Socioeconomic Inequalities in Neglected Tropical Diseases: A Systematic Review

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Abstract

Background

Neglected tropical diseases (NTDs) are generally assumed to be concentrated in poor populations, but evidence on this remains scattered. We describe within-country socioeconomic inequalities in nine NTDs listed in the London Declaration for intensified control and/or elimination: lymphatic filariasis (LF), onchocerciasis, schistosomiasis, soil-transmitted helminthiasis (STH), trachoma, Chagas’ disease, human African trypanosomiasis (HAT), leprosy, and visceral leishmaniasis (VL).

Methodology

We conducted a systematic literature review, including publications between 2004–2013 found in Embase, Medline (OvidSP), Cochrane Central, Web of Science, Popline, Lilacs, and Scielo. We included publications in international peer-reviewed journals on studies concerning the top 20 countries in terms of the burden of the NTD under study.

Principal findings

We identified 5,516 publications, of which 93 met the inclusion criteria. Of these, 59 papers reported substantial and statistically significant socioeconomic inequalities in NTD distribution, with higher odds of infection or disease among poor and less-educated people compared with better-off groups. The findings were mixed in 23 studies, and 11 studies showed no substantial or statistically significant inequality. Most information was available for STH, VL, schistosomiasis, and, to a lesser extent, for trachoma. For the other NTDs, evidence on their socioeconomic distribution was scarce.

The magnitude of inequality varied, but often, the odds of infection or disease were twice as high among socioeconomically disadvantaged groups compared with better-off strata. Inequalities often took the form of a gradient, with higher odds of infection or disease each step down the socioeconomic hierarchy. Notwithstanding these inequalities, the prevalence
of some NTDs was sometimes also high among better-off groups in some highly endemic areas.

Conclusions
While recent evidence on socioeconomic inequalities is scarce for most individual NTDs, for some, there is considerable evidence of substantially higher odds of infection or disease among socioeconomically disadvantaged groups. NTD control activities as proposed in the London Declaration, when set up in a way that they reach the most in need, will benefit the poorest populations in poor countries.

Introduction
The burden of Neglected Tropical Diseases (NTDs) is heavily concentrated in low- and middle-income countries [1]. Not only between countries but also within countries, NTDs are often assumed to be concentrated in the poorest populations [2]. Poverty is usually seen as a root cause of NTDs because of its association with living and working conditions and access to preventive and curative health services [3]. In turn, NTDs have strong impoverishing effects because of the absence of social protection systems (including health insurance to protect people against catastrophic health expenditures and sickness and disability insurance to protect people against loss of income in the case of sickness or disability) in most developing countries [4].

The almost omnipresent assumption about the unequal distribution of NTD prevalence across socioeconomic strata contrasts with the scattered nature of the literature on this subject. Empirical evidence on the socioeconomic distribution of NTDs comes from studies conducted from a variety of disciplinary and methodological perspectives (cf. [5–8]). Sometimes, socioeconomic inequalities in NTD prevalence are the main study focus, but more often, socioeconomic position (SEP) is examined as one of a broad range of determinants or merely as a potential confounder of other relationships of interest. So far, there has been no effort to bring together this dispersed literature and describe, across a broad range of NTDs, the extent of socioeconomic inequalities in infection or disease prevalence.

By contrast, the literature on socioeconomic inequalities in health more generally is extensive. From this literature, we know that most health outcomes are unequally distributed, with people at the lower end of the socioeconomic ladder having lower chances of leading a long and healthy life compared with better-off groups within the same country [1,9,10]. This is the case in high-income countries, where the literature on socioeconomic inequalities in health is extensive [10], but especially also in low- and middle-income countries, where the body of literature is growing, in particular with regard to inequalities in child health outcomes [9]. From this body of work, we know that socioeconomic inequalities in health are often substantial. It is generally assumed that socioeconomic inequalities in NTDs are also large and that control strategies would benefit poor populations most.

We used our combined expertise in health inequalities research and NTD research to bring together the recent evidence on the distribution of NTD prevalence—and/or prevalence of the underlying infection—across socioeconomic strata within countries and to summarize the magnitude and pattern of these inequalities. With this aim, we conducted a systematic literature review for nine NTDs listed in the London Declaration for intensified control and/or elimination, including those that are controlled through preventive chemotherapy (PCT) (i.e., lymphatic filariasis [LF], onchocerciasis, schistosomiasis, soil-transmitted helminths [STH], and trachoma) and those controlled through intensified disease management (IDM) (i.e., Chagas’ disease, human African trypanosomiasis [HAT], leprosy, and visceral leishmaniasis [VL]).
Methods

We conducted a systematic literature review on the socioeconomic distribution within endemic countries of LF, onchocerciasis, schistosomiasis, STH, trachoma, Chagas’ disease, HAT, leprosy, and VL. Our search protocol is provided in S1 Supporting Information.

The search included all publications between 2004–2013 in the following databases: Embase, Medline (OvidSP), Cochrane Central, Web of Science, Popline, Lilacs, and Scielo. Also, the most relevant results from Google Scholar were screened, and we searched for articles that were present in PubMed but not yet available in Medline. The search terms used included the NTD names, types of epidemiological data, and indicators of SEP (S1 Supporting Information). The search was completed on 18 December 2013.

Papers were included based on the following predefined criteria: published in an international peer-reviewed journal (i.e., journals with an impact factor) of any language between 2004 and 2013; study period between 2000 and 2013; and reporting estimates of the association between SEP and NTD prevalence, or prevalence of the underlying infection, with a measure of statistical significance (e.g., p-value or 95% confidence interval). We only included studies about the top 20 countries in terms of burden for the NTD under study (Global Burden of Disease [GBD] 2010) in order to focus our review on the most significant endemic countries. The included countries comprised almost 90% of the global burden for the studied NTDs. We excluded papers that did not report the study period (seven papers).

First-stage screening on the basis of title and abstract was done by HKK, who, when in doubt, discussed findings with MCK and TAJH, only excluding papers that were definitely not: about the NTD under study, about the association between SEP and the NTD, or within the above-mentioned study and publication period. In the second stage, the full text of the papers was reviewed by HKK, MCK, and TAJH, with at least two of the authors involved in the decision about inclusion or exclusion. Data were extracted by HKK in a sheet including author, publication date, study aim, NTD measurement, study design, statistical methods (including whether clustering was taken into account in the statistical analyses), sampling design, sample size, indicator of SEP, NTD prevalence, and univariate and multivariate association between SEP and the NTD. The extraction sheets were checked for correctness by JAH, TAJH, and MCK. Support with translation of Spanish and Portuguese papers was provided by EL and EdV.

Analytical framework

The analytical framework (Fig 1) that we used for this review is based on the extensive literature on socioeconomic inequalities in health [9–14]. The core of the framework, and the focus of our review, is the association between SEP and NTD infection and, in particular, the distribution of disease or infection prevalence across socioeconomic strata. Here, and in the remainder of the manuscript, we use NTD infection to signify the prevalence of the NTD and/or the underlying infection.

Usually, health outcomes are unequally distributed, with worse outcomes among socioeconomically disadvantaged groups compared with better-off strata [10]. Taking infection prevalence as an example, the magnitude of inequality in infection prevalence between socioeconomic groups can be measured using the ratio of the odds (OR) of infection between lower and higher socioeconomic strata or other summary measures of inequality [15]. Sometimes, only disease or infection prevalence rates are described across socioeconomic strata, combined with tests for differences in rates between strata. We included papers reporting any measure of association between SEP and NTD infection as well as papers only presenting SEP-specific diseases or infection prevalence rates.
SEP, in the context of research on low- and middle-income countries, is typically measured using indicators of educational attainment and/or economic status [14]. In such countries, household ownership of assets is often used as measure of economic status [16]. Sometimes, other dimensions of position in the socioeconomic hierarchy are studied, such as caste or occupational class. Ecological and multilevel designs usually (also) use aggregate measures of SEP, such as village-level per capita income, the percentage of adults unemployed, or the percentage of households owning their own home in a given geographical area. We have been inclusive when selecting papers, including studies reporting any measure that, according to the authors of those papers, indicated (individual or aggregate level) position in the socioeconomic hierarchy in their specific context.

SEP influences NTD infection via more proximate determinants of such infection (Fig 1, pathway 1). These proximate determinants vary by NTD and include, for example, hygiene behaviours, access to clean water and sanitation facilities, environmental hygiene, exposure to infection through working conditions, and access to health services. The relationship between these proximate determinants and NTD infection is the subject of a broad literature and is, by itself, not the focus of our review. An implication of the causal pathway from SEP via proximate determinants to NTD infection is that statistical adjustment for such proximal determinants generally reduces the magnitude of socioeconomic inequality in NTD infection. In other words, the association between SEP and NTD infection is partially explained by intermediate variables. As our paper focuses on the description of the magnitude of socioeconomic inequality in NTD infection, we focus in our description on associations unadjusted for these proximal determinants.

Potential confounders (Fig 1, pathway 3) of the relationship between SEP and NTD infection can include, for example, age and sex. A Brazilian study found, for instance, that elderly people tend to be richer and, independently of SEP, have higher odds of having trachoma [17]. Age and sex (but also other factors) can also be effect modifiers of the relationship between SEP and NTD infection (Fig 1, pathway 2). In other words, the relationship between SEP and NTD infection can be stronger or weaker depending on age or sex. Whenever available, we
have presented data about the relationship between SEP and NTD infection stratified by age and sex. The magnitude of socioeconomic inequality in NTD prevalence can also vary by the specific outcome studied (e.g., for trachoma, whether trachomatous inflammation—follicular [TF], trachomatous inflammation—intense [TI], trachomatous scarring [TS], or trachomatous trichiasis [TT] was studied) and the detection method used (e.g., based on blood samples or stool samples). We have presented findings stratified by specific outcome and detection method whenever available.

Finally, SEP not only influences the odds of NTD infection; NTD infection can also affect SEP through income lost because of illness, costs of medical care, and, in the case of children, impeded school attendance and performance (Fig 1, pathway 4). The socioeconomic consequences of NTDs are the subject of separate papers in this series [4].

This review is compliant with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist (S2 Supporting Information) [18].

Results

Overview

5,516 unique papers published between 2004 and 2013 in international peer-reviewed journals were identified (Fig 2). Of these, 4,769 were excluded on the basis of title and abstract. The full text of the remaining 747 papers was reviewed, of which 93 papers met the inclusion criteria. Of the 93 studies that presented data on the socioeconomic distribution of NTDs, almost two-thirds (59/93) reported substantial and statistically significant inequalities (Fig 3). The findings were mixed in a quarter of the studies (23/93), and over 10% (11/93) showed no substantial or statistically significant inequality in NTD distribution. Most information was available for STH (34 papers), schistosomiasis (16 papers), VL (17 papers), and, to a lesser extent, for trachoma (11 papers). For the other NTDs, there is a paucity of evidence, with no recent papers identified for onchocerciasis and HAT.

Lymphatic filariasis

Four papers on lymphatic filariasis met the inclusion criteria (India [GBD #1] two [19,20]; Tanzania [GBD #11] two [21,22]) (S1 Table). All were cross-sectional studies, representing the general population in a defined geographical area: Chennai city in Tamil Nadu (mass drug administration [MDA] status not reported) and endemic villages in Andhra Pradesh (Fig 4) (MDA since 2004, 58% of respondents participated) in India and the cities of Tanga (59% of respondents participated in MDAs) and Dar es Salaam (MDA in 2006 and 2007, 19% of respondents participated) in Tanzania [21,22]. The Tanzanian papers also included schoolchildren in addition to the general population.

In the Indian studies, the prevalence of *Wuchereria bancrofti* microfilaria was nearly twice as high among poor villagers (4.9%) and in poor urban neighbourhoods (1.3%) compared with richer ones (rural: 2.6%, p = 0.02; urban: 0.5%, p = 0.01). The prevalence was also higher among less-educated people (in the rural study) than among the better educated, but this association became only statistically significant after adjusting for age and several intermediate determinants.

The Tanzanian studies reported that the prevalence of circulating filarial antigens (CFA) among school children was higher in children from poorer neighbourhoods (Fig 5). Among community members (aged ≥ ten years), such neighbourhood-level association was only found in one study [21,22]. At the household level, no association was found between economic status and CFA prevalence (only examined for school children). The authors suggested that this was perhaps due to the importance of community-level factors like poor water and sanitation conditions.
In sum, there is a paucity of evidence on socioeconomic inequalities in LF prevalence. The currently available studies, from India and Tanzania only, present a mixed picture, with an association between SEP and LF prevalence in some age groups when using some SEP measures but not in other age groups and/or when using other SEP measures.

**Onchocerciasis**

No relevant papers were found.
Schistosomiasis

16 relevant papers were found (Nigeria [GBD #1] one, on *Schistosoma haematobium* [23]; China [GBD#2] six, on *S. japonicum* [24–28]; Sudan [GBD#5] two [one on *S. haematobium*, one on *S. mansoni*] [6,29]; Côte d’Ivoire [GBD #14] five, on *S. mansoni* [30–34]; Uganda [GBD #15] two, on *S. mansoni* [35,36]) (S2 Table). Moreover, we found two relevant studies about combined schistosomiasis and STH infection (reported on under STH). Four papers from Côte d’Ivoire report about the same study population [31–34]; the same is true for two studies from China [25,26]. All studies used a cross-sectional design.
S. japonicum

The Chinese studies were conducted in the general population, usually sampling villages in specific geographic settings. These studies found a strong spatial clustering of infection, but the association with SEP appeared to depend on the interlinkage between geography, economy, and occupational structure. In the *S. japonicum* surveillance sites of Hunan province, representing different geographic and epidemiologic conditions, strong village-level clustering of infection was observed, with higher infection levels in lake and embankment areas and among fishermen. Here, infection prevalence was higher in poorer villages (4.7%) than in richer ones (2.5%) (*p* < 0.001) [Fig 6] [28]. Conversely, in mountainous Eryuan county, the socioeconomically better-off plain areas with irrigated farmland had higher seroprevalence levels [26]. However, within the endemic (mostly plain area) villages of Eryuan, a higher SEP was protective against seroconversion (OR least poor versus poor: 0.48, 95% CI: 0.32–0.73) [25]. Similarly, two other papers report much greater odds of infection among illiterate and poor people within villages after adjusting for a range of intermediate variables such as water contact [27,37].

S. mansoni

Four studies among school children in Côte d’Ivoire—by the same authors using the same data—found similar odds of infection in all wealth groups except for lower odds among the least poor group [31–34]. A systematic socioeconomic gradient in infection prevalence was observed—with prevalences increasing from 39% among those with secondary education to 48% and 57% among those with primary education and no schooling, respectively (*p* = 0.01)—in a study among all age groups in Côte d’Ivoire. This association was found among farming
households (irrigated rice cultivation) only; no association was found among nonfarming households (Fig 7) [30]. A study among school children in lakeside and island communities in Uganda reported a drop in the infection risk for each additional household asset owned (electricity, solar power, latrine, landline, mobile phone) (OR 0.74, \( p = 0.001 \)) [35]. A very strong and systematic gradient in *S. mansoni* infection among teenagers was also found in a Ugandan study [36]. The odds of infection were 54.5 times higher in the poorest than in the richest households (next-poor: 15.9 times; next-rich: 3.5 times) (\( p < 0.001 \)). This study also reported a systematic gradient in infection intensity, with a twice-as-high intensity in poor households compared with richer households. Substantial inequalities were also reported in a study among pregnant women in a secondary care hospital in central Sudan [29]. Here, the odds of infection were six times higher among women with no education than among those with secondary education or higher (OR 5.9, 95% CI: 2.8–12.3).

**S. haematobium**

The two studies on *S. haematobium* reported substantial inequalities in infection prevalence. In South Kordofan State, Sudan, the odds of infection were more than three times as high among adults with a low educational attainment (\( \leq \) primary school) than among those with a higher educational attainment (OR 3.07, 95% CI: 1.29–7.32) (Fig 8) [6]. In a study in Nigeria, the prevalence of overall and moderate or high infection (excreting >50 eggs/10 ml urine) was much higher among the poor than among the less poor (73% versus 1.5% for overall infection;

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**Fig 5. Example of socioeconomic inequalities in LF: association between filarial antigen prevalence (%) and family wealth in the city of Tanga, eastern Tanzania (2012) [22].** The study was conducted in the city of Tanga in eastern Tanzania, along the Indian Ocean, where a mass drug administration program (MDA) has been active since 2004. Two urban and one peri-urban ward were selected as being representative for the city, and 960 children aged five to 16 years from public primary schools were examined for circulating filarial antigens (CFA) in their blood. Of these children, 895 also filled in a questionnaire. Ownership of a fridge and TV was used as indicator of family wealth; this information was aggregated to constitute a measure of wealth at the ward level. To create the figure, we divided the wards into three groups according to the family wealth measures: “most poor,” “intermediate poor,” and “least poor.” The overall CFA prevalence was 5.5%. CFA prevalence was statistically significantly lower in the least poor as compared to the intermediate and most poor (\( p = 0.04 \)).

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Fig 6. Example of socioeconomic inequalities in schistosomiasis: association between *S. japonicum* and income per capita at village level in Hunan province, China (2005) [28]. The studied villages represent four types of areas: lake-embankment, lake-beach, inside embankment, and hill areas. A total of 10,245 residents aged six years and older from 16 villages were included in the study. The presence of antibodies to *S. japonicum* was screened using the indirect hemagglutination (IHA) test, and stool samples were examined for IHA-positive cases. Per capita income was measured at the village level (currency rate used: 1 Yuan = US$0.121, July 2005). The overall infection prevalence was 4.1%. Infection prevalence was higher in poorer villages, (*p* < 0.001).

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Fig 7. Example of socioeconomic inequalities in schistosomiasis: association between *S. mansoni* prevalence (%) and educational attainment of household head in the town of Man, western Côte d’Ivoire (2004–2005) [30]. The study was conducted in urban farming communities in the town of Man, western Côte d’Ivoire. A total of 113 farming households (586 individuals from all ages) and 21 nonfarming households (130 individuals from all ages) from six agricultural zones were interviewed, and stool samples were examined for *S. mansoni*. Infection prevalence was 51.4% in farming households and 44.6% in nonfarming households. Lower educational attainment was associated with higher infection prevalence in farming households (*p* = 0.008) but not in nonfarming households. Infection prevalence was higher in poorer households but not statistically significantly so.

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40% versus 0% for moderate or high infection, for monthly household incomes of <US$50 with ≥US$140, respectively). This study found no educational differences in overall infection prevalence and somewhat higher levels of moderate or high infection among the better educated but higher infection intensity among the less educated [23].

In sum, socioeconomic inequalities in schistosomiasis infection were usually (very) large. Nevertheless, the strength and direction of the association appears to be dependent on the intersection of geography and the occupational and socioeconomic structure of the study population. This is related to the strongly spatially clustered nature of the infection, which has to do with fishing and irrigated agriculture, among other causes.

Soil-transmitted helminths (STH)

34 relevant papers on STH (Ascaris lumbricoides, Trichuris trichiura, hookworm disease) were found (China [GBD #1] four [25,37–39]; India [GBD #2] three [40–42]; Vietnam [GBD #5] five [43–47]; Malaysia [GBD #6] three [48–50]; Nigeria [GBD #8] three [51–53]; Brazil [GBD #9] ten [5,54–62]; Nepal [GBD #14] one [63]; Pakistan [GBD #15] one [64]; Ethiopia [GBD #16] one [65]; Colombia [GBD #18] one [66]; Tanzania [GBD #19] one [67]; Thailand [GBD #20] one) (S3 Table) [68]. In addition, we found two relevant studies (Ethiopia, Nigeria) about combined STH and schistosomiasis infection [69,70]. Two studies examined STH reinfection using a cohort design, and one paper reported on an ecological study; the other studies used a cross-sectional design. 18 studies included children, two included pregnant women or women of reproductive age, and 16 studies included all ages.

Preschool- and school-aged children

STH prevalence was systematically and substantially higher among children from socioeconomically deprived households. Evidence comes from a range of settings (rural and urban...
areas in China, India, Pakistan, Nigeria, Brazil, and Malaysia) using a variety of SEP measures. While ORs varied strongly—from around 1.5 to 9—typically, infection prevalence was about twice as high among socioeconomically worse-off children. A comparatively large study in Brazil, for instance, found that 41% of poor children had any STH infection, compared with 22% of richer children [58]. Often, there was a systematic socioeconomic gradient in infection. A study in rural Nigeria, for example, found that ascariasis infection prevalence ranged from 10% when both parents had at least primary education to 31% when only the mother had such education and 53% when only the father had such education to 96% when neither parent had a primary education (Fig 9) [53]. Even in children under two years of age, STH prevalence was much higher among those from deprived backgrounds [52,66]. Ascariasis prevalence among children aged 0–25 months, for instance, was 27% among those whose father was a farmer, compared with 11%–13% among those whose father had a professional occupation or was a businessman [52]. Despite these inequalities, infection prevalence was sometimes also very high among better-off children. A study among schoolchildren in Kashmir, India, for instance, found that the prevalence of intestinal helminthiasis was 84% among children of illiterate mothers and 60% among children of mothers with secondary education [41].

There were minor variations to this overall pattern of inequality in STH prevalence in children. In rural Malaysian aboriginal children, the odds of infection were twice as high among poor children, but no association with parental education was found, nor was an association between household income and reinfection with any STH three and six months after treatment observed [48,49]. Conversely, among children attending outpatient clinics in Brazil, a higher prevalence of parasitic intestinal infection was associated with low parental educational attainment but not with income level [60]. A study among schoolchildren in Vietnam found no differences in parental income or education between children that were highly positive for ascariasis (egg per gram count (EPG) of >2,000) or trichuriasis (EPG of >600) and those without these infections [47].

Fig 9. Example of socioeconomic inequalities in STH: parental education and ascariasis infection in a rural community in Osun State, Nigeria (2005–2006) [71]. A total of 440 children <16 years of age from randomly selected households were included. Information on parental education was collected through a questionnaire, and faecal samples were examined for the presence of Ascaris eggs. The prevalence of ascariasis was statistically significantly higher among children of parents without a primary education (p < 0.001).
Other risk groups

The two studies among pregnant women (Thailand) and women of reproductive age (Vietnam) found strong associations between STH prevalence and SEP; the odds of infection were three to nine times higher among women from lower socioeconomic strata (Thai study: OR any STH 3.2 [95% CI, 2.0–5.3]; Vietnamese study OR any STH 7.5 [95% CI, 3.4–16.4], OR ascariasis 9.0 [95% CI, 3.6–22.7], OR trichuriasis 3.7 [95% CI, 1.5–9.1]).

In tea estates in Assam, India, STH prevalence was very strongly associated with educational attainment and occupational status. The prevalence of ascariasis, trichuriasis, and hookworm disease was 5%–6% among staff (teachers, health workers, factory workers) and ranged between 45%–52% among workers (tea-pickers) ($p < 0.001$). Similarly strong associations with educational attainment, in turn strongly associated with occupational status, were found [40].

General population

Evidence from China, Brazil, and Malaysia indicates that also in the general population (all ages), the association between SEP and STH prevalence is often strong [5, 50, 54]. For example, in remote poor rural villages in five Malaysian states, the prevalence of intestinal parasitic infections was 83.5% among people from low-income households and 40% among richer people ($p < 0.001$) [50]. Findings were similar in a study on hookworm in southeastern Brazil, illustrating that, despite large inequalities, infection prevalence can remain high among the better off. A study in Hunan province, China, found that the odds of any STH infection were up to five times lower in wealthier households, after adjusting for a range of intermediate determinants such as hygienic behaviors [37]. Reinfection 12 months after successful treatment was found to be twice as high among the poor than among the least poor in a study in southeastern Brazil [56].

At the same time, the strength and direction of the association sometimes differed by type of parasite and SEP indicator [25, 44–46, 62, 65]. For example, a study in an endemic urban area in Brazil found that household income was associated with hookworm infection (prevalence among those with no wage: 16%; >US$396: 5%, $p = 0.005$) but not with ascariasis or trichuriasis [62]. In mountainous Eryuan county, China, the odds of ascariasis infection were about twice as high among the poor and less educated, but trichuriasis was almost exclusively found in areas below 2,150 m, where richer people lived [25]. A Vietnamese study in an agricultural community where the intensely polluted Nhue Rivier was used for irrigation and where excreta were used as fertiliser found an association between STH infection and lower educational attainment but found no association with household economic status [44]. The authors hypothesize that this is due to the relatively homogeneous study population, but it is perhaps also related to the inclusion of rice fields and fish ponds—risk factors for STH—as indicator of economic status. A study among podoconiosis patients and healthy controls in Ethiopia found no association between educational attainment and STH [65].

Combined STH and schistosomiasis infection

Two studies, one from Ethiopia [69] and one from Nigeria [70], measured the association between SEP and intestinal parasite status. The study among schoolchildren in Ethiopia found no association between family income and intestinal parasite infection (A. lumbricoides, T. trichiura, hookworm, S. mansoni). The Nigerian study, conducted among adult nomadic Fulanis, found that 95% of people with no schooling were infected with an intestinal parasite (A. lumbricoides, T. trichiura, and S. mansoni, among others) compared with a minority among those with some education [70]. Findings were similar when using housing type (hut, brick, cement) as SEP indicator.
In sum, socioeconomic inequalities in STH prevalence are often large, with ORs of two or greater, both among children and pregnant women or women of reproductive age and in the general population. Despite these inequalities, infection prevalence sometimes remained also high among better-off people. In several cases, the specific pattern depended on the measure of socioeconomic status used and the type of STH infection.

Trachoma

11 relevant papers were found (Ethiopia [GBD #4] six [72–77]; Brazil [GBD #6] one [17]; Sudan [GBD #9] one [78]; Tanzania [GBD #11] three [79–81]) (S4 Table). All studies used a cross-sectional design, describing trachoma prevalence by SEP in specific age groups (especially young children) or the general population within a defined geographical area. MDA coverage was generally not reported.

All studies examined the association between SEP and active trachoma (TF only, TF and/or TI), usually in young children. Seven studies (Ethiopia five, Tanzania two) reported statistically significant associations between SEP and the prevalence of active trachoma, with higher prevalences among lower strata (Fig 10) [73–77,79,80]. The strength of the association varied between and within (by the SEP indicator used) studies, from moderate (OR between 1.3–1.9) [74,75,79] to strong (OR between 4 and 9) [73,75,76], with one study reporting a concentration index (CI) (-0.0942) instead of an OR. Two studies (Ethiopia, Sudan) reported associations in the same direction but with fairly wide confidence intervals, including the reference value [72,78]. One study reported no clear pattern between active trachoma and SEP [81].

Two papers also reported on the prevalence of ocular *Chlamydia trachomatis* infection, with similar findings on the association with SEP as for active trachoma [72,79]. One study (Ethiopia) also reported on TS and TT (> ten years of age) and found increasing

Fig 10. Example of socioeconomic inequalities in trachoma: association between educational attainment of household head and follicular trachoma in young children in Tanzania (2008) [79]. The study was conducted among children aged 0–5 years in 36 communities (3,122 children) in 2008. The eyes of the children were examined for active trachoma, and information on the educational attainment of the household head was collected through a questionnaire. The prevalence of follicular trachoma was 30.9%. Lower educational attainment of the household head was associated with a higher risk of follicular trachoma among children in both countries (Tanzania $p = 0.001$).

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socioeconomic inequalities between illiterate and literate people with increasing trachoma severity, with ORs rising from 1.9 (95% CI 1.65–2.26) for TF or TI to 2.57 (95% CI 2.1–3.2) for TS and 4.2 (95% CI 2.4–6.9) for TT [74]. Findings from another study on TT were in the same direction, but confidence intervals were wide, including the reference value [78].

One study (Brazil, all ages) compared the distribution of education and income between people with and without trachoma (any trachoma including *C. trachomatis* infection). People with trachoma were more likely to be less educated, but this pattern was not consistently statistically significant. Richer people in this study were more likely to have trachoma than poorer people, which the authors attributed to the association between age on the one hand and trachoma and wealth on the other, with older people being richer (in this study population) and having a higher likelihood of having trachoma [17].

In sum, the odds of trachoma in children and adults were usually substantially higher among poor and less-educated people and households. These socioeconomic inequalities possibly increase with increasing trachoma severity, but this finding could also be due to confounding by age, as more severe forms of trachoma are more common in older people who are also more likely to be illiterate. As trachoma prevalence rises with age, a positive association between income and trachoma prevalence may be observed in areas where older people are richer.

Chagas’ disease

Four papers on Chagas’ disease met the inclusion criteria (Brazil [GDB #1] two [82,83]; Argentina [GDB #3] one [84]; Colombia [GDB #5] one [85]) (S5 Table). Two studies represented the general population in a specific geographical area using a cross-sectional design [82,84], while two focused on pregnant women (cross-sectional) [85] or women who recently gave birth (case control) [83], reflecting the importance of vertical infection transmission.

The three studies that tested for anti-*Trypanosoma cruzi* seropositivity found strong and statistically significant associations between SEP and infection, with two to three times higher odds of infection among lower than among higher strata. A study among pregnant women in Colombia even reported an OR of 19.6 (95% CI 2.5–152.2) comparing women without completed primary education to university-educated women [85]. The Argentinian study compared self-reported Chagas and vector presence by SEP. In a region with horizontal control strategies, they found lower reported vector prevalence, higher self-reported Chagas prevalence, and smaller (and not statistically significant) educational inequalities in such prevalence than in a region with vertical control strategies [84]. The authors concluded that horizontal control strategies reduced vector prevalence, raised awareness of Chagas’ disease in all strata, and reduced inequalities in such awareness.

In sum, there is a paucity of evidence on socioeconomic inequalities in Chagas prevalence. The available evidence suggests that the odds of Chagas’ disease are much greater for lower socioeconomic groups than for higher strata, both in the general population and particularly in pregnant women.

Human African trypanosomiasis (HAT)

No relevant papers were found.

Leprosy

Seven papers on leprosy met the inclusion criteria (Brazil [GBD #2] six [7,8,86–91]; Bangladesh [GBD #7] one [92]) (S6 Table). Four studies represented the general population in a defined geographical area (two population-based or ecological studies [90,88], two case control studies
Three studies focused on specific groups: patients with no leprosy contacts (case-control study) [91], contacts of newly diagnosed leprosy patients (cohort study) [8], and past-five-year migrants (case-control study) [86].

The Brazilian studies all showed a strong association between SEP and leprosy, with a systematic gradient in studies comparing more than two strata. The odds of leprosy were at least twice as high in poorer and less-educated people as in the better off. When using water and sanitation facilities as proxy for (household or community level) SEP, the ORs varied strongly between studies, from 1.17 (95% CI 0.96–1.43) and 1.44 (95% CI 0.95–2.80) [91] to 3.1 (95% CI 1.1–10.02) [86]. The odds of being poor were nearly five times higher among leprosy patients than among controls [90]. Among coprevalent contacts of newly diagnosed leprosy patients, the odds of having leprosy were strongly associated with the educational attainment and income level of the patient. The association with SEP of the contact itself was weaker and not always statistically significant. After follow up of contacts, the odds of leprosy were not systematically associated with educational attainment or income level of the leprosy patient or the contact, except perhaps with a low income of the contact [8].

In the Bangladeshi study, the odds of leprosy were systematically higher among poorer people when using household assets as wealth indicator (borderline statistically significant) [92]. There was no association between income level and educational attainment on the one hand and the odds of leprosy on the other. Conversely, food shortage in the past year was associated with clinical signs of leprosy.

In sum, recent evidence on socioeconomic inequalities in leprosy prevalence is scarce. In Brazil, socioeconomic inequalities in leprosy were large. In Bangladesh, the association remains ambiguous. There is no evidence from other countries.

### Visceral leishmaniasis (VL)

17 papers on VL met the inclusion criteria (India [GBD #1] six [93–98]; Bangladesh [GBD #2] two [99,100]; Ethiopia [GBD #5] one [101]; Uganda and Kenya [GBD #11, #14] one [102]; Brazil [GBD #19] seven [103–108]) (S7 Table). The studies were typically conducted in (highly) endemic areas among the general population and all age groups. One study examined risk factors among people living in mud-wall houses [100], one compared migrants with residents [101], and one examined VL prevalence in household contacts of VL patients [106]. Four of the Brazilian studies were conducted in state capital cities. Study designs varied from ecological to cross-sectional, cohort, and case-control.

The ecological studies (two from India; three from Brazil) found moderate to strong associations between low SEP (poverty, unemployment, low educational attainment) and higher VL incidence [93,94,104,105]. The individual-level studies found that the risk of VL infection was as high or higher in lower strata than in the better off, although findings were not universally consistent across SEP indicators. Details about these individual-level studies are reported below.

The South Asian studies reported 1.6 to 3.4 times higher odds of VL in the poorest groups compared with the less poor [96–98,100]. One study also reported a 2.9 (95% CI 1.3–6.8) times higher odds of infection for the Mushahar caste, who are among the poorest of the poor, after adjusting for wealth quintile and a range of intermediate factors [98]. Two of these studies also examined literacy and found no association with VL infection [96,100]. Conversely, two other studies found no association between poverty and infection, while one of these also examined literacy and found a 1.66 times (95% CI 1.10–2.51) higher odds of VL infection among illiterate people [95,99]. One of these studies attributes the lack of association between poverty and VL to the homogenous high-risk study population [99]. Yet, even among people living in mud-
wall houses in rural Bangladesh, the poorest villagers (without electricity) had a 3.40 (95% CI 1.76–6.59) higher odds of VL infection than less-poor villagers (with electricity) [100].

Among the Brazilian studies, two report moderate to strong associations between educational attainment and VL infection [103,107]. Conversely, another study reports no income differences between people with and without VL infection [108]. Among household members of VL patients, the risk of infection was not associated with income level, but it was statistically significantly lower among those owning many household assets [106].

The two African studies report large socioeconomic inequalities in VL. In a study population largely consisting of pastoralists in Uganda and Kenya, a very strong and systematic association between household wealth and VL was observed, with 13 times higher odds of infection in the poorest quintile and five times greater odds in the next-poorest quintile than in the least poor group [102]. The Ethiopian study found a 1.4 to nearly 3 times higher odds of VL among less-educated and poor people and those with a low occupational status compared with the better off, but no association was found with land ownership.

In sum, most (15 out of 17) studies found a (much) higher odds of VL infection among lower socioeconomic strata along at least one indicator of SEP, but findings were not universally consistent across SEP indicators. Several studies found inequalities in VL along one SEP indicator but not along another. Only two studies found no association between SEP and VL.

Discussion

Our systematic review exhibited a paucity of recent evidence on within-country socioeconomic inequalities in several NTDs—onchocerciasis, HAT, LF, and Chagas, in particular—even for high-burden countries. Yet, for STH, schistosomiasis, VL, and, to a lesser extent, for trachoma, there is considerable evidence of substantial socioeconomic inequalities in the odds of infection, with often (much) higher odds among socioeconomically disadvantaged groups. While the magnitude of inequality varied, the odds of infection were usually at least twice as high among disadvantaged groups compared with better-off people. Inequalities often took the form of a gradient in studies comparing more than two strata, with subsequently greater odds of infection each step down the socioeconomic hierarchy. Notwithstanding these inequalities, the prevalence of some NTDs remained high also among better-off groups in some highly endemic areas.

Limitations of the study

Search strategy. The limited number of obtained studies might be partly a result of our search strategy, which only included international publications in the last decade and studies conducted in the 20 countries with the highest burden for each NTD. The rapid changes in many countries—in NTD prevalence [109], economic growth, and concurrent changes in inequality in income and educational attainment—underscore the importance of having up-to-date evidence. While the magnitude and pattern of socioeconomic inequalities in NTDs may have been different in earlier decades, this will be difficult to assess, given the incomparable methodologies used in the studies we reviewed (see below). The included countries comprised nearly 90% of the global burden for the studied NTDs (ranging from around 83%–86% for schistosomiasis and STH to >90% for LF, VL, and leprosy, 97.5% for trachoma, and 100% for Chagas, HAT, and onchocerciasis).

Publication and review bias. The substantial proportion of papers reporting inequalities in infection risk could be due to publication bias, with studies reporting statistically significant associations being more likely to get published. Yet, most studies were not specifically set up to assess inequalities, and the fact that we rarely found statistically significantly higher odds of
infection among rich people supports the plausibility of our conclusions. Review bias may have led to the exclusion of studies finding no association between SEP and infection, as these did not always report a measure of association.

**Methods used to assess the relationship between SEP and NTD prevalence in the original studies.** Many of the included studies were not designed to describe socioeconomic inequalities in infection prevalence. Often, SEP was examined as one of a wide range of risk factors or only as a potential confounder, using a broad range of methods from a variety of disciplinary backgrounds. The SEP measures used were not always well described or well constructed, and the distribution of the study population across SEP categories was not always described. Sometimes, ORs were only provided after adjustment for intermediate variables (which generally leads to a downward bias in the magnitude of inequality), and potential confounders such as age were not always taken into account. Also, sampling design (clustering) was infrequently taken into account in the included studies, potentially leading to too narrow confidence intervals. Furthermore, studies were usually highly local in nature rather than taken from a nationally representative population sample. For these reasons, one cannot directly compare the magnitude of socioeconomic inequality in NTDs between studies. We will discuss the implications of two problems in more detail: potential confounding by age and sex and the highly local nature of many studies.

Third factors, especially age and sex, may sometimes have biased the reported magnitude of inequality. Inequalities may be overestimated for (more severe forms of) trachoma and Chagas, which are more common among elderly people, who are often less educated. This possibly contributed to the larger inequalities in more severe forms of trachoma than in less-severe forms of trachoma in an Ethiopian study [74]. Conversely, in some settings, older people tend to be richer, arguably explaining the finding of a Brazilian study that people with trachoma (mainly TS) were wealthier and less educated [17]. Nevertheless, studies of trachoma in children generally found substantial inequalities by household (head) SEP, and studies on Chagas that adjusted for age also reported large inequalities, suggesting that confounding by age does not explain the overall pattern of socioeconomic inequality in NTD prevalence. Conversely, some other infections like STH and schistosomiasis are more common among younger people, with usually a higher educational attainment, possibly leading to an underestimation of the magnitude of inequality.

The reported inequalities are probably an underestimation of inequalities within countries as a whole. Most studies were small and conducted in relatively homogeneous, high-risk populations and/or highly endemic areas, leaving country-level heterogeneity in wealth and infection risk underexposed. Several studies that found no or small inequalities cite study population homogeneity as explanation [92,99]. Nevertheless, even within small, highly endemic areas, inequalities were often substantial [26]. Furthermore, several studies that found no statistically significant association between SEP and infection lacked statistical power to detect substantial inequalities. Finally, the reported socioeconomic inequalities in overall prevalence of worm infections may only partly reflect inequalities in intensity of these infections. Most morbidity due to macroparasitic infections is directly associated with intensity of infection at the individual level and thus only indirectly with prevalence of infection at the community level. Infection intensity is known to strongly vary between individuals within communities [110]. While few papers report on the socioeconomic distribution of infection intensity, it is likely that reported inequalities in overall prevalence of worm infection hide even larger inequalities in intensity of infection. The same would arguably hold for multiple parasitic infections in the same individual and/or community.
Explaining inequalities

Spatial clustering of infection because of geographic conditions, among other causes, is typical for most NTDs. While poor people conceivably tend to live in areas that are conducive to NTD transmission, studies unravelling the contribution of such conditions to socioeconomic inequalities in NTDs are rare. The relationship between spatial infection clustering and SEP may be context-specific, depending on the intersection of economy, geography, and occupational structure. A Chinese study, for instance, found higher schistosomiasis prevalence in richer plain areas with irrigated agriculture, but greater odds of infection among lower SEP groups within these richer areas [26].

Living conditions associated with poverty play an major role in NTD transmission and undoubtedly also in explaining the association between SEP and infection [3]. Scarce multivariate analyses indeed suggest that living conditions are important intermediates in the pathway between SEP and infection [36,38,46,74]. Furthermore, difficulties in accessing preventive and curative care increase the odds that poor people become infected and, once infected, are left untreated [3,111–113]. The costs of care can be substantial for, for example, treatment of trichiasis and advanced stages of Chagas. But even when care is free, other barriers, including distance, low quality of care, and other costs (for transport, working time forgone) can hamper early diagnosis and treatment, which is critical for the prevention of advanced stages of, for instance, leprosy, trachoma, and Chagas. At the same time, MDA for PCT NTDs has the potential to reduce infection prevalence across all socioeconomic layers by reducing barriers to treatment and through herd effects. Unfortunately, the coverage of NTD control programs was not systematically reported, limiting our ability to draw conclusions about their impact on inequalities in infection prevalence. Further research on the socioeconomic distribution of the coverage of NTD control programs will be important to understand the extent to which these efforts help reduce socioeconomic inequalities in NTDs.

SEP clearly influences the odds of NTD infection. The effects of infection on poverty and educational attainment in children are equally clear [4,114]. The paucity of cohort studies makes it difficult to unravel the relative importance of either direction of causality, and evidence from available cohort studies is inconsistent [8,48,56,100]. However, it is clear that reverse causation cannot fully explain inequalities in infection. Many studies report greater odds of infection in children of less-educated parents and greater odds of infection among less-educated adults, while in these cases reverse causation can hardly play a role.

Implications for policy making and research

The public health impact of socioeconomic inequalities in NTDs is large. The short- and long-term consequences of STH infection, for instance, include anaemia in pregnant women and impaired nutritional status, growth, and cognitive development of children, and it has effects on school attendance and performance, with long-term consequences for health, educational attainment, productivity, and income levels [4,114]. Addressing these inequalities will contribute to more equal life prospects and freedom to lead a flourishing life.

Improving the social determinants of health—the conditions in which people grow, live, work, and age, and the structural drivers of these conditions—is critical for a sustainable reduction in socioeconomic inequalities in NTDs [3,36]. Thus, interventions to reduce inequalities in NTDs should include poverty reduction and improving educational attainment as well as improving housing, water, and sanitation conditions for poor people. Hence, action on inequalities in NTDs in not just the responsibility of the ministry of health and (international) organisations specialized in vector control and mass drug administration. Rather, it requires
action across nongovernmental organisations and government departments, including, among others, public works, urban planning, agriculture, finance, and education [3,113].

Reducing inequalities in NTDs requires equitable access to preventive and curative health care without the risk of suffering from catastrophic health expenditures. While treatment of infection is free or inexpensive for NTDs, the costs of care can remain high for some, especially for more advanced disease stages [3], and are compounded by other costs and barriers to seeking care [3]. Universal health coverage for NTDs requires health system strengthening—the penetration of good quality care and vector control programs in poor and remote areas, reaching the poor where they live, with prevention, early detection, and affordable, quality treatment.

Action on inequalities in NTDs requires that monitoring and surveillance systems include equity indicators for disease prevalence and burden and the reach and impact of interventions. Such systems should include, at the minimum, disaggregated data by educational attainment and wealth quintile, further stratified by age and sex where relevant, with measures of statistical uncertainty. The emphasis on reducing inequalities in the Sustainable Development Goals can provide an impetus to the NTD community to incorporate indicators of socioeconomic position in routine monitoring and evaluation.

Evidence-based action would also benefit from more systematic equity research on NTDs to describe the socioeconomic distribution of infection—adjusted for confounders like age and sex—and to measure the contribution of intermediary determinants and the equity impact of interventions. Such research is hampered by the dependence on intensive field data collection because of the lack of national registration data for most NTDs in most countries. Prediction modelling has therefore become important in NTD research, and the evidence presented in our paper can be used to take socioeconomic heterogeneity into account in such models.

**Conclusion**

While commonly seen as diseases of the poor, recent evidence on socioeconomic inequalities in several individual NTDs—in particular, onchocerciasis, HAT, LF, and Chagas—remains scarce, and more systematic research on the link between socioeconomic position and NTD infection is warranted. Yet, for some NTDs—in particular, STH, schistosomiasis, and VL—there is considerable evidence of substantially higher odds of infection among socioeconomically disadvantaged groups. Addressing these inequalities in NTDs will contribute to more equal life prospects and freedom to lead a flourishing life. NTD control activities, when set up such that they reach the most in need, will benefit the poorest populations within poor countries. This requires action across government departments and across nongovernmental organisations to improve the social determinants of health and ensure universal access to preventive and curative care. It is recommended that NTD monitoring and surveillance systems include equity indicators to help ensure that interventions reach the most in need.

**Key Learning Points**

- We found that evidence on the relationship between socioeconomic position and infection or disease prevalence remains scarce for several individual NTDs. Yet, for some NTDs—in particular, STH, schistosomiasis, and VL—there is considerable evidence that poor and less-educated people are at a much higher risk of getting the infection or disease than better-off people.
• The magnitude of this inequality varies, but the risk of infection or disease is often about twice as high among disadvantaged groups compared with better-off people. These inequalities often run across the entire society, with a subsequently higher risk of getting an NTD each step down the socioeconomic hierarchy.

• Notwithstanding these socioeconomic inequalities, the prevalence of some NTDs can also be high among the better off in some highly endemic areas, especially for STH.

• It is recommended that NTD monitoring and surveillance systems include equity indicators to help ensure that interventions reach the most in need.

Top Five Papers
1. World Health Organization. Investing to overcome the global impact of neglected tropical diseases: Third WHO report on neglected tropical diseases. Geneva: World Health Organization, 2015.
2. Aagaard-Hansen J, Chaignat CL. Neglected tropical diseases: equity and social determinants. In: Blas E, Sivasankara Kurup A, editors. Equity, social determinants and public health programmes Geneva: World Health Organization; 2010. p. 135–57.
3. Hotez PJ. The Disease Next Door. Foreign Policy. 2013;March 25.
4. Dunn C, Callahan K, Katabarwa M, Richards F, Hopkins D, Withers PC, Jr., et al. The Contributions of Onchocerciasis Control and Elimination Programs toward the Achievement of the Millennium Development Goals. PLoS Negl Trop Dis. 2015;9(5): e0003703. doi: 10.1371/journal.pntd.0003703. PubMed PMID: 25996946; PubMed Central PMCID: PMC4440802
5. Commission on Social Determinants of Health. Closing the gap in a generation: health equity through action on the social determinants of health. Final Report of the Commission on Social Determinants of Health. Geneva: World Health Organization. 2008.

Supporting Information
S1 Supporting Information. Systematic search protocol.
(DOCX)

S2 Supporting Information. PRISMA checklist.
(DOCX)

S1 Table. Summary of the literature on socioeconomic inequalities in LF, 2004–2013.
(DOCX)

S2 Table. Summary of the literature on socioeconomic inequalities in schistosomiasis, 2004–2013.
(DOCX)
S3 Table. Summary of the literature on socioeconomic inequalities in STH, 2004–2013.

(DOCX)

S4 Table. Summary of the literature on socioeconomic inequalities in trachoma, 2004–2013.

(DOCX)

S5 Table. Summary of the literature on socioeconomic inequalities in Chagas’ disease, 2004–2013.

(DOCX)

S6 Table. Summary of the literature on socioeconomic inequalities in leprosy, 2004–2013.

(DOCX)

S7 Table. Summary of the literature on socioeconomic inequalities in visceral leishmaniasis, 2004–2013.

(DOCX)

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References

1. Stolk W, Kulik M, le Rutte E, Jacobson J, Richardus J, de Vlas S, et al. Between-country inequalities in the neglected tropical disease burden in 1990 and 2010, with projections for 2020. PLoS Negl Trop Dis. 2016. In press. doi: 10.1371/journal.pntd.0004560

2. Hotez PJ. The Disease Next Door. Foreign Policy. 2013;March 25.

3. Aagaard-Hansen J, Chaignat CL. Neglected tropical diseases: equity and social determinants. In: Blas E, Sivasankara Kurup A, editors. Equity, social determinants and public health programmes Geneva: World Health Organization; 2010. p. 135–57.

4. Lenk EJ, Redekop WK, Luyendijk M, Rijnsburger AJ, Severens JL. Productivity Loss Related to Neglected Tropical Diseases Eligible for Preventive Chemotherapy: A Systematic Literature Review. PLoS Negl Trop Dis. 2016; 10(2):e0004397. doi: 10.1371/journal.pntd.0004397 PubMed PMID: 26890487; PubMed Central PMCID: PMC4758606.

5. Pullan RL, Bethony JM, Geiger SM, Cundill B, Correa-Oliveira R, Quinnell RJ, et al. Human helminth coinfection: Analysis of spatial patterns and risk factors in a Brazilian community. PLoS Negl Trop Dis. 2008; 2(12): e352. doi: 10.1371/journal.pntd.0000352 PubMed PMID: 986.

6. Abou-Zeid AHA, Abkar TA, Mohamed RO. Schistosomiasis and soil-transmitted helminths among an adult population in a war affected area, Southern Kordofan state, Sudan. Parasites Vectors. 2012; 5(1). doi: 10.1186/1756-3305-5-133 PubMed PMID: 155.

7. Imbiriba EN, Silva Neto AL, Souza WV, Pedrosa V, Cunha MG, Gamelo L. Social inequality, urban growth and leprosy in Manaus: a spatial approach. Rev Saude Publica. 2009; 43(4):656–65. PubMed PMID: 503.

8. Sales AM, Ponce de Leon A, Duppre NC, Hacker MA, Nery JAC, Sarno EN, et al. Leprosy among patient contacts: A multilevel study of risk factors. PLoS Negl Trop Dis. 2011; 5(3): e1013. doi: 10.1371/journal.pntd.0001013 PubMed PMID: 1873.

9. Houweling TA, Kunst AE. Socio-economic inequalities in childhood mortality in low and middle income countries: a review of the international evidence. British Medical Bulletin. 2010; 93(1):7–26.

10. Commission on Social Determinants of Health. Closing the gap in a generation: health equity through action on the social determinants of health. Final Report of the Commission on Social Determinants of Health.
11. Mosley WH, Chen LC. An analytical framework for the study of child survival in developing countries: Child Survival: Strategies for Research. Population and Development Review. 1984; 10, Suppl.:25–45.

12. Bartley M. Health Inequality: An Introduction to Theories, Concepts and Methods. Cambridge: Polity Press; 2004.

13. Evans T, Whitehead M, Diderichsen F, Bhuiya A, Wirth M, editors. Challenging inequities in health: From ethics to action. Oxford: Oxford University Press; 2001.

14. Houweling TAJ. Socio-economic inequalities in childhood mortality in low and middle income countries [PhD thesis]. Erasmus University Rotterdam; 2007. http://hdl.handle.net/1765/11023.

15. Kunst AE, Mackenbach JP. Measuring Socio-economic inequalities in health. Copenhagen: WHO, Regional Office for Europe, 1994.

16. Filmer D, Pritchett L. Estimating Wealth Effects without Expenditure Data—or Tears: An Application to Educational Enrollment in States of India; WB Policy Research Working Paper No. 194. Washington DC: Development Economics Research Group (DECRG) The World Bank, 1998 September 1. Report No.

17. Da Rocha Lucena A, Cruz AAV, Akaishi P. Epidemiology of trachoma in the village of Araripe plateau —Ceara State. Arq Bras Oftalmol. 2010; 73(3):271–5. doi:10.1590/s0004-27492010000300012 PubMed PMID: 2278.

18. Moher D, Liberati A, Tetzlaff J, Altman DG, Group P. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. PLoS Med. 2009; 6(7):e1000097. doi:10.1371/journal.pmed.1000097 PMID: 19621072; PubMed Central PMCID: PMC2707599.

19. Kumar KNV, Ramaiah KD. Usage of personal-protection measures against mosquitoes and the low prevalences of Wuchereria bancrofti microfilaraemia in the Indian city of Chennai. Ann Trop Med Parasitol. 2008; 102(5):391–7. doi:10.1179/136485908x278892 PubMed PMID: 705.

20.ynchonk Ugbomoiko US, Ofoezie IE, Okoye IC, Heukelbach J. Factors associated with urinary schistosomiasis in two peri-urban communities in south-western Nigeria. Ann Trop Med Parasitol. 2010; 104(5):409–19. doi:10.1179/136485910x12743554760469 PubMed PMID: 215.

21. Xu JF, Xu J, Li SZ, Jia TW, Huang XB, Zhang HM, et al. Transmission Risks of Schistosomiasis Japonica: Extraction from Back-propagation Artificial Neural Network and Logistic Regression Model. PLoS Negl Trop Dis. 2013; 7(3):e2123. doi:10.1371/journal.pntd.0002123 PubMed PMID: 141.

22. Yang J, Zhao Z, Li Y, Krewski D, Wen SW. A multi-level analysis of risk factors for Schistosoma japonicum infection in China. Int J Infect Dis. 2009; 13(6):e407–e12. doi:10.1016/j.ijid.2009.02.005 PubMed PMID: 782.

23. Khalid A, Abdelgadir MA, Ashmaig A, Ibrahim AM, Ahmed AAM, Adam I. Schistosoma mansoni infection among prenatal attendees at a secondary-care hospital in central Sudan. Int J Gynecol Obstet. 2012; 116(1):10–2. doi:10.1016/j.ijgo.2011.08.018 PubMed PMID: 1334.

24. Matthys B, Tschannen AB, Tian-Bi NT, Comoe H, Diabate S, Traore M, et al. Risk factors for Schistosoma mansoni and hookworm in urban farming communities in western Cote d’Ivoire. Trop Med Int Health. 2007; 12(6):709–23. doi:10.1111/j.1365-3156.2007.01841.x PubMed PMID: 129.
31. Raso G, Matthys B, N’Goran EK, Tanner M, Vounatsou P, Utzinger J. Spatial risk prediction and mapping of Schistosoma mansoni infections among schoolchildren living in western Cote d’Ivoire. Parasitology. 2005; 131(1):97–108. doi:10.1017/s0031182005007432 PubMed PMID: 1305.

32. Raso G, Utechinger J, Silue KD, Ouattara M, Yapi A, Toty A, et al. Disparities in parasitic infections, perceived ill health and access to health care among poorer and less poor schoolchildren of rural Cote d’Ivoire. Trop Med Int Health. 2005; (10):42–57. doi:10.1111/j.1365-3156.2004.01352.x PubMed PMID: 203.

33. Raso G, Vounatsou P, Singer BH, N’Goran EK, Tanner M, Utzinger J. An integrated approach for risk profiling and spatial prediction of Schistosoma mansoni-hookworm coinfection. Proc Natl Acad Sci U S A. 2006; 103(18):6934–9. doi:10.1073/pnas.0601559103 PubMed PMID: 416.

34. Vounatsou P, Raso G, Tanner M, N’Goran EK, Utzinger J. Bayesian geostatistical modelling for mapping schistosomiasis transmission. PARASITOLOGY. 2009; 136(13):1695–705. PMID:19490724. doi: 10.1017/S003118200900599X

35. Kabatereine NB, Standley CJ, Sousa-Figueiredo JC, Fleming FM, Stothard JR, Talisuna A, et al. Integrated prevalence mapping of schistosomiasis, soil-transmitted helminthiasis and malaria in lakeside and island communities in Lake Victoria, Uganda. Parasites & Vectors. 2011; 4: 232. PMID: WOS:000300371700001.

36. Muhumuza S, Kitimbo G, Oryema-Lalobo M, Nuwaha F. Association between socio economic status and schistosomiasis infection in Jinja District, Uganda. Trop Med Int Health. 2009; 14(6):612–9. doi: 10.1111/j.1365-3156.2009.02273.x PubMed PMID: 266.

37. Balen RJ, Raso G, Li YS, Zhao ZY, Yuan LP, Williams GM, et al. Risk factors for helminth infections in a rural and a peri-urban setting of the Dongting Lake area, People’s Republic of China. Int J Parasitiol. 2011; 41(1):1165–73. doi: 10.1016/j.ijpara.2011.07.006 PubMed PMID: 60.

38. Wang X, Zhang L, Luo R, Wang G, Chen Y, Medina A, et al. Soil-Transmitted Helminth Infections and Correlated Risk Factors in Preschool and School-Aged Children in Rural Southwest China. PLoS ONE. 2012; 7(9): e49393. doi:1371/journal.pone.0049393 PubMed PMID: 136.

39. Zhang LX, Cai YP, Wang XB, Ma XC, Medina A, Smith DS, et al. Neglected Rural Public Health Issue: The Case of Intestinal Roundworms. China & World Economy. 2013; 21(3):25–43. PMID: WOS:000319212800003.

40. Traub RJ, Robertson ID, Irwin P, Mencke N, Thompson RCA. The prevalence, intensities and risk factors associated with geohelminth infection in tea-growing communities of Assam, India. Trop Med Int Health. 2004; 9(6):668–701. doi: 10.1111/j.1365-3156.2004.01252.x PubMed PMID: 333.

41. Wani S, Ahmad F. Intestinal helminths and associated risk factors in children of district Pulwama, Kashmir, India. Indian J Med Microbiol. 2009; 27(1):81–2. PubMed PMID: 4721.

42. Wani SA, Ahmad F, Zargar SA, Ahmad Z, Ahmad P, Tak H. Prevalence of intestinal parasites and associated risk factors among schoolchildren in Srinagar City, Kashmir, India. J PARASITOL. 2007; 93(6):1541–3. PMID:18314713. doi:10.1645/GE-1255.1

43. Mihrshahi S, Casey GJ, Montresor A, Phuc TQ, Thach DTC, Tien NT, et al. The effectiveness of 4 monthly albendazole treatment in the reduction of soil-transmitted helminth infections in women of reproductive age in Viet Nam. Int J Parasitiol. 2009; 39(9):1037–43. doi: 10.1016/j.ijpara.2009.01.013 PubMed PMID: 385.

44. Pham-Duc P, Nguyen-Viet H, Hattendorf J, Zinsstag J, Phung-Dac C, Zurbrugg C, et al. Ascaris lumbricoides and Trichuris trichiura infections associated with wastewater and human excreta use in agriculture in Vietnam. Parasitol Int. 2013; 62(2):321–31. doi:10.2166/wh.2012.007 PubMed PMID: 220.

45. Trang DT, Molbak K, Cam PD, Dalsgaard A. Helminth infections among people using wastewater and human excreta in peri-urban agriculture and aquaculture in Hanoi, Vietnam. Trop Med Int Health. 2007; 12(SUPPL. 2):82–90. doi: 10.1111/j.1365-3156.2007.01945.x PubMed PMID: 415.

46. Trang DT, van der Hoek W, Cam PD, Vinh KT, Van Hoa N, Dalsgaard A. Low risk for helminth infection in wastewater-fed rice cultivation in Vietnam. J Water Health. 2006; 4(3):321–31. doi: 10.2166/wh.2006.013 PubMed PMID: 154.

47. Uga S, Hoa NTV, Thuan LK, Noda S, Fujimaki Y. Intestinal parasitic infections in schoolchildren in a suburban area of Hanoi, Vietnam. Southeast Asian J Trop Med Public Health. 2005; 36(4):1407–11. PubMed PMID: 3682.

48. Hesham Al-Mekhlafi M, Surin J, Atiya AS, Ariffin WA, Mohammed Mahdy AK, Che Abdullah H. Pattern and predictors of soil-transmitted helminth reinfection among aboriginal schoolchildren in rural Peninsular Malaysia. Acta Trop. 2008; 107(2):200–4. doi:10.1016/j.actatropica.2008.05.022 PubMed PMID: 114.
49. Hesham Al-Mekhlafi MS, Atiya AS, Lim YAL, Mohammed Mahdy AK, Wan Ariffin WA, Che Abdullah H, et al. An unceasing problem: Soil-transmitted helminthiases in rural Malaysian communities. Southeast Asian J Trop Med Public Health. 2007; 38(6):998–1007. PubMed PMID: 205.

50. Ngui R, Ishak S, Chuen CS, Mahmud R, Lim YAL. Prevalence and risk factors of intestinal parasitism in rural and remote West Malaysia. PLoS Negl Trop Dis. 2011; 5(3): e974. doi: 10.1371/journal.pntd.0000974 PubMed PMID: 1337.

51. Ekpo UF, Odoemene SN, Maflana CF, Sam-Wobo SO. Helminthiasis and hygiene conditions of schools in Ikeneke, Ogun State, Nigeria. PLoS Negl Trop Dis. 2008; 2(1): e146. doi: 10.1371/journal.pntd.0000146 PubMed PMID: 282.

52. Kirwan P, Asaolu SO, Abiona TC, Jackson AL, Smith HV, Holland CV. Soil-transmitted helminth infections in Nigerian children aged 0–25 months. J HELMINTHOL. 2009; 83(3):261–6. PMID: 19356265. doi: 10.1017/S0022149X08201252.

53. Ugbomoiko US, Dalumo V, Ofosiezie IE, Obiezie RNN. Socio-environmental factors and ascariasis infection among school-aged children in Ilobu, Osun State, Nigeria. Trans R Soc Trop Med Hyg. 2008; 103(3):223–8. doi: 10.1016/j.trstmh.2008.05.012 PubMed PMID: 406.

54. Brooker S, Jardim-Botelho A, Quinell RJ, Geiger SM, Caldas IR, Fleming F, et al. Age-related changes in hookworm infection, anaemia and iron deficiency in an area of high Nectoracrus americanus hookworm transmission in south-eastern Brazil. Trans R Soc Trop Med Hyg. 2007; 101(2):146–54. doi: 10.1016/j.trstmh.2006.05.012 PubMed PMID: 702.

55. Carvalho-Costa FA, Goncalves AQ, Lassance SL, Da Silva Neto LM, Salmazo CAA, Boia MN. Giardia lamblia and other intestinal parasitic infections and their relationships with nutritional status in children in Brazilian Amazon. Rev Inst Med Trop Sao Paulo. 2007; 49(3):147–53. PubMed PMID: 1904.

56. Cundill B, Alexander N, Bethony JM, Diemert D, Pullan RL, Brooker S. Rates and intensity of re-infection with human helminths after treatment and the influence of individual, household, and environmental factors in a Brazilian community. Parasitology. 2011; 138(11):1406–16. doi: 10.1017/ s0031182011001132 PubMed PMID: 1200.

57. de Carvalho TB, de Carvalho LR, Mascarini LM. Occurrence of enteroparasites in day care centers in Botucatu (Sao Paulo State, Brazil) with emphasis on Cryptosporidium sp., Giardia duodenalis and Enterobius vermicularis. REV INST MED TROP SAO PAULO. 2006; 48(5):269–73. PMID: 17086314.

58. Fonseca EOL, Teixeira MG, Barreto ML, Carneiro EH, Costa MCN. Prevalence and factors associated with geohelminth infections in children living in municipalities with low HDI in North and Northeast Brazil. Cad Saude Publica. 2010; 26(1):143–52. PubMed PMID: 508.

59. Lander RL, Lander AG, Houghton L, Williams SM, Costa-Ribeiro H, Barreto DL, et al. Factors influencing growth and intestinal parasitic infections in preschoolers attending philanthropic daycare centers in Salvador, Northeast Region of Brazil. Cad Saude Publica. 2012; 28(11):2177–88. doi: 10.1590/ s0102-311x2012001100017 PubMed PMID: 1504.

60. Maia MMM, Fausto MA, Vieira ELM, Benetol MFLN, Cameiro M. Intestinal parasitic infection and associated risk factors, among children presenting at outpatient clinics in Manaus, Amazonas state, Brazil. Ann Trop Med Parasitol. 2009; 103(7):983–91. doi: 10.1179/000349809x12459740922417 PubMed PMID: 946.

61. Scholte RGC, Schur N, Bavia ME, Carvalho EM, Chammartin F, Utzinger J, et al. Spatial analysis and risk mapping of soil-transmitted helminth infections in Brazil, using Bayesian geostatistical models. Geospat Health. 2013; 8(1):97–110. PMID: 24258887.

62. Valverde JG, Gomes-Silva A, de Carvalho CJM, de Souza DL, Jaeger LH, Martins PP, et al. Prevalence and epidemiology of intestinal parasitism, as revealed by three distinct techniques in an endemic area in the Brazilian Amazon. Ann Trop Med Parasitol. 2011; 105(6):413–26. doi: 10.1179/1364859411y.0000000034 PubMed PMID: 143.

63. Parajuli RP, Umezaki M, Watanabe C. Behavioral and nutritional factors and geohelminth infection among two ethnic groups in the Terai region, Nepal. Am J Hum Biol. 2009; 21(1):98–104. doi: 10.1002/ajhb.20825 PubMed PMID: 906.

64. Mehraj V, Hatcher J, Akhtar S, Rafique G, Beg MA. Prevalence and Factors Associated with Intestinal Parasitic Infection among Children in an Urban Slum of Karachi. PLoS ONE. 2008; 3(11): e3680. PMID: WOS:000265166500003. doi: 10.1371/journal.pone.0003680.

65. Taye B, Alemayehu B, Birhanu A, Desta K, Addisus S, Petros B, et al. Podocytosis and Soil-Transmitted Helminths (STHs): Double Burden of Neglected Tropical Diseases in Wolaita Zone, Rural Southern Ethiopia. PLoS Negl Trop Dis. 2013; 7(3): e2128. doi: 10.1371/journal.pntd.0002128 PubMed PMID: 1380.

66. Alvarado BE, Vasquez LR. Social determinants, feeding practices and nutritional consequences of intestinal parasitism in young children. Biomedica. 2006; 26(1):82–94. PubMed PMID: 4048.
67. Knopp S, Mohammed KA, Stothard JR, Khamis IS, Rollinson D, Marti H, et al. Patterns and risk factors of helminthiasis and anemia in a rural and a peri-urban community in Zanzibar, in the context of helminth control programs. PLoS Negl Trop Dis. 2010; 4(5): e681. doi:10.1371/journal.pntd.0000681 PubMed PMID: 230.

68. Liabsuetrakul T, Chaikongkeit P, Korviwattanagarn S, Petrueng C, Chaiya S, Hanvattanakul C, et al. Epidemiology and the effect of treatment of soil-transmitted helminthiasis in pregnant women in Southern Thailand. Southeast Asian J Trop Med Public Health. 2009; 40(2):211–22. PubMed PMID: 2080.

69. Gelaw A, Anagaw B, Nigussie B, Silesh B, Yirga A, Alem M, et al. Prevalence of intestinal parasitic infections and risk factors among schoolchildren at the University of Gonder Community School, Northwest Ethiopia: a cross-sectional study. BMC Public Health. 2013; 13:304. doi:10.1186/1471-2458-13-304 PubMed PMID: 2080.

70. Jombo GT, Damen JG, Safiyanu H, Odey F, Mbaawuaga EM. Human intestinal parasitism, potable water availability and methods of sewage disposal among nomadic Fulanis in Kuraje rural settlement of Zamfara state. Asian Pac J Trop Med. 2010; 3(6):491–81. doi:10.1016/s1995-7645(10)60118-6 PubMed PMID: 1856.

71. Ugbomoiko US, Dalumo V, Ofoezie IE, Obiezue RNN. Socio-environmental factors and ascariasis infection among school-aged children in Ilobu, Osun State, Nigeria. Transactions of The Royal Society of Tropical Medicine and Hygiene. 2009; 103(3):223–8. doi:10.1186/1471-2458-13-304 PubMed PMID: 2080.

72. Regassa K, Teshome T. Trachoma among adults in Damot Gale District, South Ethiopia. Ophthalmic Epidemiol. 2004; 11(1):9–16. doi:10.1076/opep.11.1.9.26440 PubMed PMID: 2219.

73. Vinke C, Lonergan S. Social and environmental risk factors for trachoma: a mixed methods approach in the Kembata Zone of southern Ethiopia. Canadian Journal of Development Studies-Revue Canadienne D Etudes Du Developpement. 2011; 32(3):254–68. PMID: WOS:000299594500003.

74. Edwards T, Smith J, Sturrock HJW, Kur LW, Sabasio A, Finn TP, et al. Prevalence of Trachoma in Unity State, South Sudan: Results from a large-scale population-based survey and potential implications for further surveys. PLoS Negl Trop Dis. 2012; 6(4): e1585. doi:10.1371/journal.pntd.0001585 PubMed PMID: 734.

75. Harding-Esch EM, Edwards T, Mkocha H, Munoz B, Holland MJ, Burr SE, et al. Trachoma prevalence and associated risk factors in the Gambia and Tanzania: Baseline results of a cluster randomised controlled trial. PLoS Negl Trop Dis. 2010; 4(11): e861. 0.1371/journal.pntd.0000861 PubMed PMID: 858. doi:10.1371/journal.pntd.0000861

76. Jansen E, Baltussen RMP, Van Doorslaer E, Ngirawamungu E, Nguyen MP, Kilima PM. An eye for inequality: How trachoma relates to poverty in Tanzania and Vietnam. Ophthalmic Epidemiol. 2007; 14(5):278–87. doi:10.1080/09286580701299403 PubMed PMID: 164.

77. Polack S, Kuper H, Solomon AW, Massae PA, Abuelo C, Cameron E, et al. The relationship between prevalence of active trachoma, water availability and its use in a Tanzanian village. TRANS R SOC TROP MED HYG. 2006; 100(11):1075–83. PMID: 16546229.

78. Borges-Pereira J, de Castro JAF, da Silva AG, Zauza PL, Bulhoes TP, Goncalves ME, et al. [Sero-prevalence of Chagas disease infection in the State of Piaui, 2002] Portuguese. Rev Soc Bras Med Trop. 2006; 39(6):530–9. PMID: 17308697.

79. Pinto FS, de Andrade GMQ, Januario JN, Maia MCA, Gontijo ED. Epidemiological profile of Trypanosoma cruzi-infected mothers and live birth conditions in the State of Minas Gerais, Brazil. Revista Da Sociedade Brasileira De Medicina Tropical. 2013; 46(2):196–9. PMID: WOS:000319625400014.
84. Llovet I, Dinardi G, de Maio FG. Mitigating social and health inequities: Community participation and Chagas disease in rural Argentina. Global Public Health. 2011; 6(4):371–84. doi: 10.1080/17441692.2010.539572 PubMed PMID: 753.

85. Cucunuba ZM, Florez AC, Cardenas A, Pavia P, Montilla M, Aldana R, et al. Prevalence and risk factors for chagas disease in pregnant women in Casanare, Colombia. Am J Trop Med Hyg. 2012; 87(5):837–42. doi: 10.4269/ajtmh.2012.12–0086 PubMed PMID: 841.

86. Murto C, Chammartin F, Schwarz K, da Costa LMM, Kaplan C, Heukelbach J. Patterns of Migration and Risks Associated with Leprosy among Migrants in Maranhao, Brazil. PLoS Negl Trop Dis. 2013; 7(9):e2422. doi: 10.1371/journal.pntd.0002422 PubMed PMID: 1560.

87. Dos Santos DS, Duppre NC, Sales AM, Nery JADC, Sarno EN, Hacker MA. Kinship and leprosy in the contacts of leprosy patients: Cohort at the Souza Araujo outpatient clinic, Rio de Janeiro, RJ, 1987–2010. J Trop Med. 2013. doi: 10.1155/2013/596316 PubMed PMID: 1844.

88. Hacker MdA, Duppre NC, Nery JAC, Sales AM, Sarno EN. Characteristics of leprosy diagnosed through the surveillance of contacts: a comparison with index cases in Rio de Janeiro, 1987–2010. Mem Inst Oswaldo Cruz. 2012; 107 Suppl 1:49–54. PMID: 23283453.

89. Silva DRX, Ignotti E, Reinaldo SS, De Souza Hacon S. Hansen's disease, social conditions, and deforestation in the Brazilian Amazon. Rev Panam Salud Publica Pan Am J Public Health. 2010; 27(4):268–75. PubMed PMID: 2358.

90. Schmitt JV, Dechandt IT, Dopke G, Ribas ML, Cerci FB, Viesi JMZ, et al. Armadillo meat intake was not associated with leprosy in a case control study, Curtiba (Brazil). Mem Inst Oswaldo Cruz. 2010; 105(7):857–62. PubMed PMID: 1951.

91. Kerr-Pontes LRS, Barreto ML, Evangelista CMN, Rodrigues LC, Feldmeier H. Socio-economic, environmental, and behavioural risk factors for leprosy in North-east Brazil: Results of a case-control study. Int J Epidemiol. 2006; 35(4):994–1000. doi: 10.1093/ije/dyq672 PubMed PMID: 968.

92. Feenstra SG, Nahar Q, Pahan D, Oskam L, Richardus JH. Recent food shortage is associated with leprosy disease in Bangladesh: A case-control study. PLoS Negl Trop Dis. 2011; 5(5):e1029. doi: 10.1371/journal.pntd.0001029 PubMed PMID: 344.

93. Bhunia GS, Chatterjee N, Kumar V, Siddiqui NA, Mandal R, Das P, et al. Delimitation of kala-azar risk areas in the district of Vaishali in Bihar (India) using a geo-environmental approach. Mem Inst Oswaldo Cruz. 2012; 107(5):609–20. doi: 10.1590/s0074-02762012000500007 PubMed PMID: 2183.

94. Boelart M, Meheus F, Sanchez A, Singh SP, Vanliverbergh V, Picado A, et al. The poorest of the poor: A poverty appraisal of households affected by visceral leishmaniasis in Bihar, India. Trop Med Int Health. 2009; 14(1):63–44. doi: 10.1111/j.1365-3156.2009.02279.x PubMed PMID: 595.

95. Ranjan A, Sur D, Singh VP, Siddiqui NA, Manna B, Lal CS, et al. Risk factors for Indian kala-azar. AM J TROP MED HYG. 2005; 73(4):268–74. doi: 10.4269/ajtmh.2005.73.44. PMID: 15814837.

96. Saha S, Ramachandran R, Hutin YJF, Gupte MD. Visceral leishmaniasis is preventable in a highly endemic village in West Bengal, India. Trans R Soc Trop Med Hyg. 2009; 103(7):737–42. doi: 10.1016/j.trstmh.2008.10.006 PubMed PMID: 1210.

97. Singh SP, Hasker E, Picado A, Gidwani K, Malaviya P, Singh RP, et al. Risk factors for visceral leishmaniasis in India: Further evidence on the role of domestic animals. Trop Med Int Health. 2010; 15(7):74–8. PMID: 16014837.

98. Hasker E, Singh SP, Malaviya P, Picado A, Gidwani K, Singh RP, et al. Visceral leishmaniasis in rural Bihar, India. Emerg Infect Dis. 2012; 18(10):1662–4. doi: 10.3201/eid1810.120162 PubMed PMID: 3875.

99. Bern C, Hightower AW, Chowdhury R, Ali M, Amann J, Wagatsuma Y, et al. Risk factors for kala-azar in Bangladesh. Emerg Infect Dis. 2005; 11(5):655–62. PubMed PMID: 1604.

100. Ferdousi F, Alam MS, Hossain MS, Ma E, Itoh M, Mondal D, et al. Visceral leishmaniasis eradication is a reality: Data from a community-based active Surveillance in Bangladesh. Trop Med Health. 2012; 40(4):133–9. doi: 10.2149/1h.2012.12–25 PubMed PMID: 976.

101. Argaw D, Mulugeta A, Herrero M, Nombela N, Teklu T, Tefera T, et al. Risk Factors for Visceral Leishmaniasis among Residents and Migrants in Kafta-Humera, Ethiopia. PLoS Negl Trop Dis. 2013; 7(11):e2453. PMID: 24244778. doi: 10.1371/journal.pntd.0002453

102. Kolaczinski JH, Reithinger R, Worku DT, Ochong A, Kasirimo J, Kabaterine N, et al. Risk factors of visceral leishmaniasis in East Africa: A case-control study in Pokot territory of Kenya and Uganda. Int J Epidemiol. 2008; 37(2):344–52. doi: 10.1093/ije/dym275 PubMed PMID: 1387.

103. Borges BKA, Da Silva JA, Haddad JPA, Moreira EC, De Magalhaes DF, Ribeiro LML, et al. Assessment of knowledge and preventive attitudes concerning visceral leishmaniasis in Belo Horizonte,
104. De Almeida AS, Medronho RDA, Werneck GL. Identification of risk areas for visceral leishmaniasis in Teresina, Piauí State, Brazil. Am J Trop Med Hyg. 2011; 84(5):681–7. doi:10.4269/ajtmh.2011.10–0325 PubMed PMID: 2341.

105. de Araujo VEM, Pinheiro LC, de Almeida MCM, de Menezes FC, Morais MHF, Reis IA, et al. Relative Risk of Visceral Leishmaniasis in Brazil: A Spatial Analysis in Urban Area. PLoS Negl Trop Dis. 2013; 7(11): e2540. doi:10.1371/journal.pntd.0002540 PubMed PMID: 2360.

106. De Oliveira ALL, Paniago AMM, Sanches MA, Dorval MEC, Oshiro ET, Leal CRB, et al. Asymptomatic infection in family contacts of patients with human visceral leishmaniasis in Tres Lagoas, Mato Grosso do Sul State, Brazil. Cad Saude Publica. 2008; 24(12):2827–33. doi:10.1590/s0102-311x2008001200011 PubMed PMID: 3500.

107. Gouvea MV, Werneck GL, Costa CHN, de Amorim Carvalho FA. Factors associated to Montenegro skin test positivity in Teresina, Brazil. Acta Trop. 2007; 104(2–3):99–107. doi:10.1016/j.actatropica.2007.07.010 PubMed PMID: 3532.

108. Lima ID, Queiroz JW, Lacerda HG, Queiroz PVS, Pontes NN, Barbosa JDA, et al. Leishmania infantum chagasi in Northeastern Brazil: Asymptomatic infection at the urban perimeter. Am J Trop Med Hyg. 2012; 86(1):99–107. doi:10.4269/ajtmh.2012.10–0492 PubMed PMID: 260.

109. de Vlas SJ, Stolk WA, le Rutte EA, Hontelez JA, Bakker R, Blok DJ, et al. Concerted Efforts to Control or Eliminate Neglected Tropical Diseases: How Much Health Will Be Gained? PLoS Negl Trop Dis. 2016; 10(2):e0004386. doi:10.1371/journal.pntd.0004386 PMID: 26890362; PubMed Central PMCID: PMC4758649.

110. Anderson RM, May RM. Infectious diseases of humans: dynamics and control. New York: Oxford University Press; 1992.

111. Alvar J, Yactayo S, Bern C. Leishmaniasis and poverty. Trends Parasitol. 2006; 22(12):552–7. doi:10.1016/j.pt.2006.09.004 PubMed PMID: 228.

112. Gwatkin DR, Wagstaff A, Yazbeck AS, editors. Reaching the poor with Health, Nutrition, and Population Services: What Works, What Doesn’t, and Why. Gwatkin D, Wagstaff A, Yazbeck A, editors. Washington: World Bank; 2005.

113. World Health Organisation. Investing to overcome the global impact of neglected tropical diseases: Third WHO report on neglected tropical diseases. Geneva: World Health Organization, 2015.

114. Bethony J, Brooker S, Albonico M, Geiger SM, Loukas A, Diemert D, et al. Soil-transmitted helminth infections: ascariasis, trichuriasis, and hookworm. Lancet. 2006; 367(9521):1521–32. doi:10.1016/S0140-6736(06)68653-4 PMID: 16679166.