CIGARETTE SMOKING AND ITS RELATION TO PULMONARY TUBERCULOSIS IN ADULTS

Niorn Ariyothai[1], Amornrath Podhipak[2], Pasakorn Akarasewi[1], Songpol Tornee[3], Saijai Smithtikarn[1] and Pittaya Thongprathum[1]

[1]Tuberculosis Division, Ministry of Public Health; [2]Department of Epidemiology, Faculty of Public Health, Mahidol University; [3]Department of Health Education, Srinakarinwirot University, Bangkok, Thailand

Abstract. The purpose of this hospital-based case-control study is to determine the effect of passive and active smoking on pulmonary TB in adults. The study subjects were 100 new pulmonary TB cases diagnosed at TB Division, and age-sex matched 100 non-TB cases from patients admitted to Taksin Hospital and healthy subjects who came for annual physical check-up at either the outpatient clinic of the TB division or Taksin Hospital, during May 2001 to October 2001. All subjects had blood tests and only persons who were HIV-negative, DM-negative and free of other lung diseases were included. Data were collected by direct interview using questionnaires. Multivariate analysis of cigarette smoking related to pulmonary TB in adults was performed. The factors related to pulmonary TB in adults were current active smoking regardless of passive smoking exposure. There was a significant association between early age at initiation of smoking and TB. Active (current + ex-active) smokers who started smoking at age 15-20 years had a higher risk of pulmonary TB compared to others (OR = 3.18, 95% CI = 1.15-8.77); as well as the long duration of smoking: persons who had smoked >10 years had a higher risk of pulmonary TB (OR = 2.96, 95% CI = 1.06-8.22). There was a relationship between pulmonary TB and the amount of smoking exposure. Those who smoked >10 cigarettes/day (OR = 3.98, 95% CI = 1.26-12.60) or >3 days/week (OR = 2.68, 95% CI = 1.01-7.09) had higher risk of pulmonary TB compared to non-smokers. Passive smokers who were exposed to tobacco smoke >3 times/week outside the home had a higher risk of pulmonary TB than those with exposure ≤ 3 times/week (OR = 3.13, 95% CI = 1.07-9.17). It was also found that the effects of passive smoking in the office and/or neighborhood were strong. Persons with such exposures had a higher risk of pulmonary TB than no exposure or exposure ≤ 3 times/week from either or both places (OR = 4.62, 95% CI = 1.68-14.98). Therefore, an effective anti-smoking campaign is expected to have a positive repercussion on TB incidence. Smoking cessation must be considered and promoted by all levels of health care providers.

INTRODUCTION

Practically, anybody can get tuberculosis infection during a lifetime. It is potentially rapidly spread because it is airborne and spreads like the common cold, when infectious people cough, spit, talk or sneeze (WHO, 1998). Some individuals may develop disease soon after they become infected, others may get sick later when their immune system becomes weak. Lowered immunity may associated with some health-deteriorating factors, such as cigarette smoking (Pio and Chaulet, 1998; Doll, 1999; Rajanapithayakorn and Narain, 1999; Yach, 2000).

Tobacco is a risk factor for tuberculosis and TB death. TB patients have extremely high smoking rates (Yach, 2000). Several studies have shown that smoking increases the rates of TB infection in both the incidence and severity. They also found a dose-response relationship between the number of cigarettes smoked daily, duration of smoking and the risk of active pulmonary TB (Bam, 1991; Baskin *et al*, 1994; Alcaide *et al*, 1996; Yach, 2000). Other Spanish studies have found a relationship between passive smoking by family members and TB in children (Altet *et al* 1994; 1996). In Doll's study, which investigated

Correspondence: Amornrath Podhipak, Department of Epidemiology, Faculty of Public Health, Mahidol University, Bangkok 10400. Thailand.
E-mail: phapp@mahidol.ac.th

the impact of tobacco on total and cause-specific mortality, the ratio of death for tuberculous smokers to non-smokers was 2.8, compared with the ratio of 2.0 for tuberculous ex-smokers (Doll, 1999). This is consistent with the knowledge that long-term exposure to cigarette smoking has an adverse effect on the lung's defense mechanisms (Yach, 2000).

Although smoking is one of the literature-cited factors, the association between smoking and pulmonary tuberculosis has been assessed in very few studies. Using WHO guidelines, there are effective TB control programs and highly effective treatment is available, however if people are not aware of its seriousness and prevention, especially the behavioral risk factors, it will remain be a public health problem in Thailand. For these reasons, this study is interested in cigarette smoking as a risk factor associated with TB manifestation in adults. This knowledge can be utilized to develop a program for TB prevention and control.

MATERIALS AND METHOD

Selection of cases and controls

The study was conceptualized as a hospital-based case-control study. The study subjects were 100 new pulmonary TB cases diagnosed at the TB Division, and age-sex matched 100 non-TB cases from patients admitted to Taksin Hospital and healthy subjects who came for annual physical check-ups at either the outpatient clinic of the TB division or Taksin Hospital, during May 2001 to October 2001. In order to eliminate manifestations related to HIV-infection and DM, all subjects (blood) were tested and only HIV-negative, DM-negative persons were included. X-rays and physical examinations showed that the comparison groups were also free of signs of other lung diseases. Data were collected by using questionnaires to interview personal risk behavior, and lab results were coded from data in the medical records.

Study definition

Case: a new pulmonary TB case, diagnosed by physician, aged ≥15 years who attended the outpatient clinic of the TB Division, and/or never had previous treatment for TB (new patients who has taken anti-TB drugs for less than 4 weeks were included). The TB criteria are: at least two sputum specimens positive for acid-fast bacilli (AFB) by microscopy; or with at least one sputum specimen positive for AFB and radiographic abnormalities relevant with pulmonary tuberculosis and a decision by a physician to treat with a full curative course of anti-tuberculosis chemotherapy (Pio and Chaulet, 1998; Payanandana *et al*, 1999). They must also have HIV-antibody and fasting blood sugar (FBS) results.

Control: non-TB case, aged ≥15 years attending the outpatient department for medical check-up or inpatient department of Taksin Hospital and had no evidence of lung disease. Controls were matched for sex and age (15-24 years, 25-34 years, 35-44 years, 45-54 years and ≥55 years), were diagnosed by physician with radiographic normalities, and no history of TB. They must also have HIV-antibody and FBS results negative for HIV infection and DM.

Smoking habits:

1. Non smoker: any person that has never smoked (non-active smoker) or who has never or less than 3 times/week been exposed to tobacco smoked by others at home, work, or in public places (non-passive smoker).

2. Active smoker:

2.1 Current active smoker: any person who smoked a tobacco product at the time of the study or persons who used to smoke but had stopped smoking <6 months before the interview.

2.2 Ex-active smoker: any person who used to smoke and had stopped smoking ≥ 6 months before the interview.

3. Passive smoker:

3.1 Current passive smoker: any non-smoker who was exposed to tobacco smoke >3 times/week, either at home, work, or in public places.

3.2 Ex-passive smoker: any non smoker who was exposed to tobacco smoke who had terminated such exposure ≥ 6 months before the interview.

Data was collected using questionnaires to

interview people on their demographic and behavioral factors. Medical data were recoded from the medical records. General characteristics and study variables were described by percentage, mean, and standard deviation. Smoking habits were compared among the cases and controls. Univariate analysis and multiple logistic regression models were used to assess the effects of individual variables. The odds ratio (OR) and 95% confidence intervals (CI) were calculated and adjusted for age, sex and confounding factors, and were controlled for in the final regression analysis. The level of significance was set at 0.05.

RESULTS

In the case and control groups, the number of persons in each sex and age group were similar. The majority of cases and controls were male between 15-34 years of age, married, living in

Table 1
Socio-demographic characteristics in case and control groups.

Demographic characteristics	Case n =100		Control n =100	
	Number	Percent	Number	Percent
Sex				
Male	63	63.0	63	63.0
Female	37	37.0	37	37.0
Age (years)				
15-34	57	57.0	57	57.0
35-54	37	37.0	37	37.0
≥ 55	6	6.0	6	6.0
Mean (SD)	32.94	(11.98)	33.61	(12.27)
Resident				
Bangkok and perimeter	44	44.0	36	36.0
Other provinces	56	56.0	64	64.0
Marital status				
Single	28	28.0	32	32.0
Married	54	54.0	57	57.0
Divorced, widowed, separated	18	18.0	11	11.0
Educational level				
No formal education	3	3.0	2	2.0
Primary level	53	53.0	45	45.0
Secondary level	37	37.0	37	37.0
Vocational level	3	3.0	4	4.0
University level	4	4.0	12	12.0
Income (baht/month)				
< 5,000	54	54.0	48	48.0
5,000-10,000	38	38.0	41	41.0
> 10,000	8	8.0	11	11.0
Mean (SD)	4,803.50	(5,212.34)	7,007.00	(8,626.95)
Occupation				
Employee	52	52.0	49	49.0
Trade/Self-employed	6	6.0	15	15.0
Government officer	6	6.0	14	14.0
Student	9	9.0	5	5.0
Unemployed	18	18.0	11	11.0
Others	9	9.0	6	6.0
Number of family members (persons)				
1-3	44	44.0	40	40.0
4-6	37	37.0	43	43.0
≥7	19	19.0	17	17.0
Mean (SD)	4.35	(2.58)	4.47	(2.36)

the provinces outside Bangkok, had a primary educational level, and an income < 5,000 baht/ month. The majority of cases and controls were employees and had family members between 1-3 persons (Table 1).

The evaluation of potential confounding factors were performed using the criteria for change in the OR of the smoking variable related to the risk of pulmonary TB, comparing the full model (in which all other factors were presented) and the reduced model (in which one suspected confounding factor was dropped). The change of 15% was considered as important. Table 2 shows the result of the evaluation. First, the list of potential confounding factors was obtained by performing an association test between each independent known risk factor and TB manifestation in this study sample. It was found that 5 factors were associated with pulmonary TB in this study: alcohol consumption, household environments, history of TB in the family, BMI and presence of a BCG scar. Secondly, these factors, as well as age, were brought for further analysis as potential confounders. Although in this study the subjects were matched for age, the matching scheme was quite loose (within 10-year intervals). The possible residual confounding effect of age was also put in the model. The investigator analyzed the association between smoking and pulmonary TB via the evaluation of the role of each potential confounder and made adjustments for the confounding factors. All potential confounders were included in the logistic regression analysis. This model was used as the full model (a total of 6 independent variables). A reduced model equals a full model with a potential confounder deleted. There were 6 reduced models. Each reduced model could illustrate the confounding effect of the dropped potential confounders. Only BMI was found to be a confounding factor since the OR of smoking in the full model and the reduced model changed by greater than 15%. Thus, only BMI was controlled for in the final regression analysis.

The logistic regression analysis for the effect of cigarette smoking on pulmonary TB in adults was performed, after balancing for sex, age and controlling for BMI as a potential confounder. The majority of cases were current active smokers (44.0%), whereas most persons in the control group were passive smokers (36.0%). The results showed that only current active smoking was associated with an increased risk for developing pulmonary TB in adults, with relative odds of 2.70 (95% CI = 1.04-6.97) compared to non-smokers (Table 3). Moreover the active (current + ex-active) smokers who started smoking age ≤ 20 years (OR = 3.18, 95% CI = 1.15-8.77), or had a duration of smoking >10 years (OR = 2.96, 95% CI = 1.06-8.22) had a higher risk of pulmonary TB

Table 2
Evaluation of the confounding effects of each potential confounder on cigarettes smoking and its relationship to pulmonary TB in adults.

Potential confounders	OR of active smoking			OR of passive smoking		
	Full[a] model	Reduced[b] model	% Change in OR[c]	Full[a] model	Reduced[b] model	% Change in OR[c]
Age	3.08	3.20	3.90	2.52	2.39	-5.15
Alcohol drinking	3.08	3.13	1.96	2.52	2.42	-3.97
House environments	3.08	3.47	12.67	2.52	2.71	7.54
TB patients in family	3.08	2.84	-7.79	2.52	2.45	-2.78
BMI	3.08	3.87	25.64	2.52	2.60	3.17
BCG scar	3.08	2.84	-7.79	2.52	2.31	-8.33

[a]Full model includes smoking status, BCG scar, BMI, history of TB patients in the family, household environment, alcohol drinking and age.
[b]Reduced model equals full model with a specified variable deleted.
[c]The criteria of change > 15% was used for the classification of confounders.

compared to others. There was a relationship between pulmonary TB and the amount of direct exposure to cigarettes, especially in those who smoked 1-10 cigarettes/day or smoked >3 days/ week. The risks were 3.98 times (95% CI = 1.26-12.60) and 2.68 times (95% CI = 1.01-7.09), respectively, higher than non-smokers (Table 4). Passive smokers who were exposed to tobacco

Table 3
Smoking status related to pulmonary TB in adults.

Smoking status	Case n =100 No. (%)	Control n =100 No. (%)	OR (95%CI)	Adjusted OR[a] (95%CI)
Non-smokers [(a)]	9 (9.0)	26 (26.0)	1.00	1.00
Active smokers				
Current active smokers [(b)]	44 (44.0)	29 (29.0)	4.38 (1.80-10.69)	2.70 (1.04-6.97)
Ex-active smokers [(c)]	11 (11.0)	9 (9.0)	3.53 (1.11-11.29)	2.88 (0.85-9.78)
Passive smokers (indoor and outdoor)[(d)]	36 (36.0)	36 (36.0)	2.89 (1.19-7.02)	2.37 (0.94-6.01)

[a]Adjusted for BMI.
[(a)]Non-smokers who were never or ≤ 3 times/week exposed to environmental tobacco smoke.
[(b)]Current active smokers, included smokers who were never or ≥1 times/week exposed to environmental tobacco smoke. (Current active only plus both current active and current passive smokers).
[(c)]Ex-active smokers, included ex-smokers who were never or ≥1 times/week exposed to environmental tobacco smoke. (Ex-active smokers only plus both ex-active and current passive smokers).
[(d)]Passive smokers who never smoked or were exposed to environmental tobacco smoke >3 times/ week (included 1 ex-passive smoker in the case group).

Table 4
Association of smoking with pulmonary TB in adults among active smokers.

Smoking status	Case n =100 No. (%)	Control n =100 No. (%)	OR (95%CI)	Adjusted OR[c] (95%CI)
Active smokers	n =55	n =38		
Age started smoking (years)[a]				
15-20	42 (65.6)	22 (34.4)	5.52 (2.21-13.80)	3.18 (1.15-8.77)
> 20	13 (20.3)	16 (25.0)	2.35 (0.82-6.73)	1.35 (0.42-4.39)
Duration of smoking (years)[a]				
1-10	19 (29.7)	15 (23.4)	3.66 (1.32-10.11)	1.60 (0.50-5.14)
> 10	36 (56.3)	23 (35.9)	4.52 (1.80-11.36)	2.96 (1.06-8.22)
Types of cigarettes[b]				
Non-filtered / hand-rolled	11 (20.0)	9 (23.7)	0.81 (0.30-2.19)	-
Filtered	44 (80.0)	29 (76.3)	1.00	
Number of cigarettes/day[a]				
1-10	32 (50.0)	27 (42.2)	3.42 (1.37-8.55)	1.79 (0.63-5.04)
> 10	23 (35.9)	11 (17.2)	6.04 (2.13-17.17)	3.98 (1.26-12.60)
Frequency of smoking (days/week)[a]				
1-3	3 (4.7)	6 (9.4)	1.44 (0.30-7.01)	0.95 (0.17-5.41)
> 3	52 (81.3)	32 (50.0)	4.69 (1.95-11.28)	2.68 (1.01-7.09)

[a]Reference group was non-smokers who were never or ≤ 3 times/week exposed to environmental tobacco smoke.
[b]Reference group was filtered cigarettes.
[c]adjusted for BMI.

Table 5
Association of smoking with pulmonary TB in adults among passive smokers (outdoor smoked exposure).

Passive smoking	Case n =100 No. (%)	Control n =100 No. (%)	OR (95%CI)	Adjusted OR[a] (95%CI)
Outdoor smoke exposure[d] (times/wk) (n = 25, n = 45)				
No exposure or ≤ 3 times /wk	11 (44.0)	33 (73.0)	1.00[b]	1.00
>3 times	14 (56.0)	12 (26.7)	3.50 (1.25-9.80)	3.13 (1.07-9.17)
Office and neighborhood exposure (times/wk)				
No exposure or ≤ 3 times/wk	12 (48.0)	37 (82.2)	1.00[c]	1.00
>3 times[e]	13 (52.0)	8 (17.8)	5.01 (1.68-14.98)	4.62 (1.47-14.51)

[a]Adjusted for BMI
[b]Reference group included no exposure (both indoor and outdoor) and outdoor smoking exposure 1-3 times/week.
[c]Reference group included no exposure (both office and neighborhood) and exposure ≤ 3 times/week from both places or either.
[d]Outdoor smoke exposure included passive smokers exposed to tobacco smoked outside the home only (*ie* non-smoker and no indoor exposure).
[e]Outdoor smoke exposure > 3 times/week at the office, neighborhood or both.

smoke >3 times/week outside the home had higher risk of pulmonary TB than those who were not exposed (both indoor and outdoor) or exposed ≤3 times/week outside the home, either at the office, neighborhood or both (OR = 3.13, 95% CI = 1.07-9.17). These persons had a higher risk of pulmonary TB than persons with no exposure or exposure ≤ 3 times/week from both places (OR = 4.62, 95%CI = 1.68-14.98) (Table 5). Indoor exposure (family members who smoked) did not show a statistically significant association with pulmonary TB in this study. A household smoke exposure increased the risk of pulmonary TB, but there was a small group of passive smokers (>3 times/week) with family members smoked at home (10 from 23 cases and 7 from 25 controls), thus the difference in risk did not reach statistical significance. If the sample size was larger, the association would be more clearly identified.

DISCUSSION

The present study was conducted to examine the association of cigarette smoking with active pulmonary TB in adults without the interfering effect of HIV infection or DM. The hospital-based case-control study design enabled us to summarize the relative odds of being a smoker in the diseased group as compared to the controls.

This study had some limitations due to the method in selecting the study subjects, particularly, the controls which were supposed to come from the same source population as cases. All pulmonary TB cases were diagnosed at the OPD of the TB Division, which is a specific place where people would come for lesions if TB was suspected and/or treatment for TB. One criterion for the control group was the controls must not have other lung diseases or chronic diseases associated with smoking, so it was difficult to select controls with other diseases from the same place. Only 25 healthy controls who came for annual physical check-up could be identified. In order to solve this problem and reduce selection bias, the rest of the controls were selected from the IPD (surgery, orthopedic) of Taksin Hospital, which is located near the TB Division. Approximately half of them had acute appendicitis and the other half had accidental injuries. Therefore, it was expected that they are similar to the general population in a sense that they are otherwise generally healthy persons, but had acute injuries/disease. Both cases and controls were completely normal in terms of HIV infection and DM. The exposure

characteristics of cases and controls were investigated. In the study, it was not possible to include only people who were positive to tuberculin skin test (TST, which was an indication for TB bacillus exposure in some areas). In Thailand, the BCG vaccination coverage in children under one year of age has become almost 100% since 1990 (Payanandana *et al*, 1999), therefore the skin test was not a good indication for natural infection because BCG can give a false positive result on a TB skin test as well. The lack of confirmation on exposure to a pathological agent might be a drawback for making a definite conclusion on the effect of smoking on the development of TB, if cases and controls differed in the agent exposure level. In addition, a limitation in this study was that the investigator could not measure cotinine levels as a solid indicator for smoking. The amount and duration of cigarette exposure must be totally relied on the recall of subjects in both groups. These limitations might cause some bias in the result. Therefore, TST, measurement of cotinine levels and population-based case-controls should be conducted for confirmation of the results.

The results of this study showed that only current active smoking was associated with an increased risk for developing active pulmonary TB in adults, with relative odds increased to 2.70 times by multiple logistic regression analysis, after balancing for sex, age and BMI. This result corresponded with other studies, but the risk estimated for TB was difference: Alcaide *et al* (1996) in a case-control study, showed the risk estimated for TB was higher (OR = 3.8, 95% CI = 1.5-9.8) for active smokers among young adults (15-24 years) after balancing for age, sex and social class status. In Gupta *et al*'s report (2001) of a case-control study, multiple logistic regression analysis showed OR of 5.41 (95% CI = 3.06-9.5) for 200 pulmonary TB cases as compared to 200 controls with other respiratory diseases and an OR of 4.42 (95% CI = 2.54-7.60) as compared to 200 healthy controls, after balancing for age, sex, socio-economics status and exposure to TB patients. The adjusted OR of both studies was higher compared to this study because of differences in study design, study population, sample size and potential confounders. Most studies concerned the need of adjustment for age and sex. In many populations, smoking habits vary considerably with age. Adelestein and Rimington (1967) found that smoking could be a factor in the difference in rates of TB between men and women above 35 years of age. Chan *et al* (1995) and Yu *et al* (1988) also found that the differences in sex (males) and age (old age) that were associated with a higher risk of TB were due to the smoking factor. Nisar *et al* (1993) found that smoking and male gender is associated with tuberculin test positivity.

This study found the number of cigarettes/day and duration of smoking are strongly associated with active pulmonary TB in active (current + ex-active) smokers. The results corresponded with other studies: Peng *et al* (1990) found that persons who smoked > 20 cigarettes a day had 2.50 times (95% CI = 1.11-5.60) more risk of pulmonary TB compared to non-smokers after balancing for age, sex, occupation, marriage, type of housing, history of contacting TB and wine. Yu *et al* (1988) found that the relative risk of heavy smokers ≥ 400 cigarettes a year compared with non-smokers was 2.17 (95% CI = 1.29-3.63) after balancing for age, sex, history of contact, area of housing and type of work. Nisar *et al* (1993) found that the number of pack-years, was also associated with a stronger positive reaction to the tuberculin test in current smokers than ex-smokers. Bam (1991) found that persons who smoked >2 years had 8.75 times (95% CI = 4.88-15.79) more risk for developing TB compared to non-smokers. Baskin *et al* (1994) found that the relationship was statistically significant only for those who had been smoking for ≥ 30 years (OR = 2.6, 95%CI = 1.1-5.9).

Indoor exposure when family members smoke did not show a statistically significant association with pulmonary TB in this study. The result corresponded with Alcaide *et al* (1996), but did not correspond to Altel *et al* (1996) who found that in children (under 15 years old) living in households with a patient with active pulmonary TB, passive smoking was a substantial risk factor for the development of active pulmonary TB (OR = 5.39 adjusted for age and socio-economic status, 95% CI = 2.44-11.91).

Outdoor smoke exposure was shown to be

associated with pulmonary TB in this study. This corresponded with a study by the Chest Institute (1987) and Marcus *et al* (1990) that the workplace serves as an important source of environmental tobacco smoke (ETS) exposure for most non-smokers. These results suggested that restrictive worksite smoking policies may reduce employees' exposure to ETS.

This is consistent with the understanding that long-term exposure to cigarettes has an adverse effect on the lung's defense mechanisms, namely clearance of potential pathogens, such as *M. tuberculosis* (Yach, 2000). Cilia are tiny hair-like projections which help to sweep dirt waste products out of the lungs. When impaired by exposure to cigarette smoke, the cilia can not perform this cleansing process (Holbrook, 1982). The acute inhalation of cigarette smoke results in ciliostasis and reduced mucociliary clearance. Chronic inhalation of smoke alter clearance by converting ciliated epithelium to nonciliated epithelium, by altering the amount and character of the mucus products, and by inflammatory and emphysematous narrowing of the airway (Burns, 1991; Chitanondh 1991).

Smoking affects the normal function of alveolar macrophages. Alveolar macrophages eliminate microorganisms, including *M. tuberculosis,* from the distal airway and keep the alveoli sterile. *M. tuberculosis* can survive within the macrophage, particularly if the immune system is not operating adequately (Sibille and Reynolds, 1990). Development of TB seems to be linked to an altered immune response; multiple defects in macrophage/monocyte immune responses and CD4+ lumphapenia have been described (Onwubbalili *et al*, 1987). Tobacco smoke can alter native and acquired resistance to *M. tuberculosis*. Exposure to tobacco smoke causes morphological and functional changes in alveolar macrophages. Macrophages from smokers are more numerous and are generally larger in size and highly pigmented (Sibille and Reynolds, 1990). Smoking has an inhibitive effect on phagocytosis and the bactericidal activity of the alveolar macrophage. The level of IgG and IgM in serum of smoking group is lower. Smoking has a prohibitive effect on the immunity of the body (World Conference on Lung Health, 1990). Immunity is considered to be expressed when immunomodulated macrophages suppress the growth of the TB statistically significantly compared with unmodulated control macrophages (Crowle *et al*, 1990). Therefore conceptually, smoking is likely to precipitate and/or aggravate TB in view of altered immune functions. From these results confirming the effect of passive smoking on TB manifestation in adults, effective anti-smoking campaigns can have positive repercussions on TB incidence in the country.

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