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Rate of Change in Childhood Body Mass Index and Risk of Metabolic Syndrome: Pathways and Generational Effects

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Rate of Change in Childhood Body Mass Index and Risk of Metabolic Syndrome; Pathways and generational effects

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ABSTRACT

Background/objective: We aim to understand associations and pathways of time-varying rates of change in childhood body mass index (BMI) on later metabolic syndrome (MetS) risk and assess whether associations persist across generations.

Subjects/methods: We utilized data from two birth cohorts; Northern Finland Birth Cohorts 1966 (NFBC1966; *N* = 5,647) and 1986 (NFBC1986; *N* = 5,583), with longitudinal anthropometric data from birth to the first cardiometabolic assessment at 31 years (1997–1998) and 16 years (2002–2003) for the NFBC1966 and 1986 respectively. Individual BMI trajectories from 3 weeks to 16 years were modelled using linear mixed-effects models with cubic splines. Period-specific BMI change rates were derived as slopes during infancy (0 to 5 months), early childhood (1.5 to 3.5 years), and late childhood (6.5 to 10 years). MetS was defined using sex- and age-specific international diabetes federation criteria. Associations with BMI slopes were assessed using logistic regression, adjusted for sex, birthweight, maternal pre-pregnancy BMI, maternal education, and maternal smoking during pregnancy. Cohort- and sex-stratified path analyses were conducted to evaluate direct and indirect associations between childhood BMI slopes and MetS.

Results: BMI slopes in early (OR: 1.3, 95% CI:1.2-1.5; OR:2.3, 95% CI: 2.0-2.8) and late (OR: 1.6, 95% CI:1.4-1.8; OR: 2.9, 95% CI:2.4-3.5) childhood were associated with both adult and adolescent MetS respectively. However, the magnitude of effects in the association were higher among NFBC1986 at 16 years than NFBC1966 at 31 years. BMI slope in late childhood had direct effect on MetS outcomes. Infancy and early childhood BMI slopes had indirect effects on MetS outcomes, mediated through late childhood BMI slope. **Conclusions:** Early growth rates shape future metabolic health, with late childhood being a critical window for metabolic risk programming, and effects appearing stronger in adolescence than adulthood.

Keywords: body mass index; metabolic syndrome; trajectory; childhood; slope; longitudinal

INTRODUCTION

The metabolic syndrome (MetS) has been identified as a global health burden (1,2) with a prevalence ranging from 20 to 40 % depending on population settings and age (3). In 2023, it was estimated that 1.54 billion adults were with MetS, which represented a 15 % increase in the global prevalence over one decade (4). In the European region, the prevalence of MetS ranges from 13 to 36 % (5), with 17 to 43 % prevalence in Finland (6). MetS is identified by the presence of three or more abnormal clinical diagnostics according to age and sex specific cut-off points for waist circumference (WC), blood pressure (BP), fasting blood glucose (FG), triglycerides (FTG), and high-density lipoprotein cholesterol (HDL-C) (7,8). MetS presents a comprehensive assessment of integrated metabolic burden, making it a critical predictor of type 2 diabetes (9), coronary heart disease (10), stroke (11), and premature mortality (12).

The current life-course theories suggest that chronic health conditions, such as obesity and MetS are shaped through the life-course (13–15). In line with the current hypothesis, infancy and childhood are thought to be important life-course windows when rapid changes in body composition occur (16). Notably, BMI changes rapidly during this period (17). In addition, BMI alone, measured before the age of 5 years in males and females can be very imprecise and lead to important misclassification of the risk of being with obesity. Most studies to date have treated childhood (from birth to 18 years) as a single life-course period for BMI development. However, emerging evidence from genome-wide association studies (18) and modelling approaches (Click or tap here to enter text.(19,20)Click or tap here to enter text. supports a counterhypothesis. Specifically, BMI in late childhood, typically after the adiposity rebound, may arise from distinct time-varying processes that influence weight gain and subsequent cardiometabolic outcomes. Importantly, these life-course windows of BMI development are not solely defined by the level of adiposity attained (e.g., weight-for-length or BMI). They should also be characterized by the *dynamics* of change within each window—specifically, the

rate and pattern (e.g., rapid vs. gradual) of BMI gain or decline. Moreover, it is essential to examine whether transitions between these developmental phases are predictable, and how such transitions may influence subsequent metabolic health. Together, this perspective emphasizes that both the *timing* and *velocity* of BMI change across childhood may play a critical role in shaping later metabolic risk which brings important scientific and clinical relevance. Indeed, its importance moves beyond generalized BMI measurements, by employing developmentally distinct growth trajectories (BMI slopes). This study will allow an identification of sensitive age windows, when rapid catch-up growth and accelerated adiposity gain is captured, and importantly a period during which metabolic risk is programmed.

To address this, we examined how time-varying rates of change in childhood BMI, previously developed for international genome-wide association studies (18), at three developmental stages, namely infancy (0 to 5 months), early childhood (1.5 to 3.5 years), and late childhood (6.5 to 10 years), were associated with early measures of MetS. We further constructed sex-specific path analysis models to assess whether infancy, early and late childhood windows confer distinct risks for MetS, or whether risk accumulates progressively across childhood. Finally, we used data from two consecutive birth cohorts, born 20 years apart, to evaluate potential convergence of findings and the influence of context-specific factors.

MATERIAL AND METHODS

Study participants

We used data from Northern Finland Birth Cohorts (NFBC) 1966 and 1986, established two decades apart in the provinces of Oulu and Lapland (21,22).

NFBC1966 study enrolled 12,055 pregnant women due to deliver their babies between January 1st to December 31st, 1966. Their newborns, 12,058 (96% of births in the study area during this period) were included in this study (23). Participants data at 31 years were collected through clinical visits, postal questionnaires, and national registers. The present analysis included participants with complete data on growth and MetS at age of 31 years (N=5647, males = 49% and females = 51%).

NFBC1986 study included 9362 pregnant mothers with due date within July 1st 1985 to June 30th, 1986, and their 9432 offspring, which constituted 99% of all births within the study region (24). Information on participants at 16 years were collected through postal questionnaires, clinical examinations, and health records. For this study analysis, we included participants with complete data on growth and MetS at the age of 16 years (N = 5583, males = 50.5% and females = 49.5%).

In both studies, we excluded participants born to multiple, and preterm births (< 37 gestational weeks) due to known specific early growth patterns and insufficient sample size to account for their effect in sensitivity analyses. We also excluded participants with incomplete data on all six metabolic variables which are required to define the MetS. The study selection is described in detail in the study flowchart (Supplementary Figure S1).

Outcome: Metabolic Syndrome (MetS)

We used the International Diabetes Federation (IDF) definition for adults (25)and for adolescence (26)to determine the MetS status of participants in NFBC1966 at 31 years and at

16 years in NFBC1986. According to IDF adult definition, MetS is diagnosed in 31 years old when WC ≥ 94 cm for males and ≥ 80 cm for females, along with the presence of at least two additional metabolic abnormalities: elevated blood pressure (SBP ≥ 130 mm Hg, DBP ≥ 85 mm Hg), FG levels ≥ 6.1 mmol/L, FTG levels ≥ 1.7 mmol/L, and HDL-C < 1.03 mmol/L for males, and < 1.29 mmol/L for females. Based on the IDF paediatric definition, MetS was diagnosed in 16 years old when WC ≥ 90cm for males and ≥ 80cm for females, plus the presence of at least two of these metabolic components: SBP ≥ 130 mm Hg, DBP ≥ 85 mm Hg, FG ≥ 5.6 mmol/L, FTG ≥ 1.7 mmol/L, and HDL-C< 1.03 mmol/L.

Metabolic measures were collected by trained research personals during clinical examinations (NFBC1966 at 31 years and NFBC1986 at 16 years). WC was measured while standing upright with a relaxed abdomen, at the level midway between the lowest rib margin and the iliac crest to the nearest 0.1 cm (elastic tape: Seca200). Systolic and diastolic BP were measured in a sitting position after 15 minutes of rest with the cuff on the right upper arm. An Omron sphygmomanometer (Omron Corporation, Shiokoji Horikawa, Kyoto, Japan) was used for automatic measurement, and a mercury sphygmomanometer was used when the automatic measurement failed. Two readings were taken 2 minutes apart, and the average of the measurements was used in the analysis. Blood samples were drawn for FG, FTG and HDL-C, after 12 hours of fasting by venipuncture or capillary sampling. Samples were analysed within 24 hours of sampling in the Oulu University Hospital laboratory using ongoing internal/external quality Click or tap here to enter text.control (23,27)Click or tap here to enter text.. Same protocols were used in both NFBC1966 and 1986 data collection processes.

Exposure: BMI Slopes

The method to create the BMI slopes is described elsewhere (18). We considered three developmental windows: infancy (0 to 5 months), early childhood (1.5 to 3.5 years), and late

childhood (6.5 to 10 years). Throughout the article, the corresponding exposure variables are referred to as the *infancy*, *early*, and *late* BMI slopes. These slopes represent the average rate of change in BMI for each participant within a given time window. The slopes were derived from BMI trajectories estimated using linear mixed-effects models with cubic splines. Each of these were derived by calculating the change in BMI between the start and end of each window and dividing it by the duration of that window. BMI (kg/m^2) in both cohorts was calculated from repeated measurements of weight (kg) and height (m), recorded from birth to 16 years of age. Data were obtained from primary healthcare records (covering clinical visits from birth to school age, approximately 7 years) and from school health records thereafter.

Covariates

We considered sex, birthweight, maternal pre-pregnancy BMI, maternal smoking, maternal socioeconomic factors (education and occupation) as covariates because these variables are established determinants of childhood BMI (28)and MetS (29–32). Data on sex, birthweight, and maternal pre-pregnancy BMI were obtained from primary healthcare records. Information on maternal educational level and maternal smoking status during pregnancy were obtained using a specifically designed questionnaire for the NFBC data collection. Maternal smoking during pregnancy was self-reported and categorised as "Did you smoke during pregnancy (Yes or No)?" and maternal level of education at the time of pregnancy was classified as "What is the highest level of education you have completed?" Responses were categorized as follows: Primary (Elementary or equivalent), Secondary (High school or equivalent), College (Technical or vocational training), Tertiary (University degree or higher).

Statistical analyses

All analyses were performed using R software package version R.4.4.2 (33). All the codes used in this study are published in the Supplementary Material. Categorical variables were presented

as frequencies, and continuous variables were presented as mean and standard deviation (SD). The values of each BMI slope were standardized with a mean of 0 and a standard deviation of 1, and the results were presented as a 1-unit standard deviation change in BMI slope. Logistic regression analyses were performed to calculate odds ratios (OR) and their 95% confidence intervals for the association between BMI slopes and the incidence of MetS. Three models were tested in our analysis; unadjusted, adjusted for sex, and fully adjusted for covariates. We further evaluated sex and cohort specific path analyses using *lavaan* in R package (34)to estimate the linear relationships among time specific BMI slopes and the development of MetS. It captures the direct effects (part of the effect which is not mediated), indirect effects (mediated through an intermediate variable) and total effects (sum of direct and indirect effects) (35). The pathways were modelled in two ways: path a) estimating the effects of infancy BMI on MetS, mediated through late childhood BMI, path b) estimating the effects of early childhood BMI on MetS, mediated through late childhood BMI. The model accounted for data missingness through maximum likelihood estimation. The model fitness was evaluated using root mean square error of approximation (RMSEA), comparative fit index (CFI) and Trucker-Lewis Index (TLI), where RMSEA values below 0.06, CFI and TLI values above 0.90 are suggestive of good model fit (36).

RESULTS

Characteristics of participants and prevalence of metabolic syndrome

Baseline characteristics of the study participants are shown in table 1 and the prevalence of MetS are shown in Supplementary Table S1. Of the 5,647 participants included from NFBC1966, 648 (11%) were diagnosed with MetS, and 142 (3%) of the 5,583 participants from NFBC1986 had MetS. In both cohorts, MetS was more prevalent among males than females, with 398 and 94 male cases in NFBC1966 and NFBC1986, respectively, compared with 250 and 48 female cases.

Associations between BMI slopes and metabolic syndrome

In NFBC1966 at 31years, we observed a positive association with MetS and mild increase in association when adjusted for BMI slopes in early (OR: 1.3, 95% CI:1.2-1.5) and late childhood (OR: 1.6, 95% CI:1.4-1.8). In NFBC1986 at 16 years, BMI slopes at infancy showed positive association with MetS and one-fold increase in odds ratio was observed when adjusted for covariates (OR:2.3, 95% CI:2.0-2.8). Similarly, for BMI slopes at early childhood a moderate increase in odds ratio was observed when adjusted for covariates (OR:2.3, 95% CI: 2.0-2.8). BMI slope at late childhood showed the strongest association with MetS, however a slight attenuation in odds ratio was observed when adjusted (OR: 2.9, 95% CI: 2.4-3.5) (Figure 1 and Supplementary Table S4).

Path analysis estimating direct, indirect and total effects of BMI slopes on metabolic syndrome

The sex specific path diagrams shown in figures 2 and 3, as well as tables 2 and 3 present the direct, indirect and total effects of age specific childhood BMI slopes on MetS in both cohorts. All models demonstrated a good fit to the data (RMSEA = .000; CFI = 1.00; TLI = 1.00). We

observed similar pattern of pathways between sexes; however, the magnitude of effects was observed to be higher in males than females in both cohorts.

In the male analysis, no significant direct effects of BMI slope at infancy (path a) and early childhood (path b) on MetS was observed in both cohorts. However, when mediated through late childhood BMI slope, significant indirect and total effects were observed in both pathways (Figure 2 and Table 2). In NFBC1966 at 31 years, the indirect and total effect of BMI slope at infancy on MetS were negative (β: - 0.08 and β: - 0.07; p-value = 0.001 respectively), but in early childhood the indirect and total effect were observed to be positive (β: 0.13 and β: 0.16; p-value = 0.001 respectively). Conversely, in NFBC1986 at 16 years, BMI slopes at infancy and early childhood showed positive indirect (path a β:0.41 and path b β:0.50, p-value = 0.001): and total effects (path a β:0.33 and path b β:0.40, p-value =0.001) respectively. BMI slope at late childhood showed direct effect on MetS compared to earlier time points and between cohorts, stronger effect was observed at 16 years (Path a: $\beta = 0.64$, and Path b: $\beta = 0.66$, $p < 0.001$) than 31 years (both $\beta = 0.40$, $p < 0.001$) in both pathways.

In the female analysis, a significant direct effect of BMI slopes at infancy (path a) and early childhood (path b) on MetS was observed in the cohort at 31 years (Figure 3). When mediated through late childhood BMI slopes, in NFBC1966 at 31 years, the indirect and total effect of BMI slopes at infancy on MetS were negative (β: - 0.10 and β: - 0.07; p-value = 0.001 respectively), but in early childhood the indirect and total effect were observed to be positive (β: 0.04 and β: 0.18; p-value = 0.001 respectively). In NFBC1966 at 16 years, BMI slopes at infancy and early childhood showed positive indirect (path a β:0.35 and path b β:0.41, p-value = 0.001): and total effects (path a β:0.33 and path b β:0.38, p-value =0.001) respectively. (Table 3). BMI slope at late childhood showed direct effect on MetS compared to earlier time points and between cohorts, stronger effect was observed at 16 years in both the pathways (Path a: $\beta = 0.52$, and Path b: $\beta = 0.53$, $p < 0.001$).

DISCUSSION

This study examined the association between longitudinal changes in childhood BMI and the risk of MetS in adolescence and adulthood using data from NFBC1966 and NFBC1986. Our findings suggest that BMI change during early life was associated with a risk of MetS later in life, and the strongest association was observed during late childhood. This effect was more pronounced in the adolescent and in males. BMI slope in late childhood had a direct effect on later MetS risk. BMI slopes during infancy and early childhood had indirect associations with later MetS risk through the mediation of late childhood BMI slope.

We observed that the strongest impact of the rate of change in childhood BMI on later risk of developing MetS occurred in late childhood, which is often considered as a *sensitive period*. A sensitive period refers to a developmental stage during which an exposure exerts a stronger effect on an outcome than the effect of the same exposure during other periods (35). This finding is consistent with evidence from a Belarus birth cohort study on the association between infancy and childhood growth and metabolic risk biomarkers by Zhang et al.in 2020. The study observed that growth during ages 6.5 to 11.5 years, appeared to be more sensitive to adverse metabolic consequences than growth during earlier developmental periods (36). Late childhood also corresponds to the post-adiposity rebound period, when BMI growth trajectories clearly begin to change direction, and appear to be a crucial phase of rapid growth and physiological transition (37). This stage precedes puberty and is characterized by substantial changes in body composition, insulin sensitivity, and fat distribution, which may increase vulnerability to metabolic risk later. Similarly, Yang J et al. in their study evaluating the causal relationship between childhood BMI and adult cardiometabolic traits, reported that high BMI during late childhood was associated with severe cardiometabolic outcomes later in life (38). The above

stated epidemiological evidence suggests that metabolic risk becomes more firmly established during this late childhood developmental window.

The present study revealed that the rate of increase in childhood BMI has a stronger effect on the development of MetS during adolescence (NFBC1986 at 16 years). Adolescence appears to be a critical period because puberty involves major hormonal, physiological, and body composition changes that make individuals more vulnerable to metabolic problems(39,40). Consistent with our findings, Park et al. in their study on metabolic complication of obesity among children and adolescence observed that adolescents are more likely to develop unfavourable metabolic risk profiles due to changes in childhood BMI (41). Our results extend this evidence by suggesting that the increased risk of MetS associated with the rate of childhood BMI change does not show up only during adulthood as demonstrated in the NFBC1966 cohort at 31 years. However, the magnitude of this association appeared even stronger in adolescence (NFBC1986 at 16 years). The cumulative effects of prolonged childhood BMI exposure, present stronger immediate effects in adolescence and a continual metabolic imprint in adulthood (42). The observed lower effect margin of the risk during adulthood compared to adolescence may partly reflect the growing influence of adult lifestyle and behavioural factors. In adulthood, individuals may have greater capacity to influence their health through physical activity, dietary choices, healthcare engagement, and other lifestyle modifications. Thus, while childhood BMI appears to be an important early-life determinant of MetS risk, adult lifestyle factors may play a modifying role in shaping the eventual metabolic phenotype.

Similar results were observed in the path analysis. The rate of change in BMI in late childhood was the principal direct predictor of later MetS and this result was consistent in both cohorts, both sexes and across all models. This indicates that the rate of BMI change in late childhood captures metabolic risk beyond that which is accumulated during earlier developmental

periods. Our study further observed substantial indirect effects in the associations between earlier BMI slopes and the risk of developing MetS, operating through late childhood BMI slope. Suggesting that late childhood represents a critical pathway through which the metabolic consequences of earlier growth are transmitted into adolescence and adulthood. These findings support a developmental pathway in which rapid BMI gained early in life becomes metabolically relevant when it persists into later childhood rather than through an independent effect of early childhood BMI (43).

In the models predicting MetS at 16 years, the direct effect from infancy to late childhood BMI slope was positive, following the expected pattern of positive developmental tracking (45,46). However, the developmental pathway linking infancy growth to adult metabolic risk (MetS at 31 years) appeared significantly negative in both male and female analyses. The observed differences in the direction of effects in the pathways among 1966 and 1986 cohorts may be influenced by changes in environmental and behavioural processes (45) operating after adolescence, which provides opportunity for intervention or prevention. The opposing effects further suggests that complex interplay of developmental processes, including adiposity rebound(46), compensatory growth patterns and environmental factors, which are likely to modify the long-term consequences of BMI slopes during childhood.

Based on sex-specific threshold definitions of MetS, a higher prevalence of MetS was observed among males than females in our study. We recorded higher SBP, DBP, FTG and FG levels in males than females in both cohorts. However, females in both cohorts on average had larger WC. Also, the effect estimates depicting the path relationships between childhood BMI slopes and MetS were of slightly greater magnitude in males than females. However, the qualitative patterns of the developmental pathways were similar across males and females. These sex-specific differences in metabolic traits may be partly explained by hormonal mechanisms, particularly the effects of estrogen and testosterone on fat distribution, lipid metabolism, insulin

sensitivity, and vascular function, which may contribute to sex differences in the development, progression, and long-term consequences of MetS (47).

Strengths and limitations

A major strength of this study is the availability of extensive longitudinal growth data from two large prospective birth cohorts, enabling the investigation of growth trajectories across generations. The use of repeated measurements over time allowed us to model BMI growth trajectories and quantify rates of change during distinct developmental periods rather than relying on cross-sectional age-specific measures. By summarizing multiple measurements into time-specific BMI slope parameters, we achieved a more precise and interpretable characterization of growth dynamics and identified critical growth periods relevant to later metabolic health. Furthermore, path analysis with mediation decomposition enabled a detailed examination of the developmental pathways linking childhood growth patterns to subsequent metabolic outcomes.

Our study also had certain limitations. The study population was largely homogeneous and of Finnish ancestry, which may limit the generalizability of the findings to other populations. Follow-up growth measurements were available for only 71% of eligible participants, raising the possibility of selection bias and potentially attenuated associations. The use of the IDF definition of MetS may also affect comparability, as different diagnostic criteria can yield different prevalence estimates and association strengths. Self-reported fasting status before blood sampling may have led to misclassification of fasting glucose and triglyceride levels, reducing the accuracy of the estimated associations. An important limitation is that we have chosen to analyse the association with the first available measure of MetS. It is unfortunate that no measure of the MetS was available during adolescence in the NFBC1966. Though the

prevalence of MetS was three times higher among the 31-year-olds of NFBC1966 than among the 16-year-olds of NFBC1986. We cannot draw direct conclusion on comparing prevalence.

CONCLUSION

Our findings suggest that early growth rates establish a trajectory that influences future metabolic health, making BMI growth trajectories more informative than single BMI measurements for predicting future risk. The study identifies late childhood as a critical developmental window, as BMI growth during this period shows the strongest and most consistent associations with adverse metabolic outcomes. Although BMI changes in infancy and early childhood contribute to later metabolic risk, much of their impact seems to be mediated through BMI trajectories in late childhood. These findings highlight the need for prevention and intervention efforts that extend throughout childhood rather than focusing solely on infancy. The stronger associations observed in adolescents compared with adults suggest that metabolic alterations are established early in life but may become less evident with age due to behavioral and environmental influences. Overall, the results emphasize the importance of longitudinal monitoring of BMI changes across childhood and adolescence to help prevent metabolic syndrome and related health complications later in life. Future studies should investigate whether altering BMI trajectories during late childhood can effectively reduce long-term metabolic risk and improve cardiometabolic health outcomes.

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Author Contributions SS, PC and MB conceptualised and designed the study. MB wrote the first draft and analysed the data. All authors contributed to the study design, to the interpretation of the results and to the writing, critical review of the manuscript and agree to be accountable to accuracy and integrity of the study. SS, PC and MB are the guarantors of this work and, as

such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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Competing Interests: Authors declare that they have no conflict of interest.

Ethics and Data Management and Data availability

The general study protocols for the Northern Finland Birth Cohorts 1966 (NFBC1966) and 1986 (NFBC1986) were approved by the Ethical Committee of the Northern Ostrobothnia Hospital District (decisions 94/2011, issued on 12 December 2011, and 108/2017, issued on 15 January 2018). The present study did not require separate, study-specific ethical approval. However, all procedures were conducted in accordance with the European General Data Protection Regulation (GDPR) and the ethical guidelines outlined in the Northern Finland Birth Cohorts user policies at the University of Oulu.

Permission to use the specific NFBC data for this project was obtained from the NFBC Scientific Committee. NFBC data are available from the University of Oulu, Infrastructure for Population Studies. Permission to use the data can be applied for research purposes via an electronic material request portal. In the use of data, we follow the EU general data protection regulation (679/2016) and the Finnish Data Protection Act. The use of personal data is based on a cohort participant's written informed consent in their latest follow-up study, which may

cause limitations to its use. Please, contact the NFBC project center (NFBCprojectcenter(at)oulu.fi) and visit the cohort website (www.oulu.fi/nfbc) for more information.

In addition, approval to publish the results was sought and granted prior to manuscript submission. The NFBC Scientific Committee ensures that all approved research complies with applicable ethical standards and regulatory requirements.

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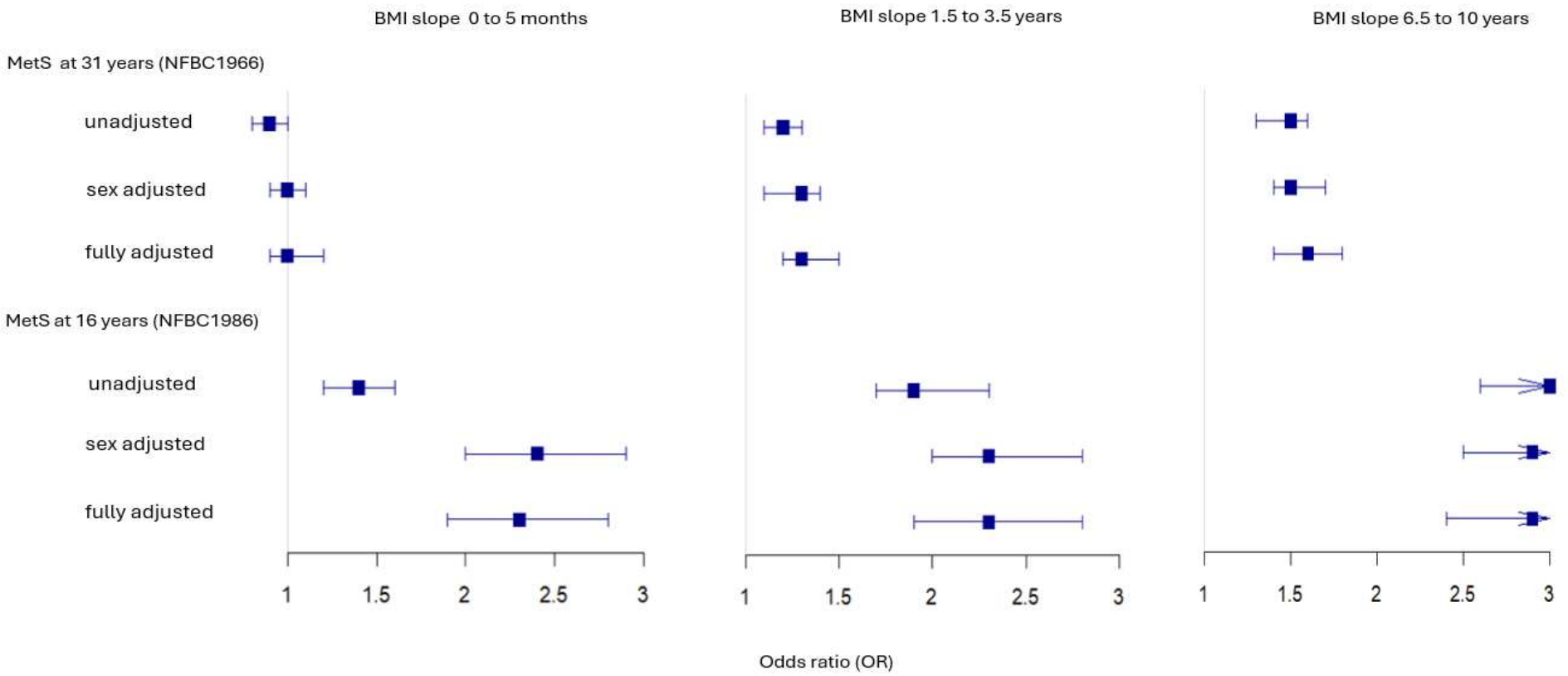


Figure 1: Association of childhood BMI slopes and metabolic syndrome among NFBC 1966 at 31 years and NFBC 1986 at 16 years

Results are presented as odds ratios (ORs) and 95% confidence intervals (CIs) for the unadjusted, sex-adjusted, and fully adjusted models, including sex, birthweight, maternal pre-pregnancy BMI, maternal education, maternal occupation, and maternal smoking during pregnancy. Results are presented as a 1-unit standard deviation change in BMI slope. Abbreviations: BMI = Body Mass Index, MetS = metabolic syndrome, NFBC = Northern Finland Birth Cohort.

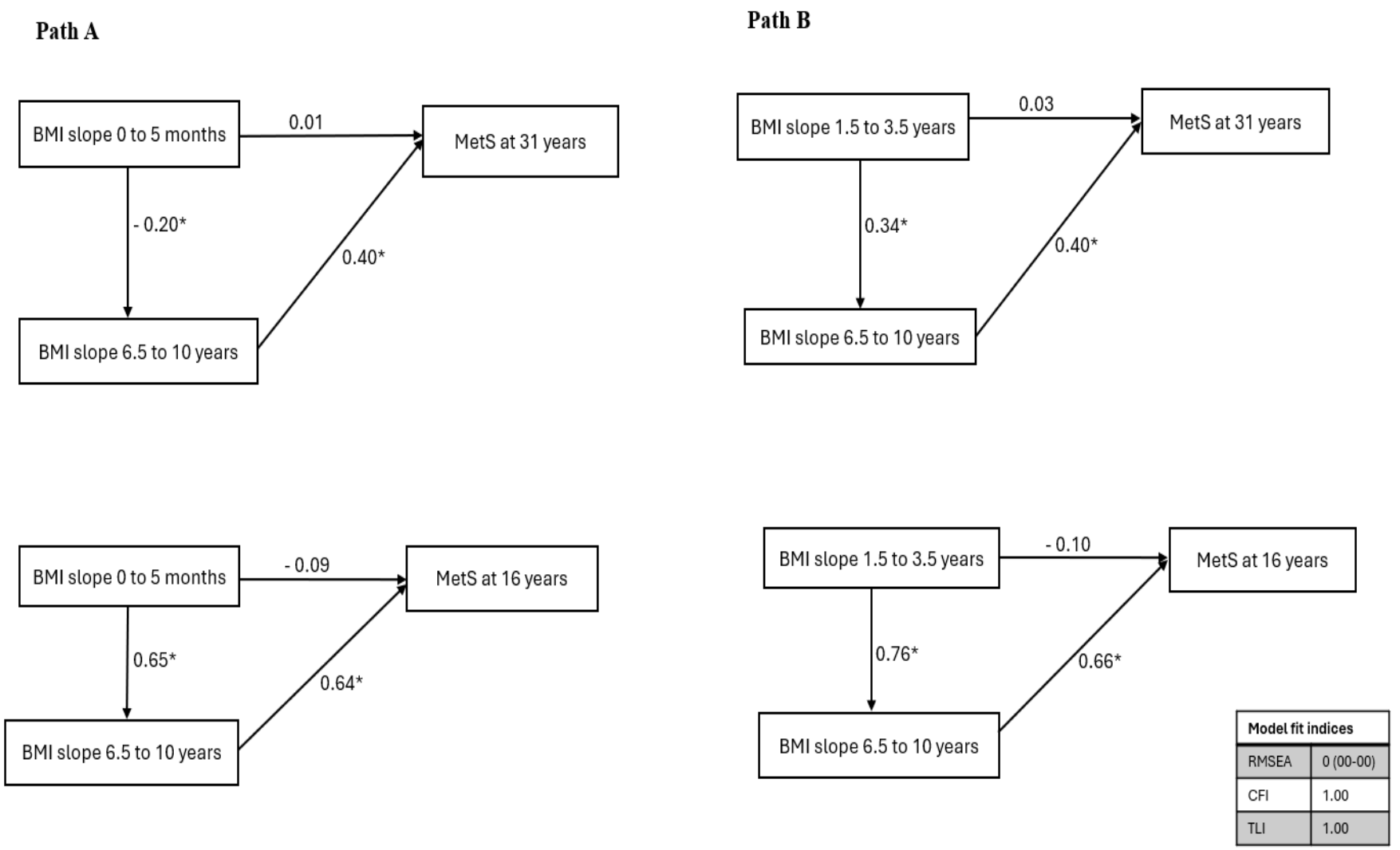


Figure 2: Direct pathways between BMI slopes and metabolic syndrome among males in NFBC1966 and NFBC1986 at 31 years and 16 years

Abbreviations: BMI = body mass index, MetS = metabolic syndrome, RMSEA = root mean square error of approximation, CFI = comparative fit index and TLI = Tucker-Lewis Index.

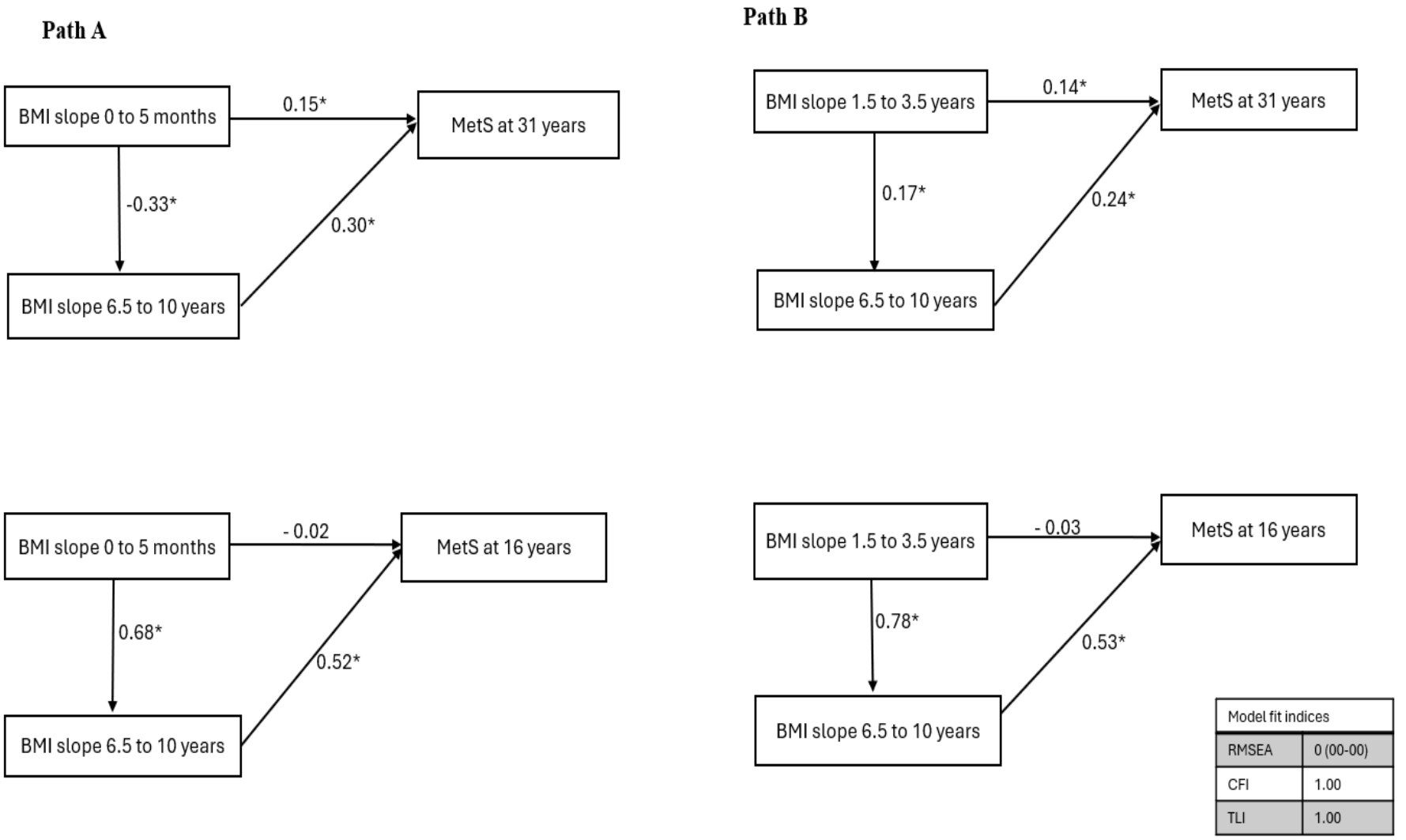


Figure 3: Direct pathways between BMI slopes and metabolic syndrome among females in NFBC1966 and NFBC1986 at 31 years and 16 years
Abbreviations: BMI = body mass index, MetS = metabolic syndrome, RMSEA = root mean square error of approximation, CFI = comparative fit index and TLI = Trucker-Lewis Index.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Table1.docx
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- Table3.docx
- Codesonanalysis.txt
- README.txt
- Supplementarymaterial.pdf