Ascariasis and childhood malnutrition

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...that common Round Worm which Children usually are troubled with...—Edward Tyson MD, 1683

In 1968, the World Health Organization published a volume entitled Interactions of Nutrition and Infection (SCRIMSHAW et al., 1968). The preface opened with these words: "That malnutrition increases susceptibility to infectious disease seems a reasonable assumption, and clinical observation in areas where malnutrition is common has generally lent support to this belief. Equally reasonable is the supposition that infectious diseases have an adverse effect on the nutritional state." The authors' analysis of the interactions between parasitism and nutrition laid the foundation for much experimental and clinical research and for many investigations in communities where parasitic disease is endemic and where malnutrition prevails. They stimulated a vigorous debate between those who concurred with the notion that parasitic infections impair human nutritional status and those who were somewhat sceptical of the evidence and the manner in which it was being obtained. Not surprisingly, the proposed role of Ascaris lumbricoides as a determinant of childhood nutritional status was discussed extensively. Was Tyson right? Does A. lumbricoides usually trouble children?

At least 4 general types of complex interaction appear to link helminth infection and host nutrition. Firstly, many types of helminth are transmitted in the host's diet often as uncooked contaminants of a range of foodstuffs (COOMBS & CROMPTON, 1991). Secondly, in addition to obtaining food and energy for themselves, all hosts at some time provide nutrients and energy for their helminths either directly from chyme and digestion products in the gut or indirectly, and more usually, at the expense of their own tissues and metabolites. Thirdly, host food intake, digestion, absorption, metabolism, growth, and development may be impaired to varying degrees during the course of a helminth infection. Fourthly, the population biology, growth, survival, and reproduction of helminths may be altered in association with the quality and quantity of food obtained and metabolized by the host (BUNDY & GOLDEN, 1987; CROMPTON, 1987). The general effects do not occur in any sequence; they are interrelated and often unpredictable in their extent or significance. The situation is made even more complex by the fact that the host's resistance to infection is affected by nutritional factors (CHANDRA & NEWBERNE, 1977; CHANDRA, 1980) and some of the agents of the immune response, particularly cytokines released during infection (KLASING, 1988), influence nutritional status.

Direct evidence for the existence of interactions between helminth infections and host nutrition is based overwhelmingly on the results of controlled experiments with laboratory rodents (PARSHAD et al., 1980; CROMPTON et al., 1981; STOrey, 1982a, 1982b; NESHEIM, 1984; SLATER & KEYMER, 1986; MICHAEL & BUNDY, 1991), domesticated pigs (FORSUM et al., 1981) and sheep (SYKES & COOP, 1977; SYMONS, 1976, 1985). The paradigm for these and many other studies has been the measurement of the daily intake of purified diets by infected and uninfected subjects before and after some form of intervention (WHO, 1981; STEPHENSON, 1987; THEIN HLAING, 1989). Under these circumstances, the nature of the study design becomes crucial because detection of causation rather than association is required. Critics of the view that a helminth infection impairs human nutrition can usually resort to a flawed study design in support of their case. Of course, flawed study designs may equally well explain why connections between infection and nutritional status have not always been detected in other investigations.

Much of my experience in this field has been gained through attempts to disprove a seemingly simple null hypothesis: infection with A. lumbricoides does not contribute to childhood malnutrition. This topic has been argued about for years and the story can be traced through the many articles and reviews cited by JOICFP (1980), SCHULTZ (1982), CROMPTON & NESHEIM (1982, 1984), CROMPTON (1984, 1985), CHAGAS & KEUSCH (1985), CROMPTON et al. (1985), STEPHENSON (1987), TENN & CROMPTON (1989), and TOMKINS & WATSON (1989). A consensus is now emerging that ascariasis is indeed a factor in the etiology and persistence of childhood malnutrition and it seems appropriate to highlight a few of the results that have helped to formulate this view.

During a study of preschool children in 4 villages in Uttar Pradesh, where ascariasis and protein malnutrition were prevalent, GUPTA et al. (1977) discovered that infected children given an anthelmintic drug showed a significant improvement in body weight gain after 8 months (P<0.01) when compared with similar children given a placebo. WILLETT et al. (1979), working in Tanzania with preschool children known to be infected with A. lumbricoides, observed that the rate of weight gain in those given monthly doses of levamisole over a year was 21% greater (P<0.03) than in those given a placebo. STEPHENSON et al. (1980) carried out a longitudinal study with Ascaris-infected preschool children in rural Kenya and again demonstrated significant improvements in their nutritional status (P<0.05-P<0.0005) following chemotherapy with levamisole. Most convincingly, THEIN HLAING et al. (1991) demonstrated significant gains in both height and weight (P<0.001) following treatment with levamisole to expel A. lumbricoides from children living in rural Myanmar. The mechanisms underlying these improvements in growth are still far from being fully explained, but malabsorption (TREATH & et al., 1972), lactose malabsorption (CARELLA et al., 1984; TAREN et al., 1987), impaired intestinal permeability (NORTHROP et al., 1987) and reduced food in-
take (Fasli Jalal, 1991) have been detected in Ascaris-infected children. Of 25 studies that have been published since that of Gupta et al. (1977) on the relationship between ascariasis and childhood malnutrition, only 5 appear not to implicate A. lumbricoides. In some cases concurrent infections of A. lumbricoides with Trichuris trichiura and hookworm exacerbate childhood malnutrition. More and more evidence has been accumulating recently to reveal the adverse effects of trichuriasis on the nutritional status of children (Cooper & Bundy, 1988; Cooper et al., 1990; Robertson et al., 1992). The pathological effects of hookworm disease have long been known (Gilles et al., 1964; Roche & Layrisse, 1966) and clearly those have profound nutritional effects (Crompton & Stephenson, 1990).

This body of information has helped to define the public health significance of ascariasis and other soil-transmitted helminthiasis. The responsibility of scientists is to acquire information by the most reliable means at their disposal and make it available and understandable in the public domain. But who is responsible for taking this information and using it for the public good? Scientists must inform, they may draw attention to matters they judge to be worthy of concern, they may plead for action, but it is not for them to formulate and implement health policies; that should be the responsibility of governments. We have seen the campaign to improve the health and nutrition of millions of children in developing countries gain considerable ground. The United Nations Subcommittee on Nutrition and the World Health Organization (ACC/SCN, 1989) endorsed the recommendation that in areas where the prevalence of moderate underweight in children is greater than 25%, and where parasites are known to be widespread, high priority should be given to deworming programmes for treatment of parasites. Savioli et al. (1992) have summarized in this journal some of the benefits that will accrue when actions are taken to combat intestinal parasitic infections. Let us share their optimism and let us encourage Ministries of Health to take up the challenge; many clinicians, epidemiologists, parasitologists and nutritionists are waiting to help. Now is the time to emphasize the twin goals of nutrition improvement and control of infection in programmes of primary health care (Scrimshaw, 1989).

References


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