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Heterogeneity in the association of BMI with type 2 diabetes across lifestyle behaviour combinations: A MAIHDA analysis of 293,825 UK Biobank participants

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Abstract

Background

Higher body mass index (BMI) is strongly associated with greater risk of type 2 diabetes (T2D), but it remains unclear how lifestyle behaviour combinations modify the BMI-T2D relationship. We applied multilevel analysis of individual heterogeneity and discriminatory accuracy (MAIHDA) to examine heterogeneity in the BMI-T2D association across lifestyle profiles.

Methods

The sample comprised 148,363 males and 145,462 females. Participants were classified into 48 strata combining five lifestyle behaviours: fruit and vegetable intake, physical activity, sleep duration, alcohol intake, and smoking. MAIHDA models were fitted with individuals nested within strata. Sex-stratified binary logistic models examined how lifestyle behaviours and BMI accounted for between-strata variation in T2D, including whether BMI associations varied across strata, and adjusted for sociodemographic confounders. Variance partition coefficient (VPC), proportional change in variance (PCV), and strata-specific BMI associations with T2D were estimated.

Results

T2D prevalence was 11.7% in males and 6.3% in females. Each 1 kg/m² higher BMI was associated with higher T2D odds in males (OR 1.18; 95% CrI 1.17–1.19) and females (OR 1.15; 95% CrI 1.15–1.16). Between-stratum variation was modest in the null models (VPC: males 6.7%; females 8.0%) and was largely explained by additive lifestyle effects (PCV: 96.8% and 97.2%, respectively). The random BMI slope SD was 0.01 (95% CrI 0.00–0.02) in both sexes, with broadly similar positive BMI-T2D associations across lifestyle strata.

Conclusion

Higher BMI was associated with greater T2D odds in both sexes, with little heterogeneity across lifestyle strata, highlighting the importance of weight management across lifestyle profiles.

Introduction

Type 2 diabetes (T2D) remains a major global health challenge, with prevalence continuing to rise across high- and low-income settings [1, 2]. In the UK, an estimated 3.6 million adults are living with T2D, which contributes substantially to morbidity, premature mortality, and healthcare expenditure [3, 4]. Obesity is a principal modifiable risk factor for T2D, with higher body mass index (BMI) strongly associated with increased T2D risk [5, 6].

However, obesity does not confer equal risk to all individuals [7, 8]. Evidence indicates that individuals with similar BMI or adiposity can exhibit markedly different T2D risks [9, 10]. The relationship between BMI and T2D therefore, may not be uniform across populations and is likely related to variation arising from underlying biological, behavioural, and contextual factors [8]. For example, a systematic review by *Orazumbekova et al.*, found stronger associations of overweight and obesity with T2D among White than Black and East Asian populations [8].

Among these factors contributing to the heterogeneity in the BMI-T2D relationship, modifiable lifestyle behaviours are particularly relevant because they influence both adiposity and T2D. Diet, physical activity, alcohol consumption, smoking, and sleep are independently associated with both obesity and T2D risk [11–18]. Higher fruit and vegetable intake and physical activity are generally associated with lower BMI and T2D risk, whereas excessive alcohol intake, smoking, and short or long sleep durations are associated with higher BMI and T2D risk [14–16, 18–23]. Beyond their independent associations with BMI and T2D, lifestyle behaviors may also modify the BMI-T2D association because they can influence metabolic pathways linking adiposity to T2D, including insulin sensitivity, glucose metabolism, and inflammation [24, 25]. For example, physical activity can improve insulin sensitivity and glucose uptake alongside its effects on adiposity, providing a potential mechanism through which the T2D risk associated with obesity may differ according to physical activity. A systematic review by *Qin et al*., found that, physical activity interacts with obesity to affect T2D risk due to obesity [24]. Similarly, insufficient sleep can promote weight gain through increased appetite and energy intake while also impairing insulin sensitivity and glucose metabolism [25] . A study by *Lee et al*. reported that, associations between sleep duration and diabetes varied by obesity status [26].

Lifestyle behaviors in real-world settings these behaviors rarely occur in isolation and instead co-occur, giving rise to distinct lifestyle profiles [27]. Such behavioural clustering jointly influences both obesity and diabetes outcomes [28–30]. For instance, in the Dutch Lifelines

cohort, a pattern of low physical activity, poor diet, and smoking was associated with higher risks of obesity and incident T2D [31]. Studying behaviors in isolation may obscure important effect modification, as the interplay between co-occurring behaviors can amplify or attenuate risk for T2D related to BMI [32, 33].

Modelling complex effect modification across multiple lifestyle combinations is analytically challenging and requires advanced statistical approaches. Existing conventional approaches are often limited in their ability to capture high-order interactions [34]. Standard approaches such as conventional logistic regression and latent class analysis (LCA), typically require specifying numerous interaction terms or may not capture complex effect modification [34, 35]. As the number of behavioural combinations increase, these models can become unstable and difficult to interpret, particularly when data is sparse [34–36].

Intersectional Multilevel Analysis of Individual Heterogeneity and Discriminatory Accuracy (I-MAIHDA) has become the gold standard method for studying intersectional inequalities. Briefly, this approach involves assigning participants to intersectional groups or strata based on combinations of individual characteristics and then fitting multilevel models in which individuals are nested within strata [37, 38]. This more general version of the method has been termed multi-categorical MAIHDA [39]. In our previous MAIHDA application, we examined variation in T2D onset across combinations of lifestyle behaviours and found that differences were largely explained by the additive associations of individual behaviours [40]. However, although that study demonstrated how T2D onset varied across lifestyle profiles, whether the association between BMI and T2D itself also varies across these profiles remains unexplored.

Extending our previous work, we apply multi-categorical MAIHDA with random BMI slopes to examine heterogeneity in the relationship between BMI and T2D across lifestyle profiles defined by five key lifestyle behaviors.

Methods

Data and sample

This study used data from the UK Biobank, a population-based cohort of over 500,000 adults aged 40-69 years recruited across the UK between 2006 and 2010. At baseline, participants provided sociodemographic, lifestyle, and health information and underwent physical assessments and biological sampling. All participants provided written informed consent.

Further details of the UK Biobank design and data collection are reported elsewhere [41]. This study was conducted under the approved project number 80843.

Measures

Lifestyle behaviors assessment

Five modifiable lifestyle behaviors were assessed at baseline using self-reported questionnaires. Four behaviors were dichotomised according to whether participants met current UK National Health Service (NHS) recommendations: fruit and vegetable intake (≥5 portions/day), physical activity (≥150 minutes of moderate-intensity or ≥75 minutes of vigorous-intensity activity per week), sleep duration (7–9 hours/day), and alcohol intake (≤14 units/week) [42–45]. Smoking status was classified into three categories: previous, never, and current smoker. Detailed descriptions of variable derivations and coding decisions are provided in Supplementary Table 1.

Lifestyle strata

Participants were classified into 48 distinct lifestyle strata reflecting all possible combinations of the five behaviors ($2 \times 2 \times 2 \times 2 \times 3$). Each stratum is represented by a unique five-digit code indicating meeting (1) or not meeting (0) to fruit and vegetable intake, physical activity, sleep duration, and alcohol intake recommendations, with smoking coded as previous (0), current (1), or never smoker (2). For example, the code 01112 denoted the stratum who did not meet fruit and vegetable intake recommendations, met physical activity, sleep duration, and alcohol intake recommendations, and were never smokers.

BMI

BMI was derived from measured weight and height at baseline (Field ID 21001) and analysed continuously in kg/m². BMI was mean centred prior to modelling (males: 27.7 kg/m²; females: 26.5 kg/m²).

Outcome: prevalent T2D

Prevalent T2D at baseline was identified using a composite algorithm, including self-reported T2D (Field ID. 20002), first occurrence of ICD-10 coded E11 (Field ID. 130708), doctor diagnosed diabetes (Field ID. 2443), glycated haemoglobin (HbA1c Field ID. 30750), and diabetes or glucose-lowering medication use (Field IDs. 20003, 6177, 6153). The clinical

HbA1c threshold of ≥48 mmol/mol was used to support diabetes classification [46]. The full case ascertainment algorithm is provided in Supplementary Figure 1.

Confounders

Potential confounders included age, ethnicity, Townsend deprivation index (TDI), and employment status. Age and ethnicity were self-reported, with ethnicity categorised as White or Other than White. TDI was derived from residential postcode, and employment was categorised as employed or not currently employed/retired. Continuous confounders were mean centred prior to modelling.

Statistical analysis

Analytical framework

Analyses were conducted separately for males and females. Bayesian Markov Chain Monte Carlo (MCMC) logistic MAIHDA models examined heterogeneity in the BMI–T2D association across lifestyle strata. Individuals (Level 1) were nested within the 48 strata (Level 2), allowing baseline T2D odds and the BMI-T2D association (random slopes) to vary across strata.

Model specification

Random-intercept models quantified between-stratum variation in T2D, while subsequent random-slope models allowed the BMI–T2D association to vary across lifestyle strata.

Five sequential MAIHDA models were fitted consistent with the MAIHDA procedures [37]:

1) Model 0 (Null model). Random intercepts; used to quantify between-strata variation in T2D,

$$y_{ij} \sim \text{Bernoulli}(\pi_{ij})$$
$$\text{logit}(\pi_{ij}) \equiv \ln\left(\frac{\pi_{ij}}{1-\pi_{ij}}\right) = \beta_0 + u_j$$
$$u_j \sim N(0, \sigma_u^2)$$

2) Model 1. Model 0 plus the five lifestyle behaviour variables added to the fixed-part predictor to the model:

$$y_{ij} \sim \text{Bernoulli}(1, \pi_{ij})$$
$$\text{logit}(\pi_{ij}) \equiv \ln\left(\frac{\pi_{ij}}{1-\pi_{ij}}\right) = \beta_0 + \beta_1 x_{1j} + \cdots + \beta_5 x_{5j} + u_j$$

$$u_j \sim N(0, \sigma_u^2)$$

3) Model 2. Model 1 with the addition of mean-centered BMI as a fixed effect;

$$y_{ij} \sim \text{Bernoulli}(1, \pi_{ij})$$
$$\text{logit}(\pi_{ij}) \equiv \ln\left(\frac{\pi_{ij}}{1-\pi_{ij}}\right) = \beta_0 + \beta_1 x_{1j} + \cdots + \beta_5 x_{5j} + \beta_6 x_{6ij} + u_j$$
$$u_j \sim N(0, \sigma_u^2)$$

4) Model 3. Unlike the previous models that assumed a fixed effect of the predictors on the outcome for every strata, Model 3 furthers Model 2 with the addition of level 2 random slopes for BMI; used to allow the effect of BMI on T2D to vary across strata.

$$y_{ij} \sim \text{Bernoulli}(1, \pi_{ij})$$
$$\text{logit}(\pi_{ij}) \equiv \ln\left(\frac{\pi_{ij}}{1-\pi_{ij}}\right) = \beta_0 + \beta_1 x_{1j} + \cdots + \beta_5 x_{5j} + \beta_6 x_{6ij} + u_{0j} + u_{6j} x_{6ij}$$
$$\begin{pmatrix} u_{0j} \\ u_{1j} \end{pmatrix} \sim N\left\{\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} \sigma_{u0}^2 & \\ \sigma_{u01} & \sigma_{u1}^2 \end{pmatrix}\right\}$$

5) Model 4 (Fully adjusted). Model 3 with the addition of the confounders as fixed effects;

$$y_{ij} \sim \text{Bernoulli}(1, \pi_{ij})$$
$$\text{logit}(\pi_{ij}) \equiv \ln\left(\frac{\pi_{ij}}{1-\pi_{ij}}\right) = \beta_0 + \beta_1 x_{1j} + \cdots + \beta_5 x_{5j} + \beta_6 x_{6ij}$$
$$+\beta_7 x_{7ij} + \cdots + +\beta_{10} x_{10ij} + u_{0j} + u_{6j} x_{6ij}$$
$$\begin{pmatrix} u_{0j} \\ u_{1j} \end{pmatrix} \sim N\left\{\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} \sigma_{u0}^2 & \\ \sigma_{u01} & \sigma_{u1}^2 \end{pmatrix}\right\}$$

Variance parameters

For models 0–2, the Variance partitioning coefficient (VPC) was estimated to quantify the proportion (%) of total variation in T2D attributable to differences between lifestyle strata. The VPC was calculated as:

$$\text{VPC} = 100 \times \frac{\sigma_u^2}{\sigma_u^2 + \sigma_{\text{residual}}^2}$$

Where σ_{u}^2 is the stratum-level random effect variance and $\sigma_{\text{residual}}^2$ is the residual variance, which in logistic models, is set equal to the variance of the standard logistic distribution $\frac{\pi}{3} \approx 3.29$ [37].

We also computed the Proportional change in variance (PCV) which quantifies the proportion (%) reduction in stratum-level variance relative to model 0 following the inclusion of fixed effects:

$$\text{PCV} = 100 \times \frac{\sigma^2_{u[\text{Model 0}]} - \sigma^2_{u[\text{Model X}]}}{\sigma^2_{u[\text{Model 0}]}}$$

Where $Model\ X$ refers to the subsequent models 1 and 2. The Model 1 PCV signifies how much of the between-strata variation is explained by additive main effects (e.g., of the lifestyle behaviour variables used to define the strata), while the remaining unexplained variation (1-PCV) reflects multiplicative interactions.

In Models 3–4, inclusion of random BMI slopes make between-stratum variance dependent on BMI, therefore, a single overall VPC or PCV cannot be meaningfully estimated, and these measures were not calculated.

Strata-specific random effects

Posterior estimates of the strata-specific random effects were extracted from the fully adjusted Model 4. Random intercepts represented differences in T2D risk at mean BMI, while random BMI slopes represented differences in the BMI-T2D association. The slope-intercept joint distribution was also examined to assess whether strata with higher risk for individuals with mean BMI exhibited stronger or weaker BMI-T2D association.

Data preparation and variable coding were undertaken using Stata version 17 (StataCorp LLC, College Station, TX). Bayesian models were fitted in R version 4.5.0 using the brms package. Convergence was evaluated using effective sample sizes and R-hat statistics. Relative model fit was assessed using the leave-one-out information criterion (LOOIC).

Results

Participants characteristics

Among the final analytical sample (n=293,825), 17,341 (11.7%) males and 9,162 (6.3%) females had T2D. Of the initial 502,356 participants, 208,531 were excluded because of missing data on lifestyle behaviors (n = 183,816), confounders (n = 3,380), T2D (n = 20,379), and BMI (n = 956). Details of missingness and the rationale for complete-case analysis are provided in the Supplementary Materials [40].

Most participants met physical activity (82% males; 83% females) and sleep recommendations (75%; 76%), whereas fewer met fruit and vegetable recommendations (28%; 39%). Alcohol recommendations were met by 34% of males and 60% of females. 49% of males and 59% females never smoked. Mean BMI was 27.7 kg/m² in males and 26.5 kg/m² in females. Full descriptive characteristics are presented in Table 1.

MAIHDA results

Associations of lifestyle behaviors and BMI with T2D

In Model 1 (Tables 2 and 3), meeting physical activity and sleep recommendations and never smoking were associated with lower T2D odds in both sexes. For example, among males, meeting physical activity guidelines was associated with a 0.66 times lower odds of T2D. In contrast, meeting alcohol and fruit and vegetable intake recommendations were associated with higher odds of T2D in both sexes. Current smoking (versus never smoking) showed opposite directions of association by sex, with an OR of 0.92 (95% CrI: 0.84, 1.00) for males and 1.26 (95% CrI: 1.13, 1.40) for females.

In Model 2, a one-unit (1 kg/m^2) increase in BMI was associated with a 1.18 (95% CrI: 1.17, 1.19) higher T2D odds in males and 1.15 (95% CrI: 1.15, 1.16) higher odds in females. To put this 1 kg/m² difference in context, BMI typically varied considerably more within lifestyle strata: the median within-stratum SD was 4.47 kg/m² (IQR 4.01–4.83) in males and 5.06 kg/m² (IQR 4.66–5.45) in females (Supplementary Table 2). Thus, the reported association reflects the higher T2D odds associated with a relatively small increment in BMI. Following BMI adjustment, lifestyle–T2D associations changed modestly from Model 1 in both sexes, with full estimates presented in Tables 2 and 3.

Fixed effect estimates remained broadly stable across Models 3 and 4, although adjustment for confounders attenuated the association of current smoking among males (OR 0.75, 95% CrI 0.69-0.79)

Variance parameters

In the null model, the stratum-level SD was 0.48 in males, with a VPC of 6.7%, indicating that approximately 7% of variation in T2D propensity was attributable to differences between lifestyle strata (Table 2). Among females (Table 3), the stratum random effect SD (0.53) and VPC (7.9%) were marginally higher.

The stratum random effect SDs (males: 0.09, females: 0.09) and the resulting VPCs (males: 0.23%, females: 0.24%) in Model 1 were substantially lower than in Model 0, indicating that little between-stratum variation in the propensity to be T2D remained after including the lifestyle behaviours as additive fixed effects. High PCVs (males: 96.8%, females: 97.2%) in Model 1 indicated that lifestyle behaviors explained 97% of the between-stratum variation in T2D risk.

For both sexes, after adding BMI in Model 2, the VPCs decreased further (males: 0.12%, females: 0.17%), while the PCVs increased (males: 98.3%, females: 97.9%).

The stratum intercept random effect SD in Models 3 and 4 was similar to the random effect SD in Model 2 for both sexes.

Heterogeneity in the BMI-T2D associations across strata

Stratum-specific random intercepts from Model 4 illustrate residual between-stratum variation in T2D risk at mean BMI after full adjustment (Supplementary Figures 2 and 3).

The BMI slope SD was 0.01 for both sexes in Models 3 and 4, indicating limited variation in the BMI- T2D association across lifestyle strata. Allowing BMI slopes to vary in Model 3 provided little improvement in predictive performance over Model 2 in males (LOOIC 96,389.3 vs 96,390.6; SE 2.3) or females (61,213.8 vs 61,220.8; SE 3.3). Full LOOIC results for all models are provided in Supplementary Table 3. Stratum-specific predicted probabilities similarly showed broadly positive BMI-T2D associations across strata within their observed BMI ranges (Supplementary Figures 4 and 5). Figures 1 and 2 present Model 4 stratum-specific BMI slopes for males and females, respectively, showing the extent to which each stratum-specific BMI association differed from the overall BMI effect.

Model 4 intercept-slope correlations were close to zero in both sexes (males: 0.07 in Model 3 and 0.01; females: -0.16 in Model 3 and -0.12 in Model 4). Supplementary Figures 6 and 7 illustrate the Model 4 relationship between stratum-specific random intercepts (baseline T2D risks) and BMI slopes (BMI effects) for males and females, respectively.

Discussion

In this study, we applied MAIHDA within a Bayesian binary logistic regression framework to examine heterogeneity in the BMI-T2D association across 48 lifestyle behaviour strata. The key finding was that BMI was positively associated with T2D risk consistently across models.

However, there was little evidence of heterogeneity in the BMI-T2D association across lifestyle strata. We also found that, most of the variation in T2D risk between strata was attributable to differences between individuals rather than differences between strata.

In Model 2, each 1 kg/m² increase in BMI was associated with approximately 18% and 15% higher T2D odds in males and females. These estimates remained almost unchanged in subsequent models 3 (random slopes) and 4 (adjusted for sociodemographic confounders). Previous research has similarly shown that overweight (BMI 25.0-29.9 kg/m^2) and obesity (BMI $\geq$ 30 kg/m^2) remain strongly associated with T2D after adjusting for lifestyle behaviors [47, 48]. Our findings extend this evidence by showing that BMI-T2D association remained consistent across models, following adjustment for sociodemographics. While causality cannot be established from our study and residual confounding may remain, evidence from causal studies suggests that the BMI–T2D association is unlikely to be explained entirely by confounding [49].

The small BMI slope SDs (0.01) in both sexes for Models 3 and 4 indicated that the BMI-T2D association was very similar across 48 strata. Existing evidence examining how combined lifestyle behaviors modify the adiposity-diabetes relationship is limited. However, Studies of individual behaviours suggest either modest modification or, particularly for physical activity, stronger interactions with adiposity [50–52]. While our findings might differ from some of this previous evidence, it is important to emphasise that our analysis examined combined lifestyle profiles, rather than individual behaviors. Overall, there was little evidence that combined lifestyle behavior profiles meaningfully modify the BMI-T2D association.

The low intercept–slope correlations in Models 3 and 4 (males: 0.07 and 0.01; females: -0.16 and -0.12) indicated that strata with higher T2D risk at mean BMI did not consistently have stronger or weaker BMI-T2D associations. As such, the strength of the BMI-T2D association was relatively stable across lifestyle combinations. Our interpretation is consistent with prior work suggesting that lifestyle behaviors may primarily influence absolute diabetes risk rather than substantially modifying adiposity-related diabetes risk [47, 48, 50, 53, 54].

We also noticed the substantial drop in VPCs for both sexes in Model 1 (males: 0.23%, females: 0.24%) and Model 2 (males: 0.12%, females: 0.17%) compared to the null model (males: 6.7%, females: 7.9%), consistent with previous MAIHDA studies, showing large reductions after including stratum-defining variables as fixed effects [37, 55–57]. Model 1 PCVs were high (i.e., 97%), indicating that little between-stratum variation in T2D propensity remained after

accounting for individual lifestyle behaviours and BMI as fixed effects, and that most of the variation observed in the null model was explained by additive fixed effects. These findings provide little evidence of multiplicative associations between lifestyle behaviours on T2D odds.

One unexpected finding from the fixed effects of the models was that meeting recommendations for fruit and vegetable and alcohol intake were positively associated with T2D odds. The odds were substantially high among both sexes meeting the low alcohol intake recommendation. While this may seem counterintuitive, there are multiple reasons that might have led to this finding. First, the categorisation of alcohol intake variable to meeting or not meeting recommendations, a requirement for strata creation, might have obscured some heterogeneity around cut points (e.g. 13.9 vs 14.1 units per week). This also doesn't factor in the type of alcoholic drink, patterns such as binge drinking, and the frequency. Additionally, previous studies have shown alcohol-T2D associations to be highly non-linear and heterogeneous across populations and beverage type. These findings should therefore not be interpreted as evidence that exceeding alcohol recommendations is beneficial but rather may reflect limitations in how alcohol consumption was captured and categorised in this analysis, as well as the fact that these associations were estimated conditional on the other lifestyle behaviours included in the model.

From a public health perspective, the broadly similar positive BMI-T2D association across lifestyle strata suggests that prevention strategies should prioritise healthy weight maintenance across all lifestyle profiles, rather than focusing on specific behavioural clusters. At the same time, our results do not diminish the importance of lifestyle behaviors for T2D prevention.

Strengths and limitations

This study has several key strengths. First, the large UK Biobank sample enabled the application of MAIHDA in order to estimate the heterogeneity across lifestyle strata. Second, the MAIHDA approach allowed robust modelling of lifestyle strata, allowing heterogeneity to be quantified without specifying numerous interaction terms, and benefitting from partial pooling to stabilise estimates for sparsely populated strata. Third, the Bayesian framework further strengthened the inference by providing full posterior uncertainty for both fixed effects and variance components. Finally, lifestyle strata were defined using standardised UK public health recommendations, therefore improving interpretability and policy relevance.

Several limitations of the study should be acknowledged. UK Biobank participants are generally healthier and less socioeconomically deprived than the wider UK population, potentially limiting generalisability through selection bias [58]. Second, the cross-sectional nature of the study limits causal inferences and prevents conclusions about incident T2D risk. Changes in BMI and lifestyle behaviours following T2D diagnosis may also have influenced the observed associations across lifestyle profiles. Although our previous study examined T2D onset using Bayesian survival MAIHDA, extending this framework to incorporate random BMI slopes would introduce additional computational complexity. Future longitudinal analyses could investigate whether similar patterns of heterogeneity are observed for incident T2D. Furthermore, while the dichotomisation of lifestyle behaviors required for MAIHDA modelling may improve interpretability, it may obscure dose-response relationships with continuous outcomes. Finally, in studies where sex differences are of primary interest, joint modelling of the relevant male and female models would allow direct Bayesian inference on these differences via posterior distributions of the relevant parameter contrasts [59].

Conclusions

Using a Bayesian MAIHDA, we examined heterogeneity in the BMI-T2D association across lifestyle behavior combinations. We found that higher BMI was consistently associated with greater T2D odds in both sexes. However, there was little heterogeneity in the BMI-T2D association across strata, suggesting that lifestyle behaviour combinations did not meaningfully modify the BMI-T2D association. From a public health perspective, the findings suggest that the association between higher BMI and T2D risk is consistent across lifestyle profiles, and that weight management should remain a key focus of T2D prevention efforts irrespective of behavioural profile.

CRediT authorship contribution statement

Swain Ansuman: Conceptualization, Methodology, Formal analysis, Investigation, Visualization, Writing - original draft, Writing - review & editing. Pearson Natalie: Conceptualization, Methodology, Writing - review & editing. Willis Scott: Conceptualization, Methodology, Supervision, Writing - review & editing. Leckie George: Conceptualization, Methodology, Formal analysis, Validation, Writing - review & editing. Johnson Will: Conceptualization, Methodology, Supervision, Writing - review & editing. All authors: Writing - review & editing.

Data availability statement

The data underlying this article cannot be shared publicly for the privacy of individuals that participated in the study. The data will be shared on reasonable request to the corresponding author.

Ethics approval and consent to participate

Ethical approval was covered by UK Biobank's existing approval, and all participants provided written informed consent; therefore, no separate ethical approval was required.

Competing interests declaration

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Table 1. Descriptive statistics

		Males n = 148,363	Females n = 145,462
Lifestyle variables			
Smoking			
Current	N (%)	16,719 (11.3)	11,845 (8.1)
Previous	N (%)	58,555 (39.5)	48,508 (33.3)
Never	N (%)	73,089 (49.3)	85,109 (58.5)
Fruit and vegetable			
Not meets	N (%)	106,958 (72.1)	88,187 (60.6)
Meets	N (%)	41,405 (27.9)	57,275 (39.4)
Physical activity			
Not meets	N (%)	27,086 (18.3)	24,937 (17.1)
Meets	N (%)	121,277 (81.7)	120,525 (82.9)
Alcohol			
Not meets	N (%)	97,950 (66.0)	58,864 (40.5)
Meets	N (%)	50,413 (34.0)	86,598 (59.5)
Sleep			
Not meets	N (%)	37,445 (25.2)	35,269 (24.2)
Meets	N (%)	110,918 (74.8)	110,193 (75.8)
BMI	Mean (SD)	27.7 (4.1)	26.5 (4.8)
Confounders			
Age	Mean (SD)	56.7 (8.1)	55.8 (8.0)
Townsend Deprivation Index	Mean (SD)	−1.5 (3.0)	−1.6 (2.9)
Ethnicity			
White	N (%)	136,751 (92.2)	132,485 (91.1)
Non-White	N (%)	11,612 (7.8)	12,977 (8.9)
Employment			
Employed	N (%)	92,529 (62.4)	84,824 (58.3)
Not currently employed or retired	N (%)	55,834 (37.6)	60,638 (41.7)
Outcome			
Prevalent Type 2 diabetes			
Yes	N (%)	17,341 (11.7)	9,162 (6.3)
No	N (%)	131,022 (88.3)	136,300 (93.7)

Table 2. MAIHDA logistic regression models for type 2 diabetes with random BMI slopes (Males)

	Model 0 – Null OR (95% CrI)	Model 1 – Main effects OR (95% CrI)	Model 2 – BMI as predictor OR (95% CrI)	Model 3 – Random BMI slope OR (95% CrI)	Model 4 – Confounders[a] included OR (95% CrI)
Fruit & vegetable intake					
Not meets	–	–	–	–	–
Meets	–	1.20 (1.12, 1.28)	1.17 (1.11, 1.25)	1.16 (1.09, 1.23)	1.07 (1.02, 1.14)
Physical activity					
Not meets	–	–	–	–	–
Meets	–	0.66 (0.61, 0.71)	0.80 (0.76, 0.85)	0.79 (0.74, 0.84)	0.76 (0.72, 0.80)
Sleep					
Not meets	–	–	–	–	–
Meets	–	0.76 (0.71, 0.81)	0.88 (0.83, 0.92)	0.87 (0.82, 0.92)	0.85 (0.80, 0.90)
Alcohol intake					
Not meets	–	–	–	–	–
Meets	–	1.58 (1.49, 1.70)	1.62 (1.52, 1.71)	1.62 (1.54, 1.72)	1.54 (1.46, 1.62)
Smoking status					

Previous	–	–	–	–	–
Current	–	0.92 (0.84, 1.00)	1.06 (0.99, 1.15)	1.07 (0.99, 1.16)	0.75 (0.69, 0.79)
Never	–	0.54 (0.50, 0.58)	0.61 (0.57, 0.65)	0.61 (0.57, 0.66)	0.75 (0.69, 0.77)
BMI (per unit increase)	–	–	1.18 (1.17, 1.19)	1.18 (1.17, 1.19)	1.18 (1.17, 1.20)
SD (strata intercept)	0.48 (0.39, 0.61)	0.09 (0.06, 0.12)	0.06 (0.03, 0.10)	0.06 (0.02, 0.10)	0.04 (0.01, 0.08)
SD (BMI slope)	–	–	–	0.01 (0.00, 0.02)	0.01 (0.00, 0.02)
Intercept–slope correlation	–	–	–	0.07 (-0.64, 0.79)	0.01 (-0.72, 0.78)
VPC (%)	6.65	0.23	0.12	–	–
PCV[b] (%)	–	96.80	98.28	–	–

[a]Confounders were Age, ethnicity, tdi, and employment status
[b]All estimated PCVs are relative to the null model

Table 3. MAIHDA logistic regression models for type 2 diabetes with random BMI slopes (Females)

	Model 0 – Null OR (95% CrI)	Model 1 – Main effects OR (95% CrI)	Model 2 – BMI as predictor OR (95% CrI)	Model 3 – Random BMI slope OR (95% CrI)	Model 4 – Confounders[a] included OR (95% CrI)
Fruit & vegetable intake					
Not meets	–	–	–	–	–
Meets	–	1.08 (1.01, 1.17)	1.16 (1.07, 1.25)	1.15 (1.06, 1.25)	1.05 (0.97, 1.13)
Physical activity					
Not meets	–	–	–	–	–
Meets	–	0.64 (0.59, 0.69)	0.86 (0.79, 0.93)	0.85 (0.78, 0.92)	0.79 (0.73, 0.87)
Sleep					
Not meets	–	–	–	–	–
Meets	–	0.68 (0.63, 0.73)	0.78 (0.73, 0.84)	0.78 (0.72, 0.84)	0.81 (0.76, 0.87)
Alcohol intake					
Not meets	–	–	–	–	–
Meets	–	2.01 (1.86, 2.18)	1.75 (1.62, 1.88)	1.75 (1.63, 1.90)	1.60 (1.49, 1.73)
Smoking status					

Previous	–	–	–	–	–
Current	–	1.26 (1.13, 1.40)	1.40 (1.27, 1.57)	1.40 (1.27, 1.55)	1.49 (1.35, 1.67)
Never	–	0.82 (0.76, 0.90)	0.88 (0.81, 0.95)	0.88 (0.81, 0.95)	0.97 (0.90, 1.05)
BMI (per unit increase)	–	–	1.15 (1.15, 1.16)	1.15 (1.15, 1.16)	1.15 (1.15, 1.16)
SD (strata intercept)	0.53 (0.43, 0.67)	0.09 (0.05, 0.14)	0.08 (0.04, 0.12)	0.08 (0.04, 0.13)	0.08 (0.03, 0.13)
SD (BMI slope)	–	–	–	0.01 (0.00, 0.02)	0.01 (0.00, 0.02)
Intercept–slope correlation	–	–	–	−0.16 (−0.79, 0.66)	−0.12 (−0.80, 0.70)
VPC (%)	7.96	0.24	0.17	–	–
PCV[b] (%)	–	97.20	97.98	–	–

[a]Confounders were Age, ethnicity, tdi, and employment status
[b]All estimated PCVs are relative to the null model

Figure 1. Random BMI slopes for type 2 diabetes risk across lifestyle combinations (Model 4, Males)

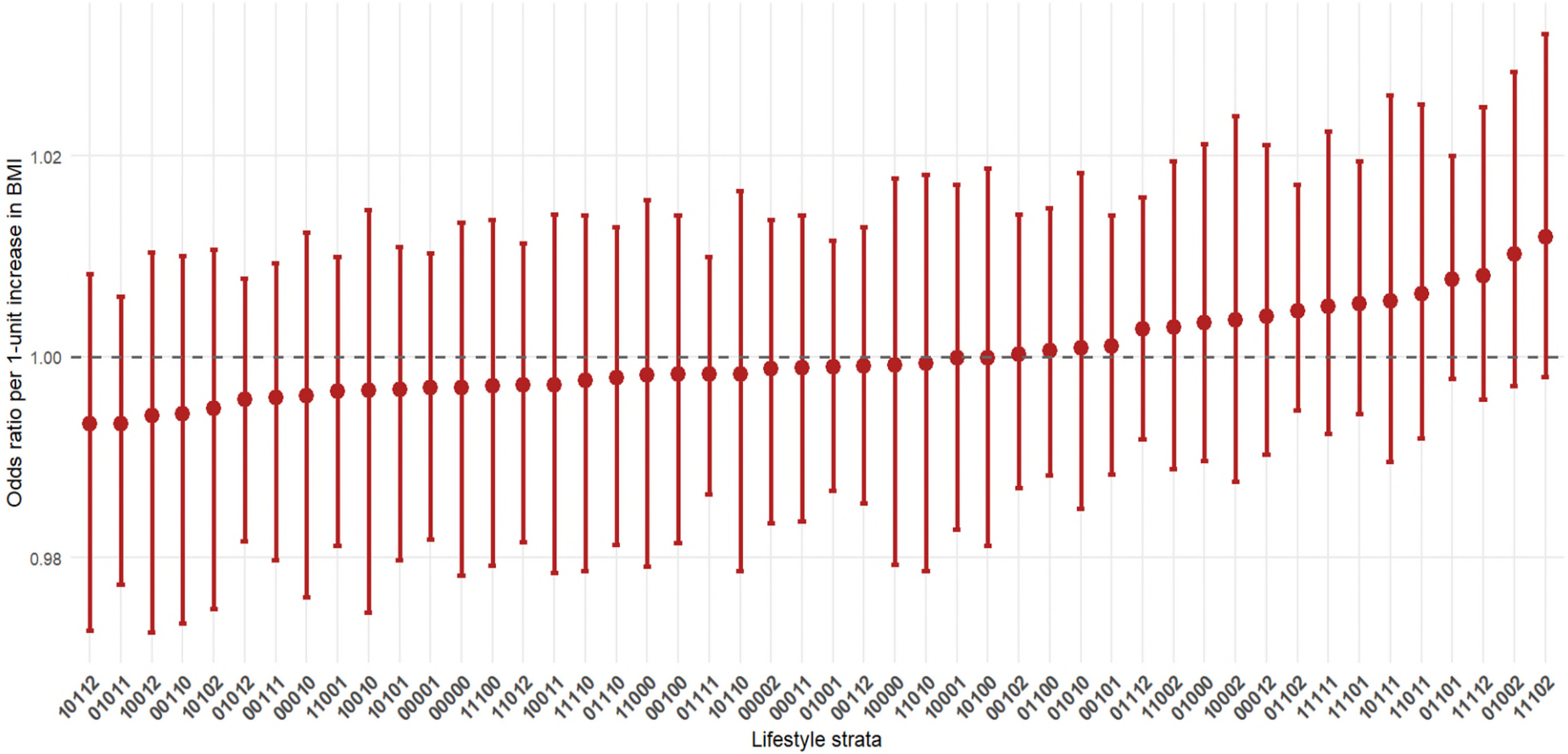
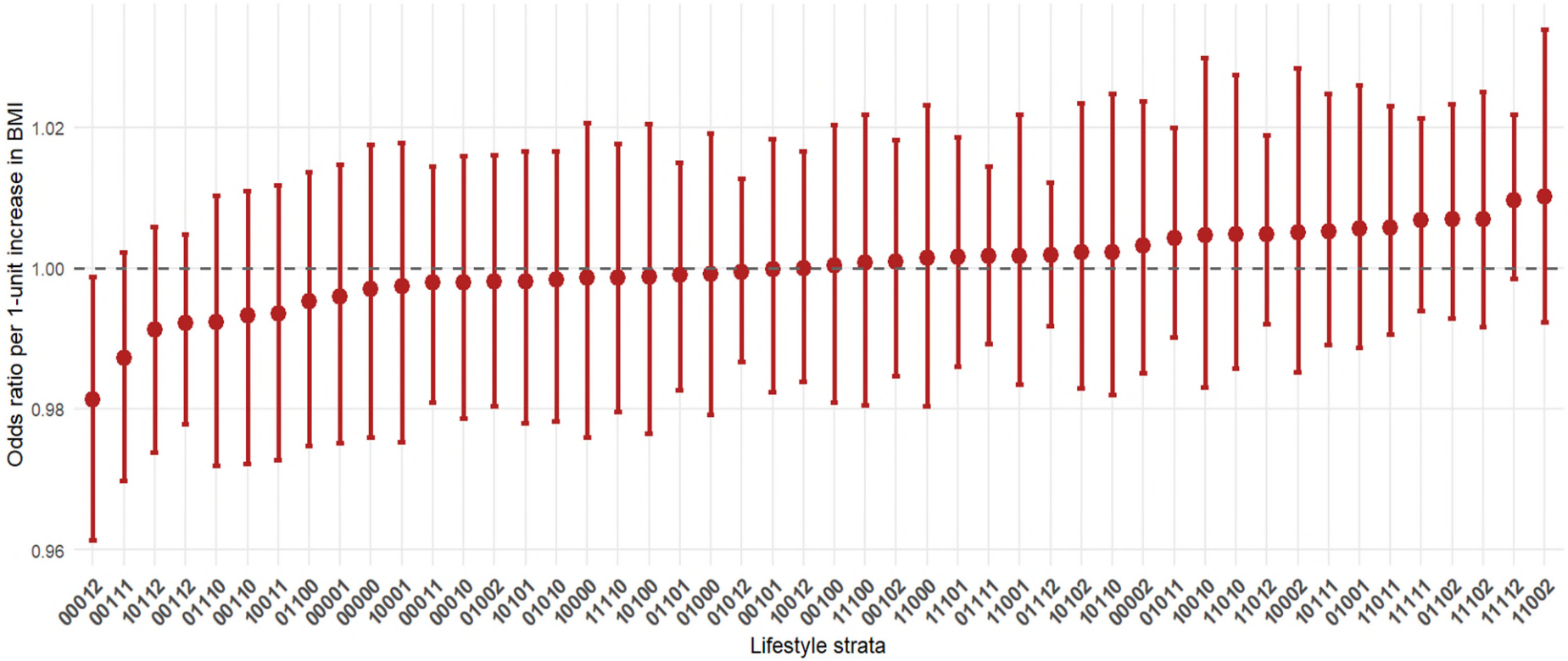


Figure 2. Random BMI slopes for type 2 diabetes risk across lifestyle combinations (Model 4, Females)



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