



Effectiveness of community participation in tuberculosis control

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To the Editor: Tuberculosis (TB) has re-emerged as an important global public health issue, particularly with the advent of HIV and AIDS.¹⁻³ Sub-Saharan Africa has the highest incidence of TB in the world, estimated at over 350 cases per 100 000.¹ With many of its provinces being rural and characterised by limited access to health services, South Africa ranked fifth globally among high TB burden countries in 2007.⁴ In low-resource settings where health systems barely cope with increased disease burdens, community participation has come to the fore as a pivotal measure for successful programming.

We sought to evaluate the performance of a community-based TB project piloted in a rural sub-district in the Eastern Cape province of South Africa. The project, implemented by an international non-governmental organisation (NGO) over 2 years, had the key objective of achieving the global target of an 85% treatment success rate¹ among TB patients registered in the sub-district. It mainly comprised advocacy measures to increase community awareness on aspects of TB control which, it was envisaged, would enhance treatment-seeking behaviours at individual, household and community levels. It was implemented in close collaboration with community entities such as rural clinics, local schools and civil society organisations (CSOs). The CSOs received support to initiate community microprojects such as community gardens to enhance nutrition among affected communities, TB contact and defaulter tracing to improve adherence to treatment, and smoking cessation campaigns. However, in undertaking these activities there was little focus on the other technical components of an effective TB programme, such as quality microscopy, timely recording and reporting.

A total of 463 new TB patients were registered in the pilot sub-district over the 2-year period, 145 in 2004 and 318 in 2005, representing a >100% increase in new caseload without any reported exogenous contributory factor other than the introduction of the community project. A comparable neighbouring sub-district ($N=631$) registered an increase of only 3.0% during this time.

The average age of the patients was 38.0 years (95% confidence interval (CI) 36.52 - 39.45). Males were older, with a mean age of 40.5 years (95% CI 38.56 - 42.3 years)

compared with 34.9 years (95% CI 32.7 - 37.2) for females. For both genders the age category with the highest proportion of patients was between 15 and 54 years; females peaked at 15 - 44 years and males at 25 - 54.

We used data from the pilot sub-district's electronic TB register to analyse the quarterly performance of successive treatment cohorts and made comparisons over the period between quarter 1, 2004 and quarter 4, 2005. Bacteriological coverage declined from 80% to 50%, new smear conversion rates dropped from 60% to 30%, and treatment success rate dropped from 60% to 38%. TB patients reported as 'not evaluated' increased to 45% in quarter 4, 2005, from insignificant proportions reported in the preceding quarters.

Analysing determinants of treatment success revealed that patients registered in 2005 were 75.4% less likely to achieve treatment success than those in 2004. Similarly, patients who transferred into the treatment centres located in the pilot sub-district during the same period were less likely to be successfully treated (Table I).

We are cognisant of limitations in this evaluation. Reliable causal inference to a specific intervention as responsible for the changes observed was difficult for a descriptive study of this nature. Reliance on secondary data, the quality of which cannot be readily ascertained, is also a weakness. However, attempts were made to mitigate for quality issues through rigorous data cleaning and collation before analysis.

We conclude that community participation alone is inadequate to improve the performance of a TB control programme. Planning a holistic and comprehensive approach should seek to strengthen the programme's technical and organisational capacity⁵ before engaging in purely community interventions. Failure to observe this logical relationship would result in suboptimal performance. Efforts to increase demand for health services should be cognisant of the health system capacity.

We recommend further research and sharing of best practices and evidence-based approaches of harnessing community-based interventions to improve health outcomes.

Table I. Determinants of treatment success (463 patients)

Treatment success	Odds ratio (95% CI)	<i>p</i> -value
Sex (male)	0.822 (0.55 - 1.22)	0.333
Transferred in patients	0.526 (0.35 - 0.79)*	0.002
Age category	1.11 (0.99 - 1.26)	0.083
Year of registration 2005	0.246 (0.16 - 0.38)	0.001

*Statistically significant odds ratios are set in bold.



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CFTR structural rearrangements are not a major mutational mechanism in black and coloured southern African patients with cystic fibrosis

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To the Editor: Exon copy number variants (CNVs) in the CFTR gene have been identified as a new type of mutation in cystic fibrosis (CF) patients.¹ Most of these mutations have been identified in CF patients of European origin; however, a deletion of the promoter and exons 1 and 2 of the CFTR gene was detected in two African American families.²

To increase the CFTR mutation detection rate in black and coloured South African CF patients for molecular diagnosis, we tested patients with a clinical phenotype of CF, and at least one unidentified CFTR mutation, for exon CNVs in the CFTR gene: 24 coloured patients (of Khoisan, Malay, European and African admixture), most of whom originate from the Western Cape, and 18 black patients (sub-Saharan African origin) were tested. Multiplex ligation-dependent probe amplification (MLPA) that detects deletions and duplications based on the principles of complementary probe binding, ligation and amplification with a comparison of patient data to control data, was used (CFTR P091 kit).³ Controls were matched for ethnicity and geographical region of origin.

A heterozygous deletion of exon 2 was detected in a single black CF patient, using both MLPA and semi-quantitative fluorescent PCR. The deletion breakpoints were determined as c.54-1161_c.164+1603del2875 and a mutation mechanism was suggested based on a small direct repeat at the 5' and 3' breakpoints.⁴ This mutation causes the deletion of amino acids 91 to 163, corresponding to the deletion of the first two transmembrane regions, two cytoplasmic topological domains

and one extracellular topological domain of the chloride channel (<http://www.expasy.org>, <http://www.genet.sickkids.on.ca/cftr/>). The anticipated consequence would be complete loss of function. This patient had one positive sweat test and other symptoms suggestive of CF, including respiratory failure. He has since been lost to follow-up.

This mutation was not detected in any of the other patients, and may be unique to this family. No other CNVs were detected. It would therefore not be cost effective to include it in the diagnostic panel of mutations in black and coloured patients with symptoms suggestive of CF. Although the sample size was small, this report suggests that exon copy number variants of CFTR is not a major mutational mechanism giving rise to CF in black and coloured southern African CF patients.

Conflict of interest. The authors declare that there are no sources of conflict of interest to the above research.

Ethnic classification. The rationale for ethnic classification is that the study was done specifically to identify mutations in ethnic groups in which CFTR mutation detection in South Africa is low.

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