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Transmission Patterns and the Epidemiology of Hookworm Infection

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Chan M S (The Wellcome Trust Centre for the Epidemiology of Infectious Disease, Department of Zoology, South Parks Road, Oxford OX1 3PS, UK), Bradley M and Bundy D A P. Transmission patterns and the epidemiology of hookworm infection. *International Journal of Epidemiology* 1997; **26:** 1392–1400.

Background. This paper presents a suite of models of hookworm transmission dynamics which vary the mixing patterns and rates of contamination and infection between children and adults. In this context mixing refers to the degree of epidemiological communication between children and adults, for example, whether adults are likely to get infected from infective material passed by children.

Methods. Three models are described which represent random mixing, no mixing and restricted mixing respectively. Child, adult and population targeted chemotherapy programmes are examined and compared between these models. Data from a hookworm control programme in Zimbabwe were analysed with respect to their fit to the various models.

Results. The analysis suggests that some mixing does occur and that in this study location, the sites where adults deposit faeces are more likely to lead to subsequent contamination than the sites children use.

Conclusions. Mixing patterns may have a profound effect on transmission dynamics and should be considered in relation to design of control programmes.

Keywords: *Necator americanus*, mathematical model, population dynamics, parasite ecology

Hookworm infection is a major disease in many countries of the developing world[1–2] and is an important cause of anaemia[2] in endemic areas. There are two main species which infect humans, *Necator americanus* and *Ancyclostoma duodenale,* and since the distributions overlap many people suffer from mixed infections. As with other intestinal helminths, the distribution of infection and disease is strongly age dependent. However, in contrast to the other common intestinal helminths such as *Ascaris lumbricoides* (large roundworm) and *Trichuris trichiura* (whipworm) and also schistosomes where children are more heavily infected, hookworm is generally more common in adults. This means that the child targeted chemotherapy programmes advocated for the treatment of the other species may be less appropriate in the community control of hookworm disease.

It is generally thought that the differences in levels of hookworm infection in children and adults are due to exposure differences, as hookworm is generally transmitted in the fields as opposed to near houses as are *A. lumbricoides* and *T. trichiura*. However, the impact of a treatment programme which is either directed at the whole population or only at children or adults will also depend on two other factors.

1) The potential contamination from a population group (such as adults or children) depends not only on the egg output of that group but also on its behaviour. In the case of hookworm infection this depends on the defaecation sites of individuals in each group, whether infective stages are likely to survive at the site and whether these sites are likely to lead to further exposure. The general view is that children are more indiscriminate in their defaecation behaviour and may therefore produce more contamination. However, for the study site in Zimbabwe which is analysed in this paper, adults appear to deposit faeces in sites more conducive to larval development.[3]

2) The impact of treatment programmes will depend on the 'mixing patterns' between adults and children. This refers to the relative probabilities of the members of one group being infected with infective stages that originally come from the same or a different group. The influence of such mixing patterns on transmission dynamics of infectious diseases has been well formulated and studied for sexually transmitted diseases.[4] Here we extend this idea to hookworm infection.

In this paper the influence of the above factors on the epidemiology of hookworm infection and the consequences of control programmes will be investigated using mathematical models. Mathematical models for intestinal

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helminth infections have been developed by Anderson.[5] This was formulated for the control situation by Medley *et al.*[6] and age structure in this model was added by Chan *et al.*[7] The aims of the study are to investigate the influence of mixing patterns using this model framework and also to examine the patterns observed in field data.

Field data from a hookworm control programme in Zimbabwe[8] will be compared with the output of the model. This will allow both the estimation of the pattern of mixing occurring in this population and also the magnitude of the differences in contamination potential between adults and children.

CONCEPTUAL FRAMEWORK

In this paper, three types of transmission patterns are investigated which involve three different models as follows:

Model 1. One transmission site (random mixing model). In this model it is assumed that adults and children get infected from the same transmission site and that they are equally likely to be infected with worms from either adults or children. This is equivalent to random mixing in population genetics or epidemiological models of sexually transmitted diseases.

Model 2. Separate transmission sites (no mixing model). This model assumes that children and adults acquire their infections from different sites. This means there are actually two separate transmission systems which are unconnected. This is equivalent to total assortative mixing.

Model 3. Two sites (restricted mixing model). In this case, there are again two sites but one of the groups can use both sites. This means that there is an epidemiological connection between the groups but still a degree of isolation between them. This describes a situation in between the first two models.

The concept of the latter two models are derived from the field situation in the Zimbabwean study site in Burma Valley.[8] Adults in the study villages are employed in banana plantations. However, children do not usually enter these plantations and stay near the houses. The two sites in the models are therefore equivalent to the houses and plantation respectively. This gives a potential for a nonrandom transmission pattern. The models are formulated to describe this situation and are illustrated in Figure 1(a-c).

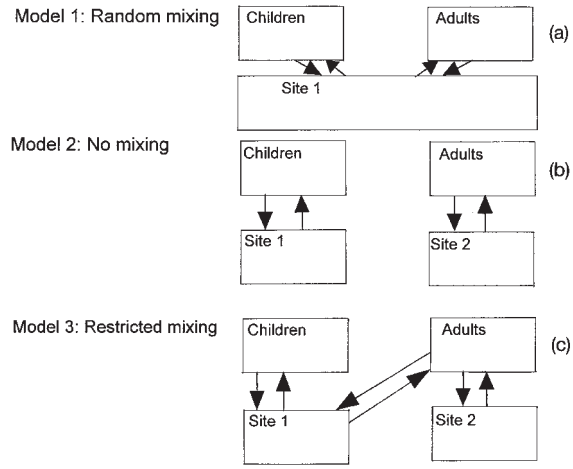

FIGURE 1 *Flow charts to show the mixing structure of the models used. 1a. Random mixing model. 1b. No mixing model. 1c. Restricted mixing model*

MODEL DEVELOPMENT

All the models are based on the differential equation framework of the directly transmitted intestinal helminth model described by Anderson.[5] In general terms, the rate of change of mean worm burden (W) in a population can be described by the following differential equation:

$$\frac{dW}{dt} = \mu R_0 f(W) - \mu W \qquad (1)$$

where μ is the mortality rate of the worms (inverse of the average lifespan), R_0 is the basic reproductive number and $f(W)$ is a density dependent fecundity function.

Heterogeneity in the population in infection rates is included using a framework developed by Medley *et al.*[6] In this model, the population is divided into a number of host types with different rates of infection. These differences may be due to behavioural or immunological factors but the model does not explicitly describe one or the other of these. The negative binomial distribution of worm burden which is usually observed in human communities[9,10] is reconstructed in this framework by the use of a gamma distribution of average infection rate between host types and a Poisson distribution of actual infection rates within each host type. The framework explicitly simulates the changing worm burden distribution across host types during the course of a community chemotherapy programme. Further details of this framework are given in Medley *et al.*[6] It is noted that a much simpler framework using a fixed negative binomial distribution gives virtually identical

projections for mean worm burden.[11,12] In this paper, the more complex framework is used because it is more accurate in predictions of infection prevalence which is the measure used in the field studies.

Age structure has been also added to the framework by Chan *et al.*[7] In this model two fixed age groups are used which represent adults and children respectively. These two groups differ in their contact rates with infection (ρ_C and ρ_A), contamination rates (K_C and K_A) and the proportional representation in the population (π_C and π_A). These differences result in a different mean worm burden at equilibrium (W_C and W_A) and basic reproductive number (R_{0C} and R_{0A}) for worms in each group of hosts. The rate of change of mean worm burden by age group can then be generalized to (using a subscript *i* for the age group under consideration and the subscript j for summation over all age groups):

$$\frac{dW_i}{dt} = \rho_i \mu \frac{\sum_j R_{0j}\pi_j f(W_j)}{\sum_j \rho_j \pi_j} - \mu W_i \qquad (2)$$

The basic reproductive number for each age group refers to the biological definition of this quantity, namely the lifetime number of surviving offspring a worm in a particular host age group produces in the absence of density dependent constraints. Of particular interest to this study is the ratio of this number between different age groups since this gives a measure of the contribution of each age group to the overall transmission. Further details of this model are described by Chan *et al.*[7]

The Chan *et al.*[7] model assumes there is a single transmission site from which individuals get infected randomly with respect to the origin of the infective stages. This model was therefore used as Model 1 in the current paper (random mixing). Two runs of the Medley *et al.*[6] model were used for the two groups to simulate Model 2 (no mixing). For Model 3 in which there are two sites but one group can use two sites (restricted mixing) a new framework was formulated.

The form of Model 3 is shown in Figure 1c. Suppose a proportion *x* of defaecation in adults occurs in the plantation (site 2) and a proportion (*1 – x*) occurs in the housing compound. Children are assumed not to enter the plantation. The model is asymmetrical with respect to the two groups and the rate of change of mean worm burden can be given by the following differential equations:

$$\frac{dW_C}{dt} = \frac{\rho_C \mu R_{0C}(\pi_C f(W_C) + ((1-x)\pi_A f(W_A)))}{\rho_C \pi_C + (1-x)\,\rho_A \pi_A} - \mu W_C \qquad (3)$$

$$\frac{dW_A}{dt} = \mu(R_{0A} - (1-x)R_{0C}) f(W_A) + \frac{(1-x)\rho_A \mu R_{0C}(\pi_C f(W_C) + (1-x)\pi_A f(W_A))}{\rho_C + (1-x)\rho_A \pi_A} - \mu W_A \qquad (4)$$

Note that in this model the difference in reproductive number is due solely to a different survival rate for infective stages in the different sites. The full derivation of the model is shown in the Appendix. Details of the computer programming and implementation of the models are described by Medley *et al.*[6] and Chan *et al.*[7]

MODEL TESTING

The model was tested using data from a hookworm control programme in Zimbabwe. This programme was carried out in the Burma Valley, a commercial farming area on the border between Zimbabwe and Mozambique. Mass chemotherapy programmes were carried out on three occasions at yearly intervals with both adults and children being treated on all occasions.[8] Treatment was with 400mg of albendazole.

The parameters used in the analysis were those obtained in the study and are shown in Table 1. The coverage was set at 60% as estimated from the data. It is assumed to be constant for the model although it did vary between rounds of treatment in the field. Some explanation is required however about how the initial endemic situation was set. Firstly, prevalence measures had to be rescaled because reported prevalence values measure egg prevalence (prevalence of individuals passing eggs in their faeces) whereas the model calculates worm prevalence (the true prevalence of parasites in the community). The reported prevalence has to be corrected to include infections with worms of the same sex (only mated female hookworms produce eggs) by use of a model developed by Guyatt[13] which assumes a negative binomial distribution for the worms. Secondly, since worm expulsion data are not available from this study site the prevalence measures were used for comparing the model with data. It is, however, important to use a reasonable initial mean worm burden to run the model since the prevalence is a relatively insensitive measure of worm intensity. The initial mean worm burdens were therefore obtained from another study in a nearby area[14] where the age prevalence curves and mean egg count are similar. However, the actual prevalence values at the study site were used and the *k* value of the negative binomial was fitted for each age group from the prevalence and mean worm burden. For the restricted mixing model the value of *x* was set at 0.7

TABLE 1 *Parameter values used in analysis*

Parameter	Symbol	Children	Adults	Source
Mean worm burden	W	5	8	Bradley *et al.* 1993
Initial egg prevalence	P_e (%)	56	72	Bradley *et al.* 1993
Initial worm prevalence	P_w (%)			
	using $k = 0.34$	71	81	Bradley *et al.* 1993; Guyatt 1992
Proportion of population	π	0.5	0.5	Bradley *et al.* 1993
Worm lifespan	$1/\mu$	2.5	2.5	Anderson & May, 1991
Basic reproductive number	R_0 (overall)	2		Bradley *et al.* 1993
Treatment coverage	c (%)	60	60	Bradley *et al.* 1993
Treatment schedule		years 1,2,3	years 1,2,3	Bradley *et al.* 1993
Proportion of contacts in site 2	x (model 3 only)	0	0.7	Bradley, unpubl

from observations made at the study site (Bradley, unpublished data).

The models were first used to look at the consequences of different (hypothetical) treatment strategies. For each model, simulations were run for the treatment of children only, adults only or both children and adults. In all other respects the parameters used were the same as in the study site. The trajectories predicted by these different simulations can then be compared. The mean reduction in worm burden for adults and children over the time of the simulation was also calculated and compared between the different simulations. The ratio of R_{0A}/R_{0C} was kept at 2.0 for these simulations which means that the contamination rate for adults is twice that for children. This value was chosen because it is thought that the ratio should be greater than one because of the higher levels of infection in adults and a conservative value was used to avoid loss of generality. If this ratio is higher, similar qualitative patterns would be observed although if $R_{0C} < 1$, infection in children can only be sustained through mixing and the 'no mixing' model will not be valid.

The field data were used to ascertain the type of mixing pattern present in the study site and estimate the relative contamination rates of adults and children. This was done by varying these parameters and comparing the model output to the observed data. A maximum likelihood analysis was carried out to determine which of the simulations best fits the observed data. Prevalences are assumed to be binomially distributed. For every data point i, we observe s_i individuals with worms and d_i individuals without worms. Using the expected value for the proportion with worms calculated from the model, p_i, the log-likelihood function, L, is calculated as follows:

$$L = \sum_i [d_i \ln(1 - p_i) + s_i \ln(p_i)] \quad (5)$$

The higher the value the better the fit. Ninety-five per cent confidence intervals are calculated for the observed values using a Normal approximation.

RESULTS

The results of the simulations for the treatment of children only are shown in Figure 2 and Table 2. For the random mixing model there is a moderate reduction in mean worm burden in children and also a small reduction in adults due solely to the reduction in transmission. For the no mixing model, there is a much larger reduction in child worm burden and hardly any reinfection during and after the programme finishes. In this case there is, by definition, no change in the adult worm burden. The overall reduction in worm burden (average for children and adults) is slightly greater for the no mixing case. For the restricted mixing model, there is virtually no reduction in mean worm burden in the adults since transmission continues to occur at the second site. Furthermore, the reduction for children is considerably less than for the no mixing model due to the continued presence of heavily infected adults. Therefore, this model shows the lowest overall reduction in mean worm burden.

Figure 3 and Table 2 show the results for the treatment of adults only. Note that the adult group has both higher initial worm burdens than the child group and higher contamination rates in these simulations. This means that the impact of treating adults with the same coverage will lead to greater reductions in overall mean worm burden (Table 2). Also, there is much less variation in the trajectory of the mean worm burden curve for adults between the models indicating the lesser epidemiological importance of children in this case. In the random mixing case there is a small reduction in the worm burden of children and the overall reduction is

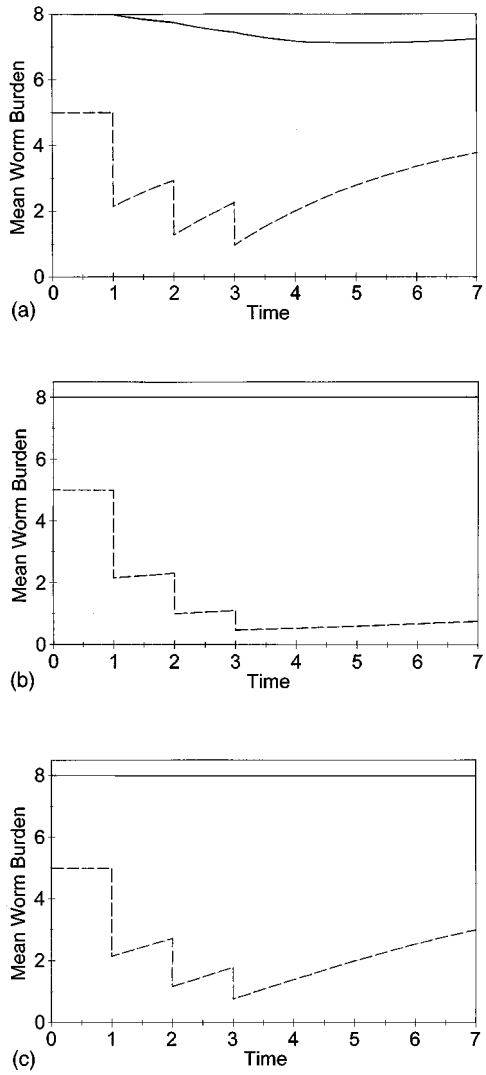

FIGURE 2 *Results of simulation of a hookworm control programme treating children only. The mean worm burden for each group (adults solid line, children dotted line) is plotted over time. 2a. Random mixing model. 2b. No mixing model. 2c. Restricted mixing model*

greater for this case compared with the no mixing case. The restricted mixing model gives the highest reduction. In general, with targeted treatment, mixing slightly increases the reduction in overall worm burden due to the transmission reduction for the untreated group.

The results for the treatment of both adults and children are shown in Figure 4 and Table 2. This is the same treatment programme used in the actual field study site.

TABLE 2 *Reductions in mean worm burden calculated from simulations*

Model	Treated population	Reduction in mean worm burden		
		Children	Adults	All
Random	Children	2.147	0.537	1.342
Random	Adults	0.834	4.098	2.466
Random	All	3.004	4.806	3.905
No mixing	Children	3.472	0.000	1.736
No mixing	Adults	0.000	4.333	2.167
No mixing	All	3.472	4.333	3.903
Restricted mixing	Children	2.609	0.006	1.308
Restricted mixing	Adults	0.441	4.681	2.561
Restricted mixing	All	3.074	4.691	3.882

The treatment of both groups leads to an increased reduction in overall mean worm burden as expected. For the no mixing model, it is exactly the sum of the child and adult targeted treatment programmes and for the other models it is slightly higher due to reductions in overall transmission. If the random mixing and no mixing models are compared, the overall reduction in mean worm burden is almost exactly equal. However, when there is no mixing, the reduction in children is larger and the reduction in adults is smaller. This is because of the greater epidemiological importance of adults. In the presence of adults the child group experiences higher infection rates. The restricted mixing model shows an intermediate transmission pattern.

The results of the maximum likelihood analysis comparing the observed data with the different simulations are shown in Figure 5. Not all the parameter combinations lead to endemic infections in both children and adults in the no mixing and restricted mixing models. Figure 5 clearly shows that higher likelihood values are obtained when the reproductive number of worms in adults is higher than that in children. In this area of parameter space ($R_{0A}/R_{0C} = 5$, $R_{0A}/R_{0C} = 10$), an endemic infection in children is not sustained with the no mixing model. This means that mixing does occur and that the endemic infection in children is sustained solely through mixing with adults (the reproductive rate in children is less than 1). Of the two other models, the random mixing model shows a very slightly higher likelihood value than the two site restricted mixing model. Studies of larval counts in soil at another site in Zimbabwe would indicate that a restricted mixing model with $R_{0A}/R_{0C} = 10$ would be most appropriate which is consistent with the above result.[3]

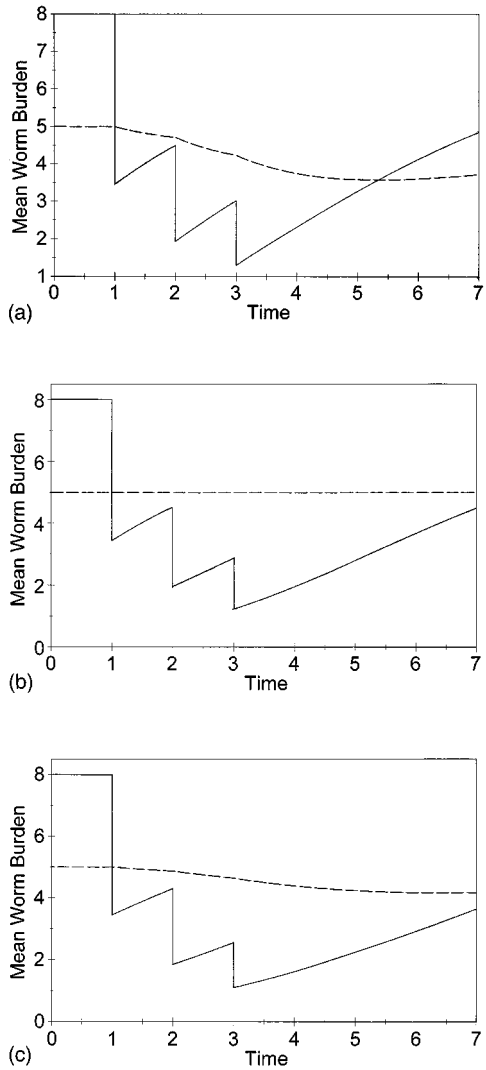

FIGURE 3 *Results of simulation of a hookworm control programme treating adults only. The mean worm burden for each group (adults solid line, children dotted line) is plotted over time. 3a. Random mixing model. 3b. No mixing model. 3c. Restricted mixing model*

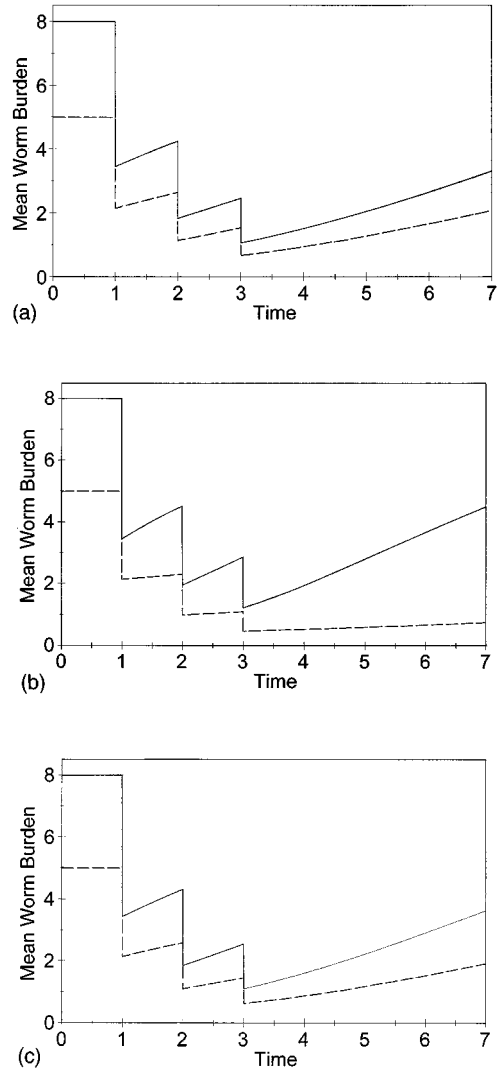

FIGURE 4 *Results of simulation of a hookworm control programme treating children and adults. The mean worm burden for each group (adults solid line, children dotted line) is plotted over time. 4a. Random mixing model. 4b. No mixing model. 4c. Restricted mixing model*

The results are more clearly examined in Figure 6 which compares the results of the model simulations with the actual data. The maximum likelihood fitted model (random mixing, R_{0A}/R_{0C} = 10) is shown in Figure 6a. The model and data fit very well with the model predictions generally falling within the confidence limits of the data. The discrepancies observed could be due to variable coverages for the different treatments. The fitted model can be compared to a variety of different models. Figure 6b shows the model with equal reproductive rates in adults and children. The model overpredicts the reduction in prevalence achieved by the control programme because the overall transmission rate is lower (the contamination by adults, who have more worms has been reduced). In the absence of mixing (Figure 6c) the prevalence in children

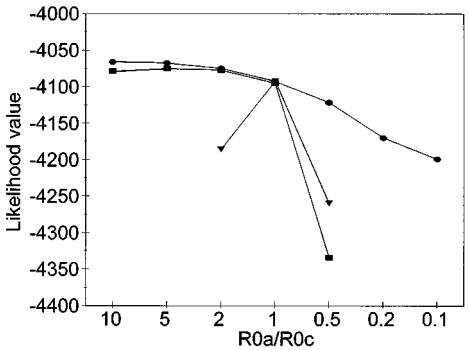

FIGURE 5 *Maximum likelihood values for the comparison of the field data with the model. The likelihood value is plotted against the ratio of reproductive numbers in adults and children for the three models, random mixing (circle), no mixing (triangle) and restricted mixing (square)*

is underestimated and that in adults is overestimated. This is due to the separation of two groups with different transmission rates. The two site restricted mixing model shows an intermediate situation between the random and no mixing models (Figure 6d) and also fits the data reasonably well.

DISCUSSION

In the models described in this paper fixed age groups were used. This means ageing of individuals (passing from child to adult age groups) is not taken into account. This assumption is justifiable when the rate of turnover of worms is high, such that the actual population of worms is continually changing and the rate of worms passing between the age groups is small. Therefore it is probably appropriate to use a fixed age group model for intestinal helminths (such as hookworms) which live for 1–3 years but not for schistosomes or filarial worms with average lifespans of up to 10 years.

In the theoretical part of this paper, the influence of mixing patterns on the impact of chemotherapy programmes was investigated. For targeted treatment it was observed that mixing can lead to a transmission effect such that treating one group leads to a reduction in the mean worm burden of the other group. This effect has been observed in field data.[15] Mixing, however, also reduces the impact of treatment on the treated group due to the presence of an untreated reservoir. The net impact of these two effects may lead to either a beneficial or detrimental effect of mixing. It is also observed that the effect of treating the two groups is asymmetrical

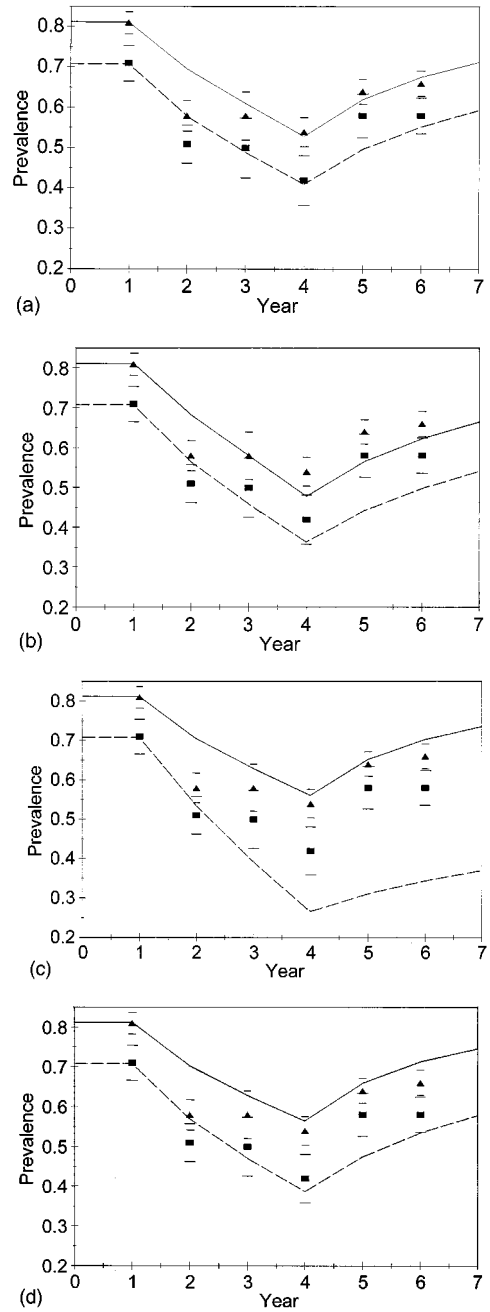

FIGURE 6 *Comparison of data and model for different model formulations. Data points show infection prevalence (corrected) with triangles for adults and squares for children, the bars showing 95% confidence limits. Solid line shows the model prediction for adults and the dotted line that for children. 6a. Random mixing model, R_{0A}/R_{0C} = 10 (the fitted model). 6b. Random mixing model, R_{0A}/R_{0C} = 1. 6c. No mixing model, R_{0A}/R_{0C} = 2. 6d. Restricted mixing model, R_{0A}/R_{0C} = 10*

with the more heavily infected and more contaminating group (adults) having a bigger impact on the other group than the other way round. With mass treatment, the overall reduction in mean worm burden is similar for all the models. However, the distribution of the benefit is evened out in the models where mixing occurs.

An analysis of mixing patterns for schistosomiasis transmission was carried out by Woolhouse *et al.*[16] In contrast to the current paper, they use multiple sites which are identical. It was found that a higher variance in contacts between sites or between hosts leads to a higher basic reproductive rate and hence less effective control. They also observed that control is least effective if people with high contact rates are associated with several sites.

In fitting the data to the model, mean worm burden values actually measured in Zimbabwe were used and probably do reflect worm loads at the study site. However, these values are very low compared with mean worm burden values estimated in other parts of the world.[2] However, this is unlikely to affect the qualititative patterns observed in the results.

Analysis of the field data suggests that the reproductive number of the parasite is higher in adults than in children. This can be due to two factors. Firstly, adults may be more contaminative in their behaviour which is probably unlikely.[17,18] It is much more likely to be due to the fact that adults deposit their faeces in sites which are more suitable for growth of larvae,[3] and has indeed been observed in a similar study area (Bradley, unpublished data).

The data also indicate that mixing between adults and children probably occurs since transmission rates in children are too low to sustain infection in isolation. However, there are probably insufficient data at present and the behaviour of the model is not sensitive enough to distinguish between random and restricted mixing.

In terms of practical implications of the results, it is important to note that drugs used for treatment of hookworm (Albendazole, Mebendazole) are also used for the treatment of *A. lumbricoides* and *T. trichiura* infection which are also generally prevalent in the same endemic areas. Since most chemotherapy programmes target the three species together, any practical recommendations should take this into account. In areas where the rates of hookworm infection and contamination are higher in adults than in children, as in this study area, the results of the current analysis suggest that treatment of children alone (as done in most intestinal helminth programmes) may be insufficient even to limit levels of infection in children. In addition, such a strategy may be unlikely to limit morbidity in adults which may be considerable, one particular potential risk group being pregnant women.[19, 20] Therefore, in areas of high hookworm prevalence, treatment of adults, perhaps targeted to particular risk groups, may be advisable in addition to the usual child-based programme.

Considering the study area in Zimbabwe in particular, we observe a situation where prevalence of infection is very high in adults and almost as high in children. Observation of the field situation suggests there may be a difference in transmission sites between adults and children. The current analysis indicates that 'mixing' does occur between adults and children and infection in children is probably sustained solely through mixing with adults. Where there is high prevalence of infection in children, children should be treated to reduce morbidity and potential developmental effects of hookworm infection.

However, in this case, treatment of children alone is unlikely to reduce transmission and may be unlikely to decrease substantially morbidity levels in children. Therefore treatment of adults is recommended in addition to treatment of children in this area. This programme would have the added benefit of decreasing morbidity in adults.

This study has shown that mixing patterns can often lead to both complex and not always intuitive results. This has obvious implications for control programmes when trying to predict the impact of interventions on both the treated groups and the community as a whole. The results also highlight the need to understand human behaviour patterns with respect to contact and contamination in hookworm epidemiology. In the absence of detailed information on this, the study has constructed a set of epidemiological models which can be used together when planning interventions and which can be extended to other helminth parasite systems.

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APPENDIX

DERIVATION OF TWO SITE RESTRICTED MIXING MODEL (MODEL 3)

In terms of the populations of infective stages L_1 in site 1 and L_2 in site 2, the differential equations for the mean worm burden in children and adults and for the infective stages in sites 1 and 2 can be written as follows:

$$\frac{dW_C}{dt} = \beta_C L_1 - \mu W_C \qquad \text{A1}$$

$$\frac{dW_A}{dt} = x\beta_A L_2 + (1-x)\beta_A L_1 - \mu W_A \qquad \text{A2}$$

$$\frac{dL_1}{dt} = \pi_C \lambda f(W_C) + \pi_A(1-x)\lambda f(W_A) - \pi_C \beta_C L_1 - \pi_A(1-x)\beta_A L_1 - \mu_{L1} L_1 \qquad \text{A3}$$

$$\frac{dL_2}{dt} = \pi_A x \lambda f(W_A) - \pi_A x \beta_A L_2 - \mu_{L2} L_2 \qquad \text{A4}$$

This formulation assumes that the fecundity of worms (λ)is the same in adults and children but that the mortality of infective stages is different in the two sites (μ_{L1} and μ_{L2}). β_C and β_A are the unscaled (absolute) values for the contact rates for adults and children respectively. Other parameters are as in the main text. The basic reproductive number of worms in children and adults respectively can be defined as:

$$R_{0C} = \frac{\lambda}{\mu} \cdot \frac{\pi_C \beta_C + (1-x)\pi_A \beta_A}{\mu_{L1} + \pi_C \beta_C + (1-x)\pi_A \beta_A} \qquad \text{A5}$$

$$R_{0A} = \frac{x\lambda}{\mu} \cdot \frac{\pi_A \beta_A x}{\mu_{L2} + \pi_A \beta_A x} + (1-x)R_{0C} \qquad \text{A6}$$

By setting the numbers of infective stages to be always at equilibrium and rearranging, equations (3) and (4) in the main text for the rate of change of mean worm burden can be obtained.