

by older men clearly points to this conclusion. The fact that the risk of tuberculous infection has been slowly declining³ may well mean that the natural decline constitutes a more powerful force than an increase in the number of sources of infection – chronic cases produced by an ineffective treatment programme. Even very poor treatment, however, saves many lives and reduces suffering.⁴

2. The rather low ('not alarming') prevalence of initial drug resistance may in part be due to the fact that many chronic cases of tuberculosis in India do not harbour resistant bacilli having defaulted early in the course of previous therapy, and in part to the fact that during the decline phase of a tuberculosis epidemic new cases increasingly arise from remote infections (acquired often in the pre- or early anti-microbial era) rather than from recent infections.
3. Using Styblo's formula of 49 new smear-positive cases being equivalent to 1% of the risk of infection,⁵ the estimated number of new smear-positive cases in India would be less than 300 000 and the total bacteriologically proven cases less than 500 000 using the risk of infection of 0.6%. Even allowing for the large number of chronic cases produced by the ineffective treatment programme the estimate of 2 million bacteriologically positive cases appears to be excessive. It is difficult to understand Chakraborty's estimate of another 11.2 million sputum-negative cases requiring treatment as bacteriological confirmation of activity is well over 50% in most jurisdictions and over 75% in many.
4. The task facing decision-makers in India appears to be simpler than Dr. Chakraborty's letter would indicate. It is to give supervised, intermittent treatment consisting of some 50 doses (or perhaps fewer if further research so indicates) to some 30 smear-positive patients for each population of 100 000. Such treatment, coupled with the special effort to cure chronic bacillary excretors, would accelerate significantly the decline of tuberculosis in India.⁶

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Staining for acid-fast bacilli: hot or cold?

Since 1991 we have been carrying out so-called 'external quality controls' on direct sputum-smears in our laboratory,¹ and have compared the reading results between the service and reference laboratories according to the reading schedule recommended by the IUATLD.² We have frequently noticed a poor staining quality¹ and have always urged the project laboratories to perform the hot carbol-fuchsin stain (= heating of the slides) for acid-fast bacilli (AFB).²

Back in 1991 we carried out parallel stainings at our Institute for hot and cold (= Kinyoun) carbol-fuchsin stains (non-heating of the slides). We found that some batches of carbol-fuchsin purchased from different companies were unsuitable for the cold stain (the bacilli stained too pale to be able to detect them comfortably), especially if only a few bacilli were present (1–10 AFB/100 fields and 10–100 AFB/100 fields). We felt that this may lead to false negative results.

The purchase of bulk carbol-fuchsin stains for the programmes is not carried out by trained bacteriologists. Furthermore, often the cheapest product is bought to save costs. It is not common knowledge that not all carbol-fuchsin stains from all chemical companies are suitable for the cold carbol-fuchsin stain. We would therefore strongly suggest that all programmes adhere to the original recommendations given by the IUATLD² and that only the hot carbol-fuchsin stain be used. We would also like to recommend that the staining time for the hot carbol-fuchsin stain be not less than 15 min. This will ensure the adequate staining of AFB.

This may be a rather moot point. However, it may be important enough to consider in the laboratory diagnosis of tuberculosis in developing countries, where reliance on direct sputum-smear examinations is still of paramount importance.

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