with onset of lameness under 6 years was 21 months (table II). After adjusting for the male : female school enrolment, the male : female ratio of cases with onset under 6 years was 1·65, a male predominance that has been noted in other studies.

Calendar year of onset was calculated as follows: 1973 minus current age plus age of onset. The figure shows the trend of age-adjusted incidence rates for the years 1957 to 1967. No epidemic trend is noticed nor do any of the rates exceed two standard deviations of the expected rate based on a constant annual risk to children under 6. Because of some imprecision in reporting current age or age at onset, somewhat greater fluctuations in annual incidence may have been obscured.



Age adjusted incidence rates of paralytic poliomyelitis based on histories from Ghanaian school study.

An estimate of the prevalence ($\hat{P}r$) of lameness attributable to poliomyelitis for school aged children may be calculated using the same sensitivity (S_1) and specificity (S_2) derived during the Danfa Study[4 6]

$$\hat{P}r = \frac{\hat{P}rr + S_2 - 1}{S_1 + S_2 - 1}$$

where $\hat{P}rr$ (prevalence of all cases of lameness for school aged children) $= 0.00783$ (see table I for standardised prevalence of all cases of lameness in school children), $S_1 = 0.831$, $S_2 = 0.997$, and the standard error (SE) of $\hat{P}r = \frac{\text{SE of } \hat{P}rr}{S_1 + S_2 - 1}$. The estimated prevalence of lameness attributable to poliomyelitis ($\hat{P}r$) for all of Ghana was 5·8 (SE = 0·59) per 1000 school-aged children standardised for school-aged population by strata. The standard error was somewhat underestimated due to S_1 and S_2 being assumed constant. The prevalence for rural areas was 6·9 per 1000 school-aged children, a rate similar to that found by examining the school children in the Danfa district (7·2 per 1000).[4] Note that the prevalence is better expressed per 1000 *school-aged* children rather than per 1000 *schoolchildren*. The assumption that the prevalence of lameness due to poliomyelitis in schoolchildren is similar to that in non-school children is an important one and is supported by the Danfa study.[4]

The estimated prevalence reported here depends to a large extent on whether the sensitivity (S_1) and specificity (S_2) of headteacher performance in this study were similar to that in the Danfa study. It seems reasonable to assume so for three reasons. Firstly, in other studies done in tropical developing countries poliomyelitis is by far the leading cause of lameness.[5 7] Secondly, to a certain extent headteachers are randomly assigned to schools in Ghana, which diminishes the bias of the Danfa sample. Thirdly, after completing this study we examined children in survey schools in urban Accra. We found the S_1 of headteacher performance there to be 0·765, and the S_2 to be 0·998. Ideally, one would like to determine the S_1 and S_2 for a random sample of each population studied. This, however, may need extensive resources.

Discussion

These results confirm the findings of the Danfa study[4] and indicate that the prevalence of lameness attributable to poliomyelitis is high throughout Ghana.

The lower urban rate was due to a lower prevalence among urban primary school children. This appears to be a recent development since urban middle schools have a rate similar to rural primary or rural middle schools. The reason for this change

TABLE II—*Age of onset of all cases of lameness**

Age group (months)	<12	12-	24-	36-	48-	>59
No (%) of cases	143 (28·0)	91 (17·8)	86 (16·9)	44 (8·6)	22 (4·3)	124 (24·3)
Cumulative percentage	28·0	45·8	62·7	71·3	75·6	100

*For cases where age of onset was reported.

is not clear. It seems unlikely that the prevalence of wild poliovirus has diminished, resulting in the gradual accumulation of susceptibles, since Pasca and Afoakwa[1] recently found that in Accra 86, 88, and 93% of children had antibodies to poliovirus types 1, 2, and 3 respectively by 4 to 6 years of age. Another explanation may be the more widespread use of polio vaccine in urban areas in the past 10 years. It is estimated that up to 20% of children in Accra may have received oral polio vaccine. Faecal contamination could have spread the vaccine virus to other children.

The age group 0-4 years represents 20% of the total population in Ghana. Given a prevalence of 5·8 per 1000 school-aged children by age 5, there must be about 2100 new cases of paralytic poliomyelitis each year, an incidence of 116 per 100 000 children 0-4 years or 23 per 100 000 population. If those who had fatal cases or recovered completely are included the incidence rate would be even higher. Why is this incidence so much higher than that previously presumed for Ghana and other endemic countries?

The hypothesis most commonly proposed to explain the low incidence rates reported from these countries is that children infected early in life are partially protected by maternal antibodies, breastmilk factors, or other age-related mechanisms. In 1955 Sabin[8] presented strong arguments refuting this hypothesis and suggested instead that the virulence of the wild poliovirus may have been low in the past in endemic countries but may have increased in recent years in those countries experiencing epidemics. Although the severity of some epidemics may be related to the virulence of the prevalent poliovirus, there is little epidemiological evidence to support this hypothesis as the explanation for the low rates reported previously from endemic countries, and it is a difficult one to test.

Much less attention has been paid to the hypothesis that cases from tropical developing countries may be unrecognised and seriously underreported. The association that Paul[9] and Cockburn[10] found between decreasing infant mortality rates and increased reporting of poliomyelitis may be due much more often to the better reporting of all diseases that accompanies the improved living standards rather than to an actual increase in the disease. We have shown that at least 90% of cases of paralytic poliomyelitis in Ghana are not reported.

It does not follow that when a disease becomes epidemic its incidence must be greater than during a period when it was endemic. The cumulative incidence of new cases during a period in which epidemics occur intermittently may be the same or even less than during a period of sustained endemicity. The mean annual age adjusted incidence rate in Ghana for the period 1957-67 as determined by this study was 22 per 100 000 population. In the USA for the period 1944-54 the rate was 20 per 100 000.

We therefore suggest that the mean annual incidence rates of paralytic poliomyelitis in tropical endemic countries have always been as great, or even greater, than those experienced by temperate countries during epidemic periods in the twentieth century and that the problem was not recognised and reported. If this is true we estimate that the total number of cases of paralytic poliomyelitis occurring in the world each year has been reduced by only 25% since the advent of polio vaccine. We recommend that vaccination against poliomyelitis should be given a high priority in tropical developing countries and that similar studies be carried out in other countries to test our hypothesis. We found the teacher questionnaire to be valuable for estimating the impact of poliomyelitis in Ghana. We think it may also be used as one method of evaluating the success of the immunisation effort.

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Is retinoblastoma inherited?

Retinoblastoma, which is inherited in some instances, is a malignant tumour of the retina affecting infants and young children. It affects one eye in about 25% of cases and is bilateral in about 75%. About 15 to 20% of sporadic unilateral cases are dominant mutants. As the gene has a penetrance of about 80%, 6-8% of the children of patients with sporadic unilateral retinoblastoma will be affected. Probably all sporadic bilateral cases are dominant mutants and, therefore, with a penetrance of 80%, 40% of the children of patients with sporadic bilateral retinoblastoma will be affected. From these figures the risks of more distant relatives of a patient with retinoblastoma inheriting the disease may be calculated, and these calculations have been summarised by Vogel.[1]

[1] Vogel, F, in *Modern Trends in Ophthalmology 4*, ed A Sorsby, p 34. London, Butterworths, 1967.

Sensory fibres for pain and temperature ascend the spinal cord contralaterally, while touch fibres go up the same side. Is vibration sense conducted up different fibres and if so, do they cross in the cord? Why is it advised that a non-hairy part of the skin be used when testing for light touch?

It is true that the sensory fibres for pain and temperature ascend the spinal cord contralaterally. Clinically it is accepted that touch fibres go up the same side There is some argument, however, about this at the highly sophisticated neurophysiological level. Practically it is best to consider that both vibration sense and light touch are conducted in the spinal cord by the posterior columns—cuneate and gracile nuclei. These do not cross in the cord. It is taught, and there is some scientific evidence to suggest, that the touching of a bare area of skin is stimulating a different sensory pathway than when hair is stimulated. The touching of bare skin is a genuine test of feeling light touch. The touching of hair is described as a "tickle sensation." A light touch sensation is thought to be carried in the posterior columns. The tickle sensation may well go up the contralateral spinothalamic tracts. It is difficult, however, to touch hairs without touching the skin as well, which tends to confuse the issue. To test light touch it is therefore better to touch non-hairy portions of the skin, but it is seldom that the difference between the sensation of the two will have a significant clinical importance.

Are chilblains a known side effect of propranolol?

This is not a recognised side effect, although I suspect that there may be a real association. Further evaluation is needed to prove that the chilblains are not a coincidental finding in a patient who is being treated with a beta-blocker. If the association is real then it is uncommon—unlike the well-recognised side effect of cold extremities that affect some individuals on beta-blocker treatment.