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ORIGINAL ARTICLE

BMI as a predictor of progression from TB infection to active TB in PLHIV

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SUMMARY

BACKGROUND: Low body mass index (BMI) is a globally important risk factor for TB progression. Little is known about this association in people living with HIV (PLHIV) and the functional form of the BMI-TB incidence curve.

METHODS: Secondary analysis of a randomised controlled trial of TB preventive therapy among PLHIV in South Africa, Mozambique, and Ethiopia. Participants received 3 months of weekly high-dose rifapentine-isoniazid given once or twice over a period of 2 years. Multivariable fractional polynomials (MFPs) were used to investigate functional forms of BMI. Time to incident TB was modelled using Cox's proportional hazard regression.

RESULTS: A total of 76 TB events were documented, giving an overall TB incidence rate of 1.2 per 100 person-years (95%CI 1.0–1.6). Baseline BMI <18.5 kg/m^2 was associated with a 2.6-fold increased hazard of TB compared with BMI 18.5–24.9 kg/m^2 (aHR 2.6, 95% CI 1.4–4.8, $P < 0.001$). BMI ≥30 kg/m^2 was associated with a lower hazard of TB (aHR 0.5, 95% CI 0.2–1.0). Continuous and categorical BMI showed weak evidence of quadratic dose-response relationships ($P = 0.08$ and $P = 0.09$, respectively). MFP analysis was consistent with a decline in TB incidence for increasing BMI to around 25 kg/m^2, followed by a less steep decline in TB incidence for increasing BMI >25 kg/m^2.

CONCLUSIONS: In PLHIV, BMI showed an inverse log-linear association with TB incidence. The MFP approach showed that the relationship is more complex than a simple log-linear association.

KEY WORDS: BMI; TB infection; PLHIV; fractional polynomials

TB remains a global health challenge, with an estimated 10.8 million new cases in 2023, 6.1% of which were co-infected with HIV.[1] In the same year, there were an estimated 1.25 million deaths attributable to TB.[1] The sub-Saharan region accounts for almost one-quarter of all TB cases globally, of which over 60% have co-infections with HIV.[1] In this region, HIV-associated TB may be a barrier to meeting the targets to end the TB epidemic.

Around one-quarter of the world's population is infected with *Mycobacterium tuberculosis* (M.tb).[2] The risk of developing TB following infection is increased in immunosuppressed individuals, particularly among people living with HIV (PLHIV), or if other risk factors such as undernutrition, diabetes, smoking, or alcohol consumption are present.[3,4] TB preventive therapy (TPT) can prevent progression from infection to disease.

Undernutrition is common among PLHIV and is an independent predictor of death even in the era of highly active antiretroviral therapy.[5,6] Body mass index (BMI) is the most widely used indicator to measure nutritional status. Several studies have attempted to explain the relationship between low BMI and TB prevalence and/or incidence.[7,8] A systematic review observed a consistent and log-linear inverse dose-response relationship between BMI and TB incidence within the BMI range of 18.5–30 kg/m^2 among HIV-negative people.[9] This finding was confirmed in a recent cohort study with 99.9% HIV-negative participants.[10] Additionally, some studies suggest that low BMI increases the risk of progressing from TB infection (TBI) to active disease due to impairment of cellular immunity.[11]

Little is known about the functional form of the BMI-TB incidence relationship among PLHIV. Therefore, the present study aimed to examine the relationship between BMI measured at study enrolment (baseline) with the risk of developing active TB during 24 months of follow-up in PLHIV adults who received 3 months of weekly high-dose rifapentine-isoniazid once or twice over a period of 2 years in a large TPT drug trial. To further characterise the association between BMI and incident TB, we evaluated

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the functional forms of BMI as a continuous variable using multivariable fractional polynomials (MFP).

METHODS

Study design of parent study

This analysis used data from the WHIP3TB trial,[12] a three-arm, open-label study among PLHIV receiving antiretroviral therapy (ART) in South Africa, Mozambique, and Ethiopia. Participants were randomly assigned (ratio 2:9:9) to 6 months of daily-isoniazid therapy (6H arm), 3 months of weekly rifapentine–isoniazid (3HP) therapy once (3HP arm), or annual 3HP therapy (p3HP arm).[12,13] Participants in both 3HP arms were followed for 24 months, and 6H participants were followed for 12 months. The WHO 4-symptom screening algorithm was used to exclude TB at baseline, 12 and 24 months. At 12 months, all participants submitted sputum (irrespective of symptoms) for Xpert MTB/RIF and mycobacterial culture and had chest radiography; the same procedures were repeated at 24 months in the 3HP groups. Data were collected from November 2016 to November 2017, with 4027 HIV-positive individuals enrolled.

Participant consent

The WHIP3TB study received ethical approval from the University of the Witwatersrand-South Africa (Johannesburg, South Africa), Addis Ababa University-Ethiopia (Addis Ababa, Ethiopia), Mozambican National Ethics Committee-Mozambique (Maputo, Mozambique) and the London School of Hygiene & Tropical Medicine Ethics Committee (London, UK). Written informed consent was obtained from all participants before enrolment in the WHIP3TB study.

Data collection methods

Our analysis sought to quantify the association between BMI and TB incidence over 24 months and, therefore, is restricted to the two 3HP arms of the parent study. The analysis is further restricted to adults aged $\geq$18 years. Height and weight were recorded at baseline (randomisation visit). BMI was calculated by dividing the weight in kilograms (kg) by the square of height in meters (m^2) and categorised using the standard WHO definitions.[14,15] Baseline covariates were sociodemographic factors (age, sex, education, country of enrolment) and clinical characteristics (CD4 count, ART regimen, time on ART, previous TB episodes that occurred more than a year before enrolment, and previous isoniazid preventive therapy (IPT) received more than a year before enrolment). The latest CD4 count before enrolment and ART regimen were abstracted from the participant's clinical records. Clinical records were also reviewed for prior TB treatment, IPT, and TB episodes. Data collection methods and laboratory techniques are described elsewhere.[12]

Statistical analysis

The primary exposure hypothesised to be associated with the outcome of incident TB was BMI. Rates of incident TB were expressed per 100 person-years. Cox regression was used in univariable and multivariable analyses to examine the association between each exposure and the time to incident TB. Two approaches were taken to examine the relationship between BMI and incident TB: 1) BMI grouped as a categorical variable (four levels defined as underweight: $<$18.5 kg/m^2; normal: 18.5–24.9 kg/m^2; overweight: 25.0–29.9 kg/m^2; obese: $\geq$30 kg/m^2); and 2) BMI as a continuous variable. For BMI categories, the overall effect, linear trend, and deviations from the linear trend were evaluated with BMI treated as a continuous variable, using fractional polynomials. Likelihood ratio test (LRT) was carried out for overall associations, linear trends, and departure from linearity.

Bivariate analysis with Cox regression was used to assess the presence of confounding (Supplementary Table S2). The final multivariable model included confounding variables if present. As there were few events in our dataset, we used the rule of 10 to avoid a sparsity bias.[16] Moreover, we examined if the effect of BMI on TB incidence was modified by pre-specified variables such as CD4 count, country of enrolment, age, sex education and time on ART by fitting interaction terms.

Linear trends and any deviations from linearity were analysed for the BMI groups. The simplest departure from a linear relationship between the log(hazard) and a quantitative exposure is a quadratic relationship. For the continuous BMI, the linear trend and departure from linearity were assessed similarly to those for the BMI categories. In addition, we analysed the BMI continuously using the MFP approach.

Multivariable second-degree fractional polynomials were used to model BMI, allowing a maximum of 2 degrees of freedom and adjusting for the same variables as in the multivariable model with BMI categories. MFP modelling is a flexible method that reveals non-linear associations. MFPs offer greater flexibility than standard polynomial models (such as including a quadratic term along with the linear term for a covariate). They enable the selection of the best-fitting polynomial functions based on the data, rather than relying on predefined functional forms, thereby providing a more precise description of the BMI-TB association.[17]

We assessed the proportional hazards assumption of the effect of BMI on TB incidence using the Schoenfeld test. Analyses were conducted using Stata/SE v.17 (Stata Corporation, College Station, TX, USA).

RESULTS

Study population

Between November 2016 and November 2017, 3,610 PLHIV were enrolled into the 3HP arms, of whom 3593 were aged ≥18 years. Around 69% were female (2,496/3,593); the median age of the participants was 41.0 years (interquartile range [IQR] 35.0–49.0), and respectively 14.6% (526/3,593), 21.9% (787/3,593), 63.5% (2,280/3,593) were from Mozambique, Ethiopia, and South Africa (Supplementary Table S1). All participants were receiving ART (as per inclusion criteria), the median CD4 count was 483 cells/mm^3 (IQR 313–693), and the median time on ART was 4.4 years (IQR 2.2–7.1).

At enrolment, the median BMI was 24.1 kg/m^2 (IQR 21.0–28.2); 7.8% (272/3,593) of participants had a BMI <18.5 kg/m^2; and 15% (538/3,593) reported taking IPT more than a year before enrolment into the trial. The distribution of baseline variables differed by country of enrolment (Supplementary Table S1). Most variables had very few missing values (<6%).

Overall, there were 76 incident TB events among 3,593 individuals (6,133 person-years of follow-up), giving an overall TB incidence rate of 1.2 per 100 person-years (95% confidence interval [CI] 1.0–1.6). From these, 8 TB events were rifampicin-resistant, with 4 events in the 3HP arm and 4 in the p3HP arm. No cases of isoniazid-resistant TB were diagnosed. The proportional hazards assumption was met for the risk factors (Schoenfeld test $P = 0.84$). Incident TB differed by BMI level, with BMI <18.5 kg/m^2 associated with a shorter time to develop TB (Figure 1; $P = 0.002$). The hazard of TB decreases with increasing BMI. Low BMI (<18.5 kg/m^2) was associated with a 2.5-fold increased hazard of TB (hazard ratio [HR] 2.5, 95% CI 1.4–4.6) compared with a BMI of 18.5–24.9 kg/m^2 (Table 1). There was strong evidence for a linear trend for the effect of BMI on the log(hazard) of TB ($P < 0.001$) and no evidence for departure from linearity ($P = 0.23$). Although the confidence intervals included one, BMI ≥30 kg/m^2 may be associated with a 40% lower risk of developing TB than the normal range (Table 1).

Univariable analyses of other baseline sociodemographic measures showed that age, sex, and country of enrolment were associated with TB incidence rates. Data were consistent with no association between incident TB and ART regimen, time on ART, CD4 count, prior TB treatment, or IPT prior to enrolment (Table 1). In multivariable analyses adjusted for age (categorical variable) and country of enrolment, BMI (categorical variable) remained strongly associated with TB incidence. Being underweight (BMI <18.5 kg/m^2) was associated with a 2.6-fold increased hazard of TB compared to those with normal BMI (18.5–24.9 kg/m^2). Individuals with a BMI ≥30 kg/m^2 had a 50% lower hazard of incident TB than individuals with a normal BMI (18.5–24.9 kg/m^2). Assuming a linear effect for BMI as a continuous variable, a BMI increase of 10 kg/m^2 resulted in an HR of 0.4 (95% CI 0.3–0.7) (Table 2). There was weak evidence for a quadratic dose-response relationship between BMI and the hazard of TB for BMI grouped ($P = 0.09$) and BMI continuous ($P = 0.08$) (Table 2). The primary MFP model, adjusting for country of enrolment and age group, found the FP power of -1. A steep decline in the log hazard of TB was observed with increasing BMI, becoming less steep for BMI >25 kg/m^2 (Figure 2).

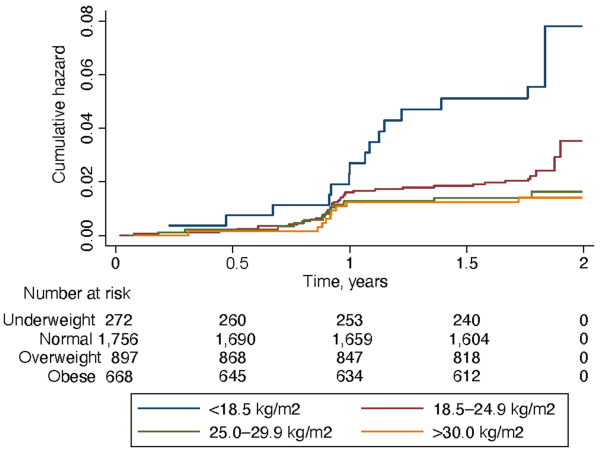

Figure 1. Kaplan–Meier plot of cumulative hazard of TB by BMI. Blue line (underweight) = lower BMI (<18.4 kg/m^2); red line (normal) = BMI (18.5–24.9 kg/m^2); green line (overweight) = BMI (25.0–29.9 kg/m^2); orange line (obese) = BMI >30 kg/m^2; log-rank test $P < 0.001$. BMI = body mass index.

DISCUSSION

In this trial population of 3,593 HIV-positive adults, BMI showed an inverse log-linear association with TB incidence with no evidence for departure from linearity. The risk of incident TB was lower in individuals with a BMI ≥30 kg/m^2 than in individuals with a normal BMI. The MFP approach showed a non-linear relationship between BMI and TB incidence. Low baseline BMI was associated with a slightly stronger effect on TB risk than the unadjusted estimate, as shown by the increased HR from 2.5 to 2.6 after adjustment. Consequently, the confounders slightly masked the true effect of low baseline BMI on TB risk. This small increase suggests that the relationship between BMI and TB is robust and is not heavily influenced by the confounders in our model.

Our results are consistent with previous findings supporting that a low BMI is associated with an increased risk of TB.[6] The association between BMI and TB that had been described for HIV-negative individuals also exists for PLHIV, suggesting a mechanism that is independent of HIV infection. Overall, the

Table 1. Distribution of sociodemographic and clinical factors at baseline, and rates and univariable hazard ratios for time to incident TB (n = 3,593, 76 TB cases).

	n (column %)	Events/PY	Rate/100 PY	HR (95% CI)	*P*-values*
Overall	3,593 (100)	76/6,133	1.2		
BMI, kg/m^2					
<18.5	272 (7.8)	15/456.5	3.3	2.5 (1.4–4.6)	0.002
18.5–24.9	1,756 (48.9)	39/3,000	1.3	1	<0.001†
25.0–29.9	897 (25.0)	13/1,500	0.8	0.6 (0.4–1.2)	
>30.0	668 (18.6)	9/1,100	0.8	0.6 (0.3–1.2)	
Country of enrolment					
South Africa	2,280 (63.5)	59/3,900	1.5	1	
Ethiopia	787 (22.0)	6/1,400	0.4	0.3 (0.1–0.7)	0.004
Mozambique	526 (14.6)	11/895.2	1.2	0.8 (0.4–1.5)	
Age, years					
18–29	401 (11.2)	10/653.5	1.5	2.1 (1.0–4.8)	
30–39	1,086 (30.2)	29/1,900	1.6	2.2 (1.2–4.1)	0.039
40–49	1,243 (34.6)	15/2,100	0.7	1	0.517‡
>50	863 (24.0)	22/1,500	1.5	2.2 (1.1–4.1)	
Sex					
Male	1,097 (30.5)	33/1,900	1.8	1.8 (1.1–2.8)	0.017
Female	2,496 (69.5)	43/4,300	1.0	1	
Education					
Non/primary	1,264 (35.2)	22/2,200	1.0	1	0.261
Secondary/tertiary	2,329 (64.8)	54/4,000	1.4	1.3 (0.8–2.1)	
ART					
TDF+FTC/3TC+EFV	3,406 (94.8)	74/5,800	1.3	1	0.278
Other	1,817 (5.2)	2/320.1	0.6	0.5 (0.1–2.0)	
Time on ART, years					
>2	763 (21.2)	21/1,300	1.6	1.4 (0.8–2.4)	0.401
2–4	880 (24.5)	17/1,500	1.1	1.0 (0.6–1.8)	0.234‡
>4	1,950 (54.3)	38/3,400	1.1	1	
CD4 count, cells/mm^3					
>200	419 (11.7)	13/701.2	1.9	2.0 (1.0–3.9)	
201–500	1,480 (41.2)	33/2,500	1.3	1.4 (0.8–2.3)	0.137
>501	1,488 (41.4)	24/2,600	0.9	1 (0.3–1.0)	0.046‡
Missing	206 (5.7)	6/349	1.7	—	—
Previous TB treatment					
No	2,734 (76.1)	56/4,700	1.2		0.630
Yes	859 (23.9)	20/1,500	1.4	1.1 (0.7–1.9)	
Previous IPT					
No	3,055 (85.0)	67/5,200	1.3	1	0.390
Yes	538 (15.0)	9/925.3	1.0	0.7 (0.4–1.5)	

*From the LRT. †$P < 0.001$ from LRT for linear association (no evidence for departure from linearity, $P = 0.23$). ‡LRT for trend;
PY = person-years at risk; HR = hazard ratio; CI = confidence interval; BMI = body mass index; ART = antiretroviral therapy; TDF = tenofovir; FTC = emtricitabine; 3TC = lamivudine; EFV = efavirenz; IPT = isoniazid preventive therapy; LRT = likelihood ratio test.

finding that the association between BMI and TB exists in both HIV-negative and HIV-positive individuals hints at the importance of addressing common risk factors (socio-economic status, undernutrition, inadequate healthcare access, or environmental factors) and underlying mechanisms contributing to TB risk in diverse populations. Therefore, there is a need for comprehensive public health strategies and interventions to reduce TB burdens and improve overall health.

Like our study, recent studies have shown that individuals with a BMI ≥30 kg/m^2 might have a lower risk of TB than individuals with a normal and underweight BMI. However, the effect of high BMI on incident TB, especially BMI ≥30kg/m^2 is uncertain. A linear inverse relationship between log(TB incidence) and BMI was reportedly uncertain for BMI ≥30 kg/m^2.[9] Furthermore, very high BMI (≥30 kg/m^2) did not reduce the risk of TB in certain subgroups, including young females and participants with diabetes mellitus in a Korean population.[18] Additionally, Zhang et al. found that a BMI exceeding 28 kg/m^2 was independently associated with an increased risk of TB in rural China.[7] Although we observed the same effect of high BMI on TB risk, our study settings differed from these studies, which were conducted in a low TB endemic area with low HIV prevalence.

Uncertainty persists about the biological mechanisms behind the increased risk of TB among individuals with a low BMI. Undernutrition alters immune homeostasis, increasing an individual's susceptibility to infections or progression of infection to disease. In addition, undernutrition impairs cell-mediated and humoral immune responses.[19] Both innate and adaptive immune cytokines are important mediators of immune responses against

Table 2. Adjusted HRs for TB incidence from multivariate analysis for linear and quadratic dose-response relationships (n = 3,593; 76 TB events).

Baseline BMI	HR*	(95% CI)	*P*-value
Categorical, kg/m^2			
<18.4	2.6	(1.4–4.8)	<0.001[†]
18.5–24.9	1		
25.0–29.9	0.6	(0.3–1.1)	
>30	0.5	(0.2–1.0)	
Categorical			
Categorical: linear[§]	0.6	(0.5–0.8)	<0.001[†]
Categorical: linear and quadratic[¶]			0.09[‡]
Linear	0.3	(0.2–0.7)	<0.001
Quadratic	1.2	(0.9–1.5)	
Continuous			
Continuous: linear[#]**	0.9	(0.9–1.0)	<0.001[†]
Continuous: linear and quadratic[††]			0.08[§]
Linear	0.8	(0.7–0.9)	<0.001
Quadratic	1.0	(1.0–1.0)	

*Adjusted for age and country of enrolment. [†]From LRT. [‡]LRT for departure from linearity; [§]Linear effect assumed for BMI grouped under four levels. [¶]Linear and quadratic effects assumed for BMI grouped under four levels. [#]An increase in BMI of 10 kg/m^2 represents a HR of 0.4 (95% CI 0.3–0.7). **Linear effect assumed for BMI ungrouped (continuous). [††]Linear and quadratic effects assumed for BMI ungrouped (continuous)
HR = hazard ratio; BMI = body mass index; CI = confidence interval; LRT = likelihood ratio test.

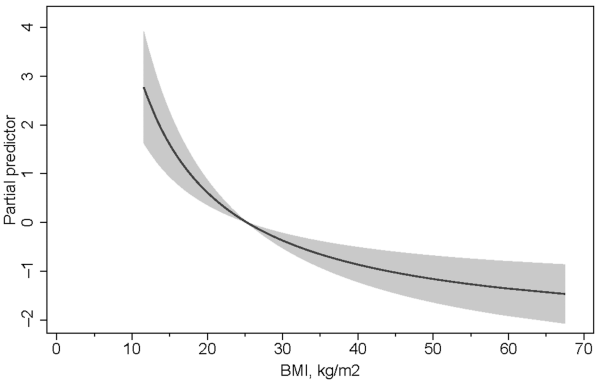

Figure 2. Plot of the fractional polynomial (–1) Cox regression model adjusted for age and country of enrolment. Shaded regions denote 95% confidence intervals for the fractional polynomial model. BMI = body mass index.

M.tb.[19] Recent studies have shown associations between low BMI and inflammatory cytokines of TB.[20,21] Anuradha et al. found that low BMI is associated with decreased levels of pro-inflammatory cytokines (interferon-gamma [IFN-γ]/tumour necrosis factor-alpha [TNF-α]/interleukin [IL] 4/IL-22/IL-1α/IL-1β/IL-6),[22] but higher levels of regulatory cytokines (IL-5/IL-13/IL-10/transforming growth factor-beta [TGF-β]) in patients with latent TB infection (LTBI). In another study, they also reported that high BMI was positively correlated with circulating levels of pro-inflammatory cytokines (IFN-γ/TNF-α/IL-22/IL-1α/IL-12/granulocyte-macrophage colony-stimulating factor [GM-CSF]), while low BMI was negatively correlated with circulating levels of anti-inflammatory cytokines (IL-4/IL-5/TGF-β)[23] in patients with LTBI. High BMI may protect against disease progression from TB infection by altering an individual's cytokine environment. Kumar et al.[21] showed that low BMI was associated with diminished plasma cytokines (IFN-γ/TNF-α/IL-2/IL-17A/IL-22/IL-1β/IL-6/IL-12/GM-CSF/IL-4/IL-5/IL-13/IL-10/TGF-β) and chemokines (CCL1/CCL2/CCL3/CCL4/CCL-11/CXC/CXCL1/CXCL2/CXCL9/CXCL10/CXCL11) in both active and latent TB patients. Although BMI is often used to measure adiposity, it is not solely responsible for modulating the immune system.[24] Instead, the physiological processes associated with excess adiposity, particularly fat accumulation, are believed to influence immune function.[25] Excess adipose tissue, especially visceral fat, is related to activating pro-inflammatory adipokines and chronic low-grade inflammation. This can lead to immune dysregulation and an increased susceptibility to inflammatory diseases. In turn, obesity suppresses the secretion of anti-inflammatory adipokines.[26] In this study, the biological mechanisms underlying the harmful effect of underweight BMI on TB are not explored. Further research is needed to clarify these mechanisms.

The MFP approach showed a sharp decrease in log (TB incidence) at BMI <25 kg/m^2 and a plateau in log (TB incidence) at BMI >25 kg/m^2. This pattern indicates a non-linear relationship between BMI and TB incidence and is consistent with weak evidence of a quadratic effect for BMI, whether grouped or continuous. The MFP approach provides a robust and precise method for determining the functional form of BMI covariate and its relationship with incident TB. This approach has many advantages over other ways of modelling BMI and has been used to study various associations in the past. Although the use of fractional polynomials to establish a relationship between BMI and TB incidence has not been documented previously, its use in this current dataset strengthens the robustness of these analyses.

This study had some limitations. The data set for this analysis was small in terms of the number of TB events (n = 76), which limited multivariable adjustment. Observed associations between low BMI and incident TB could be biased by uncontrolled confounding, which is one limitation of this study. Due to data limitations, we could not fully account for factors like nutritional intake and socio-economic status. We, therefore, believe that our findings may be limited in their internal validity by residual confounding.

BMI is a simple, rapid, and accurate way to determine a person's metabolic status. However, it has some limitations. BMI does not distinguish between adipose fat, muscle, bone, and water, which are important factors in assessing health risks and nutritional status, particularly in TB. Hence, BMI should be supplemented with other nutritional measures in TB studies to assess nutritional status[18,27] accurately.

CONCLUSIONS

Lower BMI was independently associated with a higher risk of TB development among PLHIV on

ART living in TB endemic areas. Individuals with BMI <18.5 kg/m^2 are at increased risk of developing TB vs those with BMI ≥ 18.5 kg/m^2. Across multiple methods for modelling the association between BMI and TB incidence (linear, non-linear, and categorical), BMI showed a log-linear association with TB incidence. However, the MFP approach revealed that the relationship is more complex than a simple log-linear association.

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RÉSUMÉ

CONTEXTE: Un faible indice de masse corporelle (BMI) constitue un facteur de risque significatif pour l'évolution de la TB à l'échelle mondiale. Les connaissances concernant cette relation chez les personnes vivant avec le VIH (PLHIV) et la forme fonctionnelle de la courbe d'incidence du BMI-TB demeurent limitées.

MÉTHODES: Analyse secondaire d'un essai contrôlé randomisé portant sur le traitement préventif de la TB chez les PLHIV en Afrique du Sud, au Mozambique et en Éthiopie. Les participants ont reçu un traitement hebdomadaire de 3 mois avec de la rifapentine-isoniazide à haute dose, administré une ou deux fois sur une période de 2 ans. Des polynômes fractionnaires multivariables (MFP) ont été employés pour examiner les formes fonctionnelles du BMI. Le délai avant l'apparition de la TB a été modélisé à l'aide de la régression des risques proportionnels de Cox.

RÉSULTATS: Un total de 76 cas de TB a été enregistré, ce qui se traduit par un taux d'incidence global de TB de 1,2 pour 100 personnes-années (IC à 95% 1,0–1,6). Le BMI de base <18,5 kg/m^2 était associé à un risque de TB multiplié par 2,6 par rapport au BMI 18,5–24,9 kg/m^2 (aHR 2,6 ; IC à 95% 1,4-4,8 ; $P < 0,001$). En revanche, un BMI ≥30 kg/m^2 était lié à un risque réduit de TB (aHR 0,5 ; IC à 95% 0,2–1,0). Les analyses du BMI, tant en continu qu'en catégories, ont révélé des preuves limitées de relations dose-réponse quadratiques ($P = 0,08$ et $P = 0,09$, respectivement). L'analyse de la MFP a révélé une corrélation entre la diminution de l'incidence de la TB et l'augmentation du BMI jusqu'à environ 25 kg/m^2, suivie d'une diminution moins prononcée de l'incidence de la TB pour une augmentation du BMI >25 kg/m^2.

CONCLUSIONS: Chez les PLHIV, le BMI a révélé une corrélation log-linéaire inverse avec l'incidence de la TB. L'analyse par MFP a indiqué que cette relation est plus nuancée qu'une simple association log-linéaire.