Sun, J, Xi, B, Yang, L, Zhao, M, Juonala, M, Magnussen, CG. Weight change from childhood to adulthood and cardiovascular risk factors and outcomes in adulthood: A systematic review of the literature. *Obesity Reviews*. 2020; 1– 15.

DOI https://doi.org/10.1111/obr.13138

Weight change from childhood to adulthood and cardiovascular risk factors and outcomes in adulthood: A systematic review of the literature

Short title: Life-course weight change and adult disease

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FUNDING INFORMATION

This work was supported by the National Natural Science Foundation of China (81673195)

and the Youth Team of Humanistic and Social Science of Shandong University. The funders had no role in considering the study design or in the collection, analysis, interpretation of data, writing of the report, or decision to submit the article for publication.

CONFLICT OF INTEREST

No conflict of interest was declared.

AUTHOR CONTRIBUTIONS

B.X. and C.G.M. designed the research. J.S. and L.Y. conducted the research. B.X. and J.S. performed or assisted in performing the statistical analysis of the data, J.S., B.X. and C.G.M. wrote the manuscript draft. J.S., B.X., M.Z., C.G.M., and M.J. contributed to the critical revision of the manuscript for important intellectual content. All authors have approved the final draft of the manuscript.

Summary

The magnitude of the associations between life-course change in weight status and health outcomes in adulthood has been inconsistent. This study aims to examine the associations between weight change from childhood to adulthood and cardiovascular disease (CVD) risk factors and outcomes in adulthood. PubMed, Embase, and ISI Web of Science between August 1, 1953 and July 13, 2020 were searched for potential publications and a total of 52 eligible articles were included. The systematic review strongly supported significant associations between the life-course increase in BMI and high odds of outcomes in adulthood. In the meta-analyses, normal weight in childhood but excess weight in adulthood, or excess weight in both periods was associated with increased odds of adult CVD risk factors and outcomes. However, those who had excess weight in childhood but were normal-weight in adulthood did not have increased odds of adult CVD outcomes, although were at slightly increased odds of adult type 2 diabetes. This systematic review and meta-analysis suggest that individuals who developed excess weight in adulthood or had excess weight in both periods had higher odds of developing CVD outcomes in adulthood. In contrast, the probability of these outcomes could be limited or eliminated for children with excess weight who are able to become adults with normal weight.

Keywords: childhood, adolescence, adulthood, body mass index, trajectory, diabetes, cardiovascular disease

1 INTRODUCTION

The prevalence of overweight and obesity has increased in both children and adults worldwide.[1, 2] Overweight and obesity are the main drivers of the decline in life quality and expectancy [3] due to the associated increase in a myriad of adverse health consequences including type 2 diabetes (T2D), hypertension, metabolic syndrome (MetS), subclinical markers of cardiovascular disease (CVD) (high carotid intima-median thickness [cIMT], left ventricular hypertrophy [LVH]), and CVD. In 2010, 3.8% of disability-adjusted life years, 3.9% of years of life lost, and 3.4 million deaths were attributed to overweight and obesity.[4]

Overweight and obesity in childhood or adolescence (herein termed "childhood") tracks, or persists, to adulthood,[5] and is independently associated with the onset of subclinical and clinical CVD in adulthood.[6, 7] However, it remains unclear whether a child with obesity who becomes normal weight in adulthood is able to reverse their risk of adult outcomes. Although several long-term observational studies have examined the association between weight change or weight trajectory from childhood to adulthood with CVD risk factors, and subclinical and hard CVD outcomes in adulthood,[8-79] the findings have been inconsistent. For example, while several studies show the risk of T2D in adulthood is reversible if children who were overweight or obese become normal weight in adulthood,[29, 34] others showed that the risk of T2D persisted.[54, 71]

Confirming the effect of long-term weight change from childhood on important chronic disease outcomes later in life could reinforce the need for the development of effective and tailored primordial and primary prevention strategies and interventions. To our knowledge, no systematic review and meta-analysis of the effect of weight change from childhood to adulthood on CVD risk factors, and subclinical and hard CVD outcomes in adulthood have been undertaken. Thus, we aimed to perform: 1) a meta-analysis of the associations between body mass index (BMI) status change from childhood to adulthood and adult CVD risk factors (T2D, hypertension, dyslipidemia, non-alcoholic fatty liver disease [NAFLD], MetS [a cluster of 3 or more CVD risk factors], inflammation), a subclinical marker of CVD (high cIMT), and hard CVD incidence and mortality in adulthood; 2) a meta-analysis of the associations between the

long-term burden of BMI from childhood to adulthood and adult LVH and left ventricular geometry (LV geometry); and 3) a systematic review of the effect of weight change from childhood to adulthood on adult CVD risk factors, subclinical markers of CVD (high cIMT, high pulse wave velocity [PWV]), and CVD incidence and mortality.

2 METHODS

2.1 Search strategy

In this systematic review and meta-analysis, we followed the Meta-analysis of Observational Studies in Epidemiology guidelines. PubMed, Embase, and ISI Web of Science databases were searched for literature published between August 1, 1953, and July 13, 2020. We conducted the literature search using the following keywords and strategy: ((“adiposity” or "weight" or "body mass index" or "BMI" or "overweight" or "obesity" or "trajectory" or "BMI change" or "BMI increase" or "BMI decrease" or "normal-weight" or "BMI *Z*-score" or "BMI-SDS" or "BMI percentile" or "weight change") and ("children" or "adolescents" or "childhood" or "adolescence" or "students" or "youth" or "teenagers")) and ((“non-alcohol fatty liver disease” or “non-alcoholic steatohepatitis” or “mortality" or “hypertension" or "diabetes" or "dyslipidemia" or "metabolic syndrome" or "cardiovascular disease*" or "heart disease*" or "myocardial infarction" or "stroke" or "coronary artery disease*" or "coronary heart disease*" or "left ventricular remodeling" or "left ventricular hypertrophy" or "arterial stiffness" or "pulse wave velocity" or "carotid intima-media thickness") and ("adults" or "adulthood")) and ("cohort" or "longitudinal" or "follow up" or "prospective"). Additionally, the literature in the list of references of relevant articles was manually searched. All included studies were limited to those published in English. Where articles were published using the same cohort, we included only those with the largest sample size and longer duration of follow-up. The selection process is shown in **Figure 1**.

2.2 Eligibility and exclusion criteria

Articles that met the following criteria were included: (1) assessment of the association between weight change from childhood to adulthood and adult cardiovascular risk factors (i.e., T2D, hypertension, dyslipidemia, NAFLD, MetS, and inflammation), subclinical markers of

CVD (i.e., high cIMT, LVH) or CVD incidence and mortality; (2) provision of odds ratios (ORs), hazard ratios (HRs), or relative risks (RRs) with 95% confidence intervals (CIs), or sample size used for calculating crude ORs with 95% CIs, or other estimates (e.g., β) of interest for transformation to ORs with 95% CIs; (3) age at baseline weight assessment was less than 20 years; and 4) sample size $\geq$ 500. The exclusion criteria were: 1) obvious irrelevance articles; 2) weight data only in childhood or only in adulthood; 3) BMI change started from birth; 4) weight in adulthood as a covariate rather than the exposure; 5) studies with sample size less than 500; and 5) studies without data of interest. If the studies are published repetitively, we only included the study with the largest sample size.

2.3 Study selection

Titles and abstracts found in the initial search were assessed independently by two authors (Jiahong Sun and Lili Yang). Full-text articles were evaluated by the two authors to ensure they met the eligible inclusion criteria. Any disagreements were jointly reconciled by a third author (Bo Xi).

2.4 Data extraction

The information extracted from the included studies was: (1) name of the first author; (2) publication year; (3) study name; (4) country of origin; (5) sample size; (6) sex proportion; (7) follow-up years; (8) age in childhood and adulthood; (9) definition of overweight or obesity in both childhood and adulthood; (10) definitions of adult risk factors(T2D, hypertension, dyslipidemia, MetS, NAFLD, inflammation) and subclinical markers and hard outcomes of CVD (high cIMT, LVH, LV geometry, high PWV, and CVD incidence and mortality); (11) number of cases; (12) results of interest; and (13) covariates used in adjustment.

2.5 Quality assessment

The Newcastle-Ottawa quality assessment scale (a maximum of 9 scores for each assessed study) [80] was used to assess the overall quality of the included studies. We evaluated scores of “0-3” as low, “4-6” as moderate, and “7-9” as high quality.

2.6 Statistical analysis

Meta-analysis was performed for the outcomes of T2D, hypertension, dyslipidemia, MetS, high cIMT, LVH, LV geometry, and CVD incidence and mortality in adulthood. Change in weight status from childhood to adulthood was divided into 4 groups: “normal→normal” (normal weight in both childhood and adulthood), “excess→normal” (overweight and/or obesity in childhood but normal weight in adulthood), “normal→excess” (normal weight in childhood but overweight and/or obesity in adulthood), and “excess→excess” (overweight and/or obesity in childhood and adulthood). The associations between weight status change from childhood to adulthood and T2D, hypertension, dyslipidemia, MetS, NAFLD, high cIMT, CVD in adulthood were estimated by the summary ORs with 95% CIs. Crude ORs with 95% CIs were calculated if the study provided the number of cases and sample size in each of the 4 weight change groups. It has been described in previous studies[64, 66] that the growth curves of BMI were established using a fixed-effects model and the area under the curve (AUC) was calculated using an integral calculus formula. The total AUC (AUCt) represents the long-term cumulative burden of BMI and increment of AUC (AUCi, determined by within-subject variations, i.e., adulthood BMI minus childhood BMI) represents increment burden of BMI during the follow-up period. Multivariate logistic regression analysis was usually used to examine the association of AUCt and AUCi with LVH and LV geometry[49, 64]. In this meta-analysis, we pooled the summary ORs with 95% CIs for the association. Studies with no estimates of interest or no related data for calculation were limited to the systematic review.

Between-study heterogeneity was calculated using the *Q* test and I^2 statistic. Whenever P <0.10 for the *Q* test and/or I^2 ≥50%, a random-effects model was used to calculate the summary estimates; otherwise, a fixed-effects model was used. To validate the stability of the results, sensitivity analyses that excluded one study at a time was performed. Begg’s and Egger’s tests were used to examine publication bias (with P<0.05 indicating a significant difference). Meta-regression (age in childhood or adulthood, male proportion, sample size, duration of follow-up, the definition of overweight or obesity in childhood or adulthood, definition of outcomes, adjusted covariates, race/ethnicity, and measurement of weight and height) among subgroups of different weight change patterns were performed to identify the

potential sources of between-study heterogeneity. All data analyses were conducted using Stata 11.0 (StataCorp LP, College Station, Texas, USA).

3 RESULTS

3.1 Search descriptions

The search identified 5,797 articles, from which 52 articles were potentially eligible for inclusion. Of these, 19 papers examined the association of weight change from childhood to adulthood on adult T2D (13 eligible for systematic review; 6 eligible for meta-analysis), 14 examined the association on adult hypertension (10 for systematic review; 4 for meta-analysis), 12 examined the association on adult dyslipidemia (3 for meta-analysis; 10 for systematic review [1 overlapped with an article in meta-analysis]), 4 examined the association on adult inflammation (all for systematic review only), 4 examined the association on adult MetS (2 for systematic review; 2 for meta-analysis), 2 examined the association on adult NAFLD (2 eligible for meta-analysis), 4 examined the association on adult high PWV (all systematic review only), 10 examined the association on adult high cIMT (7 for systematic review; 3 for meta-analysis), 2 examined the association on adult LVH (all for meta-analysis), 2 examined the association on adult LV geometry (all for meta-analysis only), 6 examined the association on adult CVD (2 for systematic review; 4 for meta-analysis), and 2 examined the association on adult CVD mortality (all systematic review only). The flow chart of the selection process is presented in **Figure 1**. **Tables S1-3** summarize the basic characteristics and quality of all the included articles. All articles were scored from moderate (score of 5) to high (score of 8) quality (**Table S1-3**). Related studies from our search that were not included in the systematic review and meta-analysis were due to small sample sizes, duplicate study, or data ineligible for meta-analysis (e.g., outcomes not defined by the standard criteria, lacking risk estimates or the corresponding 95% CIs, the number of weight change groups less than four) are presented in **Table S4**.

3.2 T2D

Systematic review of weight change from childhood to adulthood and adult T2D

As shown in **Table S1**, 13 articles [16, 25, 31, 37, 44, 45, 55, 56, 58, 67, 72, 76, 78] on the association between

weight change from childhood to adulthood and adult T2D were included in the systematic review. Three articles using BMI or BMI Z-score from childhood to adulthood as a continuous variable showed a significant association between BMI gain and increased fasting glucose levels [25] or T2D [16, 55] in adulthood.

Four articles assessed the association between body shape change from childhood to adulthood and T2D in adulthood.[31, 37, 58, 67] Using body shape at 10 years of age and BMI at 34 years of age as categorical variables in the Nurses' Health Study II (N=109,172), those with normal body shape in childhood but who were overweight in adulthood (OR 8.23, 95% CI 7.41-9.15) and those with increased body shape in childhood and overweight in adulthood (OR 15.10, 95% CI 13.21-17.26) had increased odds of adult T2D compared with those who were normal body shape in childhood and normal weight in adulthood; whereas children with increased body shape who were normal-weight in adulthood did not have increased odds (OR 1.02, 95% CI 0.74-1.40).[31] The other article (N=122,498) based on the Nurse's Health Study and the Health Professionals Follow-up Study found that lean-moderate increase, lean-marked increase, and heavy increase of body shape from childhood to adulthood was associated with an increase in the risk of T2D in males (HR 1.68, 2.36, and 2.80, respectively) and females (HR 3.90, 8.11, and 7.34, respectively) compared with lean stable body shape over the life-course.[67] However, no increased risk of T2D was found in the medium stable group in males (HR 1.08, 95% CI 0.89-1.30).[67]

Five articles showed significant associations between high BMI trajectory from childhood to adulthood and increased homeostatic model assessment of insulin resistance (HOMA-IR) levels [56] and T2D in adulthood.[44, 45, 72, 76] The article based on the Epidemiological Health Investigation of Teenagers in Porto (N=719) showed that HOMA-IR levels were higher among those assigned to the higher BMI trajectory groups in males (mean 2.41, 95% CI 2.06-2.82) and females (mean 2.07, 95% CI 1.84-2.32) compared with average BMI trajectory (males: mean 1.49, 95% CI 1.36-1.63; females: mean 1.67, 95% CI 1.55-1.79).[56] One article [45] based on the U.S. National Longitudinal Study of Adolescent Health (N=10,481) and one[72] in 2018 based on the Young Finns Study (N=2,631) confirmed the increased risk of adult T2D for

participants with persistent obesity, whereas the risk disappeared when children with obesity became adults with no obesity. In addition, one article from the Bogalusa Heart Study (N=1,530) reported that the total AUC of BMI from childhood (4-18 years) to adulthood (from 19 to 58.1 years of age) was higher in participants with T2D (males: 28.93 ± 5.32 kg/m^2; females: 30.62 ± 6.56 kg/m^2) compared with those without T2D in adulthood (males: 25.18 ± 4.69 kg/m^2; females: 24.72 ± 5.33 kg/m^2).[78]

Meta-analysis of weight change from childhood to adulthood and adult T2D

As shown in **Table S2**, 6 articles [29, 34, 42, 54, 71, 75] that examined the change in weight status from childhood to adulthood with adult T2D were included in the meta-analysis, with sample sizes ranging from 1,209 to 62,565. Studies were conducted in 7 countries: 1 in the U.S.,[29] 1 in the U.S., Australia, and Finland,[34] 1 in the UK,[42] 1 in China,[54] 1 in Denmark,[71] and 1 in Sweden.[75] Four articles used the U.S. centers for disease control (CDC) reference [29, 54, 71, 75] and 2 used international BMI percentile reference.[34, 42] In addition, 3 articles [29, 34, 42] used the BMI cut-off of 30 kg/m^2 to define obesity in adulthood, 1 article [54] used a BMI of 28 kg/m^2 to define obesity, 1 article[71] used a BMI of 25 kg/m^2 to define overweight (including obesity), and 1 article[75] used a BMI of 25 and 30 kg/m^2, respectively, to define overweight and obesity.

The summary results showed that compared with participants with normal weight in both childhood and adulthood (hereafter referred to as 'normal weight group'), the odds of adult T2D were 3.40 (95% CI 2.71-4.25) for those with normal weight in childhood but excess weight in adulthood (hereafter referred to as 'incident group') and 3.94 (95% CI 3.05-5.08) for those with excess weight in both childhood and adulthood (hereafter referred to as 'persistent group').The odds of adult T2D remained higher (although much lower than other two subgroups) for those who had excess weight in childhood but was normal-weight in adulthood (hereafter referred to as 'resolution group') (summary OR 1.37, 95% CI 1.10-1.70, **Table 1** and **Figure S1**); however, the odds were substantially lower than was observed for the incident and persistent groups. Sensitivity analyses that excluded one study at a time confirmed the stability of our findings. When the included studies were restricted to those where both child and adult weight were defined as overweight (including obesity) vs.

non-overweight or obesity vs. non-obesity,[29, 39, 54, 71, 75] the results were similar. When child weight was defined as overweight (including obesity) vs. non-overweight and adult weight as obesity vs. non-obesity,[34, 42] the odds were not significantly increased in the resolution group. The results were also similar in subgroup analysis that stratified by childhood and adolescence in 2 studies [42, 71] (**Table S5**).

3.3 Inflammation

Systematic review of weight change from childhood to adulthood and adult inflammation

As shown in **Table S1**, 4 articles that examined the association between weight change from childhood to adulthood with adult inflammation markers were included in the systematic review.[26, 35, 44, 55] One article (N=5,840) from the Northern Finland 1966 Birth Cohort showed that an increase in BMI from 14 years of age to 31 years of age in the top BMI tertile group at baseline explained a higher proportion of variance in C-reactive protein (CRP) levels (R^2=18%), compared to the middle (R^2=11%) and the bottom (R^2=9%) BMI tertiles at baseline.[26] An article[44] (N=13,984) based on the U.S. National Longitudinal Study of Adolescent Health from adolescence (mean of 16.9 years of age) to young adulthood (mean of 29.0 years of age) showed that BMI gain between ages 15-27 years associated with a higher risk of inflammation than stable BMI. In addition to the significant association of a 1-SD increase in BMI from childhood to adulthood with higher adult CRP in males (β 0.29, 95% CI 0.22-0.36) and females (β 0.44, 95% CI 0.35-0.52) from the New Delhi Birth Cohort (886 men and 640 women),[35] one article (N=506) from the Kaunas Cardiovascular Risk Cohort Study conducted in Lithuania showed that a 1 unit increase in BMI from childhood (12-13 years) to adulthood (48-49 years) was significantly associated with increased odds of inflammation (OR 1.23, 95% CI 1.16-1.30).[55]

3.4 Hypertension

Systematic review of weight change from childhood to adulthood and adult blood pressure and hypertension

As shown in **Table S1**, 10 articles on the association between weight gain [9, 12, 24, 25, 55] or weight trajectory [44, 56, 60, 72, 76] from childhood to adulthood and adult blood pressure levels or

hypertension were included in the systematic review. Three articles using BMI,[9, 24, 55] 1 using BMI Z-score,[25] and 1 using weight change[12] from childhood to adulthood as a continuous variable showed significant associations between BMI gain or weight gain and systolic blood pressure,[9, 12, 25]diastolic blood pressure,[9, 12] and hypertension.[24, 55] For example, data from the Young Finns Study (N=2,230)[25] showed that a 1-standard deviation (SD) increase in BMI Z-score from childhood (3-18 years) to young adulthood (24-39 years) was significantly associated with systolic blood pressure Z-score in young adulthood (standardized *β* 0.36*,* 95% CI 0.32-0.40).

Five articles using BMI trajectory groups from childhood to adulthood showed that compared with children in the persistent normal weight trajectory, those with higher BMI trajectories had higher systolic and diastolic blood pressure levels,[56] and an increased risk of hypertension in adulthood.[44, 60, 72, 76] For example, the most recent data from the Young Finns Study (N=2,631) [72] showed that compared with the stable normal weight group from childhood (6-18 years) to adulthood (34-49 years), the risk of hypertension in adulthood was 1.24 (95% CI, 1.11-1.99) for the progressively overweight group, 2.12 (95% CI, 1.15-2.89) for the progressively obese group, 2.28 (95% CI, 1.32-3.02) for the rapid overweight/obese group, and 2.98 (95% CI, 1.51-5.02) for the persistent increasing overweight/obese group; whereas those in the resolving group (i.e., from overweight/obese status to normal weight) did not have significantly increased risk (RR 0.52, 95% CI 0.13-1.32).

Meta-analysis of weight change from childhood to adulthood and adult hypertension

As shown in **Table S2**, 4 articles [29, 34, 42, 73] that examined weight status change from childhood to adulthood with adult hypertension were included in the meta-analysis, with sample sizes ranging from 2,095 to 11,447. Studies were conducted in 5 countries: 1 in the U.S.,[29]1 in the U.S., Australia, and Finland,[34] 1 in the UK,[42] and 1 in China.[73] Regarding the definition of overweight and/or obesity in childhood, 2 articles [34, 42] used international BMI percentile values, 1 used U.S. CDC BMI percentiles [29] and 1 used reference values for the Chinese population.[73] In addition, three articles [29, 34, 42] used BMI cut-offs of 30 kg/m^2 to define adult obesity, and one [73] used a BMI of 24 kg/m^2 to define adult overweight (including obesity).

The summary results showed that compared with the normal-weight group, the odds of adult hypertension was 2.69 (95% CI 2.07-3.49) for the incident group and 3.49 (95% CI 2.21-5.50) for the persistent group. However, the odds of adult hypertension were not significantly increased in the resolution group (summary OR 1.25, 95% CI 0.73-2.13) (**Table 1** and **Figure S2**). Sensitivity analyses that excluded one study at a time confirmed the stability of our findings. When the included studies [34, 42, 46] were restricted to those where adult weight status was defined as obesity vs. non-obesity, the results were similar (**Table S5**).

3.5 Dyslipidemia

Systematic review of weight change from childhood to adulthood and adult dyslipidemia

As shown in **Table S1**, 10 articles[12, 13, 21, 41, 55, 56, 65, 72, 74, 76] that examined the association between weight change from childhood to adulthood with adult lipid levels or dyslipidemia were included in the systematic review. Four articles that used BMI gain[13, 21, 55, 65] and 1 that used weight gain[12] from childhood to adulthood as a continuous variable showed significant associations with increased lipid levels including increased TC,[12, 13] TG,[13] and LDL-C,[13, 21, 65] decreased HDL-C,[13, 55, 65] and increased risk of high TG and low HDL-C.[55] For example, data from childhood (5-14 years) to young adulthood (20-29 years) in the Bogalusa Heart Study showed that BMI gain positively associated with adult levels of TC (standardized *ß* 0.2, *P*<0.001), TG (standardized *ß* 0.25, *P*<0.001), and LDL-C (standardized *ß* 0.25, *P*<0.001), and inversely associated with HDL-C (standardized *ß* -0.27, *P*<0.001).[13] One article [41] that used BMI as a categorical variable to define obese status from childhood to adulthood showed that compared with those in the never obese group, obesity onset in childhood or adulthood increased the risk of low HDL-C, high TG and high non-HDL-C in both sexes and obesity onset in adulthood increased the risk of high TC, whereas obesity only in childhood did not increase the risk of adverse lipid markers in adulthood. In addition, of 4 articles [56, 72, 74, 76] that examined BMI trajectory groups from childhood to adulthood, 3 showed that compared with the persistent normal weight trajectory group, those in higher BMI trajectory groups had increased LDL-C[56] and increased risk of dyslipidemia.[72, 76] Another article[74] (N=5,195) based on 5 cohort studies conducted in the U.S., Finland, and Australia demonstrated that total AUC

values of BMI (OR 1.61, 95% CI 1.52-1.71) and incremental AUC values (OR 1.59, 95% CI 1.50-1.69) predicted the increased risk of adult dyslipidemia.

Meta-analysis of weight change from childhood to adulthood and adult dyslipidemia

As shown in **Table S2**, 3 articles[29, 34, 74] that examined weight status change from childhood to adulthood with adult dyslipidemia were included in the meta-analysis, with sample sizes ranging from 5,195 to 10,439. Studies were conducted in 3 countries: 1 in the U.S.,[29] and 2 in the U.S., Australia and Finland.[34, 74] One article used international BMI percentile values,[34] 1 used the U.S. CDC BMI percentiles[29] and 1 used reference of the study population (U.S., Australia, and Finland)[74] to define overweight and/or obesity in childhood. Two articles[29, 34] used the BMI cut-off of 30 kg/m^2 to define obesity in adulthood and 1 article[74] used the BMI cut-off of 25 kg/m^2 to define overweight (including obesity).

Compared with participants in the normal-weight group, those in the incident (summary OR 2.42, 95% CI 1.58-3.72) and persistent (OR 2.97, 95% CI 2.35-3.75) groups had increased odds of adult dyslipidemia. In contrast, those in the resolution group did not have increased odds of adult dyslipidemia (OR 1.12, 95% CI 0.79-1.59, **Table 1** and **Figure S3**). Sensitivity analyses that excluded one study at a time confirmed these summary findings.

3.6 NAFLD

Meta-analysis of weight change from childhood to adulthood and adult NAFLD

As shown in **Table S2,** 2 articles (1 conducted in China with 1,350 participants[81] and 1 conducted in Finland with 2,020 participants[82]) that assessed weight status change from childhood to adulthood and NAFLD were included in the meta-analysis. Both of the 2 articles used International BMI percentiles to define weight status in childhood and BMI cut-offs of 25 kg/m^2 and 30 kg/m^2 to define overweight and obesity in adulthood.

The summary results showed that compared with the normal weight group, the odds of adult NAFLD was 5.19 (95% CI 2.62-10.31) for the incident group and 7.13 (95% CI 4.78-10.63) for persistent group. However, the increased risk was not statistically significant for resolution

group (summary OR 1.60, 95% CI 0.95-2.69, **Table 1** and **Figure S4**).

3.7 MetS

Systematic review of weight change from childhood to adulthood and adult MetS

As shown in **Table S1**, only 2 articles[36, 55] on the association between change in weight from childhood to adulthood and adult MetS were included in the systematic review. One article (N=5,317) in 2011 based on the Sun Project conducted in Spain showed that a 1 unit increase in weight gain (body shape) from 5 years to 20 years was associated with increased odds of MetS in males (OR 1.49, 95% CI 1.01-2.18) but not females (OR 0.97, 95% CI 0.50-1.87), suggesting sex difference in the association.[36] Another article (N=506) in 2015 based on the Kaunas Cardiovascular Risk Cohort Study conducted in Lithuania indicated that a 1 unit increase in BMI from childhood (12-13 years) to adulthood (48-49 years) was associated with increased odds of MetS (OR 1.43, 95% CI 1.33-1.53).[55]

Meta-analysis of weight change from childhood to adulthood and adult MetS

As shown in **Table S2**, 2 articles[43, 54] examining weight status change from childhood to adulthood and adult MetS were included in the meta-analysis, with sample sizes ranging from 1,209 to 1,424. Studies were conducted in 2 countries: 1 in Iran[43] and 1 in China[54]. One article used the BMI reference of Iran children and adolescents[43] and 1 used the China Obesity Task Group (WGOC) reference[54] to define overweight and/or obesity in childhood. One article [43] used the BMI cut-off of 30 kg/m^2 and 1[54] used 28 kg/m^2 to define obesity in adulthood.

Compared with those in the normal-weight group, the summary ORs of adult MetS were 10.86 (95% CI 7.54-15.63) for participants in the incident group and 10.61 (95% CI 7.51-14.99) for those in the persistent group. The odds of adult MetS was not significantly increased in the resolution group (summary OR 1.43, 95% CI 0.93-2.19, **Table 1** and **Figure S5**).

3.8 High cIMT

Systematic review of weight change from childhood to adulthood and adult high cIMT

As shown in **Table S1**, 7 articles[15, 25, 27, 50, 51, 66, 72] that examined the association between

weight change from childhood to adulthood and adult high cIMT were included in the systematic review. One article (N=2,230) from the Young Finns Study showed that a 1-SD increase in BMI Z-score from childhood (3-18 years) to young adulthood (24-39 years) was associated with 0.011 mm (95% CI 0.007-0.015) increase in adult cIMT[25]. Although one article (N=1,273) based on the 1946 British Birth Cohort found no significant association for a 1-SD increase in BMI from adolescence (15 to 20 years) with adult high cIMT (males OR 1.28, 95% CI 0.91-1.81; females OR 1.17, 95% CI 0.85-1.62),[51] a second article (N=1,273) based on the same cohort using BMI as a categorical variable showed that compared with the persistent normal weight group, those who were normal weight at birth to 11 years of age but overweight or obese in adulthood at 36-43 years of age (*β* 0.042, 95% CI 0.015, 0.069) and those with persistent overweight and obesity (*β* 0.031, 95% CI 0.007, 0.054) had higher cIMT levels in adulthood. In contrast, those who were overweight or obese in childhood but normal weight in adulthood did not have significantly higher cIMT in adulthood (*β* 0.006, 95% CI -0.019, 0.031).[50] In the Beijing Blood Pressure Cohort Study (N=1,252), total AUC values of BMI (male: OR 1.64, 95% CI 1.36-1.98; female: OR 1.51, 95% CI 1.18-1.94) and incremental AUC values (male: OR 1.56, 95% CI 1.24-1.95; female: OR 1.54, 95% CI 1.21-1.95) were associated with significantly increased odds of adult high cIMT.[66] In addition, 2 articles that examined BMI trajectories from childhood to adulthood found that compared with those assigned to the group that maintained a persistent normal weight trajectory, those assigned to higher BMI trajectory groups had higher cIMT or increased risk of high cIMT in adulthood.[15, 27] One recent article from the Young Finns Study (N=2,631) showed that participants with life-course BMI trajectories of reaching or persisting at high levels had increased risk of high cIMT in adulthood; of note, the risk reduction for cIMT in adulthood was not found for those in the resolution trajectory group (RR 3.12, 95% CI 1.51-6.03), suggesting that early obesity prevention in childhood might be more effective to attenuate the risk of atherosclerosis in adulthood.[72]

Meta-analysis of weight change from childhood to adulthood and adult high cIMT

As shown in **Table S2**, 3 articles[34, 48, 59] examining weight status change from childhood to adulthood with adult high cIMT were included in the meta-analysis, with sample sizes ranging

from 789 to 6,328. Studies were conducted in 4 countries: 1 in the U.S., Australia, and Finland[34] and 2 in China.[48, 59] One article used international BMI percentile references,[34] 1 used BMI percentiles based on a study of Chinese children,[59] and 1 used[48] the BMI reference of Taiwan (China) Department of Health to define overweight and/or obesity in childhood. One article[34] used the BMI cut-off of 30 kg/m^2 to define obesity in adulthood, one[59] used the BMI cut-off of 24 kg/m^2 to define overweight (including obesity), and 1[48] used the BMI cut-offs of 24 kg/m^2 to define overweight and 27 kg/m^2 to define obesity.

The summary results showed that there were increased odds of adult high cIMT for participants in the incident (OR 1.84, 95% CI 1.23-2.76) and persistent (OR, 2.03, 95% CI 1.30-3.17) groups compared with those in the normal-weight group. The odds of high cIMT in adulthood were not significantly increased in the resolution group (summary OR 1.36, 95% CI 0.73-2.53, **Table 1** and **Figure S6**). Sensitivity analyses that excluded one study at a time confirmed these findings. When the included studies[34, 48, 59] restricted to those where adult weight status was defined as obesity vs. non-obesity, the results were similar (**Table S5**).

3.9 LVH and LV geometry

Meta-analysis of long-term burden of BMI from childhood to adulthood and adult LVH and LV geometry

As shown in **Table S3**, 2 articles[49, 64] that examined the long-term burden of BMI from childhood to adulthood on adult LVH and LV geometry were included in the meta-analysis, with sample sizes ranging from 1,061 to 1,256. Studies were conducted in 2 countries: 1 in the U.S.[49] and 1 in China.[64]

BMI total AUC values (summary OR 2.80, 95% CI 2.03-3.85), and incremental AUC values (summary OR 2.23, 95% CI 1.90-2.64) from childhood to adulthood were significantly associated with LVH in adulthood (**Table 2** and **Figure S7**).

Only BMI incremental AUC (summary OR 1.21, 95% CI 1.06-1.37) was significantly associated with the concentric remodeling in adulthood. However, the odds of eccentric

hypertrophy and concentric hypertrophy were 2.81 (95% CI 1.67-4.72) and 2.84 (95% CI 1.95-4.13) for total AUC values, and 2.15 (95% CI 1.79-2.59) and 2.22 (95% CI 1.78-2.77) for incremental AUC values (**Table 2** and **Figure S 8-10**).

3.10 High PWV

Systematic review of weight change from childhood to adulthood and adult high PWV

As shown in **Table S1**, 4 articles[30, 59, 66, 77] on the association between weight change from childhood to adulthood and adult high PWV were included in the systematic review. In 2010, 1 article (N=1,691) from the Young Finns Study showed that adult PWV was slightly higher in the persistent high BMI group (≈8.4m/s) from childhood (3-18 years) to adulthood (30-45 years) compared with the persistent low BMI group (≈8.2m/s) and high BMI in childhood but low BMI in adulthood group (≈7.8m/s).[30] However, 2 articles based on the Beijing Blood Pressure Cohort Study (N=1,252) reported no significant association of weight status change (incident group: OR 1.00, 95% CI 0.69-1.45; persistent group: 1.02, 0.62-1.68; resolution group: 1.40, 0.49-4.02)[59] and long-term burden of BMI (increment of BMI for carotid-femoral PWV: males OR 1.17, 95% CI 0.90-1.52; females 1.18, 0.92-1.52; brachial-ankle PWV: males 0.95, 0.76-1.19; females 0.88, 0.69-1.14)[66] from childhood to adulthood with adult high PWV. In 2019, 1 article (N=1,190) based on the Bogalusa Heart Study showed a significant association of adult PWV with BMI total AUC from childhood (4-19 years of age) to adulthood (21-50 years of age) (*β* 0.081, 95% CI 0.020-0.142), but the association was not significant for incremental AUC (*β* 0.030, 95% CI -0.033, 0.093)[77].

3.11 CVD

Systematic review of weight change from childhood to adulthood and adult CVD

As shown in **Table S1**, 2 articles on the association between change in body shape from childhood to adulthood and adult CVD were included in the systematic review.[57, 67] One article in 2015 (N=50, 035) based on the Golestan Cohort Study conducted in Iran showed that compared with those who did not change body shape from 15 years of age to 30 years of age, a slight increase in body shape (≤2 scores) in males (OR 1.27, 95% CI 1.10-1.47) or major increase (>2 scores) in females (OR 1.35, 95% CI 1.11-1.62) was associated with increased

odds of heart disease.[57] The other article (N=122,498) based on the Nurse's Health Study and the Health Professionals Follow-up Study found that compared with lean stable body shape from 5 years to 55 years of age, lean-moderate increase, lean-marked increase, and a heavy increase in body shape increased the risk of CVD (HR, male: 1.16, 1.28, and 1.35, respectively; female: 1.18, 1.38, and 1.55, respectively) and coronary heart disease (HR, male: 1.25, 1.40, and 1.49, respectively; female: 1.21, 1.49, and 1.75, respectively) and stroke (HR, female:1.16, 1.29, and 1.33, respectively).[67] However, no increased risk of CVD and stroke in both sexes, and coronary heart disease in females was found in the medium stable group.[67]

Meta-analysis of weight change from childhood to adulthood and adult CVD

As shown in **Table S2**, 4 articles[40, 42, 63, 70] that examined weight status change from childhood to adulthood with adult CVD were included in the meta-analysis, with sample sizes ranging from 770 to 37,670. Studies were conducted in 3 countries: 1 in the U.S.,[40] 1 in the UK,[42] and 2 in Sweden.[63, 70] Three articles used U.S. CDC references[40, 63, 70] and 1 used international BMI percentiles[42] to define overweight (including obesity) in childhood. Two articles[40, 42] used the BMI cut-off of 30 kg/m^2 to define obesity in adulthood and 2 articles[63, 70] used the BMI cut-off of 24 kg/m^2 to define overweight (including obesity).

Compared with those with persistent normal weight, the summary odds of adult CVD was 2.77 (95% CI 1.79-4.27) for participants in the incident group and 3.04 (95% CI 1.69-5.46) for those in the persistent group. Participants in the resolution group did not have significantly increased odds of adult CVD (summary OR 1.22, 95% CI 0.92-1.62, **Table 1 and Figure S11**). Sensitivity analyses that excluded one study at a time were consistent with the main findings shown. When studies were restricted to those where adult weight status was defined as overweight (including obesity) vs. non-overweight,[63, 70] or obesity vs. non-obesity,[40, 42] the results were similar (**Table S5**).

3.12 CVD mortality

Systematic review of weight change from childhood to adulthood and adult CVD mortality

As shown in **Table S1**, 2 articles that examined the association between change in BMI status[61] and body shape status[52] from childhood to adulthood with adult CVD mortality were included in the systematic review. In 2014, 1 article (N=50,006) from the Golestan Cohort Study found that compared with those with persistent normal body shape, increased odds of adult CVD mortality was found in those with normal body shape in childhood but who had an obese body shape in adulthood (male: OR 1.57, 95% CI 1.17-2.10; female: OR 1.71, 95% CI 1.30-2.25), and persistent obese shape (female: OR 1.41, 95% CI 1.12-1.78, but not significant in male: OR 1.32, 95% CI 0.93-1.88), but the odds was not significantly increased among those who had an obese shape in childhood (at 15 years of age) and normal body shape in adulthood (at 30 years of age) (male: OR 1.29, 95% CI 0.94-1.78; female: OR 1.33, 95% CI 1.00-1.75).[52] In 2016, a population-based BMI Epidemiology Study in Sweden including 37,672 participants found that compared with participants with persistent normal weight status from childhood (8 years of age) to young adulthood (20 years of age), those who were normal weight in childhood but overweight in young adulthood (OR 2.39, 95% CI 1.86-3.09) or who were persistent overweight (OR 1.85, 95% CI 1.28-2.67) had increased odds of CVD mortality, whereas there was no increased odds of CVD mortality among those who were overweight in childhood but normal weight in young adulthood (OR 0.99, 95% CI 0.65-1.50).[61] Similar results were found at 30, 40, and 50 years of age.

3.13 Publication bias

No publication bias was found using either Begg's test or Egge's test (all P>0.05, **Table 1**).

3.14 Potential sources of between-study heterogeneity

Meta-regression analyses with consideration of sex distribution, age in childhood, age in adulthood, duration of follow-up, and sample size were performed to identify the potential sources of between-study heterogeneity (**Table S6**). Those variables listed above were not the sources of between-study heterogeneity for the majority of outcomes in adulthood. However, we observed that child age, adult age, the definition of overweight or obesity in childhood, and measurement of weight and height were four sources of between-study heterogeneity for the association between persistent excess weight and hypertension in

adulthood. After subgroup analyses by child age (<11 years and ≥11 years of age), adult age (<30 years and ≥30 years of age), definition of overweight and obesity in childhood (U.S. CDC and international BMI percentile reference), or measurement of weight and height (self-reported and measured) between-study the heterogeneity totally disappeared (**Figure S12-S15**).

4 DISCUSSION

To our knowledge, this is the first systematic review and meta-analysis to assess the relationship between weight change from childhood to adulthood with adult outcomes of CVD risk factors, markers of subclinical CVD, and CVD incidence, and mortality. Our review indicated that excess weight in adulthood alone or persistent excess weight from childhood to adulthood increases the odds of the adult outcomes we examined, with weight gain or an increasing weight trajectory also associated with these outcomes. Although we found evidence suggesting a residual or cumulative burden of BMI since childhood on markers of subclinical CVD such as LVH and LV geometry, the probability of cardiovascular risk factors in adulthood could be reduced and even removed if individuals were able to resolve their excess weight in childhood by adulthood. Our findings highlight the potential importance of prevention and intervention of excess weight from childhood and through the entire life-course to prevent the related adverse cardiovascular outcomes later in life.

Several systematic reviews and meta-analyses have shown child obesity is independently associated with T2D[83] and CVD[7] in adulthood. However, these associations may be mediated by BMI or weight status in adulthood,[84, 85] owing to the known tracking of BMI and weight status over the life-course.[86] Findings have been equivocal on the importance of mediation by adult excess weight on associations between excess weight in childhood on CVD risk factors[29, 34, 48, 54, 59]. Although the lowest odds were seen for those who maintained a normal weight from childhood to adulthood, our meta-analysis of 18 articles[29, 34, 40, 42, 43, 48, 49, 54, 59, 63, 64, 70, 71, 73-75, 81, 82] examining multiple outcomes of interest found that compared with those in the incident or persistent groups, the odds of adult CVD risk factors, subclinical and clinical CVD were substantially reduced or even removed among those able to resolve their excess weight

status in the time between childhood or adolescence and adulthood. These data are encouraging as they suggest an individual with excess weight as a child might not be destined to maintain their increased risk into adulthood, provided they are able to amend their weight status. Considered together, the available data emphasize the importance of primordial prevention of overweight and obesity to prevent or reduce T2D and cardiovascular risk factors in adulthood but also underscore the need for effective interventions targeting children with excess weight. For some outcomes (e.g., T2D), there were suggestions that a residual risk may remain for those who 'resolve' their excess childhood weight status, proposing an independent role of exposure to excess BMI in childhood on these outcomes that might reflect accumulation or a sensitive period.

Several of the studies included in our systematic review and meta-analysis only consider the change in weight status between two-time points (childhood and adulthood), which does not consider that weight change is a continuous and dynamic process, where multiple 'status changes' might occur between the observed time points. This potential for misclassification has led to an increased focus on the modeling of weight or adiposity trajectories and weight gain or rate of weight gain from childhood to adulthood.[10, 13, 14, 16, 17, 22, 25-28, 31, 36-38, 45, 46, 52, 56-60, 65-67, 72, 74, 76-78] However, it is hard to compare these studies directly as the methods used to develop the trajectories differ, and the number and visual description of any identified trajectories are diverse; hence we limited these studies to the systematic review. A total of 36 articles[9, 12, 13, 15, 16, 21, 24-27, 30, 31, 35-37, 41, 44, 45, 50-52, 55-61, 65-67, 72, 74, 76-78] on T2D, hypertension, dyslipidemia, inflammation, MetS, high cIMT, high PWV, CVD, and CVD mortality were included in the systematic review. Our systematic review shows that most included studies indicate that an increase in BMI or BMI Z-score or higher-risk BMI trajectories from childhood to adulthood associate with adult T2D, hypertension, dyslipidemia, inflammation, and high cIMT. However, the findings on weight change from childhood to adulthood and adult high PWV are inconsistent. The studies and sample sizes available for PWV were comparatively small compared with other outcomes, which suggests the need for further investigation. Although the number of studies on MetS, CVD, and CVD mortality was limited, the available data suggest greater weight change in the time between childhood and adulthood and

higher-risk weight trajectories associate with adult Mets, CVD, and CVD mortality. The views formed from our systematic review are in concert with the summary effect estimates obtained from our meta-analysis that excess weight in childhood is best avoided – underscoring the value of life-long population-based prevention and targeted intervention to reduce the burden of cardiovascular risk later in life.[87]

We know that once obesity as a chronic disease is established, it is very difficult to amend.[88] The complex environmental, physiological, behavioral, sociocultural, genetic, and epigenetic factors may pose challenges to losing weight.[89] This is reflected by many of the studies in our review where the resolution group only accounted for a small proportion of the overall sample sizes (less than 5% for most included studies, **Table S2**). This highlights the importance of keeping a healthy weight across the life course and the necessity to implement public health and population-wide measures that target factors associated with healthy weight. The factors contributing to the resolution of excess weight in the time between childhood and adulthood is critical to our understanding of how to optimize effective interventions aimed at this important period in the life-course.[90, 91] How these individuals differ from their counterparts who maintain excess weight from childhood to adulthood has not been well borne-out in the included studies and should be the focus of future enquiry. Nevertheless, our combined data from several observational studies suggest that reducing BMI in young individuals who are overweight or obese could reduce their future cardiovascular risk. Therefore, physicians should not take the pessimistic view that once childhood obesity is established, cardiovascular risk is also determined; but it could be substantially reduced if childhood obesity is successfully treated.

Several pathophysiological and metabolic mechanisms might explain the associations between weight change from childhood to adulthood and cardiovascular risk factors in adulthood. First, obesity can increase the sympathetic nervous system activity [92] which contributes to the development of cardiovascular risk factors through increasing insulin resistance[93] and decreasing endothelial function, renal and cardiac function[94]. Second, obesity promotes systematic inflammation by increasing IL-6 and TNF-α and reducing

adiponectin,[95] triggering the incidence of adverse health complications including T2D,[96] dyslipidemia,[97] MetS,[98] and other CVD risk factors.[99] Therefore, obesity in children and adults might jointly affect adult T2D and CVD through these pathways. However, weight loss exerts a function to restore the baroreflex control in cardiovascular system[100] and improve metabolic and inflammatory markers,[101] supporting our findings that substantial cardiovascular risk factors can be reversed if children with obesity become adults with normal weight.

It has been confirmed that family-based programs that limit sedentary time, increase physical activity, and promote healthy diets are effective interventions for excess weight in children.[102, 103] However, it remains a challenge to avoid weight regain.[88] In a recent systematic review and meta-analysis of 11 studies including 1,532 children and adolescents 2-18 years of age, those who received maintenance interventions after treatment for childhood overweight or obesity, such as face-to-face family therapy, having a personal physician, receiving personalized feedback, self-monitoring, and using new technologies, tended to sustain healthy weight 3 to 24 months later (weighted mean difference in BMI z-score of -0.02, 95% CI -0.09, 0.05 in the intervention group vs. 0.09, 0.00-0.18 in the control group).[104] To achieve and maintain a healthy weight, future studies that focus on barriers, facilitators, and factors that influence retention in long-term treatment interventions, response to treatment, and the potential risks associated with interventions are needed. Finally, despite the challenge, professionals should be encouraged to develop individuals' intrinsic motivation for weight loss, which is a key factor in successful intervention.[104]

Our study has important strengths. First, this is the first systematic review and meta-analysis examining the associations between weight change from childhood to adulthood with CVD risk factors. Second, the included studies were prospective cohorts with long-term follow-up and large sample sizes. Third, the included studies had moderate-to-high quality. However, there are several limitations. First, unmeasured or residual confounding might have affected the effect estimates in our meta-analysis. Second, our findings from the meta-analyses with high between-study heterogeneity should be interpreted with caution because the subgroup analyses only partly reduced the heterogeneity. Although our meta-regression analyses did

not identify the sources of between-study heterogeneity for the majority of the studied CVD risk factors and outcomes, potential factors including age in childhood or adulthood, the definition of overweight and obesity in childhood, and measurement of weight and height were found to be the possible source of heterogeneity for the association between persistent excess weight status and adult hypertension. Third, the included studies were mainly from western countries, which might affect the generalizability of our findings. Fourth, the number of articles included in the meta-analysis for each outcome in adulthood ranged from 2 to 6. However, the sample size for each outcome was relatively large, ranging from 2,633 to 128,164.

5 Conclusion

Excess weight in adulthood alone or persistent excess weight from childhood to adulthood is associated with adult CVD risk factors and outcomes; however, the risk associated with an excess weight status in childhood can be reduced or completely removed if individuals are able to obtain normal weight in adulthood. However, only a very small proportion of participants who are overweight or obese in childhood could achieve normal weight by adulthood, outlining the difficulty of resolving excess weight status in the time between childhood and adulthood and underlying the importance of maintaining a normal weight from childhood and throughout the life-course to provide the greatest reduction in risk of adult CVD risks and outcomes.

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Table 1 Meta-analysis of the effect of weight change from childhood (or adolescence) to adulthood on CVD risk factors, high cIMT, and CVD incidence and outcomes

Outcomes in adulthood	Weight change from childhood (or adolescence) to adulthood	OR	95%CI	I^2 (%)	$P_{heterogeneity}$	Statistical model	Begg's test	Egger's test
Type 2 diabetes, 6 articles (n=128,164) [29, 34, 42, 54, 71, 75]								
	Normal→ Normal	Ref.						
	Excess→ Normal	1.37	1.10-1.70	49.0	0.056	Random	0.902	0.659
	Normal→ Excess	3.40	2.71-4.25	81.2	<0.001	Random	1.000	0.961
	Excess→ Excess	3.94	3.05-5.08	81.1	<0.001	Random	0.711	0.646
Hypertension, 4 articles (n=30,309) [29, 34, 42, 73]								
	Normal→ Normal	Ref.						
	Excess→ Normal	1.25	0.73-2.13	69.4	0.020	Random	1.000	0.597
	Normal→ Excess	2.69	2.07-3.49	81.5	0.001	Random	1.000	0.833
	Excess→ Excess	3.49	2.21-5.50	89.0	<0.001	Random	1.000	0.626
Dyslipidemia, 3 articles (n=21,926) [29, 34, 74]								
	Normal→ Normal	Ref.						
	Excess→ Normal	1.12	0.79-1.59	76.8	0.013	Random	1.000	0.297
	Normal→ Excess	2.42	1.58-3.72	89.3	<0.001	Random	1.000	0.852
	Excess→ Excess	2.97	2.35-3.75	73.1	0.024	Random	0.296	0.054
NAFLD, 2 articles								

(n=3,370)[81, 82]								
	Normal→ Normal	Ref.						
	Excess→ Normal	1.60	0.95-2.69	3.0	0.356	Fixed	NA	NA
	Normal→ Excess	5.19	2.62-10.31	71.9	0.029	Random	NA	NA
	Excess→ Excess	7.13	4.78-10.63	59.6	0.084	Random	NA	NA
MetS,								
2 articles (n=2,633) [43, 54]								
	Normal→ Normal	Ref.						
	Excess→ Normal	1.43	0.93-2.19	0.0	0.532	Fixed	NA	NA
	Normal→ Excess	10.86	7.54-15.63	0.0	0.685	Fixed	NA	NA
	Excess→ Excess	10.61	7.51-14.99	0.0	0.536	Fixed	NA	NA
High cIMT,								
3 articles (n=8,342) [34, 48, 59]								
	Normal→ Normal	Ref.						
	Excess→ Normal	1.36	0.73-2.53	55.0	0.108	Random	1.000	0.180
	Normal→ Excess	1.84	1.23-2.76	73.3	0.011	Random	1.000	0.661
	Excess→ Excess	2.03	1.30-3.17	70.7	0.017	Random	1.000	0.816
CVD,								
4 articles (n=87,556) [40, 42, 63, 70]								
	Normal→ Normal	Ref.						
	Excess→ Normal	1.22	0.92-1.62	0.0	0.513	Fixed	0.089	0.090
	Normal→ Excess	2.76	1.79-4.27	70.3	0.018	Random	1.000	0.302
	Excess→ Excess	3.04	1.69-5.46	69.4	0.020	Random	0.089	0.081

Abbreviations: BMI, body mass index; cIMT, carotid intima-media thickness; CVD, cardiovascular disease; CI confidence interval; OR odds ratio; MetS, metabolic syndrome; NAFLD: non-alcoholic fatty liver disease.

NA, not available, indicating that Begg's test or Egger's test for publication bias can not be performed because of the limited number of included studies (n=2).

Table 2 Meta-analysis of the effect of long-term burden of weight change from childhood (or adolescence) to adulthood on LVH and LV geometry

Outcomes in adulthood	OR	95%CI	I^2 (%)	$P_{heterogeneity}$	Statistical model
LVH, 2 articles (n=2,317) [49, 64]					
AUCt	2.80	2.03-3.85	69.2	0.072	Random
AUCi	2.23	1.90-2.64	41.9	0.190	Fixed
LV geometry, 2 articles (n=2,317) [49, 64]					
Concentric remodeling					
AUCt	1.08	0.84-1.38	64.4	0.094	Random
AUCi	1.21	1.06-1.37	38.5	0.202	Fixed
Eccentric hypertrophy					
AUCt	2.81	1.67-4.72	77.3	0.036	Random
AUCi	2.15	1.79-2.59	0.0	0.319	Fixed
Concentric hypertrophy					
AUCt	2.84	1.95-4.13	51.5	0.151	Random
AUCi	2.22	1.78-2.77	0.0	0.361	Fixed

Abbreviations: CI confidence interval; LVH, left ventricular hypertrophy; LV geometry, left ventricular geometry; OR odds ratio. AUCt, total area under the curve (cumulative values of body mass index); AUCi, increment of area under the curve.

Figure legends

Figure 1. Flow chart of the selection process

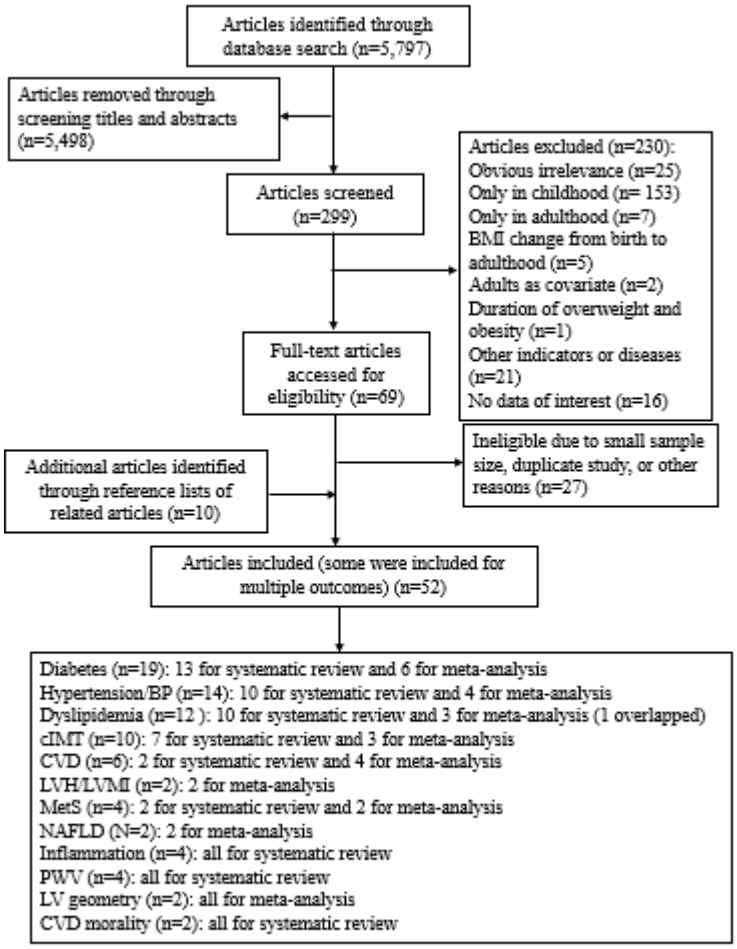

Table S1. Characteristics of studies on the effect of weight trajectory/weight change from childhood to adulthood on CVD risks factors and outcomes included in this systematic review

							Children		Adults						
	First author, Year	Study name	Country	Sample size	Sex, male (%)	Mean/median follow-up years	Age, years	Definition of overweight/obesity	Age, years	Definition of overweight/obesity	Definition of adult outcome	No. of cases / no. of samples	Main findings	Adjustment	Study quality
Type 2 diabet	Hyppönen, 2003	The 1958 British	The UK	10, 683	—	41	Birth,	—	23, 33 and	—	Self-reported	—	1 SD increase of BMI from	Sex and BMI at age 23	7

es	[1]	Birth Cohort					7, 11, and 16		41		“non–insulin-dependent diabetes that is controlled by diet or tablets”		16 to 23 years of age: Adjust for sex, birth weight, and change at other intervals OR 2.58, 95% CI (1.8-3.7) Adjust for sex and BMI at age 23 years OR 0.8, 95% CI (0.6-1.1)	years		
	Kivimaki, 2008 [2]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	2,230	45.4	21	3,6,9,12, 15, and 18	—	24-39	—	Continuous glucose level	Glucose, 2,230 HOMA index, 2,229	Glucose, 1 SD increase of age-and sex-specific BMI-Z and glucose, β 0.08 mmol/dl (0.05-0.12), P<0.0001 HOMA index, 1 SD increase of age-and sex-specific BMI-Zand HOMA index, β 0.47 mmol/dl (0.40-0.55), P<0.0001	—	6
	Yeung, 2010 [3]	The Nurses’ Health Study II	The USA	109,172	0	24	10	Recalled somatotype ≥5	34	BMI cut-off points of 25 kg/m^2	At least one of the 1997 National Diabetes Data Group diagnostic criteria: 1) one or more symptoms of weight loss, thirst, hunger, or polyuria	Normal-Normal: 503/- Overweight$_{somatotype}$-Normal: 50/- Normal-Overweight$_{BMI}$: 1,469/- Overweight$_{somatotype}$-Overweight$_{BMI}$: 441/-	Normal-Normal: Ref. Overweight$_{somatotype}$-Normal: 1.02 (0.74-1.40) Normal-Overweight$_{BMI}$: 8.23 (7.41-9.15) Overweight$_{somatotype}$-Overweight$_{BMI}$: 15.10 (13.21-17.26)	Age, race, age at birth, race, parity, smoking, parental history of diabetes, and physical activity	6

										and FPG ≥7.8 mmol /l for case befor e 1998 and FPG ≥7.0 mmol /l for case after 1998 or 2h PG ≥11.1 mmol /l; 2) no sympt om but FPG ≥7.8 mmol /l at least 2 occasi ons; 3) insuli n or hypog lycem ic medic ation use.				
Golo zar, 2011 [4]	The Golest an Cohort Study	Iran	50, 039	4 2 . 4	15	15	—	30	—	Self-r eporte d FPG ≥126 mg/dl or the use of anti-d iabeti c treat ment was used to evalu ate the specif icity and sensit ivity of self-r eporte d diabet es	Chang e in Pictogr am Male No change : 213/63 ,34 Decrea se: 101/3, 056 Increas e≤ 2: 470/8, 303 Increas e>2: 318/3, 547 Femal e No change : 366/5, 738 Decrea se: 463/71 ,114 Increas	Body shape gain PR (95% CI) **Male** No change: Ref. Decrease: 0.84 (0.65-1.08) Increase≤ 2: 1.55 (1.31-1.83) Increase>2 : 2.31 (1.90-2.81) **Female** No change: Ref. Decrease: 0.82 (0.71-0.95) Increase≤ 2: 1.26 (1.11-1.43	Age, race, educatio n, residenc e, wealth score, hyperten sion, physical activity, opium, smoking , DMFT score, and green and black tea consum ption	7

											e≤ 2: 686/8, 839 Increase>2: 836/7, 108	) Increase>2: 1.76 (1.55-2.00)		
Attard, 2013[5]	The National Longitudinal Study of Adolescent Health	The USA	Wave Ⅰ, 20, 745 Wave Ⅱ, 14, 738 Wave Ⅲ, 15, 197 Wave Ⅳ, 15, 701	52.4	12	Wave Ⅰ, mean age of 15.7 Wave Ⅱ, mean age of 16.2	1, 4 or 8 unit increase of BMI represent Small: ~20th, medium: ~60th and large: ~90th	Wave Ⅲ, mean age of 22.0 Wave Ⅳ, mean age of 28.5	1, 4 or 8 unit increase of BMI represent Small: ~20th, medium: ~60th and large: ~90th	HbA1c ≥6.5 % or reported diabetes medication use or a doctor diagnosis of diabetes	—	BMI trajectory OR (95% CI) **Men** **BMI ~ 30 kg/m ^{2} at wave Ⅳ** BMI change between waves Ⅱ-Ⅲ and Ⅲ-Ⅳ None-None: Ref. Large-Small: 2.35 (1.51-3.66) Small-Large: 1.44 (1.10-1.87) Medium-Medium: 1.84 (1.37-2.47) Small-Small: 2.33 (1.92-2.83) **BMI ~ 36 kg/m ^{2} at wave Ⅳ** None-None: Ref. Large-Large: 3.02 (1.77-5.17) Medium-Medium: 3.39 (2.53-4.55) Small-Small: 4.37 (3.07-6.22) Large-Small: 4.90 (3.10-7.74) Small-Large: 2.99 (2.20-4.08) **Women** BMI ~ 30 kg/m^2 at wave Ⅳ BMI change between waves Ⅱ-Ⅲ and Ⅲ-Ⅳ (Small:	Age at wave Ⅱ, race, region, smoking at wave Ⅱ, Ⅲ, and Ⅳ, cluster at the school level, parental history of diabetes, anti-inflammatory medication use, presence of subclinical infection markers and current infection/inflammatory disease	7

													~20th: medium: ~60th and large: ~90th) None-None: Ref. Large-Small: 1.98 (1.39-2.82) Small-Large: 1.27 (0.99-1.62) Medium-Medium: 1.58 (1.25-1.99) Small-Small: 1.70 (1.15-2.51) BMI ~ 36 kg/m ^{2} at wave Ⅳ None-None: Ref. Large-Large: 2.30 (1.52-3.49) Medium-Medium: 2.46 (1.93-3.13) Small-Small: 2.99 (2.16-4.14) Large-Small: 3.41 (2.41-4.81) Small-Large: 2.18 (1.62-2.94)		
The, 2013[6]	The National Longitudinal Study of Adolescent Health	The USA	10,481	47.7	13	12-21	—	18-33	—	Diagnosis by a health care provider or HbA1c ≥6.5 %	Never obese: 6,582 Fluctuating obesity: 2,244 Persistent obesity: 331 Incident obesity: 1,324	BMI trajectory Overall OR (95% CI) Never obese: 0.40 (0.29-0.55) Fluctuating obesity: 0.58 (0.30-1.13) Persistent obesity: 2.17 (1.55-3.05) Incident obesity: Ref. **Men** Never obese: 0.39	Obesity timing or duration, race, age, education, and parental history of diabetes.	7

												(0.25-0.63) Fluctuating obesity: 0.30 (0.11-0.80) Persistent obesity: 2.27 (1.41-3.64) Incident obesity: Ref. **Women** Never obese: 0.41 (0.27-0.62) Fluctuating obesity: 0.93 (0.40-2.19) Persistent obesity: 2.08 (1.34-3.24) Incident obesity: Ref. **White** Never obese: 0.26 (0.16-0.41) Fluctuating obesity: 0.70 (0.31-1.57) Persistent obesity: 2.37 (1.43-3.94) Incident obesity: Ref. **Blacks** Never obese: 0.65 (0.41-1.02) Fluctuating obesity: 0.28 (0.07-1.17) Persistent obesity: 1.69 (0.96-2.98) Incident obesity: Ref.		
Faghe razzi, 2015[7]	The E3N Cohort Study	France	81,110	0	≈12	8	—	20-25 and 35-40	—	—	Constantly small body shape,	Body shape trajectory HR (95% CI)	Education, smoking, physical	6

											450; Modest increase in body shape at puberty, 657; Mid-range body shape, 242; Sharp increase in body shape at puberty, 125; Upper mid-range body shape, 436; Constantly large body shape, 135	Constantly small body shape, 1.57 (1.34-1.84) Modest increase in body shape at puberty, 1.56 (1.35-1.81) Mid-range body shape, Ref. Sharp increase in body shape at puberty, 2.91 (2.35-3.62) Upper mid-range body shape, 1.44 (1.23-1.69) Constantly large body shape, 2.18 (1.76-2.69)	activity, hypercholesterolemia, hypertension, family history of diabetes, age at menarche, use of the hormone, number of children, menopausal status, use of oral contraceptives, birth weight and birth length	
Petkevicine, 2015[8]	The Kaunas Cardiovascular Risk Cohort Study	Lithuania	506	45.5	35	12-13	—	48-49	—	Diagnosed by a physician or reported glucose-lowering medication use	—	1 unit increase of BMI OR 1.15, 95% CI (1.10-1.22)	Sex, smoking, drinking, physical activity and family history of obesity	6
Arau´jo, 2015[9]	The Epidemiological Health Investigation of Teenagers in Porto	Portugal	719	45.9	8	13 and 17	—	21	—	Continuous HOMA-IR	Average BMI growth, 580 Higher BMI growth, 139	BMI trajectory Mean (95% CI) **Model 1, Female** Average BMI growth, 1.67(1.55-1.79) Higher BMI growth, 2.07 (1.84-2.32)(*P*=0.001) **Male** Average BMI growth, 1.49	Model 1, Family history of diabetes and sex Model 2, Family history of diabetes, sex and BMI at 21 years of age	6

												(1.36-1.63) Higher BMI growth, 2.41 (2.06-2.82) (*P*<0.001) **Model2, Female** Average BMI growth, 1.81 (1.68-1.94) Higher BMI growth, 1.60 (1.40-1.84) (*P*=0.147) **Male** Average BMI growth, 1.58 (1.45-1.73) Higher BMI growth, 1.75 (1.46-2.10)(*P*=0.317)			
Zheng, 2017[10]	The Nurse's Health Study and the Health Professionals Follow-up Study	The USA	122,498	30.8	Body shape, Women 34; Men, 24 Body shape trajectory-incidence of type 2 diabetes and CVD, 17	5	—	55	—	Self-reported diagnoses of type 2 diabetes	**Women** Lean stable, 246/11,049 Medium stable, 1,036/21,961 Lean-moderate increase, 1,458/16,043 Lean-marked increase, 2,138/11,983 Heavy-increase, 1,372/8,562 **Men** Lean stable, 211/5,468 Medium stable, 220/5,385	Body shape trajectory HR (95% CI) **Women** Lean stable, Ref. Medium stable, 2.14 (1.86-2.46) Lean-moderate increase, 3.90 (3.40-4.46) Lean-marked increase, 8.11 (7.10-9.27) Heavy-increase, 7.34 (6.40-8.42) **Men** Lean stable, Ref. Medium stable, 1.08 (0.89-1.30)	Height, race, regular aspirin use, pack-years of smoking, menopausal hormone therapy, physical activity, alcohol consumption, family history of diabetes and Alternate Healthy Eating Index score	7

											Lean-moderate increase, 402/5,646 Lean-marked increase, 979/10,703 Heavy-increase, 396/3,708	Lean-moderate increase, 1.68 (1.42-1.98) Lean-marked increase, 2.36 (2.04-2.74) Heavy-increase, 2.80 (2.37-3.31)		
Buscot, 2018 [11]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	2,631	45.9	31	6-18	—	34-49	—	FPG ≥ 7 mmol/l or reported the use of insulin or glucose-lowering medication, a diagnosis of type 2 diabetes by a physician and had not reported type 1 diabetes	Stable normal, 20/1,453 Resolving group, 1/43 Progressively overweight, 31/879 Progressively obese, 19/110 Rapid overweight/obese, 14/113 Persistent increasing overweight/obese, 7/33	BMI trajectory OR (95% CI) Stable normal, Ref. Resolving group, 1.93 (0.11-9.73) Progressively overweight, 2.09 (1.09-5.11) Progressively obese, 10.1 (3.13-19.72) Rapid overweight/obese, 9.33 (4.12-16.15) Persistent increasing overweight/obese, 16.5 (6.30-22.61)	Adult SES status and physical activity and family history of diabetes	7
Islam, 2019 [12]	The China Health Nutrition Survey	China	5,276	52.4	20	6-17	BMI cut-off of 85th and 95th age- and sex-specific percentile based on Chinese school-aged childr	≥18	BMI cut-offs of 24 and 28kg/m^2	FPG ≥7.0 mmol/l or self-reported a diagnosis of type 2 diabetes	Normal-Stable, -/1,180 Low normal-Normal-Stable, -/2,327 Low normal-Normal-Overweight, -/1,437 Overweight	BMI trajectory OR (95% CI) Normal-Stable, Ref. Low normal Normal-Stable, 1.7 (1.0-2.7) Low normal-Normal-Overweight, 3.2 (2.0-5.3) Overweight-Obese,	Age, sex, education, living region, smoking, alcohol consumption, unhealthy dietary pattern and physical activity at the last follow-up	8

								en for over weight and obesity				-Obese, -/332	9.1 (5.0-16.5)		
	Du, 2019 [13]	The Bogalusa Heart Study	The USA	1,530	42.9	33.1	4-18	—	19-58.1	—	FPG ≥7.0 mmol/l or HbA1c ≥6.5 % or the use of insulin or anti-diabetic medications	Men 76/1,530 Women 78/1,530	Mean± SD **Men** AUCt Non-diabetes, 25.18 kg/m ^{2} ± 4.69 Diabetes, 28.93 kg/m ^{2} ± 5.32, *P*<0.001 **Women** AUCt Non-diabetes, 24.72 kg/m ^{2} ±5.33 Diabetes, 30.62 kg/m ^{2} ± 6.56, *P*<0.001	—	5
Hypertension /Elevated BP/BP level	Bao, 1995 [14]	The Bogalusa Heart Study	The USA	1,487	43.7	≈18	5-17	—	20-31	—	Continuous BP	—	1 unit increase of BMI from childhood to adulthood SBP *B* 0.33, *P*<0.001 DBP *B* 0.39, *P*<0.0001	Baseline BP, baseline weight, height, age and sex	7
	Miura, 2001 [15]	Medical checkups performed in Ishikawa Prefecture	Japan	4,626	47.5	20	3	—	20	—	Continuous BP	—	1 percent increase of weight from 3 to 20 years was positively associated with SBP and DBP at age 20 years among men (*β* 0.30, *P*<0.0001 and *β* 0.21, *P*<0.0001) and women (*β* 0.24, *P*<0.0001 and *β* 0.13, *P*<0.0001).	—	5
	Li, 2007 [16]	The 1958 British cohort	The UK	9,297	49.8	38	7, 11, and	—	BMI 23, 33 and 45 Outco	—	SBP ≥ 140 mmHg or DBP ≥90 mmHg, or	From 16 to 23 years: 5846	1SD increase of BMI: OR 1.55, 95% CI (1.43-1.69)	Room temperature, previous BMI, and sex	7

						16		me 45		currently reported the use of anti-hypertensive medication.				
Kivimaˉki, 2008[2]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	2,230	45.4	21	3,6,9,12,15, and 18	—	24-39	—	Continuous BP	—	1 SD increase of age-and sex-specific BMI-Z and SBP-Z, β 0.36 (0.32-0.40), P<0.0001	—	6
Attard, 2013[5]	The National Longitudinal Study of Adolescent Health	The USA	Wave Ⅰ, 20,745 Wave Ⅱ, 14,738 Wave Ⅲ, 15,197 Wave Ⅳ, 15,701	52.4	12	Wave Ⅰ, mean age of 15.7 Wave Ⅱ, mean age of 16.2	1, 4 or 8 increase of BMI represent Small, ~20th, medium, ~60th and large, ~90th	Wave Ⅲ, mean age of 22.0 Wave Ⅳ, mean age of 28.5	1, 4 or 8 increase of BMI represent Small, ~20th, medium, ~60th and large, ~90th	SBP and/or DBP ≥140/90 mmHg or reported BP antihypertensive medication use or a doctor diagnosis of hypertension	—	BMI trajectory OR (95% CI) **Men BMI ~ 30 kg/m ^{2} at wave Ⅳ:** BMI change between waves Ⅱ-Ⅲ and Ⅲ-Ⅳ None-None: Ref. Large-Small: 2.13 (1.73-2.61) Small-Large: 1.76 (1.44-2.14) Medium-Medium: 1.99 (1.66: 2.38) Small-Small: 2.08 (1.81-2.39) **BMI ~ 36 kg/m ^{2} at wave Ⅳ** None-None: Ref. Large-Large: 3.38 (2.48-4.61) Medium-Medium: 3.35 (2.77-4.06) Small-Small: 3.56 (2.74-4.61) Large-Small: 3.99 (3.16-5.03) Small-Lar	Age at wave Ⅱ, race, region, smoking at wave Ⅱ, Ⅲ, and Ⅳ, cluster at the school level, parental history of diabetes, anti-inflammatory medication use, presence of subclinical infection markers and current infection /inflammatory disease	7

													ge: 3.30 (2.64-4.12) **Women** BMI ~ 30 kg/m ^{2} at wave Ⅳ BMI change waves Ⅱ-Ⅲ and Ⅲ-Ⅳ (Small: ~20th: medium: ~60th and large: ~90th) None-None: Ref. Large-Small: 2.06 (1.76-2.41) Small-Large: 1.71 (1.47-2.00) Medium-Medium: 1.93 (1.69-2.20) Small-Small: 1.79 (1.37-2.35) BMI ~ 36 kg/m ^{2} at wave Ⅳ None-None: Ref. Large-Large: 3.20 (2.54-4.02) Medium-Medium: 2.90 (2.46-3.42) Small-Small: 2.84 (2.27-3.55) Large-Small: 3.43 (2.84-4.13) Small-Large: 2.85 (2.34-3.47)			
Petkevicine, 2015[8]	The Kaunas Cardiovascular Risk Cohort Study	Lithuania	506	45.5	35	12-13	—	48-49	—	BP ≥140/90 mmHg or anti-hypertensive medication use	—	1 unit increase of BMI OR 1.26, 95% CI (1.20-1.34)	Sex, smoking, drinking, physical activity and family history of obesity	6
Arau´jo,	The Epide	Portugal	719	45	8	13 an	—	21	—	Continuous	Average	BMI trajectory	Model 1,	6

2015[9]	miological Health Investigation of Teenagers in Porto			.9		d 17				BP	BMI growth, 580 Higher BMI growth, 139	Mean (95% CI) **Model 1, SBP,** Average BMI growth, 108.3 mmHg (107.5-109.0) Higher BMI growth, 111.6 mmHg (110.0-113.1) (*P*<0.001) **DBP** Average BMI growth, 68.4 mmHg (67.8-69.0) Higher BMI growth, 71.9 mmHg (70.6-73.1) (*P*<0.001) **Model2, SBP** Average BMI growth, 109.1 mmHg (108.3-109.9) Higher BMI growth, 107.7 mmHg (105.8-109.6) (*P*=0.186) **DBP** Average BMI growth, 69.0 mmHg (68.3-69.6) Higher BMI growth, 69.5 mmHg (68.0-71.0) (*P*=0.543)	Family history of hypertension and sex Model 2, Family history of hypertension, sex and BMI at 21 years of age	
Wang, 2016[17]	A cohort study performed in Hangzhou, Shanxi Provin	China	4,211	52.9	26	6-17	BMI cut-off points of 85th and 95th based	32-43	BMI cut-off points of 24 and 28	Hypertension, SBP and/or DBP ≥ 140/90	—	BMI trajectory **Pre-HSBP** Persistent normal weight, Ref. Persistent overweigh	Time-dependent covariate	7

	ce, China						on WGOC.		kg/m^2	mmHg Pre-hypertension, SBP 130–139 mmHg or DBP 85–89 mmHg)		t, HR 1.108 95% CI (0.953, 1.287) Persistent obesity, 1.103 (0.887-1.371) **HSBP** Persistent normal weight, Ref. Persistent overweight, HR 1.811 95% CI (1.337, 2.454) Persistent obesity, 2.96 (2.077-4.22) **Pre-HDBP** Persistent normal weight, Ref. Persistent overweight, HR 1.165 95% CI (0.916, 1.482) Persistent obesity, 1.194 (1.014-1.406) **HDBP** Persistent normal weight, Ref. Persistent overweight, HR 2.023 95% CI (1.575, 2.597) Persistent obesity, 2.888 (2.128-3.92)			
Buscot, 2018[11]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	2,631	45.9	31	6-18	—	34-49	—	SBP and/or DBP ≥140/90 mmHg or reported BP antihypertensive medication use	Stable normal, 280/1,453 Resolving group, 7/43 Progressively overweight, 236/879	BMI trajectory OR (95% CI) Stable normal, Ref. Resolving group, 0.52 (0.13-1.32) Progressively overweigh	Adult SES and physical activity and family history of hypertension	7

												Progressively obese, 36/110 Rapid overweight/obese, 41/113 Persistent increasing overweight/obese, 13/33	t, 1.24 (1.11-1.99) Progressively obese, 2.12 (1.15-2.89) Rapid overweight/obese, 2.28 (1.32-3.02) Persistent increasing overweight/obese, 2.98 (1.51-5.02)		
	Islam, 2019 [12]	The China Health Nutrition Survey	China	5,276	47.6	20	6-17	BMI cut-off of 85th and 95th age- and sex-specific percentile based on Chinese school-age children for overweight and obesity	≥18	BMI cut-offs of 24 and 28 kg/m^2	SBP and/or DBP ≥140/90 mmHg or reported current use of antihypertensive medication	Normal-Stable, -/1,180 Low normal-Normal-Stable, -/2,327 Low normal-Normal-Overweight, -/1,437 Overweight-Obese, -/332	BMI trajectory OR (95% CI) Normal-Stable, Ref. Low normal-Normal-Stable, 1.6 (1.3-2.1) Low Normal-Normal-Overweight, 3.3 (2.5-4.3) Overweight-Obese, 6.6 (4.5-9.8)	Age, sex, education, living region, smoking, alcohol consumption, unhealthy dietary pattern and physical activity at the last follow-up	8
Dyslipidemia	Miura, 2001 [15]	Medical checkups performed in Ishikawa Prefecture	Japan	4,626	47.5	20	Birth, 3 months, 1, and 3	—	20	—	Continuous TC	—	1 percent increase of weight from 3 to 20 years was positively associated with TC at age 20 years among men (β 0.29, P<0.0001) and women (β 0.11, P<0.0001).	—	5
	Nicklas, 2002 [18]	The Bogalusa Heart Study	The USA	1,169	50	15	5-14	—	20-29	—	Continuous TC, TG, HDL-C, and LDL-	—	The change of BMI was associated with higher TC (Standardized β 0.20,	—	5

										C		*P*<0.001), higher TG (Standardized *β* 0.25, *P*<0.001), higher LDL-C (Standardized *β* 0.25, *P*<0.001), and lower HDL-C (Standardized *β* -0.20, *P*<0.001).		
Srinivasan, 2006 [19]	The Bogalusa Heart Study	The USA	1,163	55.4	27	5-14	—	≥27	—	Continuous Non-HDL-C and LDL-C	—	1SD increase of BMI predicted higher Non-HDL-C level Standardized *β* 0.20, *P*<0.001 1SD increase of BMI predicted higher LDL-C level Standardized *β* 0.22, *P*<0.001	—	5
Skidmore, 2007 [20]	The Medical Research Council National Survey of Health and Development	The UK	2,311	≈49	51	2,4,7, 11, and 15	—	BMI 36 Outcome 53	—	Continuous LDL-C and HDL-C	—	1 SD increase of BMI from 15 years to 36 years of age **Overall** *B* 0.097, 95% CI 0.037-0.156, *P* =0.001 **LDL-C** *B* 0.067, 95% CI 0.009-0.124, *P*=0.02 **HDL-C** Men, *β*-0.094, 95% CI -0.127, -0.060, *P* <0.001 Women, *β*-0.118, 95% CI -0.156, -0.080, *P* <0.001	Sex	7
Pinto Pereira, 2013 [21]	The 1958 British Birth Cohort	The UK	7,824	50.2	45	Birth, 7, 11, and 16	International BMI cut-offs	33 and 45	BMI cut-off points of 30 kg/m^2	Continuous TC, HDL-C, Non-HDL-C, and TG	**Men** Never: 2,880 Childhood only: 27 Childhood-onset: 59	Mean difference % (95% CI) in lipid levels of obesity onset **Men** **TC** Never: Ref.	Social class at birth and adult, education, smoking, drinking, hyperten	7

Young adulthood onset: 327 Mid-adulthood onset: 634 **Women** Never: 2,918 Childhood only: 30 Childhood-onset: 89 Young adulthood onset: 335 Mid-adulthood onset: 525	Childhood only: -2.81 (-9.92, 4.30) Childhood onset: 1.64 (-3.19, 6.46) Young adulthood onset: 1.60 (-0.70, 3.91) Mid-adulthood onset: 3.69 (2.04, 5.34) **HDL-C** Never: Ref. Childhood only: 2.75 (-5.52, 11.03) Childhood onset: -13.21 (-18.79, -7.63) Young adulthood onset: -12.09 (-14.65, -9.53) Mid-adulthood onset: -12.10 (-13.96, -10.23) **Non-HDL-C** Never: Ref. Childhood only: -2.33 (-11.62, 6.96) Childhood onset: 11.46 (8.68, 14.23) Young adulthood onset: 12.20 (10.01, 14.39) Mid-adulthood onset: 13.59 (11.28, 15.90) **TG** Never, Ref. Childhood only: -5.94 (-28.36, 16.48) Childhood onset: 30.14	sion, and women menopausal status

(15.17, 45.10)
Young adulthood onset: 26.53 (19.85, 33.21)
Mid-adulthood onset: 30.23 (25.27, 35.19)
Women
TC
Never: Ref.
Childhood only: -3.31 (-9.68, 3.07)
Childhood: 0.47 (-3.29, 4.23)
Young adulthood: 1.09 (-0.99, 3.18)
Mid-adulthood: 4.19 (2.55, 5.83)
HDL-C
Never: Ref.
Childhood only: 1.69 (-6.46, 9.85)
Childhood: -16.85 (-21.47, -12.23)
Young adulthood: -16.81 (-19.34, -14.29)
Mid-adulthood: -14.08 (-16.11, -12.06)
Non-HDL-C
Never: Ref.
Childhood only: -3.94 (-9.76, 1.89)
Childhood: 11.02 (8.62, 13.42)
Young adulthood: 13.26 (11.02, 15.49)
Mid-adulthood: 11.14 (8.95,

													13.33) **TG** Never: Ref. Childhood only: -20.56 (-39.16, -1.96) Childhood: 20.17 (9.32, 31.03) Young adulthood: 30.92 (24.83, 37.01) Mid-adulthood: 29.58 (24.82, 34.34)		
Petkevicine, 2015[8]	The Kaunas Cardiovascular Risk Cohort Study	Lithuania	506	45.5	35	12-13	—	48-49	—	TG ≥ 1.7 mmol/l HDL-C <1.03 mmol/l in men and <1.29 mmol/l for women	—	1 unit increase of BMI High TG OR 1.22, 95% CI (1.15-1.29) Low HDL-C OR 1.20, 95% CI (1.13-1.28)	Sex, smoking, drinking, physical activity and family history of obesity	6
Araújo, 2015[9]	The Epidemiological Health Investigation of Teenagers in Porto	Portugal	719	45.9	8	13 and 17	—	21	—	Continuous LDL-C, HDL-C, and TG	Average BMI growth, 580 Higher BMI growth, 139	BMI trajectory Mean (95% CI) **Model 1, LDL-C** Average BMI growth, 102.4 mg/dl (100.1-104.7) Higher BMI growth, 103.0 mg/dl (98.4-107.6)(*P*=0.821) **HDL-C** Average BMI growth, 57.0 mg/dl (56.0-58.0) Higher BMI growth, 53.3 mg/dl (51.4-55.3) (*P*=0.001) **TG** **Female** Average	Model 1, Family history of dyslipidemia and sex Model 2, Family history of dyslipidemia, sex and BMI at 21 years of age	6

BMI growth, 84.5 mg/dl (80.5-88.8)
Higher BMI growth, 83.3 mg/dl (76.0-91.4)
(*P*=0.788)
Male
Average BMI growth, 70.2 mg/dl (66.8-73.8)
Higher BMI growth, 89.0 mg/dl (80.1-99.1)
(*P*<0.001)
Model2, LDL-C
Average BMI growth, 104.3 mg/dl (101.9-106.7)
Higher BMI growth, 95.0 mg/dl (89.3-101.7)(*P*=0.005)
HDL-C
Average BMI growth, 56.7 mg/dl (55.7-57.7)
Higher BMI growth, 54.6 mg/dl (52.1-58.0)
(*P*=0.130)
TG
Female
Average BMI growth, 85.1 mg/dl (80.7-89.7)
Higher BMI growth, 81.4 mg/dl (72.5-91.3)
(*P*=0.512)
Male
Average BMI growth, 72.6 mg/dl (68.9-76.4

												) Higher BMI growth, 77.2 mg/dl (68.0-87.6) (*P*=0.402)			
Busc ot, 2018 [11]	The Longit udinal Cardio vascul ar Risk in Young Finns Study	Finla nd	2,6 31	4 5 . 9	31	6-1 8	—	34- 49	—	LDL-C ≥160 mg/dl HDL <40 mg/dl TG ≥200 mg/dl	High-r isk LDL-C Stable normal , 137/1, 453 Resolv ing group, 5/43 Progre ssively overw eight, 145/87 9 Progre ssively obese, 20/110 Rapid overw eight/ obese, 21/113 Persist ent increas ing overw eight/ obese, 7/33 High-r isk HDL-C Stable normal , 166/1, 453 Resolv ing group, 6/43 Progre ssively overw eight, 231/87 9 Progre ssivel- y obese, 46/110 Rapid overw eight/ obese, 45/113 Persist ent increas ing	BMI trajectory RR (95%CI) **High-risk LDL-C** Stable normal, Ref. Resolving group, 1.01 (0.10-1.08) Progressiv ely overweigh t, 1.12 (1.06-1.49) Progressiv ely obese, 1.30 (1.17-2.57) Rapid overweigh t/obese, 1.20 (1.11-2.30) Persistent increasing overweigh t/obese, 1.51 (1.05-2.94) **High-risk HDL-C** Stable normal, Ref. Resolving group, 1.03 (0.22-1.11) Progressiv ely overweigh t, 1.24 (1.12-1.82) Progressiv ely obese, 1.35 (1.01-12.1) Rapid overweigh t/obese, 1.41 (1.02-2.22) Persistent increasing overweigh t/obese,	Adult SES status and physical activity and family history of dyslipid emia	7

											overweight/obese, 13/33 High-risk TG Stable normal, 70/1,453 Resolving group, 2/43 Progressively overweight, 156/879 Progressively-obese, 30/110 Rapid overweight/obese, 29/113 Persistent increasing overweight/obese, 6/33	1.37 (1.26-2.60) **High-risk TG** Stable normal, Ref. Resolving group, 0.31 (0.06-2.12) Progressively overweight, 2.89 (2.02-4.08) Progressively obese, 5.11 (3.11-9.58) Rapid overweight/obese, 4.24 (3.02-7.34) Persistent increasing overweight/obese, 3.21 (1.22-9.60)		
Islam, 2019 [12]	The China Health Nutrition Survey	China	5,276	52.4	20	6-17	BMI cut-off of 85th and 95th age- and sex-specific percentile based on Chinese school-age children.for overweight and obesity	≥18	BMI cut-offs of 24 and 28kg/m^2	TC ≥6.22 mmol/L, or HDL-C <1.04 mmol/L, or LDL-C ≥4.14 mmol/L, or TG ≥2.26 mmol/L	Normal-Stable, -/1,180 Low normal-Normal-Stable, -/2,327 Low normal-Normal-Overweight, -/1,437 Overweight-Obese, -/332	BMI trajectory OR (95%CI) Normal-Stable, Ref. Low normal Normal-Stable, 2.6 (2.1-3.4) Low normal-Normal-Overweight, 4.0 (3.0-5.3) Overweight-Obese, 5.9 (3.9-8.8)	Age, sex, education, living region, smoking, alcohol consumption, unhealthy dietary pattern and physical activity at the last follow-up	8
Yan, 2019 [22]	The Bogalusa Heart Study, the Muscatine Study, the National	The USA, Finland, and Australia	5,195	39.8	27.3 ± 7.0	3-19	—	20-52	—	Dyslipidemia was defined as any of the following High LDL-	Dyslipidemia 1,916/5,195 High LDL-C 776/5,195 Low HDL-	OR (95% CI) **Dyslipidemia** Childhood 1.22 (1.15-1.29) Adulthood 1.85 (1.74-1.97	AUCt Mean age prior to regression analyses AUCi Mean age and BMI in	8

al Heart, Lung, and Blood Institute Growth and Heart Study, the Prevention of High Blood Pressure in Children Study in the US, and the Longitudinal Cardiovascular Risk in Young Finns Study in Finland.			C ≥160 mg/Dl; Low HDL-C ≤40 mg/dL; and High TG ≥200 mg/dL or taking lipid-lowering medication	C 1,355/5,195 High TG 719/5,195	) From childhood to adulthood AUCt 1.61 (1.52-1.71) AUCi 1.59 (1.50-1.69) **High LDL-C** Childhood 1.10 (102-1.19) Adulthood 1.42 (1.32-1.53) From childhood to adulthood AUCt 1.30 (1.21-1.40) AUCi 1.30 (1.21-1.40) **low HDL-C** Childhood 1.29 (1.22-1.37) Adulthood 1.82 (1.71-1.95) From childhood to adulthood AUCt 1.68 (1.57-1.79) AUCi 1.62 (1.52-1.73) **High TG** Childhood 1.13 (1.05-1.22) Adulthood 1.65 (1.53-1.77) From childhood to adulthood AUCt 1.48 (1.38-1.59) AUCi 1.50 (1.40-1.62)	childhood prior to regression analyses

Inflammation	Tzoulaki, 2008 [23]	The Northern Finland 1966 Birth Cohort study	Finland	5,840	48.1	17	14	—	31	—	Continuous CRP	—	BMI trajectory The BMI increase from 14 to 31 years of age in the top BMI tertile group at 14 years of age explained a higher variation (R^2=18%) in CRP levels, compared to the middle (R^2=11%) and bottom (R^2=9%) BMI tertiles.	Sex, total/HDL-C ratio, SBP, SES, and smoking	6
	Lakshmy, 2011 [24]	The New Delhi Birth Cohort	India	1,526	58.1	32	Birth, 2 and 11	—	26-32	—	Continuous CRP	—	1 SD increase of BMI from 11 years to adult was associated with higher hs CRP in both males (β 0.29, 95% CI 0.22-0.36, P<0.001) and female (β 0.44, 95% CI 0.35-0.52, P<0.001)	Age, alcohol consumption, smoking, SES in childhood and adulthood, education level, family history of angina, hypertension, myocardial infarction, diabetes, and stroke.	7
	Attard, 2013 [5]	The National Longitudinal Study of Adolescent Health	The USA	Wave Ⅰ, 20,745 Wave Ⅱ, 14,738 Wave Ⅲ, 15,197 Wave Ⅳ, 15,701	52.4	12	Wave Ⅰ, mean age of 15.7 Wave Ⅱ mean age of 16.2	1, 4, or 8 unit increase of BMI represent Small: ~20th, medium: ~60th and large: ~90th	Wave Ⅲ mean age of 22.0 Wave Ⅳ mean age of 28.5	1, 4, or 8 unit increase of BMI represent Small: ~20th, medium: ~60th and large: ~90th	Inflammation, CRP ≥ 3 mg/l [5]	—	BMI trajectory OR (95% CI) **Men BMI ~ 30 kg/m ^{2} at wave Ⅳ** BMI change between waves ⅡⅢ and ⅢⅣ None-None: Ref. Large-Small: 2.58 (1.88-3.56) Small-Large: 2.94 (2.24-3.87)	Age at wave Ⅱ, race, region, smoking at wave ⅡⅢ and Ⅳ, cluster at the school level, parental history of diabetes, anti-inflammatory medication use, presence of subclinical	7

	Medium-Medium: 2.98 (2.30-3.87) Small-Small: 2.60 (2.20-3.07)	infection markers and current infection /inflammatory disease
BMI ~ 36 kg/m ^{2} at wave Ⅳ	None-None: Ref. Large-Large: 6.65 (4.21-10.51) Medium-Medium: 5.93 (4.62-7.62) Small-Small: 5.24 (3.84-7.15) Large-Small: 5.87 (4.25-8.11) Small-Large: 6.68 (4.99-8.95)	
Women BMI ~ 30 kg/m ^{2} at wave Ⅳ	BMI change between waves Ⅱ-Ⅲ and Ⅲ-Ⅳ (Small: ~20th: medium: ~60th and large: ~90th) None-None: Ref. Large-Small: 3.09 (2.41-3.95) Small-Large: 4.29 (3.57-5.17) Medium-Medium: 4.07 (3.41-4.86) Small-Small: 3.14 (2.22-4.44)	

														BMI ~ 36 kg/m ^{2} at wave Ⅳ None-None: Ref. Large-Large: 11.28 (8.24-15.43) Medium-Medium: 6.55 (5.40-7.96) Small-Small: 3.80 (3.00-4.81) Large-Small: 5.81 (4.42-7.63) Small-Large: 8.08 (6.48-10.07)		
	Petkevicine, 2015[8]	The Kaunas Cardiovascular Risk Cohort Study	Lithuania	506	45.5	35	12-13	—	48-49	—	CRP ≥ 3 mg/l	—	1 unit increase of BMI OR 1.23, 95% CI (1.16-1.30)	Sex, smoking, drinking, physical activity and family history of obesity	6
MetS	Pimenta, 2011[25]	The Sun (Seguimiento Universidad de Navarra) Project	Spain	5,317	35.1	6.1	5	—	20	—	Based on the International Diabetes Federation criteria	—	1 unit increase of weight gain (body shape) from 5 to 20 years OR, 95% CI Female 0.97 (0.50-1.87), *P*=0.921 Male 1.49 (1.01-2.18), *P*=0.043	Age and adult BMI	6
	Petkevicine, 2015[8]	The Kaunas Cardiovascular Risk Cohort Study	Lithuania	506	45.5	35	12-13	—	48-49	—	WC ≥94 cm for males and ≥80 cm for female and at least 2 of the following criteria, 1) SBP and/o	—	1 unit increase of BMI OR 1.43, 95% CI (1.33-1.53)	Sex, smoking, drinking, physical activity and family history of obesity	6

												r DBP ≥ 130/85 mmHg or treatment of diagnosed hypertension; 2) HDL-C <1.03 in men and <1.29 mmol/l for women; 3)TG ≥ 1.7 mmol/l; 4) FPG ≥5.6 mmol/l or diagnosed type 2 diabetes				
High-risk cIMT	Oren, 2003 [26]	The Atherosclerosis Risk in Young Adults Study	Netherlands	750	46.9	≈15	12-16	—	27-30	—	Continuous cIMT	Low-constant over time: 174 Decreased BMI: 199 Increased BMI: 205 High-constant BMI: 172	BMI trajectory Low-constant over time: Ref. Decreased BMI: *β*=5.7 (-2.0, 13.4), *P*=0.22 Increased BMI: *β*=11.8 (4.0, 19.6), *P*=0.01 High-constant BMI: *β*=20.4 (11.9, 28.9), *P*<0.01	Gender, reader, adolescent age, adolescent BP, lumen diameter, puberty stage, adult LDL-C, BP, and relative to the low-constant category	7
	Freedman, 2008 [27]	The Bogalusa Heart Study	The USA	1,142	43.1	≈25	5-20	—	20-40	—	High IMT, at 90th percentile Low IMT, at 10th percentile	—	BMI trajectory High IMT in adulthood was associated with high BMI levels from 5 to 30 in white men, white women,	Age, sex, and race	8

												and black women (P<0.001)		
Kivimaʺki, 2008 [2]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	2,230	45.4	21	3,6,9,12,15, and 18	—	24-39	—	Continuous IMT	—	1 SD increase of age-and sex-specific BMI-Z and IMT, β 0.011mm (0.007-0.015), P<0.0001	—	6
Charakida, 2014 [28]	The National Survey of Health and Development Study (The 1946 British Birth Cohort Study)	The UK	1,273	47.3	>60	Birth, 2,4,6,7, and 11	Age- and sex-specific BMI cut-offs according to the IOTF Overweight or obesity at least one of the age points	BMI 36, 43, and 53 Outcome, 60-64	BMI cut-off points of 25 and 30 kg/m^2	Continuous cIMT	Normal-Normal: -/123 Overweight/obesity-Normal: -/302 Normal-Overweight/obesity: , -/224 Overweight/obesity - Overweight/obesity:, -/621	Normal-Normal: Ref. Overweight/obesity -Normal, β 0.006, 95% CI (-0.019, 0.031), P_{model1}=0.62and P_{model2}=0.37 Normal-Overweight/obesity: β 0.042, 95% CI (0.015, 0.069), P_{model1}=0.002 and P_{model2}=0.004 Overweight/obesity - Overweight/obesity: β 0.031, 95% CI (0.007, 0.054), P_{model1}=0.051 and P_{model2}=0.011	Model 1:sex Model 2: sex, LDL-C, heart rate, smoking, SBP at 60-64 years, and SES at 53 years	6
Johnson, 2014 [29]	The 1946 British Birth Cohort	The UK	1,273	47.4	>60	2, 4, 7, 11, and 15	—	BMI 20 Outcome, 60-65	—	Upper quartile and lower 3 quartiles of IMT	Male Upper quartile and lower 3 quartiles of IMT: 92/305 Female Upper quartile and lower 3 quartiles of IMT: 113/348	**Male,** 1SD increase of BMI (1.7 kg/m ^{2}) from 15 to 20 years: OR 1.28, 95% CI (0.91-1.81) **Female** 1SD increase of BMI (1.9 kg/m ^{2}) from 15 to 20 years: OR 1.17, 95% CI (0.85-1.62)	—	5
Yan, 2017b [30]	The Beijing Blood Pressure	China	1,252	55.3	22.9 ± 0.6	6-18	—	27-42	. —	Age- and sex-specific 75th	—	OR (95% CI) **Male** Childhood 1.41	Adult risk factors (family history	6

		Cohort Study									percentile of IMT		(1.20-1.66) Adulthood 1.77 (1.37-2.28) From childhood to adulthood AUCt 1.64 (1.36-1.98) AUCi 1.56 (1.24-1.95) Female Childhood 1.24 (1.02-1.52) Adulthood 1.68 (1.28-2.21) From childhood to adulthood AUCt 1.51(1.18-1.94) AUCi 1.54 (1.21-1.95)	of stroke and coronary heart disease, physical activity, drinking, smoking, LDL-C, HDL-C, and TG)	
	Buscot, 2018[11]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	2,631	45.9	31	6-18	—	34-49	—	cIMT ≥90th for the age- and sex-specific values	Stable normal, 113/1,453 Resolving group, 11/43 Progressively overweight, 117/879 Progressive-ly obese, 25/110 Rapid overweight/ obese, 28/113 Persistent increasing overweight/ obese, 9/33	BMI trajectory OR (95% CI) Stable normal, Ref. Resolving group, 3.12 (1.51-6.03) Progressively overweight, 1.31 (1.01-2.14) Progressively obese, 2.19 (1.31-3.90) Rapid overweight/obese, 3.10 (1.92-3.45) Persistent increasing overweight/obese, 3.14 (2.21-4.12)	Adult SES status and physical activity and family history of high-risk cIMT	7
High PWV	Aatola, 2010	The Longitudinal	Finland	1,691	45.	≈27	3-18	BMI ≥age- and	30-45	BMI ≥ag	Continuous PWV	High-high, 175	The PWV was higher in	—	5

[31]	Cardiovascular Risk in Young Finns Study			5			sex-specific 80th percentile		e- and sex-specific 80th percentile		High-low, 163 Low-High, 163 Low-low, 1,190	persistent high BMI from childhood to adulthood group compared with persistent low BMI group (P=0.002) and high BMI in childhood but low BMI in adulthood group (P=0.0002).		
Yan, 2016 [32]	The Beijing Blood Pressure Cohort Study	China	1,225	55.1	22.9 ±0.6	17.1± 3.1	BMI ≥age- and sex-specific 85th percentile based on the studied population.	34.5± 3.7	BMI cut-off points of 24 kg/m ^{2}	cfPWV ≥ age- and sex-specific 75th percentile.	Normal-Normal: -/564 Overweight/obesity-Normal: -/22 Normal-Overweight/obesity:, -/476 Overweight/obesity-Overweight/obesity:, -/163	OR (95% CI) Normal-Normal :Ref. Overweight/obesity-Normal: 1.40 (0.49-4.02) Normal-Overweight/obesity,: 1.00 (0.69-1.45) Overweight/obesity-Overweight/obesity:, 1.02 (0.62-1.68)	Age, sex, follow-up years, SBP and DBP in childhood, SBP and DBP in adulthood, taking anti-hypertensive medication or not, TG, HDL_C, LDL_C, diabetes, smoking, drinking, and physical activity	8
Yan, 2017b [30]	The Beijing Blood Pressure Cohort Study	China	1,252	55.3	22.9 ± 0.6	6-18	—	27-42	—	≥age and sex-specific 75th percentile.	—	OR (95% CI) **cfPWV** Male Childhood 1.12 (0.94-1.45) Adulthood 1.28 (0.95-1.72) AUCt 1.09 (0.88-1.27) AUCi 1.17 (0.90-1.52) Female Childhood 0.92 (0.78-1.09) Adulthood 1.18 (0.87-1.59	Adult risk factors (family history of stroke and coronary heart disease, physical activity, drinking, smoking, LDL-C, HDL-C, and TG)	6

													) AUCt 0.93 (0.72-1.20) AUCi 1.18 (0.92-1.52) **baPWV** Male Childhood 1.02 (0.87-1.19) Adulthood 0.92 (0.72-1.19) AUCt 0.79 (0.65-1.03) AUCi 0.95 (0.76-1.19) Female Childhood 0.92 (0.77-1.11) Adulthood 0.82 (0.61-1.10) AUCt 0.78 (0.60-1.01) AUCi 0.88 (0.69-1.14)		
	Liu, 2019 [33]	The Bogalusa Heart Study	The USA	1,190	43.5	30.3	4-19	—	21-50	—	Continuous afPWV	—	Childhood *B* 0.053, 95% CI (-0.012, 0.118), *P*=0.107 Adulthood *B* 0.056, 95% CI (-0.005, 0.117), *P*=0.069 From childhood to adulthood AUCt Standardized *β* 0.081, 95% CI (0.020, 0.142), *P*=0.010 AUCi Standardized *β* 0.030, 95% CI (-0.033,0.093), *P*=0.350	Adult age, sex, race, smoking, drinking, heart rate, and adult SBP. AUCt and AUCi are mutually adjusted for each other	7
CVD	Garg, 2015	The Golest	Iran	50,035	42	15	15	—	30	—	Self-reporte	Change of	Body shape gain	Pictogram score	6

[34] Heart disease	an Cohort Study		. 4							d, "Have you ever been diagnosed by a doctor as having angina, infarction, or heart failure? "	body size from childhood to adulthood Male Major decrease(>2), 13/143 Slight decrease (≤2), 138/2,906 No change, 324/6,333 Slight increase(≤2), 715/10,954 Major increase (>2), 68/863 Female Major decrease, 112/1,550 Slight decrease, 295/5,564 No change, 339/5,739 Slight increase, 791/13,000 Major increase, 249/2,942	OR (95% CI) **Male** Major decrease, 1.26 (0.66-2.39) Slight decrease, 0.85 (0.68-1.07) No change, Ref. Slight increase, 1.27 (1.10-1.47) Major increase, 1.21 (0.89-1.63) **Female** Major decrease, 1.19 (0.91-1.54) Slight decrease, 0.88 (0.74-1.05) No change, Ref. Slight increase, 1.06 (0.92-1.22) Major increase, 1.35 (1.11-1.62)	at age 15 years	
Zheng, 2017 [10] CVD	The Nurse's Health Study and the Health Professionals Follow-up Study	The USA	122,498	30.8	Women 34; Men, 24 Body shape trajectory	5	—	55	—	Self-reported diagnoses of CVD	**Women** CVD Lean stable, 742/11,183 Medium stable, 1,317/22,381 Lean-moderate increase,	Body shape trajectory HR (95% CI) **Women** CVD Lean stable, Ref. Medium stable, 0.97 (0.89-1.06) Lean-moderate	Height, race, regular aspirin use, pack-years of smoking, menopausal hormone therapy, physical activity, alcohol consum	6

y-incidence of type 2 diabetes and CVD, 17		1,279/16,534 Lean-marked increase, 979/13,201 Heavy-increase, 788/9,690 CHD Lean stable, 351/11,199 Medium stable, 641/22,418 Lean-moderate increase, 631/16,565 Lean-marked increase, 528/13,245 Heavy-increase, 448/9,717 Stroke Lean stable, 424/11,192 Medium stable, 726/22,401 Lean-moderate increase, 710/16,543 Lean-marked increase, 504/13,240 Heavy-increase, 371/9,714 **Men** CVD Lean stable,	increase, 1.18 (1.08-1.29) Lean-marked increase, 1.38 (1.25-1.52) Heavy-increase, 1.55 (1.40-1.71) CHD Lean stable, Ref. Medium stable, 0.99 (0.87-1.13) Lean-moderate increase, 1.21 (1.06-1.38) Lean-marked increase, 1.49 (1.30-1.71) Heavy-increase, 1.75 (1.52-2.02) Stroke Lean stable, Ref. Medium stable, 0.95 (0.84-1.07) Lean-moderate increase, 1.16 (1.03-1.31) Lean-marked increase, 1.29 (1.13-1.47) Heavy-increase, 1.33 (1.16-1.54) **Men** CVD Lean stable, Ref. Medium stable, 1.10 (0.98-1.23)	ption, family history of diabetes and Alternate Healthy Eating Index score

607/5,559
Medium stable, 601/5,491
Lean-moderate increase, 861/5,820
Lean-marked increase, 1,470/11,162
Heavy-increase, 463/3,938
CHD
Lean stable, 420/5,564
Medium stable, 439/5,496
Lean-moderate increase, 640/5,824
Lean-marked increase, 1,116/11,170
Heavy-increase, 354/3,942
Stroke
Lean stable, 220/5,567
Medium stable, 180/5,497
Lean-moderate increase, 264/5,831
Lean-marked

Lean-moderate increase, 1.16 (1.05-1.29)
Lean-marked increase, 1.28 (1.16-1.41)
Heavy-increase, 1.35 (1.20-1.53)
CHD
Lean stable, Ref.
Medium stable, 1.17 (1.02-1.34)
Lean-moderate increase, 1.25 (1.10-1.41)
Lean-marked increase, 1.40 (1.25-1.57)
Heavy-increase, 1.49 (1.29-1.72)
Stroke
Lean stable, Ref.
Medium stable, 0.92 (0.75-1.12)
Lean-moderate increase, 0.98 (0.82-1.18)
Lean-marked increase, 1.01 (0.86-1.19)
Heavy-increase, 1.00 (0.80-1.25)

												increase, 419/11,183 Heavy-increase, 121/3,951			
CVD mortality	Etemadi, 2014 [35]	The Golestan Cohort Study	Iran	50,006	42.4	5.1	15	Self-reportedpictogram ≥ 5	BMI:30 Outcome: 63.2 ± 9.8	Self-reportedpictogr-am ≥5	Death certificates and medical documents (ICD 10-I20-25 and I60-69)	—	HR (95% CI) **Male** Normal-Normal: Ref. Obesity-Normal: 1.29 (0.94-1.78) Normal-Obesity: 1.57 (1.17-2.10) Obesity-Obesity: 1.32 (0.93-1.88) **Female** Normal-Normal: Ref. Obesity-Normal: 1.33 (1.00-1.75) Normal-Obesity: 1.71 (1.30-2.25) Obesity-Obesity: 1.41 (1.12-1.78)	Age, SES, ethnicity, smoking, residence, opium use and education	7
	Ohlsson, 2016 [36]	A population-based BMI Epidemiology Study in Gothenburg	Sweden	37,672	100	37.8	8	BMI ≥17.9 kg/m^2	20	BMI ≥ 25 kg/m^2	390-459 in ICD 8 and 9 and I00-I99 in ICD 10	CVD mortality after age 20 years of age Normal-Normal: 591/33,514 Overweight-Normal: 23/1,368 Normal-Overweight: 66/1,800 Overweight-Overw	HR (95% CI) **CVD mortality after 20 years of age** Normal-Normal: Ref. Overweight-Normal: 0.99 (0.65-1.50) Normal-Overweight: 2.39 (1.86-3.09) Overweight-Overweight: 1.85 (1.28-2.67) **CVD mortality**	Country of birth and birth year	7

eight: 30/990 CVD mortality after age 30 years of age Normal-Normal: 570/32,590 Overweight-Normal: 23/1,329 Normal-Overweight: 66/1,757 Overweight-Overweight: 29/969 CVD mortality after age 40 years of age Normal-Normal: 536/31,491 Overweight-Normal: 23/1,282 Normal-Overweight: 65/1,721 Overweight-Overweight: 28/939 CVD mortality after age 50 years of age Normal-Normal: 424/30,272 Overweight-Normal: 18/1,2	**after 30 years of age** Normal-Normal: Ref. Overweight-Normal: 1.02 (0.68-1.55) Normal-Overweight: 2.49 (1.93-3.21) Overweight-Overweight: 1.87 (1.28-2.71) **CVD mortality after 40 years of age** Normal-Normal: Ref. Overweight-Normal: 1.09 (0.72-1.66) Normal-Overweight: 2.62 (2.03-3.40) Overweight-Overweight: 1.93 (1.32-2.83) **CVD mortality after 50 years of age** Normal-Normal: Ref. Overweight-Normal: 1.09 (0.68-1.74) Normal-Overweight: 2.14 (1.56-2.96) Overweight-Overweight: 1.78 (1.13-2.78)

									34
									Normal-Overweight: 41/1,643 Overweight-Overweight: 20/897

ATP III, The Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults; AUCi, increment of area under the curve; AUCt, total of area under the curve BMI, body mass index; afPWV, aorticfemoral pulse wave velocity; BMI, body mass index; BP, blood pressure; CRP, C-reactive protein; cIMT: carotid intima-media thickness; CHD, coronary heart disease; CVD, cardiovascular disease; cfPWV, carotid-femoral pulse wave velocity; DMFT, decayed, missing, or filled teeth; DBP, diastolic blood pressure; FPG, fasting plasma glucose; hs CRP, high-sensitivity C-reactive protein; HR: hazard ratio; HSBP, high systolic blood pressure; HDBP, high diastolic blood pressure; HOMA, homeostatic model assessment; HbA1c, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; ICD, International Classification of Disease; LVM, left ventricular mass; LVH, left ventricular hypotrophy; LDL-C, low-density lipoprotein cholesterol; MetS: Metabolism syndrome; OR: odds ratio; PR: prevalence ratio; RR: risk ratio; SD: standard deviation; SE, standard error; SEM, standard error of mean; SBP, systolic blood pressure; SES, socioeconomic status; TC, total cholesterol; TG, triglyceride; WC, waist circumference.

Table S2. Characteristics of studies on the effect of weight status change from childhood to adulthood on CVD risks factors and outcomes included in this meta-analysis

Outcome	First author, Year	Study name	Country	Sample size	Sex, male (%)	Mean/ median Follow-up years	Children		Adults		Definition of adult outcome	No. of cases / no. of samples	Main findings OR/HR/RR (95% CI)	Adjustment	Study quality
							Age, years	Definition of overweight/ obesity	Age, years	Definition of overweight/ obesity					
Type 2 diabetes	Merten, 2010 [37]	The National Longitudinal Study of Adolescent Health	The USA	10,439	51.1	7	12-19	BMI ≥age- and sex-specific 95th percentile based on the U.S. CDC reference.	19-26	BMI cut-off point of 30 kg/m ^{2}	Self-reported question: "Have you ever been diagnosed with diabetes?" (Yes/ no).	Normal-Normal : 63/7,485 Obesity-Normal (2.8%): 3/294 Normal-Obesity : 30/1,579 Obesity-Obesity : 20/1,081	Normal-Normal: Ref. Obesity-Normal: 1.38 (0.68-2.80) Normal-Obesity: 2.14 (1.63-2.92) Obesity-Obesity: 2.35 (1.65-3.32)	Sex, race, socio-economic status in both childhood and adulthood.	6
	Juonala, 2011 [38]	The Bogalusa Heart Study, the Muscatine Study, the Childhood Determinants of Adult Health study, and the Longitudinal Cardiovascular Risk in Young Finns Study.	The USA, Australia, and Finland;	6,328	46.8	23	3-18	Extrapolated according to age- and sex-specific international BMI percentiles	23-46	BMI cut-off points of 30 kg/m ^{2}	FPG ≥126 mg/dl or reported diabetes medication use.	Normal-Normal : 47/4,742 Overweight/ obesity-Normal (4.3%): 3/274 Normal-Obesity : 47/812 Overweight/ obesity-Obesity : 37/500	**Overall** Normal-Normal: Ref. Overweight/obesity -Normal: 1.3 (0.4-4.1) Normal- Obesity : 4.5 (2.9-6.8) Overweight/obesity- Obesity : 5.4 (3.4-8.5) **Male** Normal-Normal: Ref. Overweight/obesity -Normal: 3.6 (0.8-16.3) Normal- Obesity: 7.5 (3.5-16.1) Overweight/obesity - Obesity: 10.3 (4.7-22.7) **Female** Normal-Normal: Ref. Overweight/obesity -Normal: 0.5 (0.1-4.0) Normal- Obesity: 3.5 (2.1-5.9) Overweight/obesity - Obesity: 3.8 (2.1-6.8)	Age, height, length of follow-up and cohort	8
	Park, 2013 [39]	Three British National Birth Cohorts (The Medical Research	The UK	11,447	49.0	MRC NSHD, 46; NCDS, 39; BCS70, 24.	NSHD: 7 and 15 NCDS: 7 and 16 BCS70 :10 and	Extrapolated according to age- and sex-specific international BMI percentiles	BMI NSHD: 43 NCDS: 42 BCS70:34 Outcome NSHD: 53 NCDS: 42	BMI cut-off points of 30 kg/m^2	Self-reported Question: "Have you ever had diabetes"	Normal-Normal : -/8,587 Overweight/ obesity-Normal (1.6%): -/161 Normal-Obesity : -/1,144 Overweight/	**Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 1.24(0.29-5.25) Normal-Obesity: 5.47 (3.39-8.82)	Year of birth, sex, age and height in childhood, birth weight, SES position at birth, socioeconom	7

	Council National Survey of Health and Development (MRC NSHD), The National Childhood Development Survey (NCDS)and The British Cohort Study 1970 (BCS70))					16		BCS70:34			obesity-Obesity : -/266	Overweight/obesity-Obesity: 12.60 (6.61-23.98) **Childhood** Normal-Normal: Ref. Overweight/obesity-Normal: 0.99(0.35-2.80) Normal-Obesity: 5.47 (3.39-8.82) Overweight/obesity-Obesity: 4.70 (1.89-11.67) **Adolescents** Normal-Normal: Ref. Overweight/obesity-Normal: 0.88(0.31-2.50) Normal-Obesity: 5.47 (3.39-8.82) Overweight/obesity-Obesity: 6.61 (3.61-12.09)	ic position in adulthood, smoking in adulthood, and weighted sampling (NSHD).	
Liang, 2015 [40]	The Beijing Blood Pressure Cohort Study	China	1,209	45.0	22.9 ± 0.5	11.6 ± 3.7	**7-18 years:** BMI ≥age- and sex-specific 95th percentile based on the WGOC reference or left subscapular skinfold ≥ age- and sex-specific 85th percentile based on the U.S. CDC reference. **6 years:** BMI ≥age- and sex-specific 95th percentile based on the U.S. CDC reference.	≈34.5	BMI cut-off point of 28 kg/m ^{2}	FPG ≥7.0 mmol/l or 2h PG ≥11.1 mmol/l or current using glucose-lowering medication.	Normal-Normal : 38/903 Obesity-Normal (5.7%): 8/69 Normal-Obesity : 14/140 Obesity-Obesity : 18/97	Normal-Normal: Ref. Obesity-Normal: 2.8 (1.2-6.3) Normal-Obesity: 2.2 (1.2-4.4) Obesity-Obesity: 4.3 (2.2-8.1)	Age of adults, sex, physical activity, smoking, and alcohol drinking in adulthood.	8
Bjerregaard, 2018 [41]	The Copenhagen School Health Record Register	Denmark	62,565	100	1,969,165 person-years	7-13	BMI cut-off points of 17.38 kg/m ^{2} for 7 years and 21.82 kg/m ^{2} for 13 years based on the U.S. CDC reference.	BMI 17-26 Outcome 30-60	BMI cut-off points of 25 kg/m ^{2}	Type 2 diabetes: ICD, Eighth Revision (1994, code 250) and the 10th Revision (Codes E 11- E 14)	**7 years-adulthood** Normal-Normal : 2,798/54,529 Overweight/ obesity-Normal (2.4%): 70/1,437 Normal-Overweight/ obesity: 451/2,807 Overweight/ obesity-	**7 years-adulthood** Normal-Normal: Ref. Overweight/obesity-Normal: 0.99 (0.78-1.25) Normal-Overweight/ obesity: 3.24 (2.93-3.58) Overweight/obesity-Overweight/obesity: 2.55 (1.92-3.39) **13 years-**	Age at conscription, education and intelligence-test score.	6

											Overweight/ obesity: 48/374 **13 years-adulthood** Normal-Normal : 2,798/54,529 Overweight/ obesity-Normal (1.5%): 78/900 Normal-Overweight/ obesity: 451/2,870 Overweight /obesity-Overweight/ obesity: 186/956	**adulthood** Normal-Normal: Ref. Overweight/obesity-Normal: 1.70 (1.35-2.12) Normal-Overweight/ obesity: 3.24(2.93-3.58) Overweight/obesity-Overweight/obesity: 3.87 (3.33-4.49)		
Ohlsson, 2019 [42]	The BMI Epidemiology Study Gothenburg	Sweden	36,176	100	28.3	6.5-9.5	Overweight: BMI ≥17.9 kg/m	BMI 17.5-22 Outcome ≈55.7	BMI cut-off points of 25 and 30 kg/m ^{2}	ICD: E11 in ICD 10 and 250 in ICD 8 and ICD 9 (for the first time after 30 years) Information of diabetes at 55.7 years in 1,777 participants was obtained from the Swedish National Patient Register	**Type 2 diabetes (≤ 55.7 years)** Normal-Normal : 620/32,202 Overweight/ obesity-Normal (3.6%): 32/1,309 Normal-Overweight/ obesity: 152/1,718 Overweight/ obesity-Overweight/obesity: 85/947 Normal-Normal : 808/35,489 Obesity-Normal : 24/381 Normal-Obesity: 36/201 Obesity-Obesity: 21/105 **Type 2 diabetes (> 55.7 years)** Normal-Normal : 726/23,441 Overweight/ obesity-Normal (3.5%): 37/922 Normal-Overweight/ obesity: 76/1,044 Overweight/	**Type 2 diabetes (≤ 55.7 years)** Normal-Normal: Ref. Overweight/obesity-Normal: 1.28 (0.89-1.82) Normal-Overweight/obesity: 4.67 (3.90-5.58) Overweight/obesity - Overweight/obesity : 4.82 (3.84-6.05) Normal-Normal: Ref. Obesity-Normal: 2.78 (1.85-4.17) Normal-Obesity: 8.19 (5.86-11.45) Obesity-Obesity: 10.59 (6.86-16.33) **Type 2 diabetes (> 55.7 years)** Normal-Normal: Ref. Overweight/obesity-Normal: 1.35 (0.97-1.87) Normal-Overweight/obesity : 2.85 (2.25-3.61) Overweight/obesity-Overweight/obesity : 3.04 (2.27-4.06) Normal-Normal: Ref.	Country of birth and birth year.	7

												obesity-Overweight/ obesity: 49/608 Normal-Normal : 853/25,597 Obesity-Normal : 16/257 Normal-obesity: 14/109 Obesity-obesity: 5/52	Obesity-Normal: 2.18 (1.33-3.58) Normal-Obesity: 5.38 (3.17-9.12) Obesity-Obesity: 3.51 (1.46-8.45)		
Hypertension	Merten, 2010 [37]	The National Longitudinal Study of Adolescent Health	The USA	10,439	51.1	7	12-19	BMI ≥age- and sex-specific 95th percentile based on the U.S. CDC reference.	19-26	BMI cut-off point of 30 kg/m ^{2}	Self-reported question: "Have you ever been diagnosed with high BP?"(Yes/no)	Normal-Normal : 269/7,485 Obesity-Normal (2.8%): 21/294 Normal-Obesity : 170/1,579 Obesity-Obesity : 177/1,081	Normal-Normal: Ref. Obesity-Normal: 2.11 (1.46-3.06) Normal-Obesity: 3.19 (2.72-3.74) Obesity-Obesity: 5.22 (4.44-6.11)	Sex, race, socioeconomic status in both childhood and adulthood.	6
	Juonala, 2011 [38]	The Bogalusa Heart Study, the Muscatine Study, the Childhood Determinants of Adult Health study, and the Longitudinal Cardiovascular Risk in Young Finns Study.	The USA, Australia, and Finland;	6,328	46.8	23	3-18	Extrapolated according to age- and sex -specific international BMI percentiles	23-46	BMI cut-off points of 30 kg/m ^{2}	SBP and/or DBP ≥140/90 mmHg or reported BP antihypertensive medication use	Normal-Normal : 512/4,742 Overweight/ obesity-Normal (4.3%): 24/274 Normal-Obesity : 192/812 Overweight/ obesity-Obesity : 143/500	**Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 0.9 (0.6-1.4) Normal-Obesity: 2.1 (1.7-2.4) Overweight/obesity-Obesity: 2.7 (2.2-3.3) **Male** Normal-Normal: Ref. Overweight/obesity-Normal: 1.1 (0.7-1.8) Normal-Obesity: 1.8 (1.4-2.2) Overweight/obesity-Obesity: 2.5 (2.0-3.2) **Female** Normal-Normal: Ref. Overweight/obesity-Normal: 0.6 (0.2-1.4) Normal-Obesity: 2.6 (2.0-3.4) Overweight/obesity-Obesity: 3.2 (2.3-4.4)	Age, height, length of follow-up and cohort.	8
	Park, 2013 [39]	Three British National Birth Cohorts	The UK	11,447	49.0	MRC NSHD, 46; NCDS, 39; BCS70, 24.	NSHD: 7 and 15 NCDS: 7 and	Extrapolated according to age- and sex-specific international	BMI NSHD: 43 NCDS: 42 BCS70:34	BMI cut-off points of 30 kg/m ^{2}	Self-reported Question: "Have you ever had hypertension"	Normal-Normal : -/8,587 Overweight/obesity-Normal (1.6%):	**Overall** Normal-Normal: Ref. Overweight/obesity-Normal:	Year of birth, sex, age and height in childhood, birth weight,	7

		(The Medical Research Council National Survey of Health and Development (MRC NSHD), The National Childhood Development Survey (NCDS) and The British Cohort Study 1970 (BCS70))					16 BCS70 :10 and 16	BMI percentiles	Outcome NSHD: 53 NCDS: 42 BCS70:34			-/161 Normal-Obesity : -/1,144 Overweight/obesity-Obesity: -/266	1.01(0.46–2.21) Normal-Obesity: 2.28(1.76–2.95) Overweight/obesity-Obesity: 2.56 (1.40–4.68) **Childhood** Normal-Normal: Ref. Overweight/obesity-Normal: 0.87(0.54-1.40) Normal-Obesity: 2.28(1.76–2.95) Overweight/obesity-Obesity: 2.91 (1.54–5.49) **Adolescents** Normal-Normal: Ref. Overweight/obesity-Normal: 0.97(0.61-1.55) Normal-Obesity: 2.28(1.76–2.95) Overweight/obesity-Obesity: 3.01(2.11–4.29)	SES position at birth, socioeconomic position in adulthood, smoking in adulthood, and weighted sampling (NSHD).	
	Hou, 2019 [43]	The China Health and Nutrition Survey	China	2,095	59.9	11	6-17	Based on the age- and sex-specific BMI cut-off points in Chinese children aged 2-18 years	18-37	BMI cut-off points of 24 kg/m ^{2}	SBP and/or DBP ≥140/90 mmHg or current BP antihypertensive medication use	Normal-Normal : 50/1,604 Overweight/obesity-Normal (5.3%): 3/111 Normal-Overweight/obesity: 50/301 Overweight/obesity-Overweight/obesity: 11/79	Normal-Normal: Ref. Overweight/obesity-Normal: 1.05 (0.33-3.40) Normal-Overweight/obesity: 3.75 (2.49-5.64) Overweight/obesity-Overweight/obesity: 3.79(1.94-7.41)	Age, sex, SBP and DBP percentiles in childhood, smoking status and drinking in adults.	8
Dyslipidemia	Merten, 2010 [37] High cholesterol	The National Longitudinal Study of Adolescent Health	The USA	10,439	51.1	7	12-19	BMI ≥age- and sex-specific 95th percentile based on the U.S. CDC reference.	19-26	BMI cut-off point of 30 kg/m ^{2}	Self-reported question: "Have you ever been diagnosed with high cholesterol?"(Yes/no)	Normal-Normal : 266/7,485 Obesity-Normal (2.8%): 19/294 Normal-Obesity : 99/1,579 Obesity-Obesity : 111/1,081	**Overall** Normal-Normal: Ref. Obesity-Normal: 1.81 (1.22-2.75) Normal-Obesity: 1.86 (1.52-2.27) Obesity-Obesity: 3.14 (2.56-3.82) **Male** Normal-Normal: Ref. Obesity-Normal: 1.95 (1.27-3.00) Normal-Obesity: 2.72 (2.14-3.46) Obesity-Obesity: 3.86 (3.10-4.81) **Female**	Sex, race, socioeconomic status in both childhood and adulthood.	6

												Normal-Normal: Ref. Obesity-Normal: 2.14 (1.23-3.71) Normal-Obesity: 1.35 (1.06-1.72) Obesity-Obesity: 2.39 (1.86-3.06)		
Juonala, 2011 [38] High-risk LDL_C, High-risk HDL_C, and High-risk TG	The Bogalusa Heart Study, the Muscatine Study, the Childhood Determinants of Adult Health Study, and the Longitudinal Cardiovascular Risk in Young Finns Study.	The US, Australia, and Finland;	6,328	46.8	23	3-18	Extrapolated according to age- and sex-specific international BMI percentiles	23-46	BMI cut-off points of 30 kg/m ^{2}	High-risk LDL_C: ≥160 mg/dl High-risk HDL_C:<40 mg/dl High-risk TG: ≥200 mg/dl	High-risk LDL_C: Normal-Normal: 432/4,742 Overweight/obesity-Normal (4.3%): 23/274 Normal-Obesity: 119/812 Overweight/obesity-Obesity: 89/500 High-risk HDL_C: Normal-Normal: 711 /4,742 Overweight/obesity-Normal: 39/274 Normal-Obesity: 309/812 Overweight/obesity-Obesity: 196/500 High-risk TG Normal-Normal: 351/4,742 Overweight/obesity-Normal: 13/274 Normal-Obesity: 203/812 Overweight/obesity-Obesity: 117/500	**High-risk LDL_C** **Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 1.1 (0.7-1.6) Normal-Obesity: 1.5 (1.2-1.9) Overweight/obesity-Obesity: 1.8 (1.4-2.3) **Male** Normal-Normal: Ref. Overweight/obesity-Normal: 1.1 (0.7-1.9) Normal-Obesity: 1.7 (1.3-2.2) Overweight/obesity-Obesity: 1.5 (1.1-2.0) **Female** Normal-Normal: Ref. Overweight/obesity-Normal: 0.9 (0.4-1.9) Normal-Obesity: 1.3 (0.9-1.8) Overweight/obesity-Obesity: 2.7 (1.8-3.9) **High-risk HDL_C** **Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 1.0 (0.7-1.3) Normal-Obesity: 2.2 (1.9-2.6) Overweight/obesity-Obesity: 2.1 (1.8-2.5) **Male** Normal-Normal: Ref. Overweight/obesity-	Age, height, length of follow-up and cohort.	8

													Normal: 0.9 (0.6-1.3) Normal-Obesity: 2.0 (1.7-2.3) Overweight/obesity-Obesity: 1.7 (1.4-2.1) **Female** Normal-Normal: Ref. Overweight/obesity-Normal: 1.1 (0.6-2.0) Normal-Obesity: 3.0 (2.3-3.8) Overweight/obesity-Obesity: 3.3 (2.4-4.3) **High-risk TG** **Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 0.7 (0.4-1.2) Normal-Obesity: 3.2 (2.7-3.8) Overweight/obesity-Obesity: 3.0 (2.4-3.8) **Male** Normal-Normal: Ref. Overweight/obesity-Normal: 0.6 (0.3-1.2) Normal-Obesity: 3.2 (2.6-3.9) Overweight/obesity-Obesity: 3.0 (2.4-3.9) **Female** Normal-Normal: Ref. Overweight/obesity-Normal: 1.0 (0.4-2.3) Normal-Obesity: 3.2 (2.3-4.4) Overweight/obesity-Obesity: 2.9 (1.9-4.4)			
Yan, 2019 [22]	The International Childhood Cardiovascular Cohort Consortium	The US, Finland, and Australia;	5,195	39.8	27.3± 7.0	3-19	Low and high BMI was defined based on age-, sex- and race-specific	20-52	BMI cut-off points of 25 kg/m ^{2}	Dyslipidemia was defined based on one of the following criteria: High-risk HDL_C: <40	Normal-Normal: -/1,336 Overweight/obesity-Normal (12.0%): -/625 Normal-Overwe	Normal-Normal: Ref. Overweight/obesity-Normal: 0.9 (0.7-1.2) Normal-Overweight/	Age, sex, race, and cohort.	8

								medians in childhood			mg/dl; High risk TG: ≥200 mg/dl; High-risk LDL_C: ≥160 mg/dl	ight/obesity: -/1,261 Overweight/obesity-Overweight/obesity: -/1,973	obesity: 3.4 (2.8-4.1) Overweight/obesity-Overweight/obesity: 3.5 (3.0-4.2) **High LDL-C** Normal-Normal: Ref. Overweight/obesity-Normal: 0.8 (0.6-1.25) Normal-Overweight/obesity: 2.4 (1.75-3.1) Overweight/obesity-Overweight/obesity: 2.2 (1.75-2.75) **Low HDL-C** Normal-Normal: Ref. Overweight/obesity-Normal: 1.01 (0.8-1.3) Normal-Overweight/obesity: 3.3 (2.8-4.2) Overweight/obesity-Overweight/obesity: 4.0 (3.3-4.8) **High TG** Normal-Normal: Ref. Overweight/obesity-Normal: 0.5 (0.3-0.9) Normal-Overweight/obesity: 3.2 (2.4-4.2) Overweight/obesity-Overweight/obesity: 3.0 (2.3-4.0)		
NAFLD	Yan, 2017c[44]	The Beijing Blood Pressure Cohort Study	China	1,350	54.1	23.3	6-18	International BMI cut-offs for age and sex	28-45	BMI cut-off point of 25 and 30 kg/m ^{2}	fatty liver on ultrasonography (gradual attenuation of far-field echo and diffuse enhancement of nearby field echo)without heavy drinking	Male Normal-Normal: -/593 Overweight/obesity-Normal: -/32 Normal-Obesity: -/66 Overweight/obesity-Obesity: -/40 Female Normal-Normal: -/546 Overweight/obesity-Normal:	Male Normal-Normal: Ref. Overweight/obesity-Normal: 2.13 (0.98-4.65) Normal-Obesity: 3.90 (2.08-7.28) Overweight/obesity-Obesity: 3.48 (1.64-7.38) Female Normal-Normal: Ref. Overweight/obesity-Normal: 1.90	Length of follow-up, age in childhood, TG, TC, LDL-C, HDL-C, drinking, smoking, and physical activity in adulthood	8

												-/30 Normal-Obesity : -/29 Overweight/obesity-Obesity: -/14	(0.67-5.40) Normal-Obesity: 3.08 (1.11-8.58) Overweight/obesity-Obesity: 7.88 (2.03-30.55)		
	Cuthbertson, 2019[45]	The Cardiovascular Risk in Young Finns Study	Finland	2,020	45.7	31	3-18	International BMI centiles for age and sex	34-49	BMI cut-off point of 25 and 30 kg/m ^{2}	Based on the diaphragm and the visibility of vessel walls, liver parenchyma brightness, and hepatic-renal ratio	Normal-Normal 48/858 Overweight/obesity-Normal 1/20 Normal-Overweight/obesity 280/999 Overweight/obesity-Overweight/obesity 54/143 Normal-Normal 180/1,540 Overweight/obesity-Normal 6/68 Normal-obesity 148/317 Overweight/obesity-obesity 49/95	Normal-Normal Ref. Overweight/obesity-Normal 1.13 (0.14-8.92) Normal-Overweight/obesity 6.14 (4.23-8.90) Overweight/obesity-Overweight/obesity 11.3 (6.77-18.72) Normal-Normal Ref. Overweight/obesity-Normal 0.89 (0.34-2.31) Normal-Obesity 8.72 (5.86-12.10) Overweight/obesity-Obesity 9.69 (5.86-16.01)	Age, sex, physical activity, smoking, and alcohol consumption in adulthood	8
MetS	Hosseinpanah, 2013 [46]	The Tehran Lipid and Glucose Study	Iran	1,424	44.9	10.4± 1.0	14.6 ± 2.2	BMI ≥age- and sex-specific BMI 85th percentile, respectively, based on the reference of Iranian children and adolescents.	18-31	BMI cut-off point of 30 kg/m ^{2}	At least 3 of the following criteria: 1) WC ≥ 91 cm for female and ≥ 89 cm for male ; 2) BP ≥ 130/85 mmHg or antihypertensive drug use; 3) TG ≥150 mg/d or drug use; 4) HDL_C < 50 mg/dl for female and < 40 mg /dl for male or drug use; 5) FPG ≥ 100 mg/dl or drug use.	Normal-Normal : 96/1,061 Overweight/obesity-Normal (11.4%): 21/162 Normal-Obesity : 27/60 Overweight/obesity-Obesity: 66/141	**Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 1.59 (0.92-2.74) Normal-Obesity: 9.66 (4.93-18.91) Overweight/obesity-Obesity: 11.82 (7.27-19.23) **Males** Normal-Normal: Ref. Overweight/obesity-Normal: 1.62 (0.92-2.86) Normal-Obesity: 8.45 (4.18-18.73) Overweight/obesity-Obesity:	sex	7

													9.87 (5.77-16.90) **Females** Normal-Normal: Ref. Overweight/obesity-Normal: 1.32 (0.15-11.43) Normal-Obesity: 16.39 (3.68-72.96) Overweight/obesity-Obesity: 23.11(7.62-70.16)		
	Liang, 2015 [40]	The Beijing Blood Pressure Cohort Study	China	1,209	45.0	22.9 ± 0.5	11.6 ± 3.7	**7-18 years:** BMI ≥age- and sex-specific 95th percentile based on the WGOC reference or left subscapular skinfold ≥ age- and sex-specific 85th percentile based on the U.S. CDC reference. **6 years:** BMI ≥age- and sex-specific 95th percentile based on the U.S. CDC reference.	≈34.5	BMI cut-off point of 28 kg/m ^{2}	At least 3 of the following criteria: 1) WC ≥ 90 cm for male or ≥ 85 cm for female; 2) BP ≥130/85 mmHg; 3) TG ≥1.7 mmol/l; 4) HDL_C <1.04 mmol/l for male and <1.29 mmol/l for female; 5) FPG ≥ 5.6 mmol/l.	Normal-Normal : 142/903 Obesity-Normal (5.7%): 13/69 Normal-Obesity : 96/140 Obesity-Obesity : 65/97	Normal-Normal: Ref. Obesity-Normal: 1.2 (0.6-2.4) Normal-Obesity: 11.4 (7.4-17.6) Obesity-Obesity: 9.5 (5.8-15.5)	Age of adults, sex, physical activity, smoking, and alcohol drinking in adulthood	8
High-risk c-IMT	Juonala, 2011 [38]	The Bogalusa Heart Study, the Muscatine Study, the Childhood Determinants of Adult Health Study, and the Longitudinal Cardiovascular Risk in Young Finns Study.	The USA, Australia, and Finland	6,328	46.8	23	3-18	Extrapolated according to age- and sex -specific international BMI percentiles	23-46	BMI cut-off points of 30 kg/m ^{2}	cIMT ≥ age-, sex-, race-, study-year-90th percentiles of IMT	Normal-Normal : 597/4,742 Overweight/obesity-Normal (4.3%): 32/274 Normal-Obesity : 144/812 Overweight/obesity-Obesity: 101/500	**Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 0.9 (0.6-1.3) Normal-Obesity: 1.5 (1.3-1.8) Overweight/obesity-Obesity: 1.7 (1.4-2.2) **Male** Normal-Normal: Ref. Overweight/obesity-Normal: 1.2 (0.7-1.9) Normal-Obesity: 1.5 (1.1-1.9) Overweight/obesity-Obesity: 1.5 (1.1-2.2) **Female** Normal-Normal: Ref. Overweight/obesity-Normal: 0.7	Age, height, length of follow-up and cohort.	8

												(0.4-1.2) Normal-Obesity: 1.6 (1.2-2.0) Overweight/obesity-Obesity: 1.9 (1.4-2.6)		
	Yan, 2016 [32]	The Beijing Blood Pressure Cohort Study	China	1,225	55.1	22.9± 0.6	17.1±3.1	BMI ≥age- and sex-specific 85th percentile based on the studied population.	34.5±3.7	BMI cut-off points of 24 kg/m ^{2}	cIMT ≥age- and sex-specific 75th percentile.	Normal-Normal: -/564 Overweight/obesity-Normal (1.8%): -/22 Normal-Overweight/obesity: -/476 Overweight/obesity-Overweight/obesity: -/163	Normal-Normal: Ref. Overweight/obesity-Normal: 1.95 (0.68-5.53) Normal-Overweight/obesity: 2.92 (2.04 -4.18) Overweight/obesity-Overweight/obesity: 3.73 (2.37-5.85)	Age, sex, follow-up years, SBP and DBP in childhood, SBP and DBP in adulthood, taking anti-hypertensive medication or not, TG, HDL_C, LDL_C, diabetes, smoking, drinking, and physical activity.	8
	Su, 2014 [47]	A 1992-2000 mass urine screening for a renal health study in Taiwan (China)	China	789	39.7	8.5	6-18	BMI ≥age- and sex-specific 85th percentile based on the reference of Taiwan (China) Department of Health	Mean age of 21	BMI cut-off points of 24 kg/m ^{2} and 27 kg/m ^{2}	cIMT ≥75th percentile.	Normal-Normal: -/584 Overweight/obesity-Normal (3.8%): -/30 Normal-Overweight:-/ 66 Overweight/obesity-Overweight: -/32	Normal-Normal: Ref. Overweight/obesity-Normal: 2.07(0.91-4.71) Normal-Overweight: 1.67 (0.94-2.99) Overweight/obesity-Overweight: 2.82 (1.26-6.28)	Age, sex, SBP, FPG, Cholesterol, smoking, alcohol drinking and household income.	8
												Normal-Normal: -/584 Overweight/obesity-Normal: -/30 Normal-Obesity: -/23 Overweight/obesity-Obesity: -/55	Normal-Normal: Ref. Overweight/obesity-Normal: 2.07 (0.91-4.71) Normal-Obesity: 1.32 (0.51-3.44) Overweight/obesity-Obesity: 4.17 (2.21-7.85)		
CVD	Morrison, 2012 [48] CVD	The NHLBI Princeton Follow-up Study and the NHLBI Lipid Research Clinics study	The USA	770	46.0	26	12.4±3.3	BMI ≥age- and sex-specific 85th percentile based on the U.S. CDC 2000.	38.5±3.6	BMI cut-off points of 30 kg/m ^{2}	Self-reported (yes or no) defined as coronary artery bypass graft, myocardial infarction, angioplasty, carotid or peripheral artery bypass surgery, and ischemic	Normal-Normal: 4/427 Overweight/obesity-Normal (10.5%): 2/81 Normal-Obesity: 6/149 Overweight/obesity-Obesity: 7/113	Crude OR (95%CI) Normal-Normal: Ref. Overweight/obesity-Normal: 2.68 (0.48-14.87) Normal-Obesity: 4.44 (1.24-15.95) Overweight/obesity-Obesity: 6.98 (2.01-24.30)	—	6

										stroke.			
Park, 2013 [39] CHD	Three British National Birth Cohorts (The Medical Research Council National Survey of Health and Development (MRC NSHD), The National Childhood Development Survey (NCDS)and The British Cohort Study 1970 (BCS70))	The UK	11,447	49.0	MRC NSHD, 46; NCDS, 39; BCS70, 24.	NSHD: 7 and 15 NCDS: 7 and 16 BCS70 :10 and 16	Extrapolated according to age- and sex-specific international BMI percentiles	BMI NSHD: 43 NCDS: 42 BCS70:34 Outcome NSHD: 53 NCDS: 42 BCS70:34	BMI cut-off points of 30 kg/m ^{2}	Self-reported Question: "Have you ever had CHD"	Normal-Normal : -/8,587 Overweight/ obesity-Normal (1.6%): -/161 Normal-Obesity : -/1,144 Overweight/ obesity-Obesity : -/266	**Overall** Normal-Normal: Ref. Overweight/obesity-Normal: 3.43(0.60-19.64) Normal-Obesity: 3.83 (1.98-7.42) Overweight/obesity-Obesity: 6.62 (1.94-22.65) **Childhood** Normal-Normal: Ref. Overweight/obesity-Normal: 0.44(0.20-1.89) Normal-Obesity: 3.83 (1.98-7.42) Overweight/obesity-Obesity: 1.10 (0.14-8.48) **Adolescents** Normal-Normal: Ref. Overweight/obesity-Normal: 1.63(0.37-7.19) Normal-Obesity: 3.83 (1.98-7.42) Overweight/obesity-Obesity: 3.74 (1.35-10.35)	Year of birth, sex, age and height in childhood, birth weight, SES position at birth, socioeconomic position in adulthood, smoking in adulthood, and weighted sampling (NSHD).	7
Ohlsson, 2017 [49] Stroke	Archived School Health Care Records	Sweden	37,669	100	37.6	6.5-9.5	Overweight: ≥17.9 kg/m ^{2}	17.5-22	BMI cut-off points of 25 kg/m ^{2}	The stroke events were defined based on the ICD system codes	Stroke Normal-Normal : 779/33,511 Overweight/ obesity-Normal (3.6%): 36/1,368 Normal-Overweight/ obesity : 67/1,800 Overweight/ obesity - Overweight/ obesity : 36/990 Ischemic Normal-Normal : 578/33,511 Overweight/ obesity -Normal: 25/1,368 Normal-Overweight/	**Stroke** Normal-Normal: Ref. Overweight/obesity -Normal: 1.17 (0.83-1.63) Normal-Overweight/obesity : 1.81 (1.41-2.33) Overweight/obesity - Overweight/obesity : 1.71 (1.22-2.38) **Ischemic stroke** Normal-Normal: Ref. Overweight/obesity -Normal: 1.09 (0.73-1.62) Normal-Overweight/obesity : 1.48 (1.08-2.03) Overweight/obesity -	Country of birth and birth year.	7

											obesity : 41/1,800 Overweight/ obesity - Overweight/ obesity : 28/990 Intracerebral hemorrhage Normal-Normal : 169/33,511 Overweight/ obesity -Normal: 9/1,368 Normal- Overweight/ obesity : 24/1,800 Overweight/ obesity - Overweight/ obesity : 5/990	Overweight/obesity : 1.78 (1.22-2.60) **Intracerebral hemorrhage** Normal-Normal: Ref. Overweight/obesity -Normal: 1.35 (0.69-2.63) Normal- Overweight/obesity : 3.03 (1.97-4.65) Overweight/obesity - Overweight/obesity : 1.09 (0.45-2.66)		
Kindblom, 2018 [50] Heart failure	Archived School Health Care Records	Sweden	37,670	100	37.7	6.5-9.5	Overweight: ≥17.9 kg/m^2	17.5-22	BMI cut-off points of 25 kg/m ^{2}	Heart failure was defined as patients hospitalized with heart failure or death with this heart failure in a code based on the ICD system	Normal-Normal : 269/33,512 Overweight/ obesity-Normal (3.6%): 12/1,368 Normal- Overweight/ obesity : 40/1,800 Overweight/ obesity - Overweight/ obesity : 21/990	Normal-Normal: Ref. Overweight/obesity -Normal: 1.12 (0.63-2.00) Normal- Overweight/obesity : 3.14 (2.25-4.38) Overweight/obesity - Overweight/obesity : 2.85 (1.83-4.45)	Country of birth and birth year.	6

BMI, body mass index; CHD, coronary heart disease; CfPWV, carotid-femoral pulse wave velocity; DBP, diastolic blood pressure; FPG, fasting plasma glucose; HbA1c, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; ICD, International Classification of Disease; cIMT: carotid intima-media thickness; IOTF, International Obesity Task Force; LDL-C, low-density lipoprotein cholesterol; Mets: metabolism syndrome; NAFLD: non-alcoholic fatty liver disease. OR: odds ratio; RR: risk ratio; SBP, systolic blood pressure; TC, total cholesterol; TG, triglyceride; US CDC, US Center for Disease Control and Prevention; WC, waist circumference; WGOC, China Obesity Task Group; 2HPG, 2 hours postprandial blood glucose. The proportion of the resolution group is presented in brackets.

Table S3. Characteristics of studies on the effect of long-term burden of weight from childhood to adulthood on subclinical CVD included in this meta-analysis

	First author, Year	Study name	Country	Sample size	Sex, male (%),	Mean/ Median follow-up years	Children	Adults	Definition of adult outcome	No. of cases / no. of samples	Main findings OR (95% CI)			Adjustment	Study quality
							Age, years	Age, years			Childhood	Adulthood	AUC		
LVH	Lai, 2014 [51]	The Bogalusa Heart Study	The USA	1,061	42.6	28	4-18	24-46	Women>46.7 g/m $^{2.7}$ and Men> 49.2 g/m $^{2.7}$	153/1,061	1.65 (1.39-1.97)	2.53 (2.06-3.09)	AUCt 2.42 (1.98-2.95) AUCi 2.09 (1.72-2.53)	AUCt Mean adulthood age, and then Z-transformed AUCi Mean age, values in childhood and then Z-transformed	8
	Yan, 2017a [52]	The Beijing Blood Pressure Cohort Study	China	1,256	55.3	22.9 ± 0.6	6-18	27-42	Women>46.7 g/m $^{2.7}$ and Men>49.2 g/m $^{2.7}$	56/1,256	2.59 (1.86-3.60)	4.51 (3.12-6.53)	AUCt 3.36 (2.50-4.52) AUCi 2.68 (1.95-3.68)	Sex and adulthood factors (age, antihypertensive medication, diabetes status, physical inactivity, Alcohol, smoking, consumption, and length of follow-up).	8
LV geometry	Lai, 2014 [51]	The Bogalusa Heart Study	The USA	1,061	42.6	28	4-18	24-46	LVH, Women> 46.7 g/m $^{2.7}$ and Men> 49.2 g/m $^{2.7}$; Concentric LV geometry when RWT > 0.42 Concentric remodeling, RWT >0.42 with no LVH; Eccentric hypertrophy, normal RWT with LVH; Concentric hypertrophy, RWT >0.42 with LVH	Concentric remodeling, 97/1,061 Eccentric hypertrophy, 108/1,061 Concentric hypertrophy, 45/1,061	Concentric remodeling, 0.86 (0.66-1.12) Eccentric hypertrophy, 1.59 (1.30-1.93) Concentric hypertrophy, 1.66 (1.27-2.17)	Concentric remodeling, 1.04 (0.81-1.34) Eccentric hypertrophy, 2.42 (1.94-3.03) Concentric hypertrophy, 2.45 (1.75-3.43)	AUCt Concentric remodeling, 0.93 (0.72-1.20) Eccentric hypertrophy, 2.23 (1.79-2.78) Concentric hypertrophy, 2.40 (1.75-3.30) AUCi Concentric remodeling, 1.05 (0.82-1.35) Eccentric hypertrophy, 2.04(1.65-2.52) Concentric hypertrophy, 1.99 (1.44-2.75)	AUCt Mean adulthood age, and then Z-transformed AUCi Mean age, values in childhood and then Z-transformed	8
	Yan, 2017a [52]	The Beijing Blood Pressure Cohort Study	China	1,256	55.3	22.9 ± 0.6	6-18	27-42	LVH, Women> 46.7 g/m $^{2.7}$ and Men>49.2 g/m $^{2.7}$; Concentric LV geometry when RWT > 0.42 Concentric remodeling,	Concentric remodeling, 332/1,256 Eccentric hypertrophy, 25/1,256 Concentric hypertrophy, 31/1,256	Increased RWT 1.06 (0.94-1.19) Concentric remodeling, 1.06 (0.93-1.18) Eccentric hypertrophy, 3.53 (2.09-5.98) Concentric	Increased RWT 1.40 (1.21-1.63) Concentric remodeling, 1.37 (1.17-1.61) Eccentric hypertrophy, 5.91 (3.37-10.36) Concentric	Increased RWT AUCt 1.25 (1.08-1.43) AUCi 1.29 (1.11-1.49) Concentric remodeling, AUCt 1.20 (1.03-1.40) AUCi 1.27 (1.09-1.48)	Sex and adulthood factors (age, antihypertensive medication, diabetes status, physical inactivity, alcohol, smoking, consumption, and length of follow-up).	8

	RWT >0.42 with no LVH; Eccentric hypertrophy, normal RWT with LVH; Concentric hypertrophy, RWT >0.42 with LVH	hypertrophy, 2.69 (1.77-4.09)	hypertrophy, 5.68 (3.38-9.55)	Eccentric hypertrophy, AUCt 3.81 (2.43-5.96) AUCi 2.54 (1.74-3.69) Concentric hypertrophy, AUCt 3.53 (2.32-5.38) AUCi 2.45 (1.80-3.33)

AUCi, increment of area under the curve (cumulative values of body mass index); AUCt, total of area under the curve; cIMT: carotid intima-media thickness; LVMI, left ventricular mass index; LVH, left ventricular hypotrophy; HDL-C, high-density lipoprotein cholesterol LDL-C, low-density lipoprotein cholesterol; RWT, relative wall thickness.

Table S4. Characteristics of the excluded studies regarding the association of weight change from childhood to adulthood with adult CVD risk factors and outcomes in this systematic review and meta-analysis

Outcome	First author, Year	Study name	Country	Sample size	Sex, male (%)	Mean/ median follow-up years	Children		Adults		Definition of adult outcome	No. of cases / no. of samples	Main findings	Adjustment	Reasons for exclusion
							Age, years	Definition of overweight /obesity	Age, years	Definition of overweight /obesity					
Type 2 diabetes	Freedman, 2001 [53]	The Bogalusa Heart Study	The USA	2,617	43.0	17	2-17	Normal: BMI<50th and Obesity: BMI ≥95th	18-37	Normal: BMI<25 kg/m ^{2} and Obesity: BMI ≥ 30 kg/m ^{2}	Self-reported (diabetes or the use of medication for diabetes).	Normal-Normal: 0/950 Obesity-Normal: 0/12 Normal-Obesity: 3/96 Obesity-Obesity: 3/144	Crude OR **(95% CI)** Normal-Normal: Ref. Obesity-Normal: — Normal-Obesity: 71.16 (2.65-1388.15) Obesity-Obesity: 47.02 (2.42-915.11)	—	Small statistical power
	Bhargava, 2004 [54]	The study in South Delhi	India	1,526	58.1	≈27	2	BMI cut-off point of 16.1 kg/m ^{2}	Mean age of 29.2	BMI cut-off point of 22.7 kg/m^2	Impaired glucose tolerance: FPG <7.0 mmol/l and a 120-minute value ≥7.8 mmol/l Diabetes: FPG ≥7.0 mmol/l or a 120-minute ≥11.1mmol/l.	Impaired glucose or diabetes BMI<16.1kg/m^2-BMI<22.7kg/m^2: 34/340 BMI≥16.1-BMI <22.7 kg/m^2: 7/ 97 BMI<16.1kg/m^2-BMI≥22.7kg/m^2: 110/557 BMI≥16.1-BMI ≥22.7 kg/m^2: 52/364	Crude OR **(95% CI)** BMI<16.1kg/m^2-BMI<22.7kg/m^2: Ref. BMI≥16.1-BMI <22.7 kg/m^2: 0.70 (0.30-1.63) BMI<16.1kg/m^2-BMI≥22.7kg/m^2: 2.22 (1.47-3.34) BMI≥16.1-BMI ≥22.7 kg/m^2: 1.50 (0.95-2.38)	Sex and current age	Outcome including impaired glucose tolerance
	Sivanandam, 2006 [55]	A Study approved by the committee for the use of Human Subjects in Research at the University of Minnesota	The USA	132	48.5	14	13	—	27	—	Continuous insulin and glucose	Baseline thin children BMI change <5.5 kg/m ^{2}, 28 BMI change≥ 5.5 kg/m ^{2}, 38 Baseline heavy children BMI change <5.5 kg/m ^{2} , 31 BMI change≥ 5.5 kg/m ^{2}, 35	Mean±SEM Fasting insulin (mU/l) Baseline thin children < 5.5 kg/m ^{2}, 7.3 ±1.4 ≥ 5.5 kg/m ^{2}, 10.3 ±1.4 (P<0.05) Baseline heavy children <5.5 kg/m ^{2} , 7.7± 1.4 ≥ 5.5 kg/m ^{2}, 16.3 ±1.3 (P<0.05) Glucose use Baseline thin children <5.5 kg/m ^{2}, 13.0 ± 0.8 ≥ 5.5 kg/m ^{2}, 11.4 ±0.7 Baseline heavy children <5.5 kg/m ^{2} , 11.4± 0.7 ≥ 5.5 kg/m ^{2}, 8.5 ±0.8 (P<0.05)	Sex, race and Tanner stage	Small sample size
	Rademacher, 2009 [56]	The Sodium Potassium Blood Pressure Trial in Children	The USA	342	52.3	≈11	Mean age of 13	—	Mean age of 24	—	Continuous Insulin and glucose	—	1unit change of BMI from childhood to adulthood and CVD risk levels β± SE **Adjusted for baseline**	Age, sex, baseline SBP or DBP, baseline BMI, change in	Small sample size

												SBP Insulin: 0.73±0.15 (*P*<0.0001) Glucose: 0.77±0.14(*P*<0.0001) **Adjusted for baseline DBP** Insulin: 0.73±0.15 (*P*<0.0001) Glucose: 0.79±0.14(*P*<0.0001)	SBP or DBP and race	
Fonseca, 2010 [57]	The study in Universidade do Estado do Rio de Janeiro, Brazil	Brazil	115	55.7	17	A1, 12.97± 1.48	—	A2, 21.90± 1.71 A3, 30.65± 2.00	—	Glycemia ≥ 126 mg/dl	**Increased glucose** (*P*=0.004) BMI always normal at 3 assessment, 0/46 BMI varying at 3 assessment, 2/49 BMI always increased at 3 assessment, 3/20	The prevalence of increased glucose was higher in BMI always increased group compared with the other 2 groups (*P*=0.044)	—	Small sample size
Power, 2011 [58]	The 1958 British Birth Cohort	The UK	7,855	50.1	45	7,11, and 16	Extrapolated according to age- and sex-specific international BMI percentiles	BMI 23 33, and 45 Outcome 45	BMI cut-off points of 25 and 30 kg/m ^{2}	Type 2 diabetes diagnosed by doctors or HbA1c ≥7	Obesity Normal-Normal: -/5,819 Obesity-Normal: -/62 Normal-Obesity $_{young-adulthood}$: -/652 Normal-Obesity $_{mid-adulthood}$: -/1,171 Obesity-Obesity: -/151 Overweight (including obesity) Normal-Normal: -/2,366 Overweight/obesity-Normal: -/163 Normal-Overweight/obesity $_{young-adulthood}$: -/2,396 Normal-Overweight/obesity $_{mid-adulthood}$: -/1,729 Overweight/obesity-Overweight/obesity: -/1,201	**Obesity in adulthood:** Normal-Normal: Ref. Obesity-Normal: 4.95 (1.30-18.93) Normal-Obesity $_{young-adulthood}$: 3.96 (2.10-7.43) Normal-Obesity $_{mid-adulthood}$: 1.13 (0.61-2.08) Obesity-Obesity: 4.38 (1.86-10.31) **Overweight (including obesity) in adulthood:** Normal-Normal: Ref. Overweight/obesity-Normal: - Normal-Overweight/obesity $_{young-adulthood}$: 1.87 (0.79-4.45) Normal-Overweight/obesity $_{mid-adulthood}$: 0.77 (0.27-2.19) Overweight/obesity-Overweight/obesity: 3.25 (1.30-8.14)	Sex, HDL_C, TC, social class in childhood, family history of diabetes, smoking and drinking in adulthood, menopausal status, social class in adulthood, and qualifications in adulthood.	Duplicate study
Tirosh, 2011 [59]	The Metabolic, Lifestyle, and Nutrition Assessment in Young Adults	Israel	37,674	100	17.4±7.4	17	Quintile 1-5	30	Quintile 1-5	At least two FPG ≥ 7.0 mmol/l	—	Compared to the lowest BMI quintile in childhood and adulthood, the highest quartile in both childhood and adulthood had the highest hazard ratio for	Age, TG, family history of diabetes, and fasting glucose.	Limited data for meta-analysis

		Study of the Israel Defense Force Medical Corps											diabetes (HR≈4.9).		
	Hou, 2016 [60]	The Beijing Blood Pressure Cohort Study	China	1,225	55.1	22.9± 0.6	6-18	**7-18 years:** BMI ≥age- and sex-specific 95th percentile based on the WGOC reference. **6 years:** BMI ≥age- and sex-specific 95th percentile based on the U.S. CDC reference.	30-42	BMI cut-off points of 28 kg/m^2	**Diabetes diagnosed by blood glucose:** met at least 1 of the following criteria: 1) FPG ≥7.0 mmol/l; 2) 2h PG ≥11.1 mmol/l; 3) Using insulin or glucose-lowering medication. **Diabetes diagnosed by HbA1c:** HbA1c ≥6.5% or using insulin or glucose-lowering medication.	Normal-Normal: -/937 Obesity-Normal: -/40 Normal-Obesity: -/170 Obesity-Obesity: -/69	**Diabetes diagnosed by blood glucose:** Normal-Normal: Ref. Obesity-Normal: 1.90 (0.86-4.19) Normal-Obesity: 1.71 (0.50-5.79) Obesity-Obesity: 4.50 (2.22-9.14) **Diabetes diagnosed by HbA1c:** Normal-Normal: Ref. Obesity-Normal: 1.42 (0.71-2.86) Normal-Obesity: 3.13 (0.83-11.75) Obesity-Obesity: 5.93 (3.06-11.49)	Age of baseline, sex, smoking, drinking, dietary and sleep.	Duplicate study
	Zhao, 2020[61]	The National Longitudinal Study of Adolescent Health	The USA	4454	50.5	13	12-19	Overweight and obesity: BMI≥ 85th percentile based on the US CDC reference	25-32	Overweight and obesity: BMI≥ 25 kg/m^2	FPG≥126 mg/dL or no fasting glucose ≥200 mg/dL or HbA1c ≥6.5% or using insulin or glucose-lowering medication or history of diabetes diagnosed by physicians	Normal-Normal: 48/1328 Overweight/ obesity-Normal: 3/66 Normal-Overweight/obesity: 107/1983 Overweight/ obesity-Overweight/obesity: 131/1077	Normal-Normal: Ref. Overweight/obesity-Normal: 1.65 (0.45-6.05) Normal-Overweight/obesity: 1.59 (1.01-2.51) Overweight/obesity-Overweight/obesity: 3.32 (2.11-5.21)	Age, sex, race/ethnicity, smoking, drinking, physical activity in adolescence and adulthood	Duplicate study
Hypertension	Abraham, 1971 [62]	A Cohort Study in Hagerstown	The USA	715	100	≈40	9-13	Normal: relative weight<104 Overweight /obesity: ≥105	35-40	Normal: relative weight<104 Overweight /obesity: ≥105	Clinical diagnosis	Normal-Normal: 86/389 Overweight/ obesity-Normal: 9/49 Normal-Overweight/obesity: 61/190 Overweight/ obesity-Overweight/ obesity: 19/87	Crude OR (95%CI) Normal-Normal: Ref. Overweight/obesity-Normal: 0.35(0.12-1.01) Normal-Overweight/ obesity: 1.67(1.13-2.45) Overweight/obesity-Overweight/obesity: 0.98 (0.56-1.73)	—	Change in relative weight
	Srinivasan, 1996 [63]	The Bogalusa Heart Study	The USA	783	—	≈15	13-17	Overweight : BMI≥ age-, sex-, and race-	25-31	Overweight : BMI≥ age-, sex-, and race-	SBP/DBP>140/ 90 mmHg or anti-hypertensive drug use	Lean cohort: 2/ 81 Adolescent- onset of adult	The prevalence of hypertension in the overweight cohort was 8.5 times that in lean cohort.	—	Limited data for meta-analysis

							specific 75th percentile of the BMI in Bogalusa population Lean: 25-50th percentile		specific 75th percentile of the BMI in Bogalusa population Lean: 25-50th percentile		overweight: 23/110 *P*<0.001			
Sivanandam, 2006 [55]	A Study approved by the committee for the use of Human Subjects in Research at the University of Minnesota	The USA	132	48.5	14	13	—	27	—	Continuous BP	Baseline thin children BMI change <5.5 kg/m^2, 28 BMI change≥ 5.5 kg/m^2, 38 Baseline heavy children BMI change <5.5 kg/m^2 , 31 BMI change≥ 5.5 kg/m^2, 35	**SBP** (mmHg) Baseline thin children <5.5 kg/m ^{2}, 112.0± 1.9 ≥ 5.5 kg/m ^{2}, 114.7 ±1.9 Baseline heavy children <5.5 kg/m ^{2} , 113.7± 1.9 ≥ 5.5 kg/m ^{2}, 117.0 ±1.8 **DBP** (mmHg) Baseline thin children <5.5 kg/m ^{2}, 69.2± 1.9 ≥ 5.5 kg/m ^{2}, 71.3± 2.0 Baseline heavy children <5.5 kg/m ^{2} , 69.5± 1.9 ≥ 5.5 kg/m ^{2}, 72.0 ±1.8	Sex, race and Tanner stage	Small sample size
Fonseca, 2010 [57]	The Study in Universidade do Estado do Rio de Janeiro, Brazil	Brazil	115	55.7	17	A1, 12.97± 1.48	—	A2, 21.90± 1.71 A3, 30.65± 2.00	—	BP≥ 140/90 mmHg	**Increased BP** (*P*=0.004) BMI always normal at 3 assessment, 4/46 BMI varying at 3 assessment, 17/49 BMI always increased at 3 assessment, 14/20	BMI trajectory The prevalence of increased BP was higher in BMI always increased group compared with the other 2 groups (*P*<0.0001)	—	Small sample size
Suglia, 2013 [64]	The Add Health Study in the US (a National Longitudinal Study of Adolescents Health)	The USA	8,543	51.9	13	Mean age of 16	BMI ≥age- and sex-specific 85th percentile based on the U.S. CDC reference.	Mean age of 29	BMI cut-off point of 30 kg/m ^{2}	**Pre-hypertension** BP 121-140/81-89 mmHg **Hypertension** SBP and/or DBP ≥140/90 mmHg or reported BP antihypertensive medication use	Normal-Normal: -/4,752 Overweight/ obesity-Normal: -/6,26 Normal-Obesity: -/1,195 Overweight/ obesity-Obesity: -/1,966	**Male** **Pre-hypertension** **Black** Normal-Normal: Ref. Overweight/obesity-Normal: 2.08(0.8–5.8) Normal-Obesity: 1.78(0.7–4.6) Overweight/obesity-Obesity: 1.63 (0.9–2.9) **Hispanic** Normal-Normal: Ref. Overweight/obesity-Normal: 2.13 (0.8–5.9) Normal-Obesity: 3.38 (1.4–8.4) Overweight/obesity-Obesity: 2.01 (0.9–4.5) **White** Normal-Normal: Ref. Overweight/obesity-	Age, smoking, household income, and education levels.	Duplicate study

Normal: 1.44 (0.9–2.3)
Normal-Obesity: 2.32 (1.4–3.8)
Overweight/obesity-Obesity: 3.03 (1.9–4.8)
Hypertension
Black
Normal-Normal: Ref.
Overweight/obesity-Normal: 2.93 (1.2–7.3)
Normal-Obesity: 2.92 (1.1–7.5)
Overweight/obesity-Obesity: 4.36(1.9–10.0)
Hispanic
Normal-Normal: Ref.
Overweight/obesity-Normal: 1.64 (0.5–5.9)
Normal-Obesity: 4.91 (1.8–13.2)
Overweight/obesity-Obesity: 2.74 (1.1–6.7)
White
Normal-Normal: Ref.
Overweight/obesity-Normal: 1.56 (0.9–2.7)
Normal-Obesity: 3.38 (2.0–5.8)
Overweight/obesity-Obesity: 6.38 (3.9–10.3)
Female
Pre-hypertension
Black
Normal-Normal: Ref.
Overweight/obesity-Normal: 1.30 (0.6–2.8)
Normal-Obesity: 3.26 (1.8–5.8)
Overweight/obesity-Obesity: 2.33 (1.4–3.9)
Hispanic
Normal-Normal: Ref.
Overweight/obesity-Normal: 1.35 (0.3–5.2)
Normal-Obesity: 2.90 (1.5–5.5)
Overweight/obesity-Obesity: 3.06 (1.7–5.4)
White
Normal-Normal: Ref.
Overweight/obesity
-Normal: 1.54 (0.9–2.4)
Normal-Obesity: 2.83 (2.0–4.1)
Overweight/obesity-

												Obesity: 3.55 (2.6–4.8) **Hypertension** **Black** Normal-Normal: Ref. Overweight/obesity-Normal: 1.76 (0.6–5.5) Normal-Obesity: 4.99 (2.4–10.4) Overweight/obesity-Obesity: 3.37 (2.1–5.5) **Hispanic** Normal-Normal: Ref. Overweight/obesity-Normal: 3.12 (0.6–15.8) Normal-Obesity: 4.84 (1.3–18.1) Overweight/obesity-Obesity: 6.46 (2.2–19.4) **White** Normal-Normal: Ref. Overweight/obesity-Normal: 1.13 (0.5–2.4) Normal-Obesity: 4.13 (2.8–6.1) Overweight/obesity-Obesity: 5.77 (3.9–8.5)		
Su, 2014 [47]	A 1992-2000 mass urine screening for a renal health study in Taiwan (China)	China	789	39.7	8.5	6-18	BMI ≥age- and sex-specific 85th percentile based on the reference of Taiwan (China) Department of Health	Mean age of 21	BMI cut-off points of 24 kg/m ^{2} and 27 kg/m ^{2}	Hypertension: SBP and/or DBP ≥140/90 mmHg or BP antihypertensive medication use Pre-hypertension: BP 120-139/80-89 mmHg	Normal-Normal: -/584 Overweight/obesity-Normal: -/30 Normal-Overweight:-/ 66 Overweight/obesity-Overweight: -/32 Normal-Normal: -/584 Overweight/obesity-Normal: -/30 Normal-Obesity: -/23 Overweight/obesity-Obesity: -/55	Pre-hypertension and hypertension Normal-Normal: Ref. Overweight/obesity-Normal: 2.23(0.89-5.58) Normal-Overweight: 1.84 (1.02-3.30) Overweight/obesity-Overweight: 3.20 (1.40-7.33) Normal-Normal: Ref. Overweight/obesity-Normal: 2.23 (0.89-5.58) Normal-Obesity: 2.75 (1.08-6.97) Overweight/obesity-Obesity: 6.51 (3.36-12.63)	Age, sex, SBP, FPG, Cholesterol, smoking, alcohol drinking and household income.	Outcome including pre-hypertension and hypertension
Yang, 2017 [65]	The Childhood Determinants of Adult Health Study and The 1985 Australia Schools	Australia	159	47.0	28.9± 0.1	7-15	Based on the age- and sex-specific BMI cut-off points of the IOTF	36-45	BMI cut-off points of 25 kg/m^2	Hypertension SBP and/or DBP ≥140/90 mmHg.	Hypertension Normal-Normal: 1/63 Overweight-Normal: — Normal-Overweight/obesity: 9/78 Overweight-Over	**Hypertension** Crude OR (95% CI) Normal-Normal: Ref. Overweight-Normal: — Normal-Overweight/obesity: 8.09 (1.00-65.66) Overweight-Overweight/obesity: 4.43 (0.26-75.18)	—	Small sample size

		Health and Fitness Surveys						reference.					weight/obesity: 1/15			
	Wang, 2019 [66]	The China Health and Nutrition Survey (1993-2011)	China	1,431	60.3	11	6-17	WC ≥age-, sex-specific 90th percentile based on the reference of Chinese children aged 7-18 years	18-35	Male: WC ≥85 cm; Female: WC ≥80 cm	Hypertension: SBP and/or DBP ≥140/90 mmHg or current BP antihypertensive medication use or having a history of hypertension. Pre-hypertension: SBP and/or DBP ≥120/80 mmHg and <140/90 mmHg and non-antihypertensive medication use	Hypertension Normal-Normal: 26/1,057 Abdominal obesity-Normal: 1/45 Normal-Abdominal obesity: 29/289 Abdominal obesity-Abdominal obesity: 7/40 Pre-hypertension Normal-Normal: 329/1,057 Abdominal obesity-Normal: 15/45 Normal-Abdominal obesity: 157/289 Abdominal obesity-Abdominal obesity: 21/40	**Hypertension** Normal-Normal: Ref. Abdominal obesity-Normal: 1.52 (0.19-12.06) Normal- Abdominal obesity : 6.48 (3.60-11.66) Abdominal obesity - Abdominal obesity : 15.98 (5.39-47.35) **Pre-hypertension** Normal-Normal: Ref. Abdominal obesity -Normal: 1.28 (0.66-2.49) Normal- Abdominal obesity : 2.90 (2.17-3.89) Abdominal obesity - Abdominal obesity : 3.49 (1.65-7.40)	Age, sex, and hypertension in adolescents and smoking, drinking, dietary in adults.	In Chinese and WC change
	Zhao, 2020[61]	The National Longitudinal Study of Adolescent Health	The USA	4454	50.5	13	12-19	Overweight and obesity: BMI≥ 85th percentile based on the US CDC reference	25-32	Overweight and obesity: BMI≥ 25kg/m^2	SBP and/or DBP ≥140/90 mmHg or current BP antihypertensive medication use or history of hypertension diagnosed by physician	Normal-Normal: 163/1328 Overweight/obesity-Normal: 8/66 Normal-Overweight/obesity: 525/1983 Overweight/obesity-Overweight/obesity: 399/1077	Normal-Normal: Ref. Overweight/obesity-Normal: 1.37 (0.64-2.95) Normal-Overweight/obesity: 2.49 (1.98-3.15) Overweight/obesity-Overweight/obesity: 3.72 (2.86-4.84)	Age, sex, race/ethnicity, smoking, drinking, physical activity in adolescence and adulthood	Duplicate study
Dyslipidemia	Srinivasan, 1996 [63]	The Bogalusa Heart Study	The USA	783	—	≈15	13-17	Overweight, BMI≥ age-, sex-, and race-specific 75th percentile of the BMI in Bogalusa population Lean, 25-50th percentile	25-31	Overweight, BMI≥ age-, sex-, and race-specific 75th percentile of the BMI in Bogalusa population Lean, 25-50th percentile	TC >240 mg/dl; LDL-C >160 mg/dl HDL-C <35 mg/dl TG >250 mg/dl	High TC Lean cohort, 5/ 81 Adolescent-onset adult overweight, 16/110 High LDL-C Lean cohort, 5/ 81 Adolescent-onset adult overweight, 20/110. *P*<0.05 Low HDL-C Lean cohort, 2/ 81 Adolescent-onset adult overweight, 17/110. *P*<0.01 High TG Lean cohort, 1/ 81	The prevalence of high LDL-C, low HDL-C, and TG in the overweight cohort were 3.1, 5.4, and 8.3 times that in lean cohort group.	—	Limited data for meta-analysis

											Adolescent-onset adult overweight, 14/110.*P*<0.01.			
Sivanandam, 2006 [55]	A Study approved by the committee for the use of Human Subjects in Research at the University of Minnesota	The USA	132	48.5	14	13	—	27	—	Continuous TC, HDL-C	Baseline thin children BMI change <5.5 kg/m ^{2}, 28 BMI change≥ 5.5 kg/m ^{2}, 38 Baseline heavy children BMI change <5.5 kg/m ^{2} , 31 BMI change≥ 5.5 kg/m ^{2}, 35	Mean±SEM TC (mg/dl) Baseline thin children <5.5 kg/m ^{2}, 81.5± 18.6 ≥ 5.5 kg/m ^{2}, 126.6 ±19.2 Baseline heavy children <5.5 kg/m ^{2} , 105.8 ±19.0 ≥ 5.5 kg/m ^{2}, 192.4 ±18.1 (*P*<0.05) HDL-C(mg/dl) Baseline thin children <5.5 kg/m ^{2}, 52.4± 1.9 ≥ 5.5 kg/m ^{2}, 46.5± 2.0 (*P*<0.05) Baseline heavy children < 5.5 kg/m ^{2} , 46.1± 1.9 ≥ 5.5 kg/m ^{2}, 39.5± 1.8(*P*<0.05)	Sex, race and Tanner stage	Small sample size
Rademacher, 2009 [56]	The Sodium Potassium Blood Pressure Trial in Children	The USA	342	52.3	≈11	13	—	24	—	Continuous cholesterol, HDL-C, LDL-C, and TG	—	1unit change of BMI from childhood to adulthood and CVD risk levels *β*± SE **Adjusted for SBP** Cholesterol: 1.96±0.39 (*P*<0.0001) HDL-C: -0.52±0.13 (*P*<0.0001) LDL-C: 1.61±0.35 (*P*<0.0001) TG: 0.04±0.01 (*P*<0.0001) **Adjusted for DBP** Cholesterol: 1.84±0.38 (*P*<0.0001) HDL-C: -0.46±0.13 (*P*<0.001) LDL-C: 1.49±0.35 (*P*<0.0001) TG: 0.04±0.01 (*P*<0.001)	Age, sex, baseline SBP or DBP, baseline BMI, change in SBP or DBP and race	Small sample size
Fonseca, 2010 [57]	The study in Universidade do Estado do Rio de Janeiro, Brazil	Brazil	115	55.7	17	A1, 12.97± 1.48	—	A2, 21.90± 1.71 A3, 30.65± 2.00	—	IV Brazilian Directive on dyslipidemia and Atherosclerosis Prevention	**Increased TG** (*P*>0.05) BMI always normal at 3 assessment, 5/46 BMI varying at 3 assessment, 9/49 BMI always increased at 3 assessment, 6/20 **low HDL-C** (*P*=0.008) BMI always normal at 3	BMI trajectory The prevalence of low HDL-C was higher in BMI always increased group compared with the other 2 groups (*P*=0.008)	—	Small sample size

												assessment, 13/46 BMI varying at 3 assessment, 26/49 BMI always increased at 3 assessment, 13/20			
NAFLD	Sandboge, 2013[67]	The Helsinki Birth Cohort Study	Finland	1,583	40.7	60	2	BMI tertiles: <16 kg/m ^{2};16 -17 kg/m ^{2}; >17 kg/m ^{2}	62	BMI cut-off points of 25 and 30 kg/m ^{2}	NAFLD liver fat equation and score	<16 kg/m ^{2}-≤25 kg/m ^{2}: 25/207 16-17 kg/m ^{2}-≤25 kg/m ^{2}: 15/159 >17 kg/m ^{2}-≤25 kg/m ^{2}: 4/113 <16 kg/m ^{2}-25-30 kg/m ^{2}: 73/225 16-17 kg/m ^{2}-25-30 kg/m ^{2}: 65/227 >17 kg/m ^{2}-25 -30kg/m ^{2}: 80/273 <16 kg/m ^{2}->30 kg/m ^{2}: 73/108 16-17 kg/m ^{2}->30 kg/m ^{2}: 79/130 >17 kg/m ^{2}-2>30kg/m ^{2}: 73/141	<16 kg/m ^{2}-≤25 kg/m ^{2}: Ref. 16-17 kg/m ^{2}-≤25 kg/m ^{2}: 0.7 (0.4-1.4) >17 kg/m ^{2}-≤25 kg/m ^{2}: 0.3 (0.1-0.8) <16 kg/m ^{2}-25-30 kg/m ^{2}: 3.7 (2.2-6.1) 16-17 kg/m ^{2}-25-30 kg/m ^{2}: 2.6 (1.6-4.4) >17 kg/m ^{2}-25 -30kg/m ^{2}: 2.5 (1.5-4.2) <16 kg/m ^{2}->30 kg/m ^{2}: 18.5 (10.1-33.6) 16-17 kg/m ^{2}->30 kg/m ^{2}: 13.2 (7.5-23.2) >17 kg/m ^{2}-2>30kg/m ^{2}: 8.8 (5.0-15.2)	Age and sex	No definition of overweight or obesity in childhood
MetS	Vanhala, 1998 [68]	A population study for the metabolic syndrome in 1993-1994	Finland	439	49.9	>11	7	The sex-specific highest third of the BMI	≥ 18	The sex-specific highest third of the BMI	A cluster of the following criteria: 1) SBP and/or DBP ≥ 140/90 or use antihypertensive medication; 2) TG ≥ 1.70 mmol/l or HDL_C < 1.0 mmol/l in men and < 1.20 mmol/l in women or both; 3) insulin resistance based on the criteria of World Health Organization or hyperinsulinemia (≥78 pmol/l).	Normal-Normal: 2/219 Obesity-Normal: 0/74 Normal- Obesity : 7/71 Obesity - Obesity : 21/75	Normal-Normal: Ref. Obesity -Normal: - Normal-Obesity: 16.0 (2.9-159) Obesity-Obesity: 56.0(13.0-504)	—	Small sample size
High IMT	Li, 2003 [69]	The Study in the Biracial Community of Bogalusa	The USA	486	39.3	22.2	4-17	—	25-37	—	Upper quartile vs. lower 3 quartiles of IMT z-score	—	OR (95% CI) Childhood 1.25 (1.01-1.54) Adulthood 1.09 (0.86-1.39) From childhood to	Z-scores specific for age, sex, and race	Small sample size

												adulthood AUCt 1.16 (0.92-1.46)		
Freedman, 2004 [70]	The Bogalusa Heart Study	The USA	513	39.6	31	4	BMI ≥95th percentile	35	BMI cut-off points of 30 kg/m^2	High IMT, at 90th percentile, intermediate IMT at 10th -89th, and low IMT, at 10th percentile	—	The mean level of IMT was highest (834 μm) in overweight children who became obese adults.	Age, sex, and race	Limited data for meta-analysis
Juonala, 2006 [71]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	1,564	—	21	3-18	Lean, BMI <50th Overweight / obese, BMI>80th	24-39	BMI cut-off points of 25 kg/m^2	Continuous IMT	Lean-Normal:825 Overweight/ obesity-Normal: 112 Lean-Overweight/ obesity: 310 Overweight/ obesity-Overweight/obesity: 317	Lean-Normal, 0.610 mm, 95% CI (0.603-0.616) Overweight/obesity -Normal, 0.627 mm, 95% CI (0.610-0.644) Lean- Overweight/obesity , 0.634 mm, 95% CI (0.624-0.644) Overweight/obesity - Overweight/obesity , 0.642 mm, 95% CI (0.632-0.652) *P* for trend<0.0001	—	Limited data for meta-analysis
Juonala, 2010 [72]	The Longitudinal Cardiovascular Risk in Young Finns Study	Finland	1,809	43.9	27	3-18	A cut-off of 50th percentile	24-45	A cut-off of 50th percentile	Continuous IMT	Low-Low, -/655 High-Low, -/277 Low-High, -/262 High-High, -/615	Individuals with a favorable change in BMI status from childhood to adulthood had a slower IMT progression.	—	Limited data for meta-analysis
Huynh, 2013 [73]	The Childhood Determinants of Adult Health study	Australia	2,328	49.4	21	7-15	Based on the IOTF BMI cut-offs	26-36	BMI cut-off points of 25 kg/m^2	Continuous IMT	Male Normal-Normal: -/906 Overweight/ obesity-Normal: -/55 Normal-Overweight/ obesity: -/117 Overweight/ obesity -Overweight/ obesity: -/72 Female Normal-Normal: -/944 Overweight/ obesity-Normal: -/66 Normal-Overweight/ obesity: -/101 Overweight/ obesity	Mean±SD Male Normal-Normal: 0.607 mm ± 0.098 Overweight/obesity -Normal: 0.637 mm ± 0.103 Normal-Overweight/obesity: 0.622 mm ± 0.106 Overweight/obesity - Overweight/obesity: 0.614 mm ± 0.110 Female Normal-Normal: 0.576 mm ± 0.080 Overweight/obesity -Normal: 0.567 mm ± 0.067 Normal-Overweight/obesity: 0.582 mm ± 0.084 Overweight/obesity - Overweight/obesity:	Mean-corrected standard deviation scores of weight in childhood, adulthood, child height, and adult age	Limited data for meta-analysis

												-Overweight/ obesity: -/66	0.582 mm ± 0.094		
LVH/LVMI	Li, 2004 [74]	The study in the Biracial Community of Bogalusa	The USA	467	39.4	21.5	4-17	—	20-38	—	Continuous age-, sex-, and race-specific LVMI-Z score	—	Childhood *B* 0.122 (*P*=0.019) Adulthood *B* 0.231 (*P*<0.001) From childhood to adulthood (AUCt) *B* 0.204 (*P*<0.001)	SBP, HDL-C, LDL-C, and TG	Small sample size
	Tapp, 2014 [75]	The Childhood Determinants of Adult Health Study	Australia	181	53.6	≈20	9-16	Age- and sex-specific BMI cut-offs based on the British 1990 growth reference	26-36	The highest age- and sex-specific BMI quartile	Continuous LVMI and LVM	—	There was an upward trend of the mean values of LVMI across the 4 groups (Normal-Normal, Normal-Overweight/obesity, Overweight/obesity-Normal, and Overweight/obesity-Overweight/obesity, *P* for trend <0.001) **Male** 1 unit increase of BMI from childhood to adulthood was associated with 0.27 g (95% CI, 0. 04-0.51) increase of LVM in adulthood. **Female** 1 unit increase of BMI from childhood to adulthood was associated with 0.39 g (95% CI, 0. 20-0.58) increase of LVM in adulthood.	Change in adiposity, childhood obesity, covariates in adults (age, TG, TC, and fitness)	Small sample size
	Yan, 2016 [32]	The Beijing Blood Pressure Cohort Study	China	1,225	55.1	22.9±0.6	17.1±3 .1	BMI ≥age- and sex-specific 85th percentile based on the studied population.	34.5± 3.7	BMI cut-off points of 24 kg/m ^{2}	—	Normal-Normal: -/564 Overweight/ obesity-Normal: -/22 Normal-Overweight/obesity: -/476 Overweight /obesity-Overweight/obesity: -/163	OR (95% CI) Normal-Normal : Ref. Overweight/obesity-Normal: 2.43 (0.85-6.95) Normal-Overweight/obesity: 5.96 (4.01-8.86) Overweight/obesity-Overweight/obesity: 9.58 (5.92-15.49)	Age, sex, follow-up years, SBP and DBP in childhood, SBP and DBP in adulthood, taking anti-hypertensive medication or not, TG, HDL_C, LDL_C, diabetes, smoking, drinking, and physical activity	Limited articles for meta-analysis and systematic review
	Yang, 2017 [65]	The Childhood Determinants	Australia	159	47.0	28.9± 0.1	7-15	Based on the age- and sex-	36-45	BMI cut-off points of 25 kg/m^2	LVH LVMI >75 g/m $^{1.7}$ in men	LVH Normal-Normal: 4/63	**LVH** Crude OR (95% CI) Normal-Normal: Ref.	—	Small sample size

		of Adult Health Study and The 1985 Australia Schools Health and Fitness Surveys						specific BMI cut-off points of the IOTF reference.			and >57 g/m $^{1.7}$ in women	Overweight-Normal:- Normal-Overweight/ obesity: 19/78 Overweight-Overweight/ obesity: 6/15	Overweight-Normal: — Normal-Overweight/ obesity: 4.75 (1.52-14.81) Overweight-Overweight/obesity: 9.83 (2.32-41.78)		
	Zhang, 2017 [76]	The Bogalusa Heart Study	The USA	710	42.4	28	Mean age of 12	—	26-48	—	LVH Women, LVMI > 46.7 g/m $^{2.7}$ and Men, LVMI > 49.2 g/m $^{2.7}$ LV geometry, Concentric when RWT > 0.42 Eccentric hypertrophy, normal RWT with LVH; Concentric hypertrophy, RWT >0.42 with LVH	LVH 102/710 LV geometry Eccentric hypertrophy, 74/611 Concentric hypertrophy, 28/565	**LVH** Childhood 2.15 (1.76-2.65) Adulthood:— AUCt 2.71 (2.19-3.40) AUCi 2.29 (1.86-2.85) **LV geometry** Eccentric hypertrophy, Childhood: 2.25 (1.79-2.86) AUCt 2.67 (2.11-3.44) AUCi 2.25 (1.77-2.90) Concentric hypertrophy, Childhood : 1.88 (1.34-262) AUCt 2.36 (1.69-3.36) AUCi 2.08 (1.52-2.89)	AUCt Childhood-to-adulthood mean age, then Z-transformed AUCi BMI in childhood, childhood-to-adulthood mean age, then Z-transformed	Duplicate study
	Yan, 2020[77]	The Bogalusa	The USA	877	40.5	31.4	4-19	—	20-51	—	LVH Women, LVMI > 46.7 g/m $^{2.7}$ and Men, LVMI > 49.2 g/m $^{2.7}$ LV geometry, Concentric when RWT > 0.42 Concentric remodeling: RWT >0.42 with no LVH Eccentric hypertrophy, normal RWT with LVH; Concentric hypertrophy, RWT >0.42 with LVH	LVH 154/814; EH 73/733 CH 81/741	**LVH** AUCt 2.65 (2.10-3.34) AUCi 1.94 (1.56-2.41) Eccentric hypertrophy AUCt 2.48 (1.87-3.29) AUCi 1.94 (1.44-2.60) Concentric hypertrophy AUCt 2.67 (2.03-3.53) AUCi 2.17 (1.64-2.88)	SBP, sex, race, adult drinking, smoking, and age	Duplicate study
CVD	Abraham, 1971 [62]	A cohort study in Hagerstown	The USA	715	100	≈40	9-13	Normal: relative weight<104	35-40	Normal: relative weight<104	Clinical diagnosis	Normal-Normal: 149/389 Overweight/	Crude OR (95%CI) Normal-Normal: Ref. Overweight/obesity-	—	Change in relative weight

Cardiovascular renal disease							Overweight /obesity: ≥105		Overweight /obesity: ≥105		obesity-Normal: 18/49 Normal-Overweight/obesity: 88/190 Overweight/obesity-Overweight/obesity: 33/87	Normal: 0.94 (0.51-1.73) Normal-Overweight/obesity: 1.39 (0.98-1.97) Overweight/obesity-Overweight/obesity: 0.98 (0.61-1.59)		
Tirosh, 2011 [59] CHD	The Metabolic, Lifestyle, and Nutrition Assessment in Young Adults Study of the Israel Defense Force Medical Corps	Israel	37,674	100	17.4±7.4	17	Quintile 1-5	30	Quintile 1-5	Proven stenosis ≥ 50% in at least 1 coronary artery.	—	Compared to the lowest BMI quintile in childhood and adulthood, the highest quartile in both childhood and adulthood had the highest hazard ratio for CHD (HR≈9).	Age, TG, family history of diabetes, and fasting glucose.	Limited data for meta-analysis
Barbour-Tuck, 2019[78]	The Saskatchewan Pediatric Bone Mineral Accrual Study	Canada	55	45.5	24.1	11.5 ± 1.8		35.6 ± 2.2	Percentage of trunk fat mass	CVD risk scores	—	Those with higher CVD risk scores had a higher percentage of trunk fat mass trajectories compare with those with lower CVD risk scores	—	Small sample size
Islam, 2019 [12] Combined CVD risk factor	The China Health Nutrition Survey	China	5,276	52.4	20	6-17	BMI cut-off of 85th and 95th age- and sex-specific percentile based on Chinese school-age children.for overweight and obesity	≥18	BMI cut-offs of 24 and 28 kg/m^2	CVD risk factor: High BP, High FPG or dyslipidemia	Normal-Stable, -/1,180 Low normal-Normal-Stable, -/2,327 Low normal-Normal-Overweight, -/1,437 Overweight-Obese, -/332	OR (95% CI) Normal-Stable, Ref. Low normal Normal-Stable, 2.3 (1.8-2,.8) Low normal-Normal-Overweight, 4.5(3.5-5.8) Overweight-Obese, 10.0 (6.4-15.4)	Age, sex, education, living region, smoking, alcohol consumption, unhealthy dietary pattern and physical activity at the last follow-up	Combined CVD risk factors
Fan, 2020[79]	The Chinese Health and Nutrition Survey	China	541	71.9	14.9	11.4 ± 3.2	BMI ≥ 75th percentile	26.2 ± 5.0	BMI ≥ 75th percentile	CVD risk factor: overweight and obesity, high TC, hypertension, high TG, high LDL, low HDL, and high hemoglobin A1c	BMI z-score <75th percentile in both childhood and adulthood: -/332 BMI z-score <75th in childhood and ≥ 75th percentile in adulthood: -/74 BMI z-score ≥75th in childhood and < 75th percentile in adulthood: -/ 74 BMI z-score ≥75th percentile in both childhood and adulthood: -/ 61	≥ 1 CVD risk factors BMI z-score <75th percentile in both childhood and adulthood: Ref. BMI z-score <75th in childhood and ≥ 75th percentile in adulthood: 1.80 (1.58-2.06) BMI z-score ≥75th in childhood and < 75th percentile in adulthood: 1.10 (0.87-1.38) BMI z-score ≥75th percentile in both childhood and adulthood: 1.94 (1.71-2.19)	Sex, elevated blood pressure, age in childhood, follow-up duration, adult drinking, and smoking	Combined CVD risk factors; Limited data for meta-analysis

													≥ 2 CVD risk factors BMI z-score <75th percentile in both childhood and adulthood: Ref. BMI z-score <75th in childhood and ≥ 75th percentile in adulthood: 2.57 (2.03-3.25) BMI z-score ≥75th in childhood and < 75th percentile in adulthood: 0.94 (0.60-1.48) BMI z-score ≥75th percentile in both childhood and adulthood: 3.00 (2.36-3.80) ≥3 CVD risk factors BMI z-score <75th percentile in both childhood and adulthood: Ref. BMI z-score <75th in childhood and ≥ 75th percentile in adulthood: 6.17 (4.12-9.23) BMI z-score ≥75th in childhood and < 75th percentile in adulthood: 0.77 (0.32-1.86) BMI z-score ≥75th percentile in both childhood and adulthood: 5.31 (3.48-8.08)

AUCi, increment of area under the curve; AUCt, total of area under the curve; BMI, body mass index; DBP, diastolic blood pressure; FPG, fasting plasma glucose; HR, hazard ratio; HbA1c, glycosylated hemoglobin; HDL-C, high-density lipoprotein cholesterol; IOTF, International Obesity Task Force; LVMI, left ventricular mass index; LVH, left ventricular hypotrophy; NAFLD: non-alcoholic fatty liver disease; OR: odds ratio; RR: risk ratio; SBP, systolic blood pressure; TC, total cholesterol; US CDC, US Center for Disease Control and Prevention; WC, waist circumference; WGOC, China Obesity Task Group; 2HPG, 2 hours postprandial blood glucose.

Table S5 Subgroup analysis of the effect of weight change from childhood (or adolescence) to adulthood on type 2 diabetes, hypertension, high cIMT, and CVD

Outcomes in adulthood	Weight change from childhood (or adolescence) to adulthood	OR	95%CI	I^2 (%)	$P_{heterogeneity}$	Statistical model
Type 2 diabetes						
3 articles (n=106,596) [41, 42, 58]	Weight change pattern 1					
	Normal→ Normal	Ref.				
	Overweight (or obesity)→ Normal	1.31	1.01-1.70	71.7	0.014	Random
	Normal→ Overweight (or obesity)	3.03	2.26-4.06	83.8	<0.001	Random
	Overweight (or obesity) → Overweight (or obesity)	3.52	2.79-4.43	71.4	0.007	Random
2 articles (n=17,775) [38, 39]	Weight change pattern 2					
	Normal→ Normal	Ref.				
	Overweight (or obesity)→ Normal	1.28	0.52-3.16	0.0	0.960	Fixed
	Normal→ Obesity	4.91	3.57-6.74	0.0	0.550	Fixed
	Overweight (or obesity)→ Obesity	7.99	3.49-18.29	77.3	0.036	Random
3 articles (n=19,503) [37, 40, 58]	Weight change pattern 3					
	Normal→ Normal	Ref.				
	Obesity→ Normal	2.13	1.29-3.51	40.9	0.184	Fixed
	Normal→ Obesity	2.13	1.40-3.26	61.6	0.050	Random
	Obesity→ Obesity	2.84	2.13-3.80	45.5	0.160	Fixed
2 articles (n=74,012) [39, 41]	Childhood to adulthood					
	Normal→ Normal	Ref.				
	Overweight (or obesity) →Normal	0.99	0.79-1.25	0.0	1.000	Fixed
	Normal→ Excess	3.99	2.41-6.59	77.4	0.036	Random
	Overweight (or obesity) → Excess	2.69	2.05-3.53	36.7	0.209	Fixed
2 articles (n=74,012)[39, 41]	Adolescence to adulthood					
	Normal→ Normal	Ref.				
	Overweight (or obesity) → Normal	1.50	0.91-2.49	31.5	0.227	Fixed
	Normal→ Excess	3.99	2.41-6.59	77.4	0.036	Random
	Overweight (or obesity) → Excess	4.65	2.83-7.66	64.8	0.092	Random

Hypertension,						
3 articles (n=26,318) [38, 39, 64]						
	Normal→ Normal					
	Overweight (or obesity)→ Normal	1.20	0.76-1.90	62.9	0.068	Random
	Normal→ Obesity	2.66	1. 81-3.89	88.1	<0.001	Random
	Overweight (or obesity)→ Obesity	3.33	2.09-5.30	86.8	0.001	Random
High cIMT,						
3 articles (n=8,342) [32, 38, 47]						
	Normal→ Normal	Ref.				
	Overweight (or obesity)→ Normal	1.36	0.73-2.53	55.0	0.108	Random
	Normal→ Obesity	1.93	1.21-3.08	81.8	0.004	Random
	Overweight (or obesity)→ Obesity	2.18	1.30-3.65	79.2	0.008	Random
CVD						
2 articles (n=75,339) [49, 50]	Weight change pattern 1					
	Normal→ Normal	Ref.				
	Overweight (or obesity)→ Normal	1.16	0.86-1.55	0.0	0.898	Fixed
	Normal→ Overweight (or obesity)	2.21	1.81-2.70	85.1	0.010	Random
	Overweight (or obesity)→ Overweight (or obesity)	2.16	1.31-3.56	69.2	0.072	Random
2 articles (n=12,217) [39, 48]	Weight change pattern 2					
	Normal→ Normal	Ref.				
	Overweight (or obesity)→ Normal	3.02	0.89-10.27	0.0	0.843	Fixed
	Normal→ Obesity	3.95	2.20-7.10	0.0	0.841	Fixed
	Overweight (or obesity)→ Obesity	6.80	2.83-16.31	0.0	0.952	Fixed

Abbreviations: BMI, body mass index; cIMT, carotid intima-media thickness; CVD, cardiovascular disease; CI confidence interval; OR odds ratio.

Table S6. Meta-regression analysis for the potential sources of between-study heterogeneity

β (95%CI)	Weight change from childhood (or adolescence) to adulthood	Outcomes in adulthood				
		Type 2 diabetes, 6 articles(n=128,164) [37-42]	**Hypertension,** 4 articles (n=30,309) [37-39, 43]	**Dyslipidemia,** 3 articles (n=21,926) [22, 37, 38]	**High cIMT,** 3 articles (n=8,342) [32, 38, 47]	**CVD,** 4 articles (n=87,556) [39, 48-50]
Age in childhood	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	0.03 (-0.10, 0.16)	0.13 (-0.06, 0.34)	0.13 (-0.46, 0.72)	0.11 (-1.37, 1.58)	NA
	Normal→ Excess	-0.07 (-0.16, 0.01)	0.05 (-0.13, 0.24)	-0.08 (-1.13, 0.97)	0.10 (-0.29, 0.49)	0.12 (-0.64, 0.87)
	Excess→ Excess	-0.13 (-0.33, 0.07)	0.12 (0.02, 0.22)	0.03 (-0.96, 1.02)	0.10 (-1.04, 1.23)	0.22 (-0.66, 1.10)
Age in adulthood	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	-0.01 (-0.03, 0.02)	-0.05 (-0.14, 0.05)	-0.05 (-0.28, 0.17)	-0.04 (-0.67, 0.58)	NA
	Normal→ Excess	0.01 (-0.02, 0.05)	-0.02 (-0.08, 0.03)	0.04 (-0.26, 0.33)	0.02 (-0.56, 0.60)	0.02 (-0.07, 0.11)
	Excess→ Excess	0.01 (-0.05, 0.08)	-0.05 (-0.09, -0.01)	-0.01 (-0.39, 0.38)	-0.03 (-0.69, 0.63)	0.05 (-0.06, 0.16)
Sex (male proportion)	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	-0.01 (-0.02, 0.01)	0.02 (-0.26, 0.30)	0.05 (-0.42, 0.53)	-0.00 (-0.88, 0.87)	NA
	Normal→ Excess	0.00 (-0.02, 0.02)	0.05 (-0.04, 0.14)	-0.05 (-0.21, 0.10)	0.04 (-0.32, 0.41)	-0.01 (-0.05, 0.03)
	Excess→ Excess	-0.01 (-0.03, 0.02)	0.03 (-0.17, 0.22)	-0.01 (-0.48, 0.45)	0.01 (-0.75, 0.76)	-0.02 (-0.07, 0.02)
Sample size	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	-0.00 (-0.00, 0.00)	0.00 (-0.00, 0.00)	0.00 (-0.00, 0.00)	-0.00 (-0.00, 0.00)	NA
	Normal→ Excess	0.00 (-0.00, 0.00)	-0.00 (-0.00, 0.00)	-0.00 (-0.00, 0.00)	-0.00 (-0.00, 0.00)	-0.00 (-0.00, 0.00)
	Excess→ Excess	-0.00 (-0.00, 0.00)	0.00 (-0.00, 0.00)	0.00 (-0.00, 0.00)	-0.00 (-0.00, 0.00)	-0.00 (-0.00, 0.00)
Duration of follow-up	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	-0.01 (-0.05, 0.04)	-0.03 (-0.10, 0.04)	-0.04 (-0.19, 0.12)	-0.04 (-0.62, 0.53)	NA
	Normal→ Excess	0.02 (-0.01, 0.05)	-0.02 (-0.05, 0.02)	0.03 (-0.14, 0.19)	0.02 (-0.52, 0.56)	-0.05 (-0.34, 0.23)
	Excess→ Excess	0.04 (-0.02, 0.09)	-0.03 (-0.07, 0.01)	-0.00 (-0.27, 0.27)	-0.03 (-0.64, 0.59)	-0.1 (-0.41, 0.22)
Definition of overweight or obesity in	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.

childhood						
	Excess→ Normal	-0.01 (-0.23, 0.22)	-0.76 (-0.19, 0.36)	-0.19 (-2.09, 1.70)	0.30 (-1.52, 2.12)	-0.36 (-1.44, 0.72)
	Normal→ Excess	0.04 (-0.35, 0.43)	-0.41 (-0.87, 0.04)	0.21 (-0.39, 0.80)	0.07 (-2.86, 3.01)	-0.23 (-1.05, 0.59)
	Excess→ Excess	0.11 (-0.54, 0.75)	-0.65 (-1.19, -0.11)	0.06 (-1.62, 1.74)	0.29 (-0.74, 1.33)	-0.44 (-1.35, 0.46)
Definition of overweight or obesity in adulthood	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	-0.07 (-0.48, 0.34)	—	-0.18 (-3.56, 3.20)	0.30 (-1.52, 2.12)	-0.48 (-1.86, 0.90)
	Normal→ Excess	-0.04 (-0.52, 0.43)	—	0.28 (-0.56, 1.13)	0.07 (-2.86, 3.01)	-0.27 (-1.24, 0.71)
	Excess→ Excess	-0.19 (-0.93, 0.55)	—	0.13 (-1.62, 1.89)	0.29 (-0.74, 1.33)	-0.58 (-1.72, 0.57)
Definition of outcomes	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	-0.09 (-0.54, 0.36)	-0.59 (-2.34, 1.17)	-0.22 (-1.17, 0.74)	-0.05 (-0.38, 0.27)	-0.48 (-1.86, 0.90)
	Normal→ Excess	0.01 (-0.51, 0.54)	-0.02 (-1.43, 1.40)	0.14 (-1.35, 1.63)	-0.03 (-0.45, 0.40)	-0.27 (-1.24, 0.71)
	Excess→ Excess	-0.18 (-1.01, 0.66)	-0.29 (-1.95, 1.38)	-0.03 (-1.64, 1.58)	-0.05 (-0.23, 0.12)	-0.57 (-1.72, 0.57)
Adjusted covariates	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	0.15 (-0.47, 0.76)	0.51 (-1.64, 2.67)	0.66 (-2.21, 3.52)	0.81 (-4.07, 5.69)	0.11 (-2.75, 2.97)
	Normal→ Excess	-0.24 (-1.10, 0.63)	0.34 (-0.76, 1.44)	-0.42 (-4.90, 4.05)	0.38 (-5.94, 6.70)	0.08 (-1.77, 1.93)
	Excess→ Excess	-0.06 (-1.57, 1.44)	0.39 (-1.31, 2.10)	0.10 (-4.73, 4.93)	0.77 (-1.85, 3.39)	-0.02 (-2.96, 2.91)
Race/ethnicity	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	0.75 (-0.46, 1.96)	-0.20 (-3.6, 3.2)	—	0.81 (-4.07, 5.69)	—
	Normal→ Excess	-0.45 (-1.78, 0.89)	0.41 (-0.99, 1.80)	—	0.38 (-5.94, 6.70)	—
	Excess→ Excess	-0.02 (-2.03, 1.99)	0.10 (-2.35, 2.55)	—	0.77 (-1.85, 3.39)	—
Measurement of weight and height	Normal→ Normal	Ref.	Ref.	Ref.	Ref.	Ref.
	Excess→ Normal	-0.00 (-1.05, 1.04)	-0.81 (-1.94, 0.31)	-0.66 (-3.52, 2.21)	—	—
	Normal→ Excess	0.49 (-0.20, 1.19)	-0.24 (-1.59, 1.11)	0.42 (-4.05, 4.90)	—	—
	Excess→ Excess	0.75 (-0.71, 2,21)	-0.64 (-1.17, -0.10)	-0.10 (-4.93, 4.73)	—	—

Abbreviations: BMI, body mass index; cIMT, carotid intima-media thickness; CVD, cardiovascular disease; CI confidence interval; OR odds ratio;

NA, not available.

Notes Meta-regression analyses for metabolic syndrome outcomes in adulthood, as well as “Excess→ Normal” group for CVD outcome in adulthood, were not performed because there was no between-study heterogeneity.

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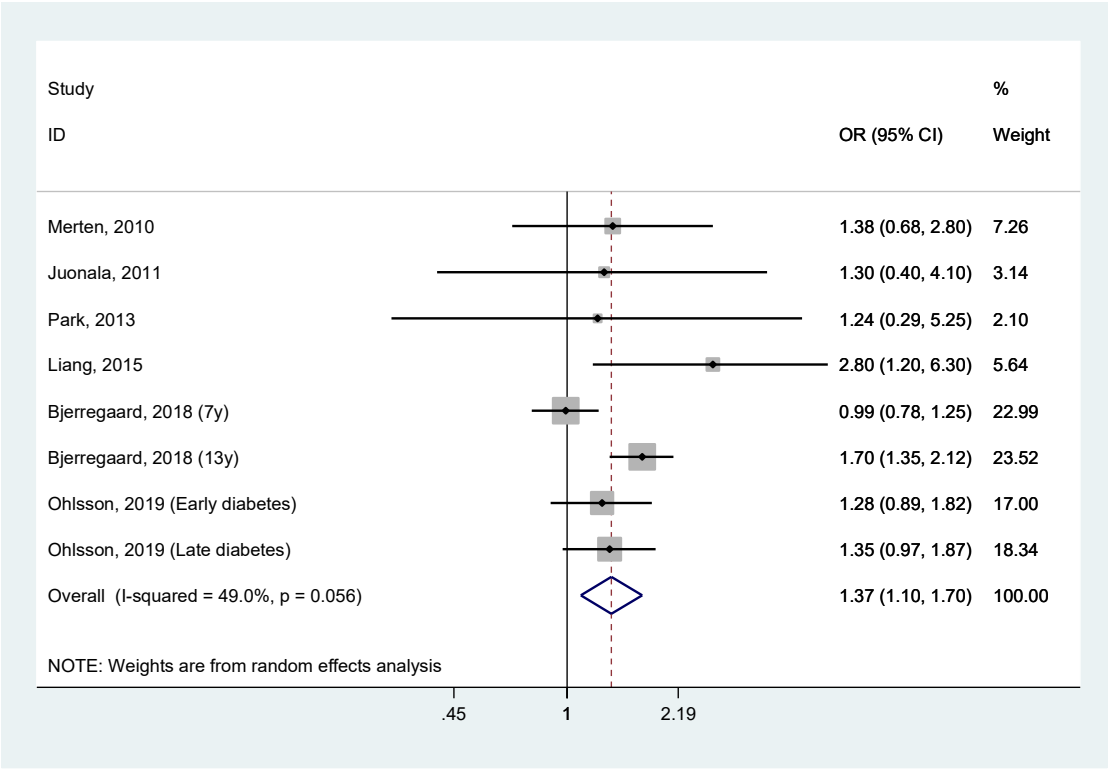
A. Excess→Normal



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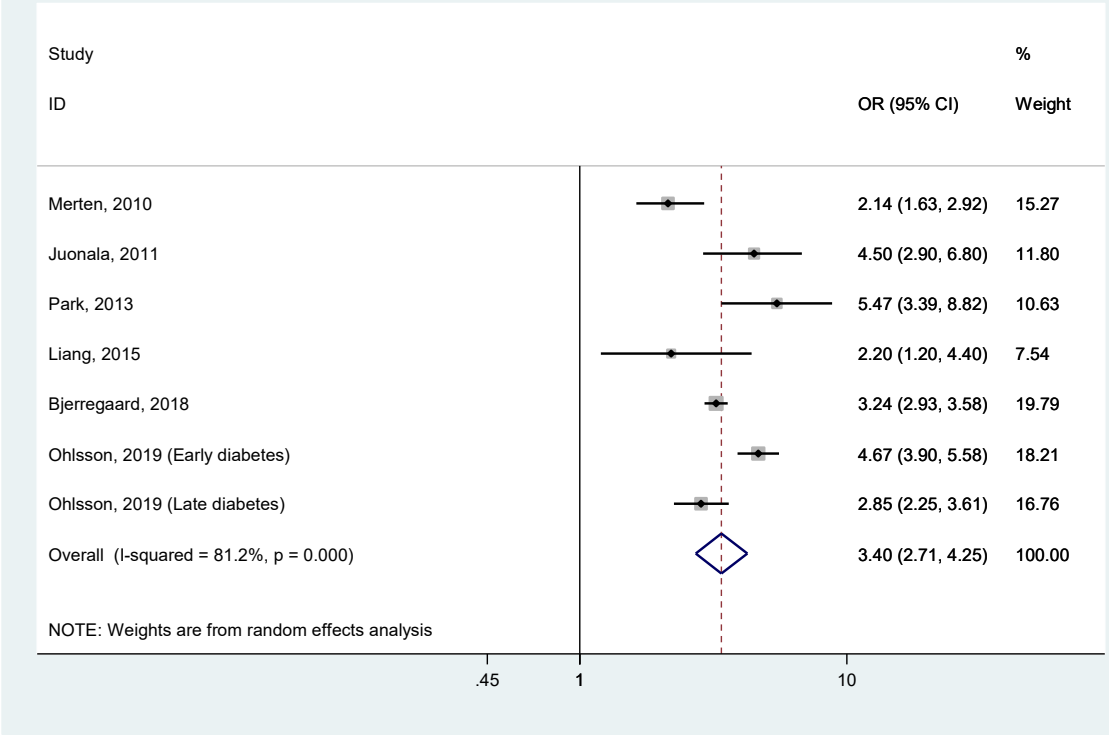

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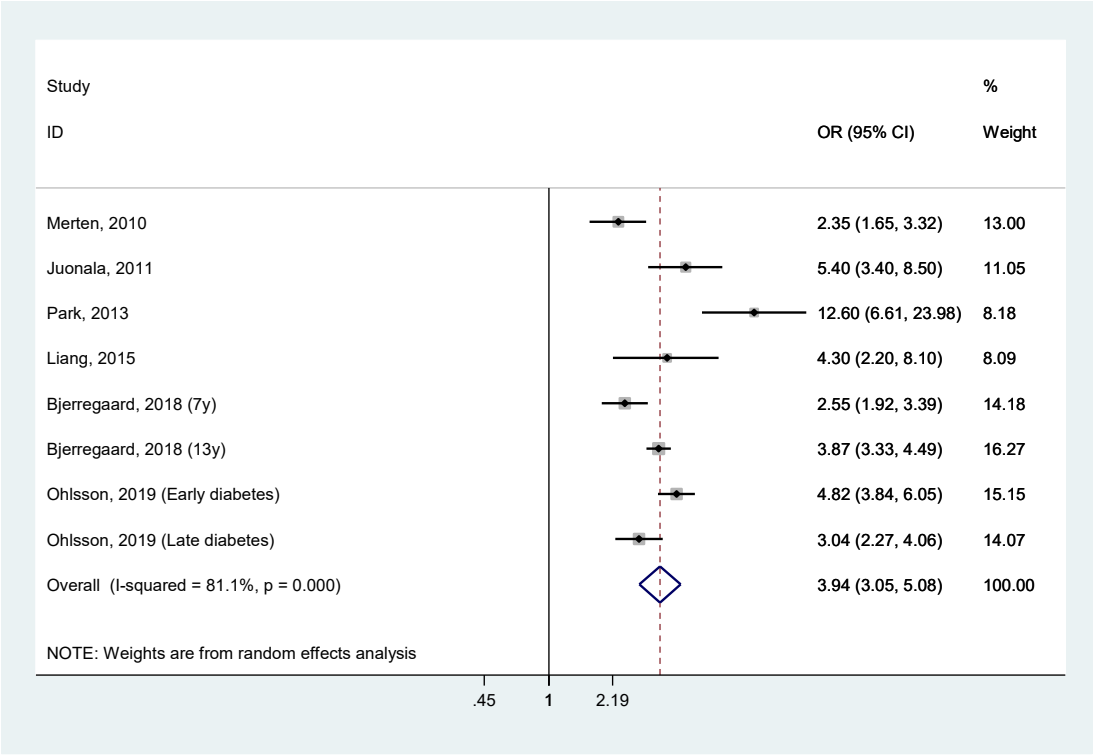
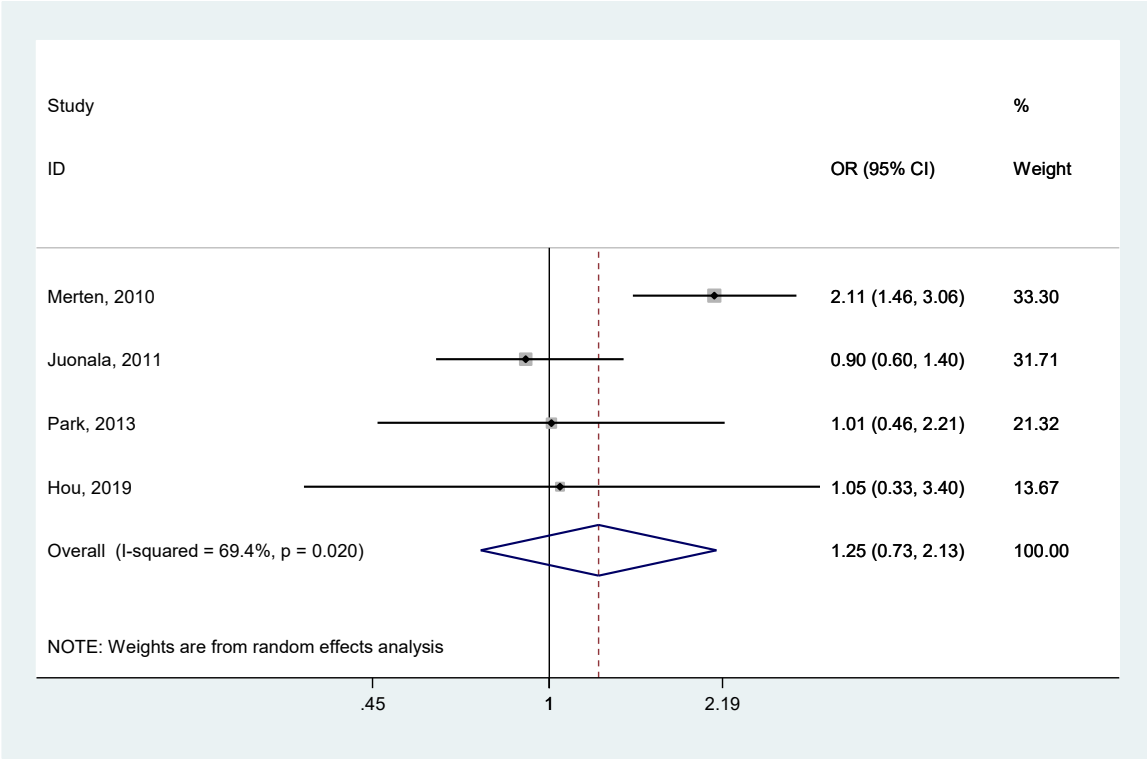


Figure S1. Meta-analysis of the association between weight status change from childhood to adulthood and type 2 diabetes in adulthood

A. Excess→Normal



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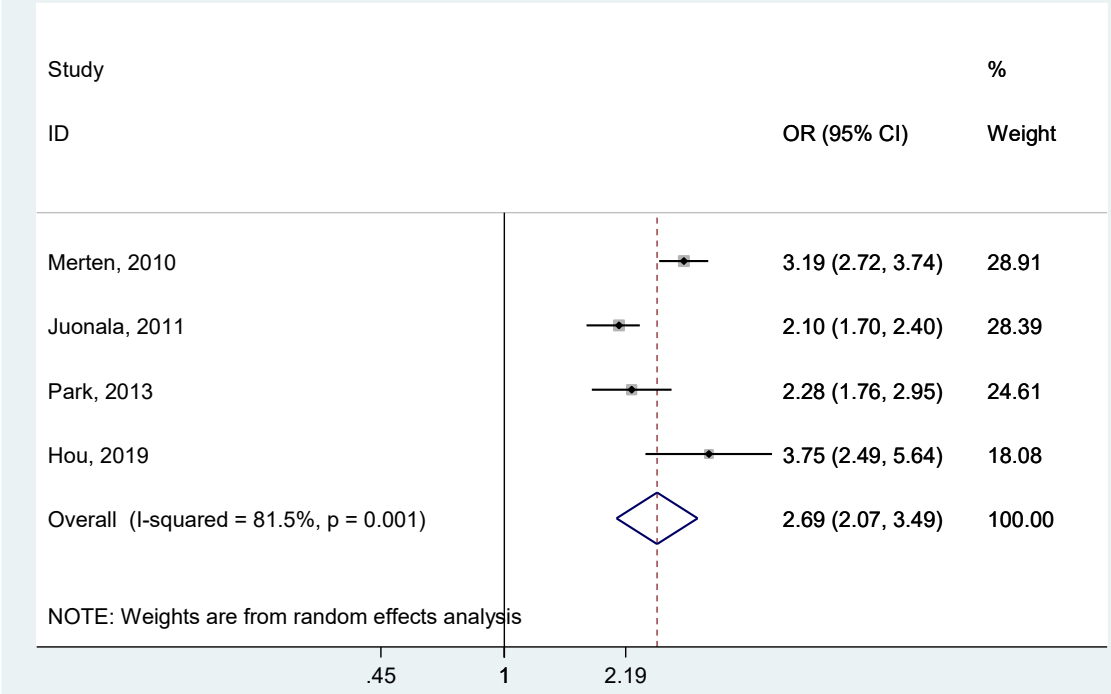

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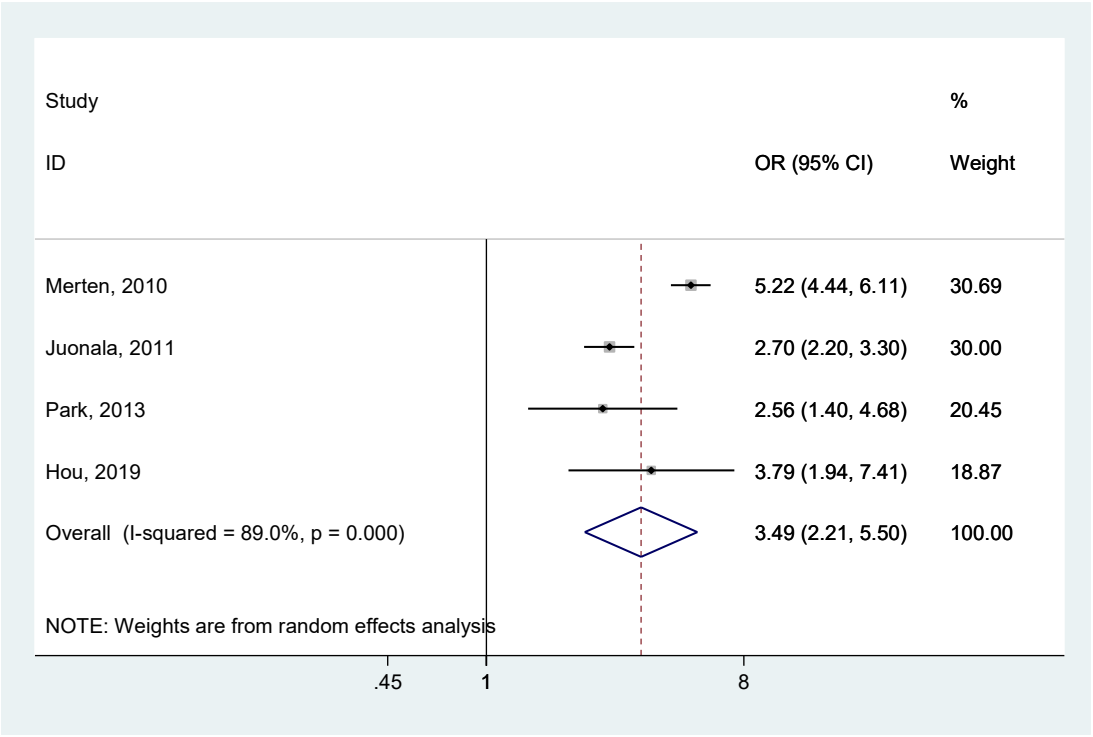
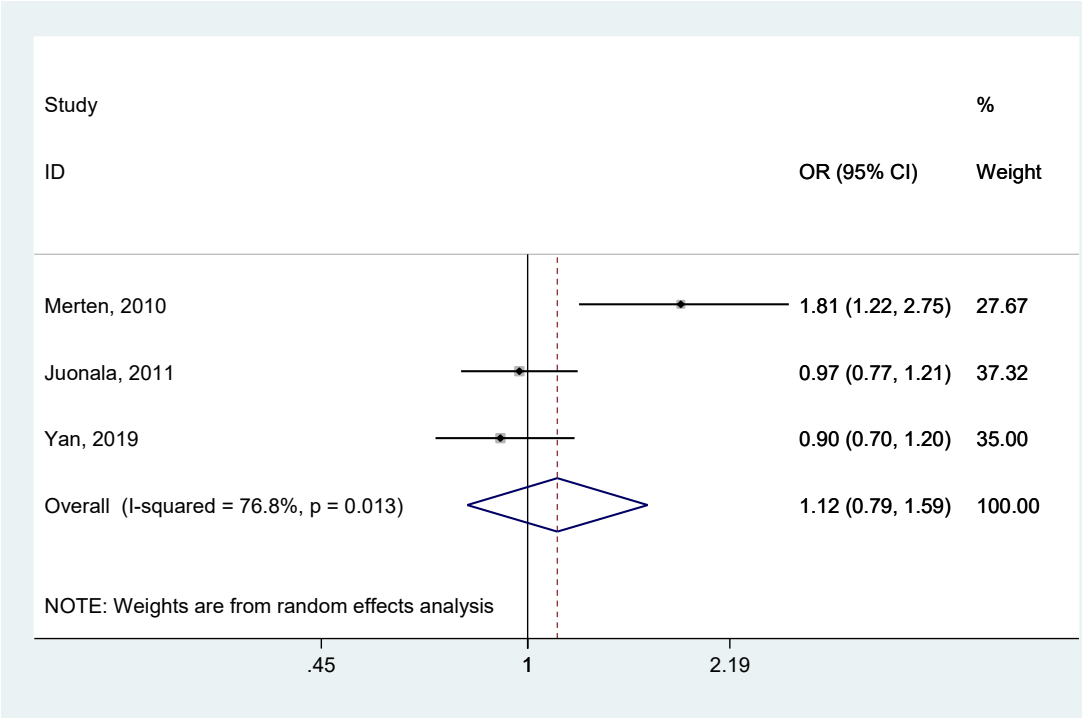


Figure S2. Meta-analysis of the association between weight status change from childhood to adulthood and hypertension in adulthood

A. Excess→Normal



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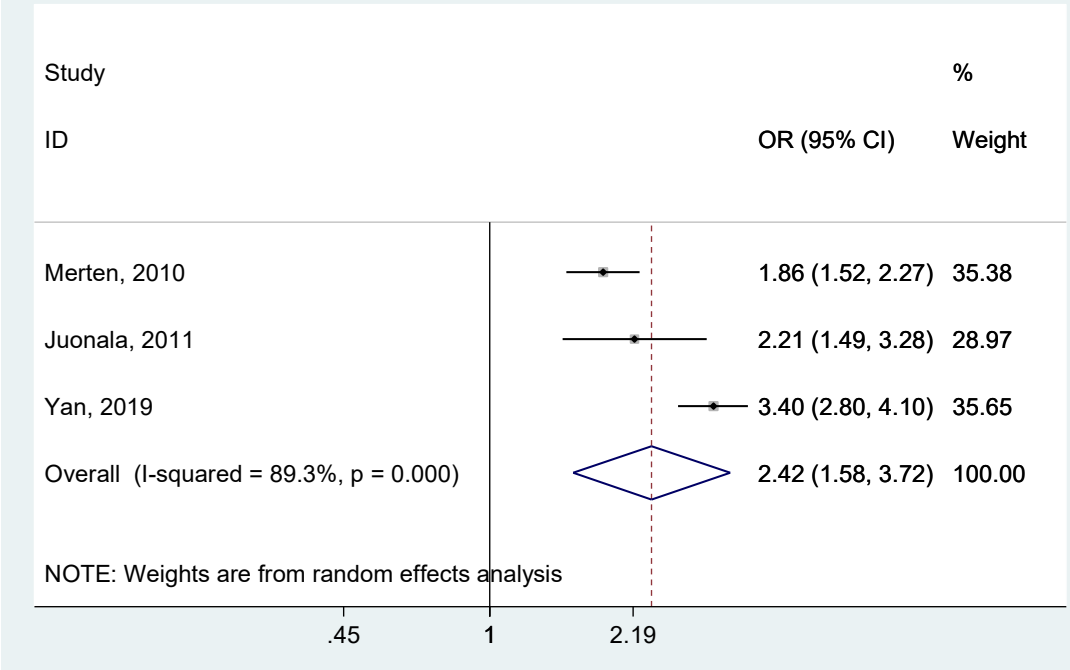

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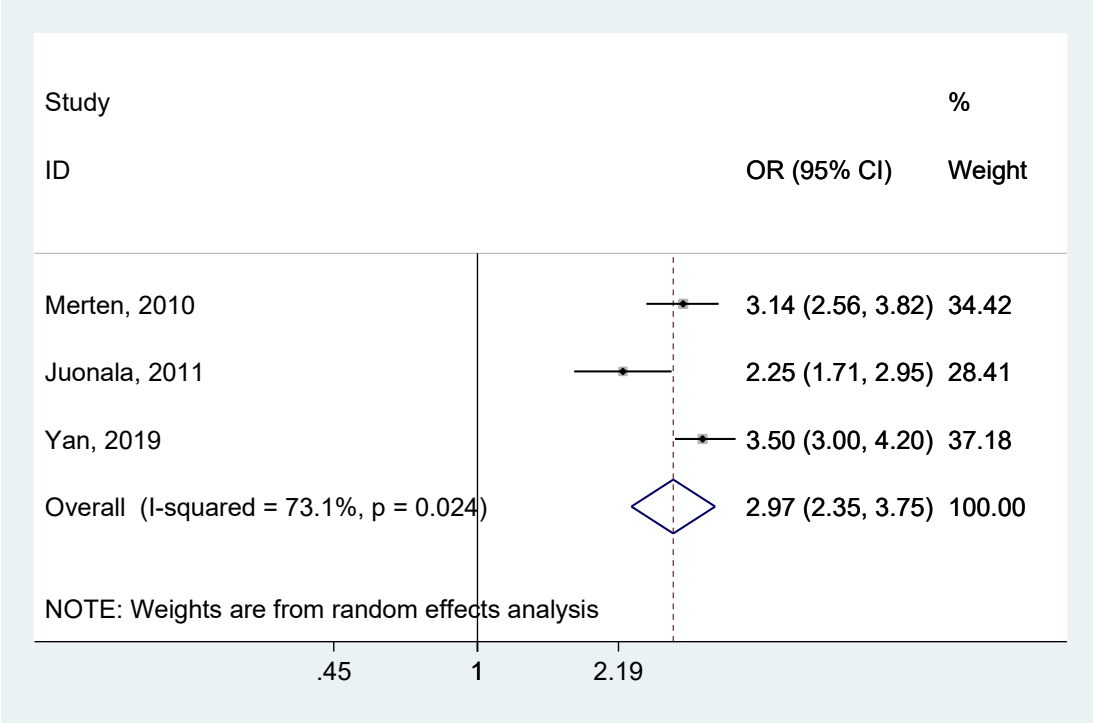
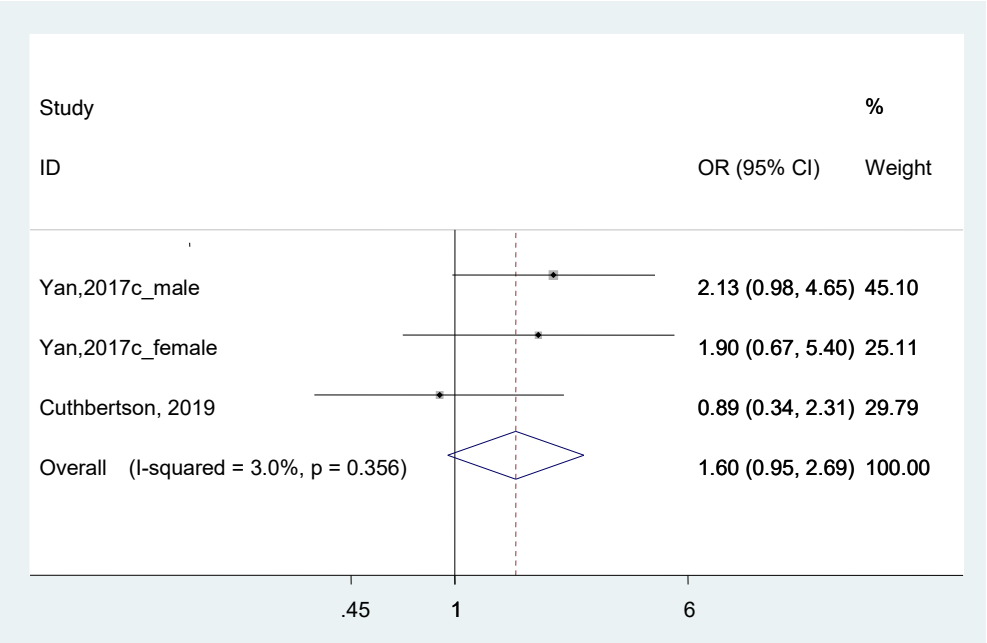


Figure S3. Meta-analysis of the association between weight status change from childhood to adulthood and dyslipidemia in adulthood

A. Excess→Normal



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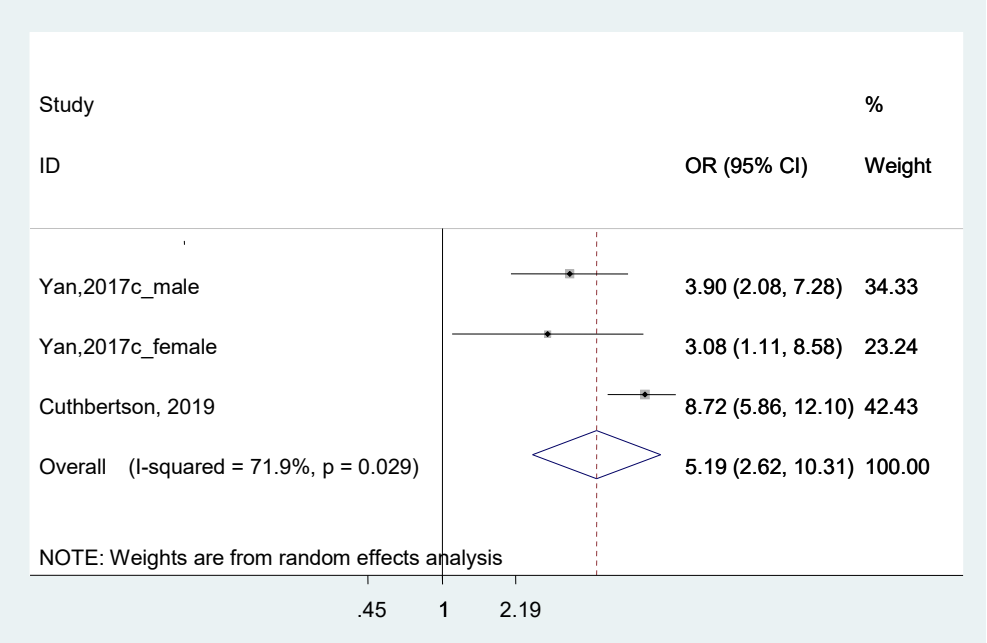

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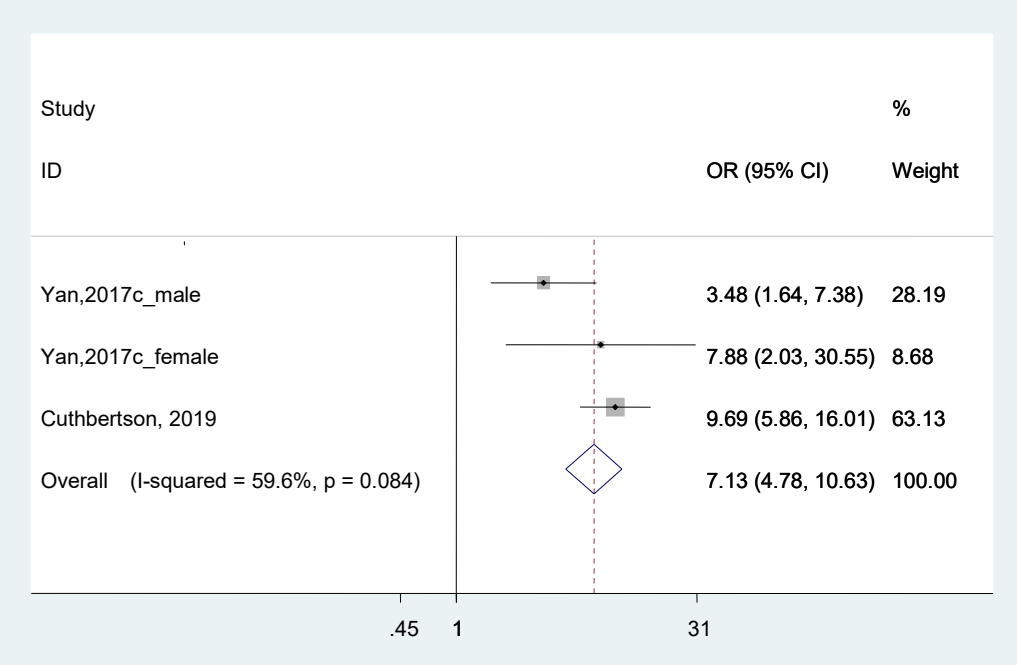
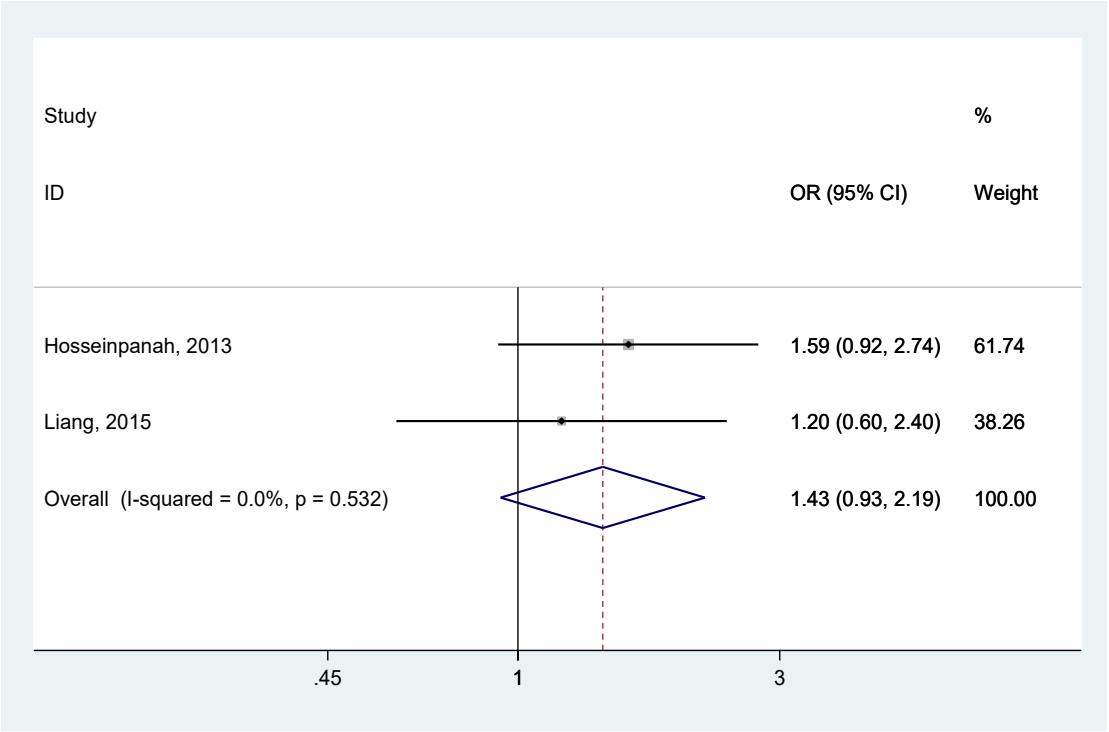


Figure S4. Meta-analysis of the association between weight status change from childhood to adulthood and non-alcoholic fatty liver disease

A. Excess→Normal



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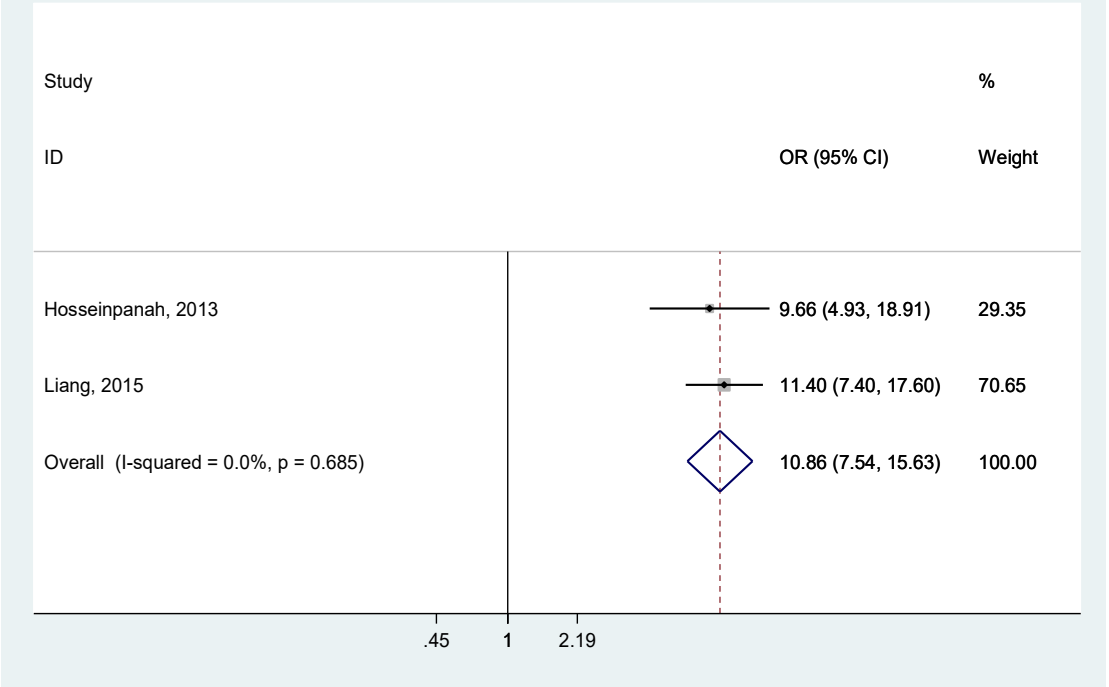

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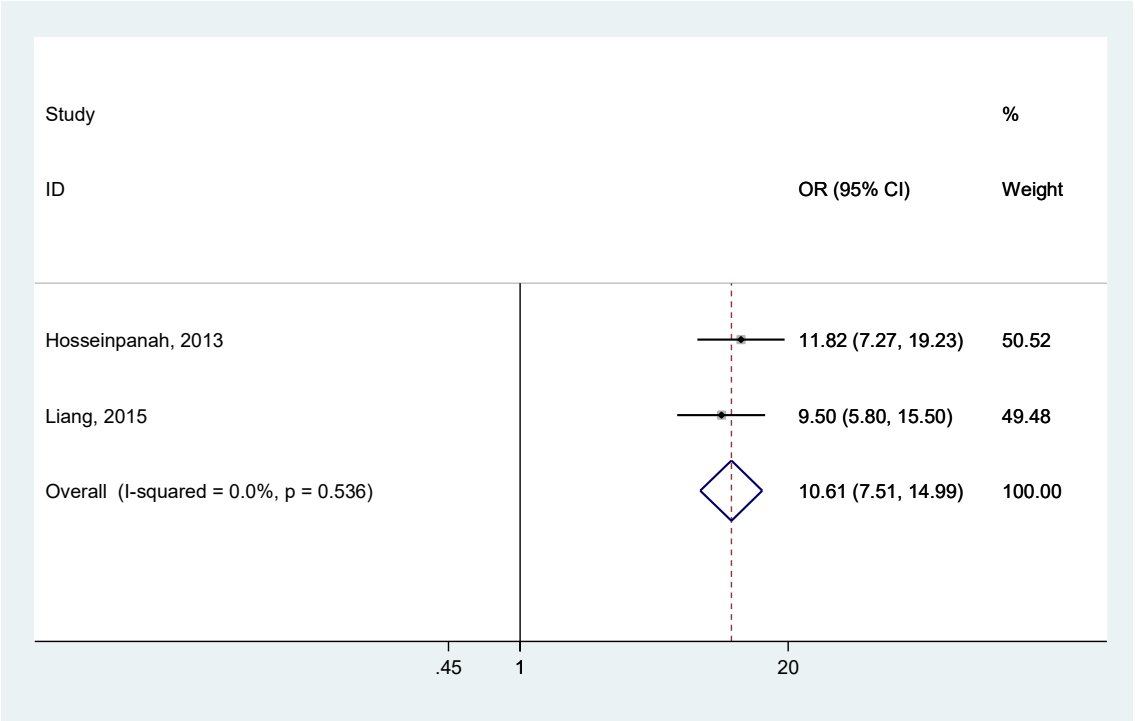
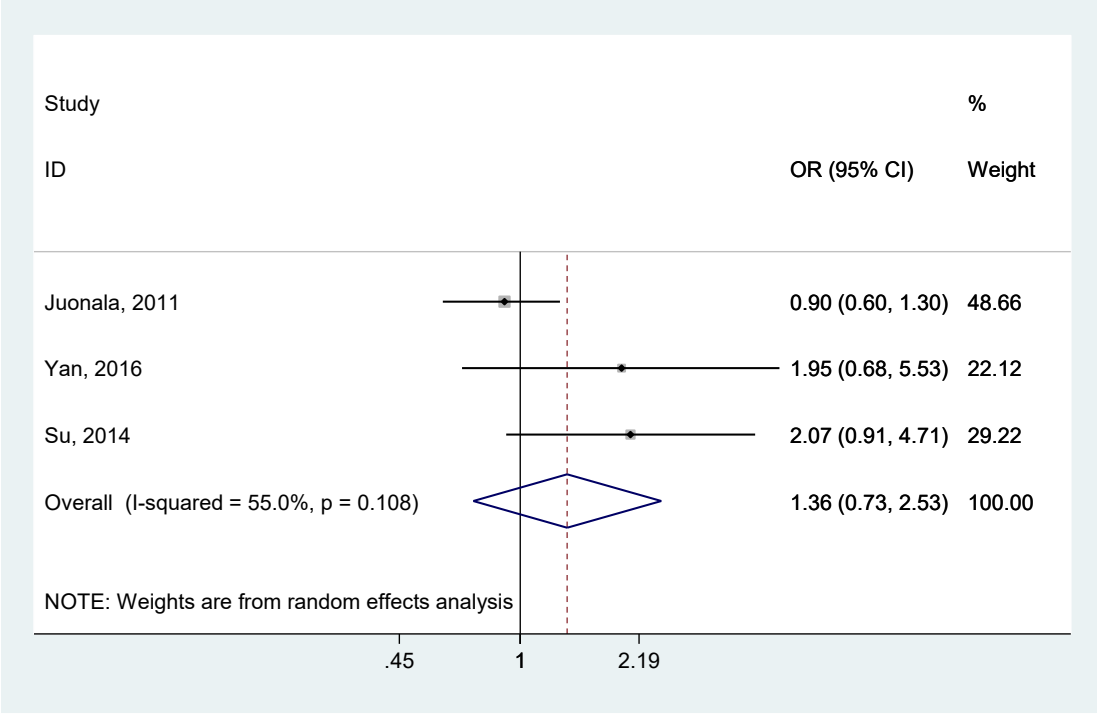
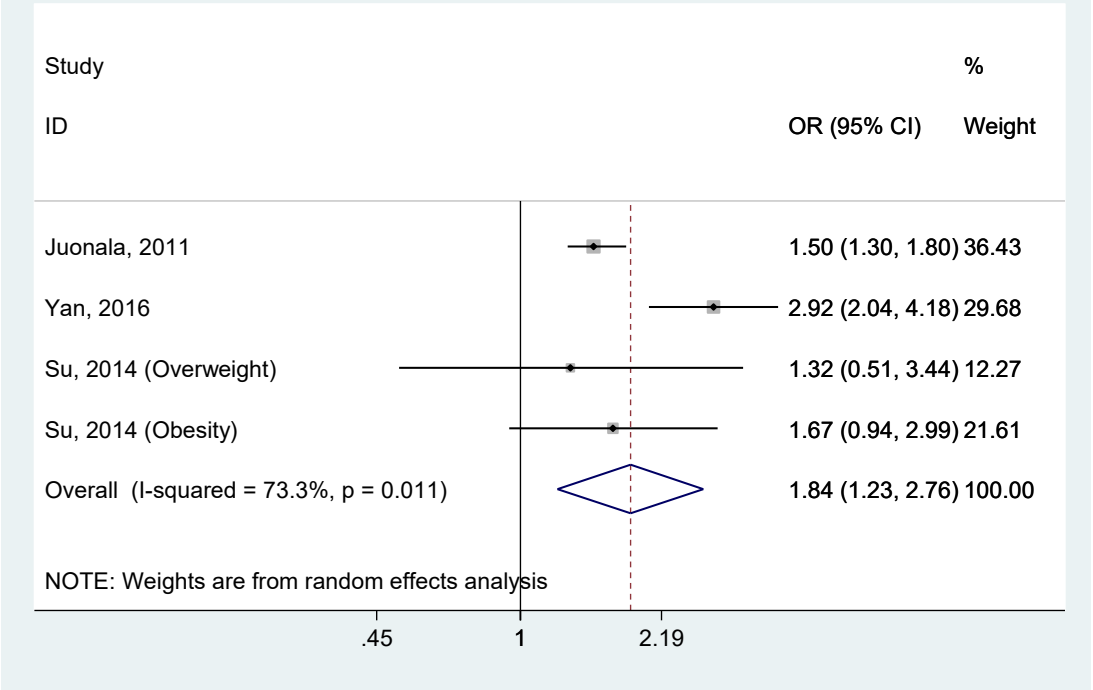


Figure S5. Meta-analysis of the association between weight status change from childhood to adulthood and metabolic syndrome in adulthood

A. Excess→Normal



B. Normal→Excess



C. Excess→Excess

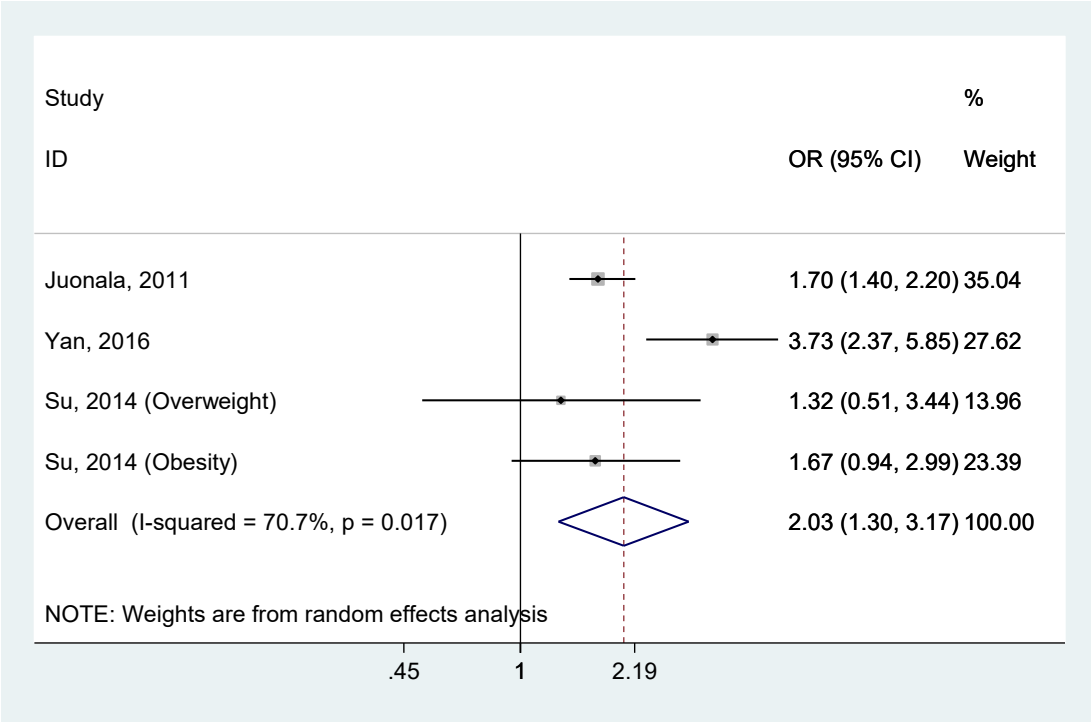
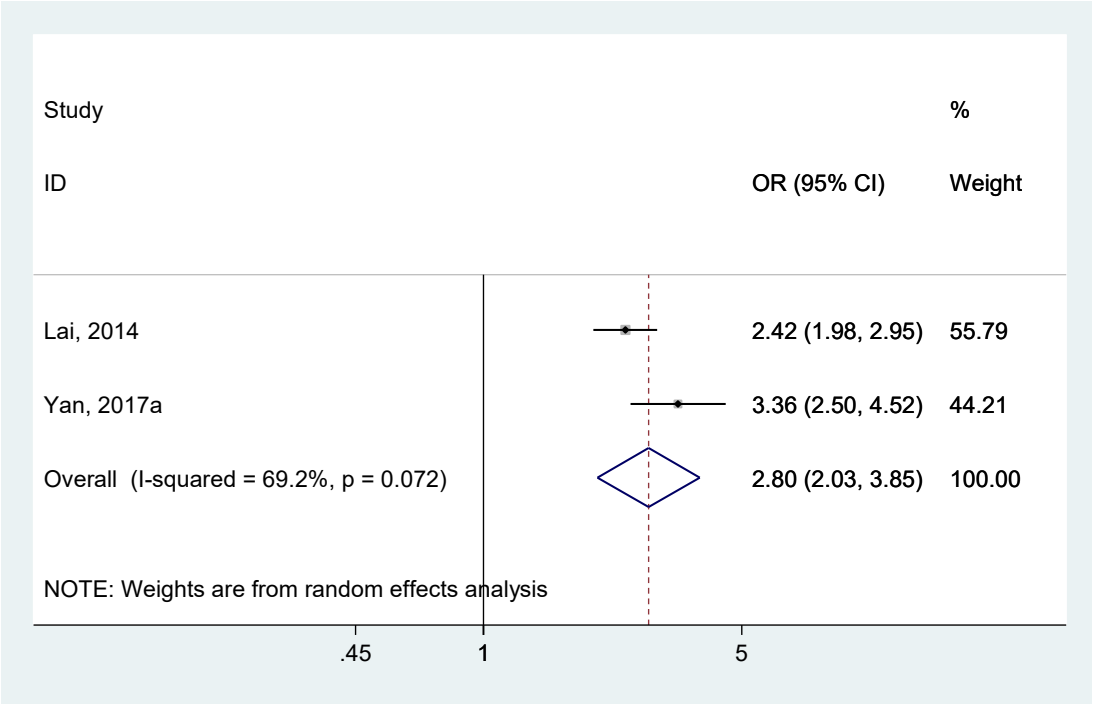


Figure S6. Meta-analysis of the association between weight status change from childhood to adulthood and high carotid intima-media thickness in adulthood

A. Total AUC



B. Increment of AUC

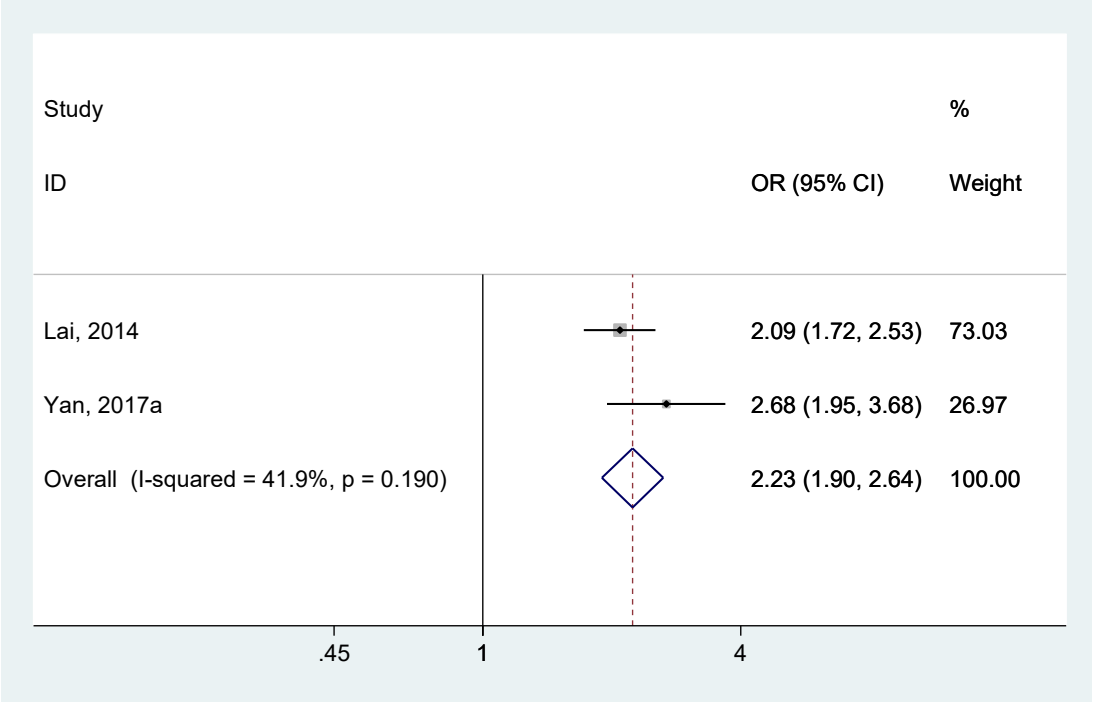
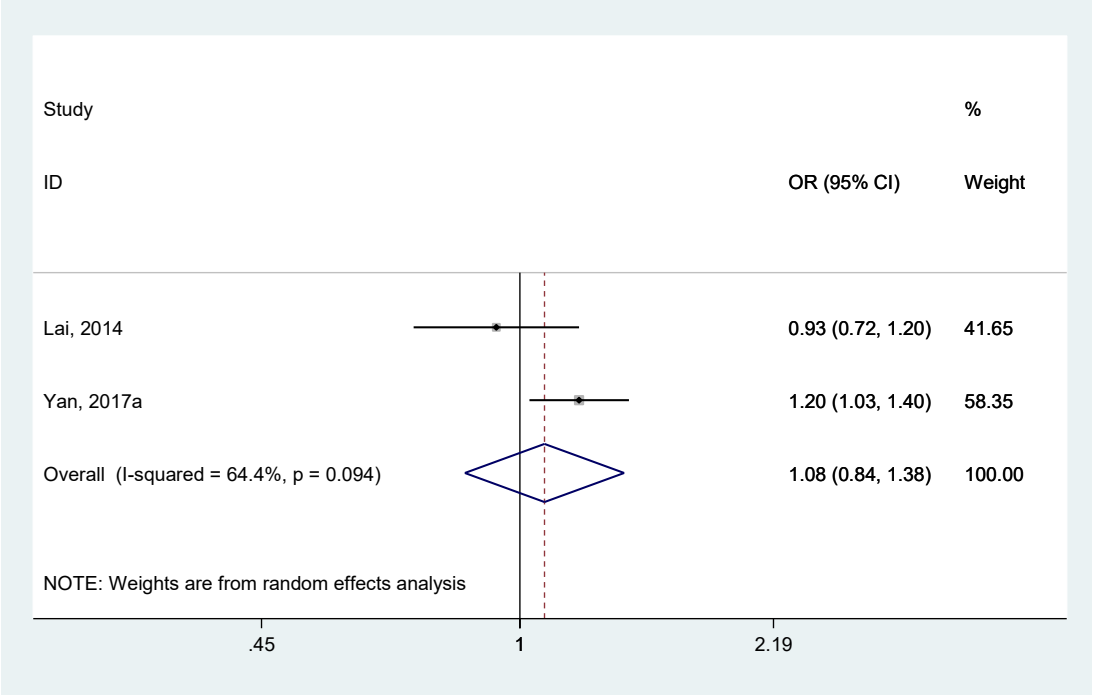


Figure S7. Meta-analysis of the association between the long-term burden of weight from childhood to adulthood and left ventricular hypertrophy in adulthood. AUC, area under the curve (cumulative values of body mass index)

Concentric remodeling

A. Total AUC



B. Increment of AUC

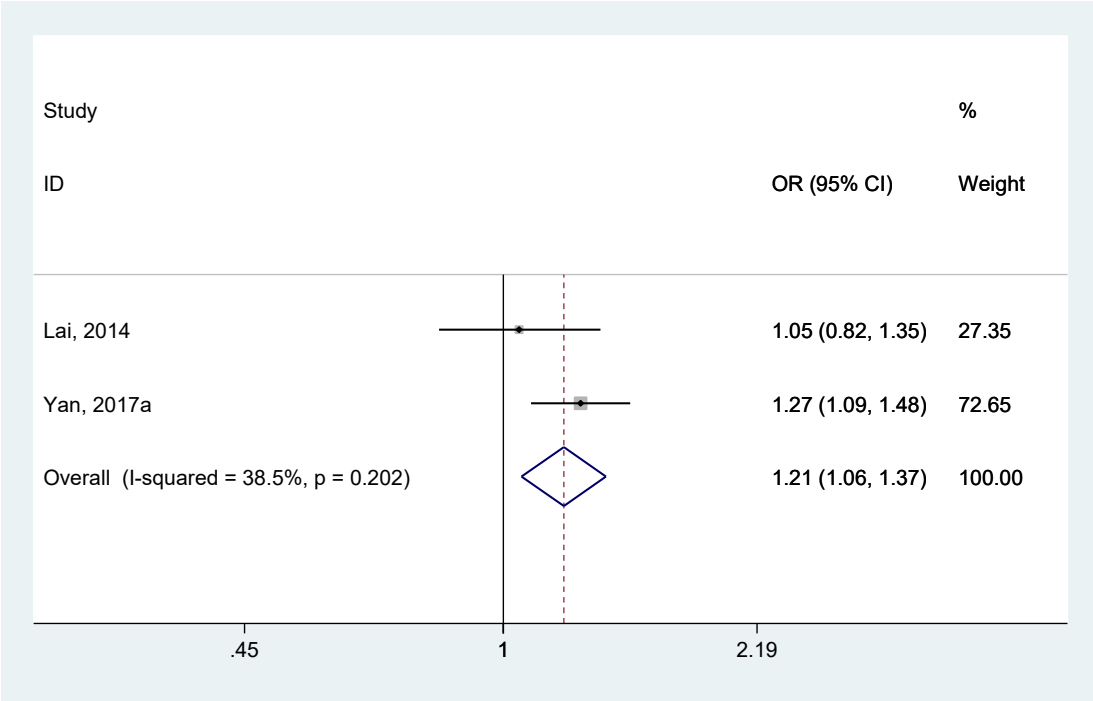
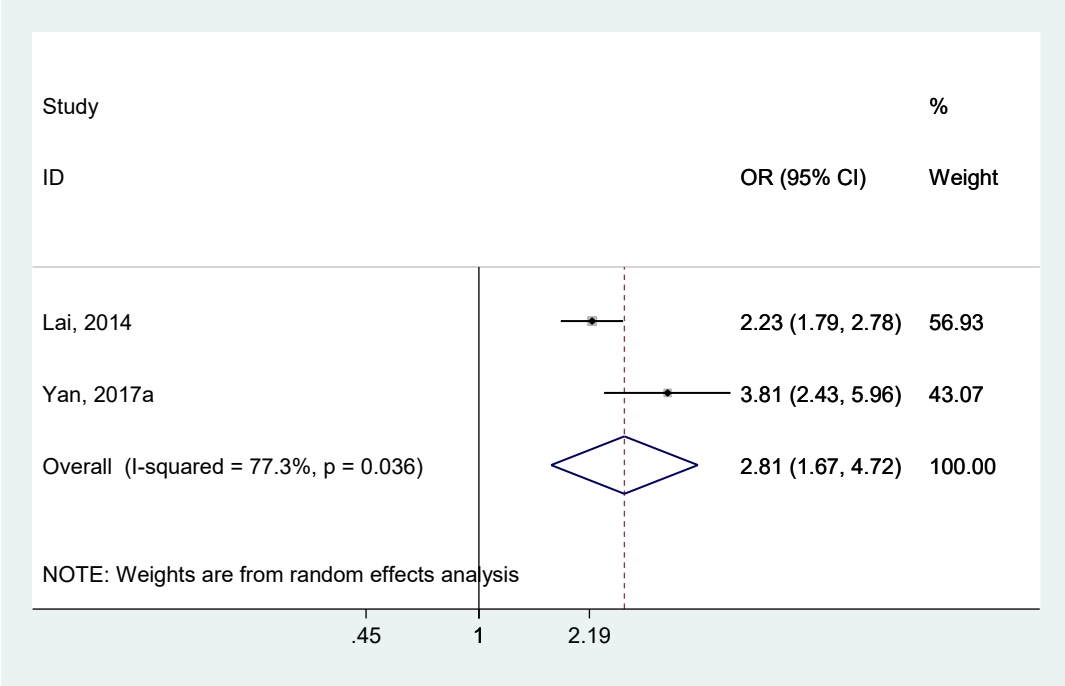


Figure S8. Meta-analysis of the association between the long-term burden of weight from childhood to adulthood and concentric remodeling in adulthood. AUC, area under the curve (cumulative values of body mass index)

Eccentric hypotrophy

A. Total AUC



B. Increment of AUC

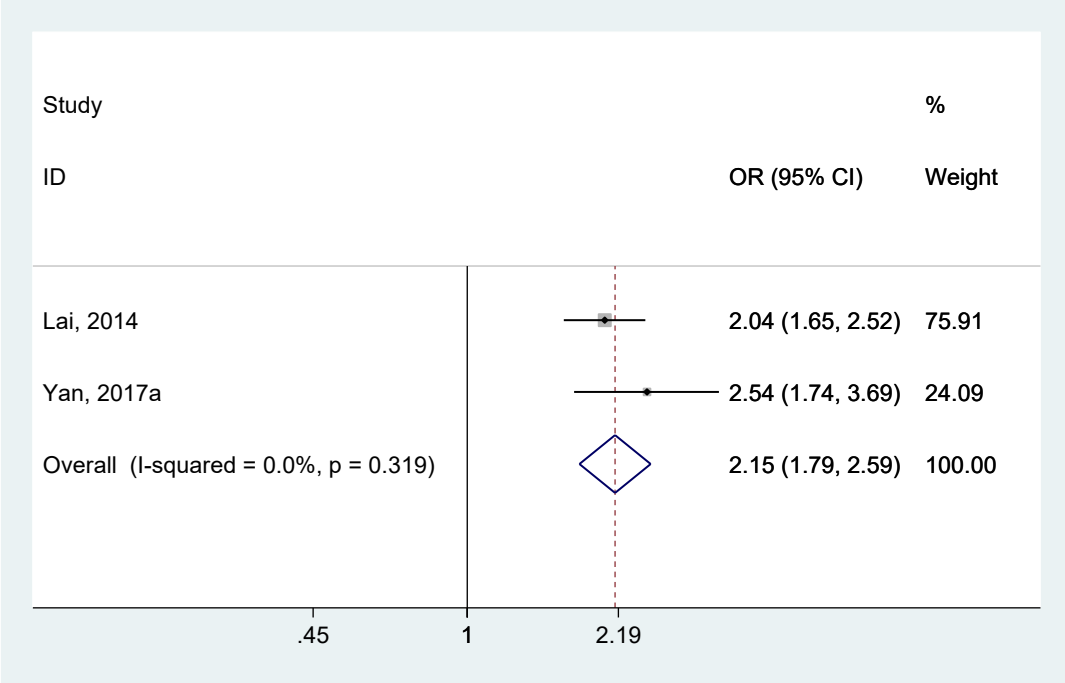
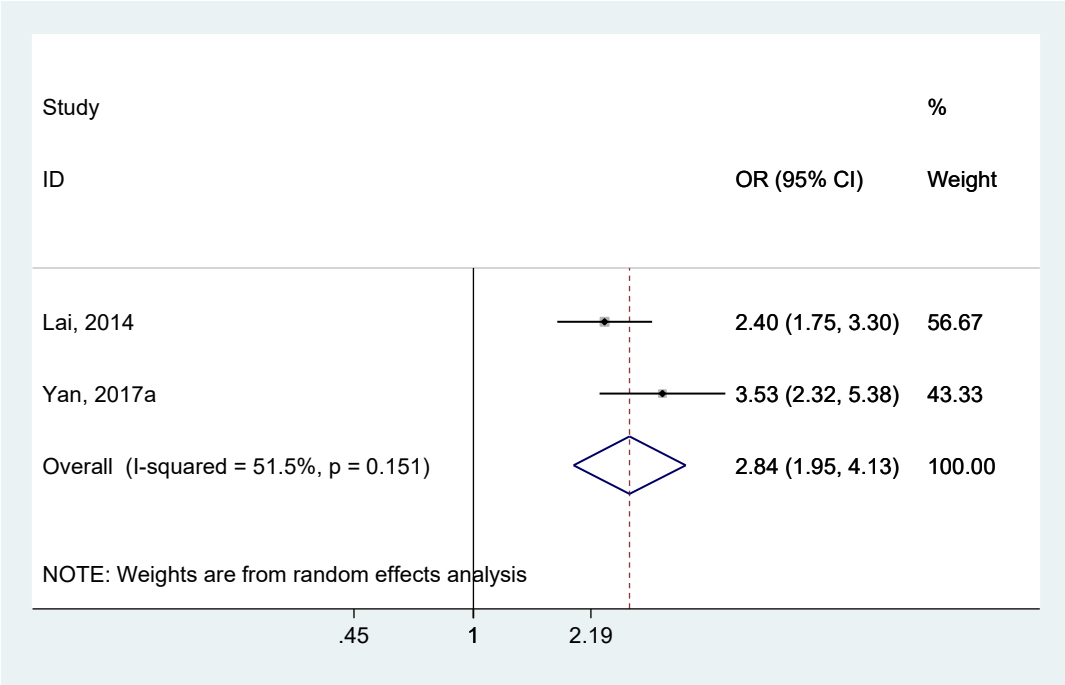


Figure S9. Meta-analysis of the association between the long-term burden of weight from childhood to adulthood and eccentric hypertrophy in adulthood. AUC, area under the curve (cumulative values of body mass index)

Concentric hypotrophy

A. Total AUC



B. Increment of AUC

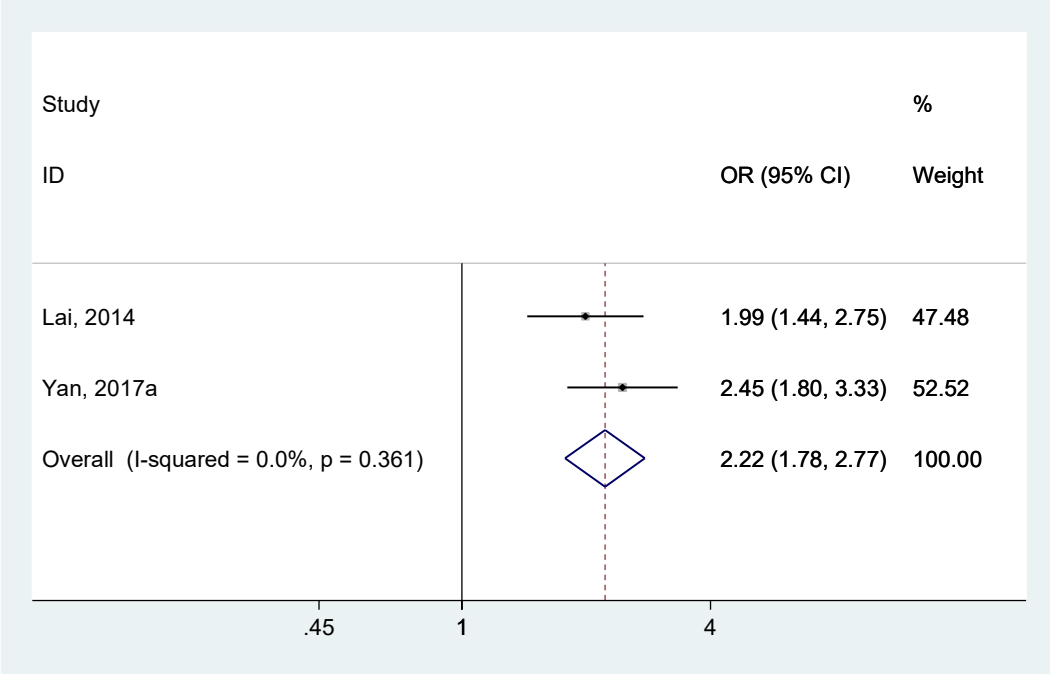
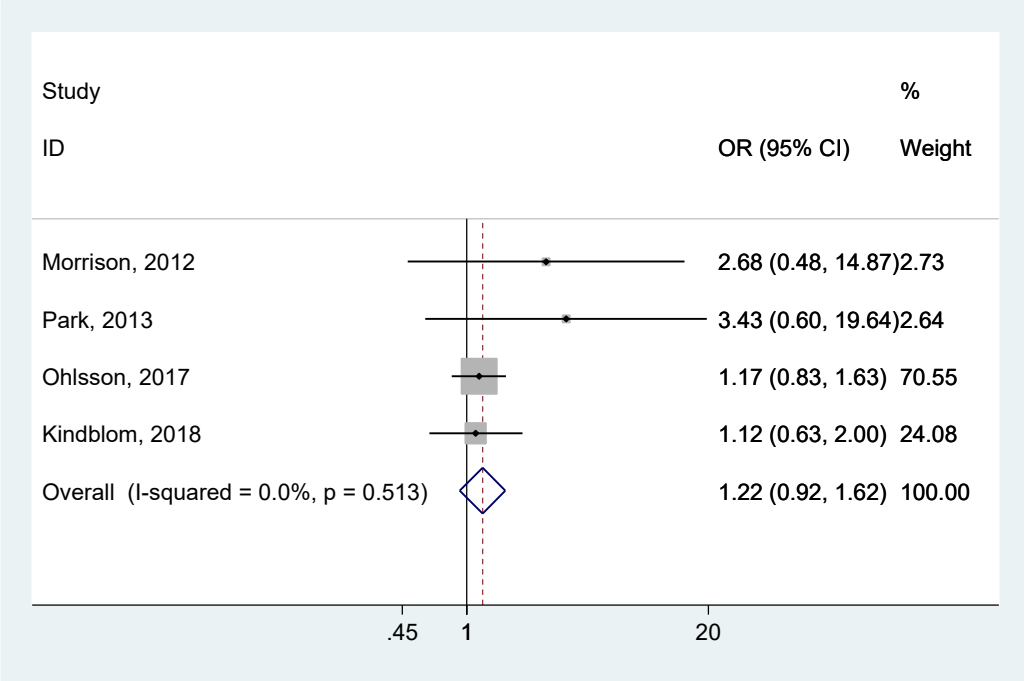
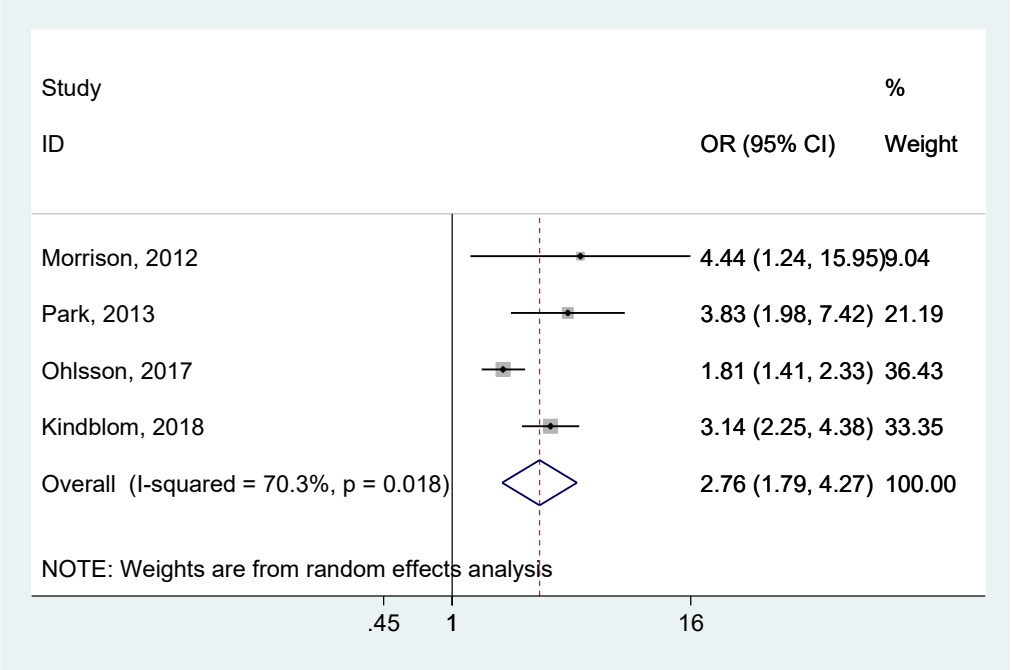


Figure S10. Meta-analysis of the association between the long-term burden of weight from childhood to adulthood and concentric hypertrophy in adulthood. AUC, area under the curve (cumulative values of body mass index)

A. Excess→Normal



B. Normal→Excess



C. Excess→Excess

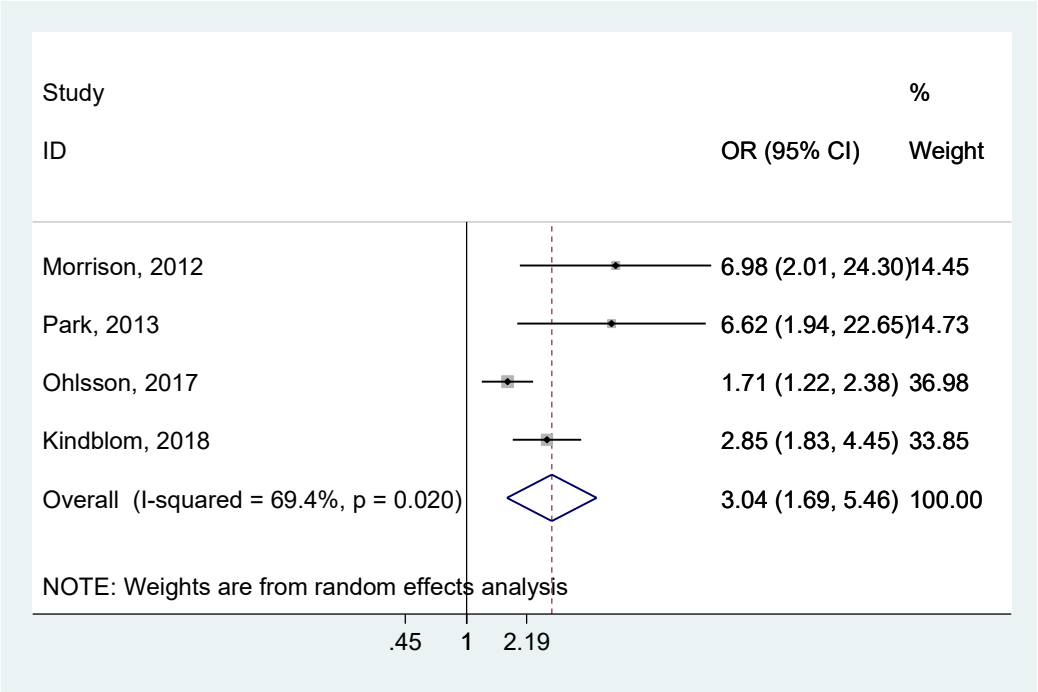


Figure S11. Meta-analysis of the association between the weight status change from childhood to adulthood and cardiovascular disease in adulthood

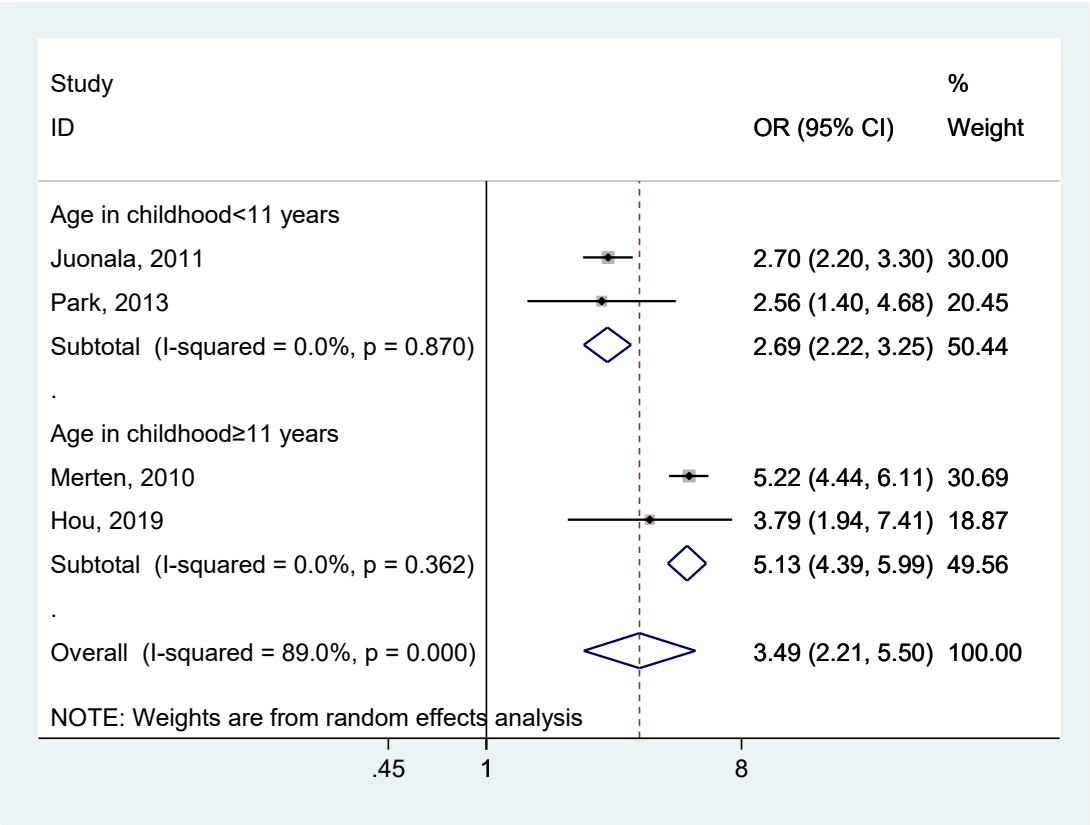


Figure S12. Subgroup analysis of the association between the persistent excess weight from childhood to adulthood and hypertension in adulthood stratified by age in childhood

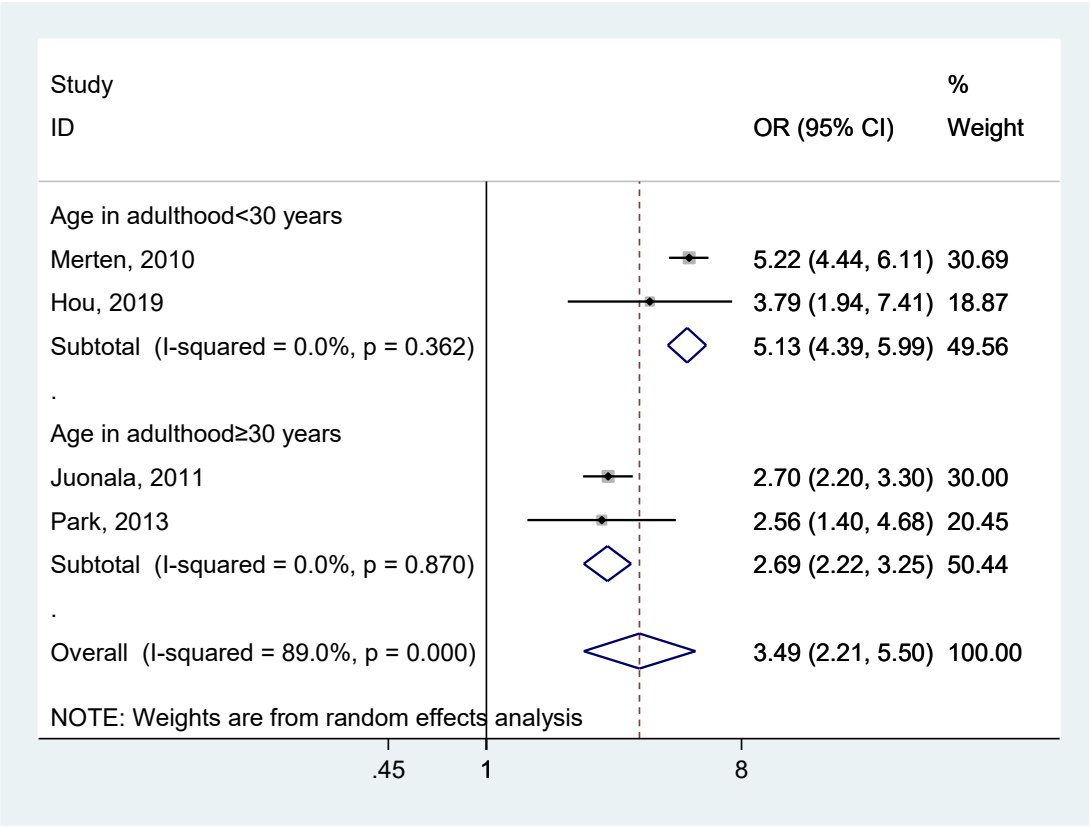


Figure S13. Subgroup analysis of the association between persistent excess weight from childhood to adulthood and hypertension in adulthood stratified by age in adulthood

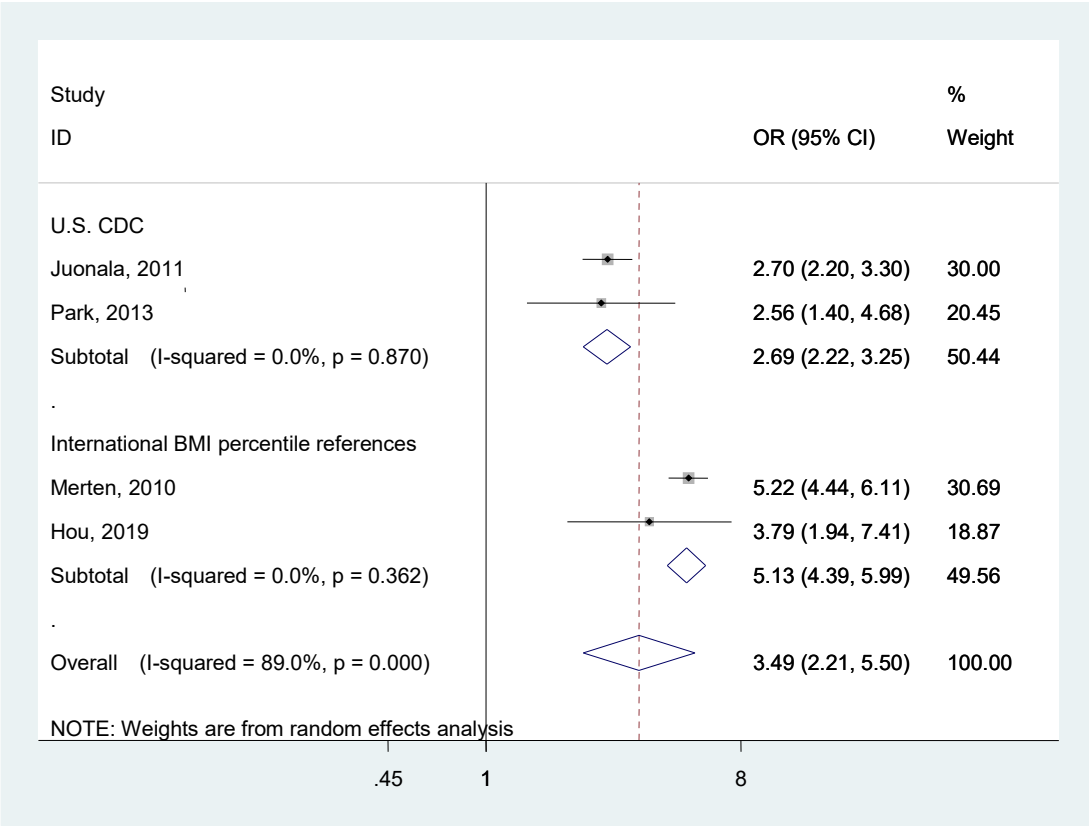


Figure S14. Subgroup analysis of the association between persistent excess weight from childhood to adulthood and hypertension in adulthood stratified by definition of overweight and obesity in childhood

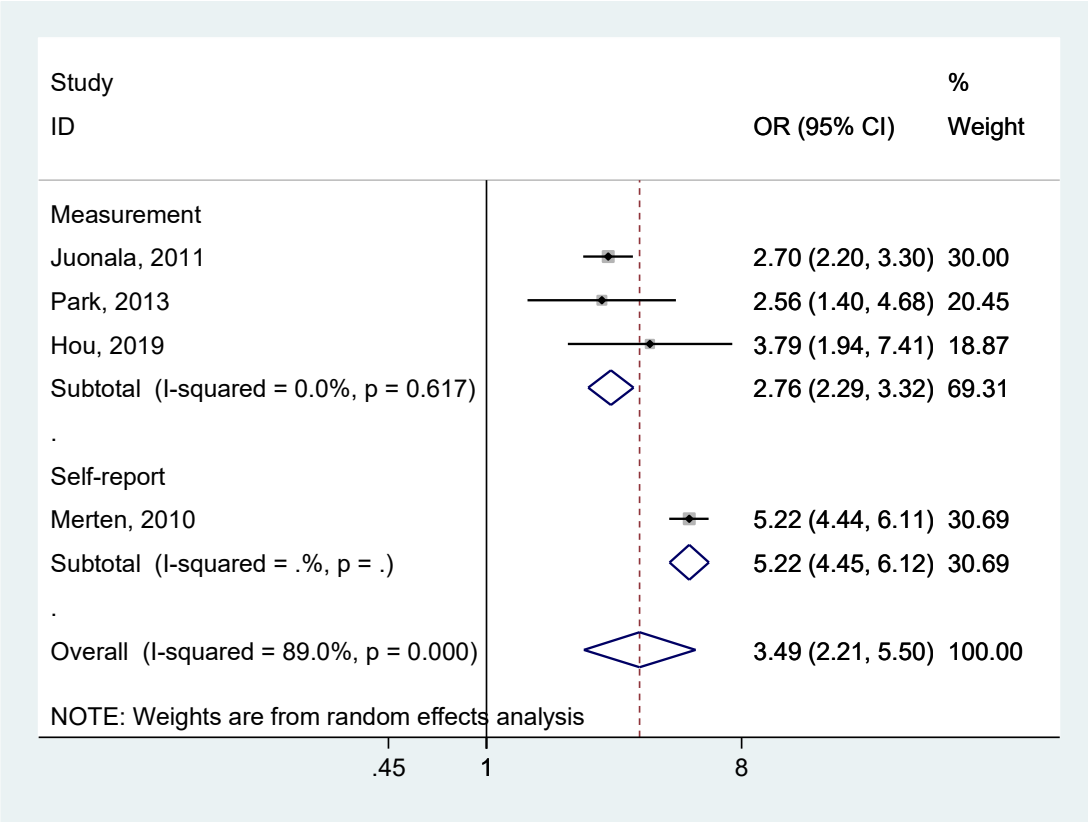


Figure S15. Subgroup analysis of the association between persistent excess weight from childhood to adulthood and hypertension in adulthood stratified by measurement of weight and height