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at 18 sentinel sites. A total of >10,000 children were tested in the three surveys. Household data included information on spraying, house construction, bednet use, illness histories and indicators of household wealth. Average prevalence of infection with *Plasmodium falciparum* at all sites combined reduced in all age groups 2 to <15 years from 45% at baseline to 31% in 2005 (p<0.001) and 26% in 2006 (p<0.03), with substantial between site variation. Reported site specific spray coverage of houses in the 2006 survey ranged from 58% to 87%. Odds of infection was significantly lower for children living in houses that had been sprayed (OR=0.67 relative to unsprayed houses, p<0.001), regardless of whether the child slept under a bednet or not. The odds of infection of an individual child decreased by a factor of 0.94 (p=0.035) for every one percent increase in spray coverage of the neighbourhood in which the child lived, independent of the spray status of her/his own home. Desire to have houses sprayed was uniformly high (mean 92%). Substantial overall reduction in prevalence of infection with *P. falciparum* in children can be achieved in equatorial settings provided a high level of spray coverage is maintained. Risk of infection is simultaneously related to the spray status of the child's home and to the spray-coverage achieved in the neighbourhood of the house.

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USING TREATMENT FAILURE TO SCREEN FOR MDR TB IS ASSOCIATED WITH RECURRENCE, DEATH AND TRANSMISSION

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In many resource-poor settings, TB drug susceptibility testing for all newly-diagnosed patients using traditional methods is not feasible. Instead, patients are given a trial of first-line drugs, and those who fail are then tested for MDR TB. This strategy seems to reduce both diagnostic and treatment costs because many MDR TB patients appear cured by first-line agents. No data exists, however, concerning the long term outcome of MDR TB patients "cured" by first-line drugs. 351 patients from a community hospital in Lima, Peru were enrolled after new diagnoses of TB disease. Patients were tested for resistance to rifampicin and isoniazid, followed throughout treatment, and interviewed a median of 60 months after treatment. This long term follow up established TB-related morbidity and mortality in both index cases and contacts. Cases of recurrence or contact TB were confirmed with health post records. Despite laboratory test results reporting 21 cases of MDR TB to patients and physicians, all 351 enrolled patients received a complete trial of first-line agents. Twelve of 21 patients(57%) with laboratory confirmed MDR TB converted to sputum smear negativity and were considered "cured" by first-line drugs alone. At long term follow up, however, patients cured of MDR TB were more likely to suffer recurrence (HR=18, 95%CI=7-45, p<0.001) and to die of TB (HR=7, 95%CI=1.4-38, p=0.018) than patients cured of non-MDR TB in Cox proportional hazard regressions accounting for differences in HIV prevalence and other risk factors for new TB infection. Among MDR TB patients, "cure" was not associated with a statistically significant reduction in long-term mortality (p=0.3). In addition, contacts of "cured" MDR TB patients suffered four times as many episodes of new TB as contacts of cured non-MDR TB patients during follow up (HR=3.8, 95%CI=2-9, p=0.001). There was similar contact TB incidence between MDR and non-MDR households prior to the the study, implying that delayed MDR TB diagnosis may have caused increased transmission. In conclusion, the majority of MDR TB patients appeared cured by first line agents, but usually this was a false cure, and MDR TB patients treated with first-line drugs were at high risk of TB relapse, death, and transmission. Using treatment failure to identify drug resistance underestimated MDR TB, and the resultant delays in optimal therapy were associated with mortality and MDR TB dissemination.

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HUMAN CELL-MEDIATED IMMUNITY AGAINST *MYCOBACTERIUM TUBERCULOSIS* ANTIGENS IS AUGMENTED BY TREATING INTESTINAL HELMINTHS

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Tuberculosis and intestinal helminth infections frequently co-exist and helminths cause immunosuppression, micronutrient deficiency and anergy. We hypothesized that treating intestinal helminths may augment antimycobacterial immunity. A double-blind, randomized, placebo-controlled trial in 140 healthy adults living in the Peruvian Amazon. Anti-mycobacterial immunity was assessed by measuring the size of cutaneous induration 48 hours after a 5 unit intradermal tuberculin skin test and by quantifying γ-interferon secretion following whole-blood stimulation with the specific *Mycobacterium tuberculosis* antigens ESAT-6 and CFP-10 (the Quantiferon in-the-tube assay). These *in vivo* and *in vitro* tests were performed at recruitment and 4 weeks after treatment with 3 daily doses of placebo or 400mg albendazole. Stools were examined by direct and concentrated quantitative microscopy. A stool examination at recruitment diagnosed intestinal helminths in 48% of 126 participants. 40% were infected with *Ascaris lumbricoides*, 12% *Trichuris trichuria*, 6.3% hookworms, and 3.2% *Strongyloides stercoralis*. Albendazole therapy caused helminth prevalence to fall to 6.9% (4/58) 2 weeks later (P=0.003) and to 15% (7/48) 4 weeks later (P<0.001). Eosinophil counts fell from median 271 cells/mm^3 at recruitment to 201 cells/mm^3 4 weeks after albendazole therapy (P=0.002). At recruitment, 39% (52/135) of Quantiferon assays were positive and this did not change significantly with albendazole therapy. In contrast, 56% (78/139) of participants were tuberculin skin test positive at recruitment and albendazole therapy caused tuberculin skin tests to increase in size compared wth placebo (P=0.03). In conclusion, treating intestinal helminths significantly augmented antimycobacterial immunity *in vivo* over a one month interval. Therefore, the interpretation of tuberculin skin test results may be complicated by antihelminthic treatment. Prevention or treatment of intestinal helminths warrants evaluation as a potential strategy for reducing tuberculosis susceptibility.