

Childhood obesity as a predictor of type 2 diabetes mellitus in adults: a systematic review and meta-analysis

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Abstract

Background Despite government-developed prevention programs, type 2 diabetes mellitus (DM) has continued to increase, suggesting that the programs are ineffective. Other potential risk factors, such as childhood obesity, may influence adult-onset diabetes.

Objective To assess for a potential association between childhood obesity and adult type 2 DM by meta-analysis of the literature.

Methods This review was conducted according to the *PRISMA Statements' Flow Diagram and Checklist* to improve quality of reporting. Cohort studies were chosen for their long-term follow-up. *Newcastle-Ottawa Scale for Cohorts (NOS-Cohort)* was used to assess for bias and quality of the included studies, in addition to the *Cochrane Handbook*. Analysis was done with forest and funnel plots using *RevMan 5.3* software for *Macintosh*.

Results A total of 237 records with 73,533 participants were retrieved, of which 10 studies were included in our systematic review and 5 studies were included in the meta-analysis. The most common bias based on NOS-Cohort was inadequate follow-up. Forest plot revealed a significant association between childhood obesity and adult diabetes (OR 3.89; 95%CI 2.97 to 5.09; $P < 0.00001$). Individuals with childhood obesity were 3.89 times more likely to have adult-onset diabetes. Funnel plot assessment was symmetrical. Studies suggested that childhood obesity led to early insulin resistance and adiposity rebound, which promotes adulthood obesity, a diabetic risk factor.

Conclusion Childhood obesity can be used as a predictor for adult-onset diabetes. Early diabetes screening and prevention guidelines should include childhood obesity as a plausible risk factor. [Paediatr Indones. 2022;62:1020-9 DOI: 10.14238/pi62.1.2022.120-9].

Keywords: childhood obesity; adult diabetes mellitus type 2; predictor

Long acclaimed as a silent killer, type 2 diabetes mellitus (DM) is rising to the point of becoming a global emergency. This non-communicable disease, characterized by the body's insensitivity to insulin, impaired hormonal production by the pancreas, and resulting hyperglycemia, had become 9th leading cause of death worldwide, with a whopping 1.5 million deaths in 2019.¹ The WHO estimated that 422 million adults globally were burdened with diabetes in 2014, beyond double the prevalence recorded in 1980.² Data gathered across the world also showed a 5% rise in premature mortality from diabetes between 2000 and 2016.¹ This indisputable burden of type 2 DM is further corroborated by an increase in disability-adjusted life years, generated by diabetic complications such as nephropathy and neuropathy, of 49.7 million in 1990 to 64.1 million in 2015.³ Unfortunately, collateral damage on the economy was US\$966 billion in losses in 2021 according to the *International Diabetes Foundation*. With such economic fallout, type 2 DM is likely to impede national and global development if not handled effectively.⁴

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The epidemic surge in type 2 DM prevalence worldwide has led to the development of curative and preventive programs. The UN has set an ambitious goal to reduce premature mortality by one-third before 2030, as part of the *Sustainable Development Goals*.¹ Among the myriad of screening tools, obesity is often considered, as it is the greatest risk factor of type 2 DM, with over 90% of patients being obese or overweight.⁵ However, the seemingly unaffected escalation in type 2 DM prevalence suggests the ineffectiveness of these programs in curbing the disease, therefore, necessitating the modification of the focus of these programs to an earlier point of DM development. As such, many studies have been performed to assess the relationship between childhood obesity and adult-onset diabetes, and whether prevention of childhood obesity can reduce the risk of diabetes during adulthood.⁶⁻¹⁹

To our knowledge, this is the first systematic review and meta-analysis conducted to draw a sound and statistically significant conclusion on the link between childhood obesity and diabetes in adulthood by comparing the occurrence of type 2 DM across two variables: adults with and without childhood obesity. To ensure international applicability, studies from various countries on different continents were reviewed. Furthermore, retrospective and prospective cohort studies were specifically chosen, to allow for exploration of causative factors in type 2 DM, along with long term, follow-up studies.

Through these endeavors, we hope to increase public awareness on the importance of dealing with

childhood obesity along with its implication on future diabetes comorbidity. Furthermore, we hope that the results of this review can be implemented into the pre-existing, practical, evidence-based guidelines for early diabetes screening and prevention. We believe that by applying the objectives stated above, this review will stand in the forefront of new strategies to combat type 2 DM as a silent threat in the achievement of the global *Sustainable Development Goals* by 2030.

Methods

This systematic review and meta-analysis were conducted based on *PRISMA Statements' Flow Diagram and Checklist* to improve quality of reporting. The flow diagram had four phases, while the checklist consisted of 27 items pertaining to the content of systematic review and meta-analysis, including the title, abstract, introduction, methods, results, discussion, and funding.⁵

A range of databases, including *PubMed*, *Scopus*, *PLOS*, *Cochrane*, *Science Direct*, *Clinical Key*, *ProQuest*, and *Wiley*, was searched up to October 14, 2018. The search strategy was structured using the following concept of keywords, summarized in **Table 1**.

The concept was then modified, based on each database's boolean terms and conditions. Cohort studies were used for this review as their time-approach design was more appropriate for identifying and following-up the potential association between childhood obesity

Table 1. Database search strategy keywords

Database	Search keywords
PubMed	(((((adult-onset diabetes mellitus OR diabetes mellitus type 2 OR DM type 2 OR DM OR diabetes mellitus[MeSH Terms])) AND (abdominal obesity OR central obesity OR visceral obesity[MeSH Terms])) AND (children[Title/Abstract] OR child[Title/Abstract] OR pediatric[Title/abstract] OR kid[Title/Abstract] OR Childhood[Title/Abstract])) AND (cohort Studies OR cohort) AND (Risk OR Factor OR etiology))
Scopus	((“diabetes mellitus type 2” OR diabetes mellitus”) AND (“abdominal obesity” OR “central obesity” OR “visceral obesity”) AND (“children” OR “child” OR “pediatric” OR “childhood”) AND (“cohort”) AND (“risk” OR “factor”))
PLOS, Cochrane, Science Direct, Clinical Key, ProQuest, Wiley	(adult-onset diabetes mellitus OR diabetes mellitus type 2 OR DM type 2 OR DM OR diabetes mellitus) AND (abdominal obesity OR central obesity OR visceral Obesity) AND (children OR child OR pediatric OR kid OR childhood) AND (cohort) AND (risk OR factor)

Keywords and boolean search terms applied for literature searches conducted in various databases to ensure comprehensive exploration of all existing studies. Different use of terms was a result of adaptation to each database's search engine requirements.

and adult diabetes. Only published studies with full text availability were searched. Additional records were identified from manual searches, similar articles suggested, and bibliographies from other studies not previously identified in electronic searches.

The study selection process followed the *PRISMA Statements' Flow Diagram*. Duplicates were removed from the retrieved records. The remaining studies were screened based on titles and abstracts; those irrelevant to the topic or objectives of this review were excluded. After screening, full-text articles were assessed for eligibility. The inclusion criteria were cohort studies with minimum 1,000 participants at follow up, age of participants at baseline ≤ 18 years, measurement of obesity and type 2 DM indicators using any method (such as BMI or *CDC Growth Chart* for obesity and HbA1c or fasting plasma glucose for diabetes), and assessment of long-term association between childhood obesity and adult-onset type 2 DM. The sample size restriction was to ensure higher quality studies (as larger size reduces the risk of loss to follow-up and has greater chance of detecting any association between childhood obesity and adult-onset diabetes). Exclusion criteria were languages other than Indonesian and English, incomplete articles or insufficient data, and publication year older than 2000. Records that were incompatible with inclusion criteria or that met the exclusion criteria were excluded, resulting in the studies directly included in the qualitative synthesis. Only some of those were included in quantitative synthesis or meta-analysis due to definition differences of obesity or type 2 DM from our study, and unavailable number of controls, exposed, positive outcome, and negative outcome groups.

Data were extracted by two reviewers using standardized forms, then they were independently checked and confirmed by a third reviewer. Duplicates had already been removed in the prior process, therefore, identical data were not extracted twice. Extraction of study characteristics included study details (author, publication year, study design, definition of childhood obesity and adult diabetes, study location, statistical analysis, sample size, and outcome of study) and participants' details (baseline age or child age, follow-up age or adult age, as well as numbers of control, exposure, positive outcome, and negative outcome groups). When available, adjusted outcome for potential confounders was used if the value was adjusted by exact age and gender only, without

adjustment from other factors.

Quality and bias assessments within studies were done after data extraction from all included studies. The *Newcastle-Ottawa Scale tools designed for Cohort Studies (NOS-Cohort)* was used to assess quality and bias of each study. This scale uses a "star system" in which a study was judged on three broad perspectives: the selection of the study, the comparability of the groups, and the ascertainment of either the exposure of interest. Each perspective had several categories, for which only a star can be given to each category.⁶ The quality of individual studies was assessed by two reviewers, then independently checked by a third reviewer. Thus, studies with lesser bias and higher quality were given more consideration in qualitative analysis, however, no primary included study was excluded. To assess bias, we used the *Cochrane Handbook* regarding summary assessment of risk of bias in 4 levels: outcome, domains, studies, and study as a whole.

Studies included in the qualitative analysis may have reported a variety of metabolic outcomes, such as coronary heart disease, stroke, and hypertension. Those outcomes were ignored and only data regarding type 2 DM was taken. A variety of definitions of obesity and diabetes was also found. Any definition was accepted for qualitative analysis. However, only those with similar sex and age groups were included in meta-analysis to allow calculation of pooled ORs.

The accepted definitions for obesity were BMI $\geq 95^{\text{th}}$ percentile (*CDC Growth Chart 2000*) and BMI $\geq +3\text{SD}$ Z-score in BMI for age per sex chart. The accepted definitions for adult-onset type 2 diabetes were fasting glucose plasma > 7.0 mmol/L, 2-hr plasma glucose > 11.1 mmol/L, and self-reported diagnosis by a physician.⁸ The BMI standard was acknowledged in this meta-analysis based on the assumption and evidence that BMI follows a normal distribution. Diabetes standard was also acknowledged because it was a standardized *WHO* recommendation applicable to everyone.¹ Self-reported diagnosis by physician was accepted with the assumption that the physician also followed *WHO* guidelines or other proven clinical guidelines. Studies that used any other definition were excluded, unless it was not essential or accompanied by other accepted definitions.

Result were reported in a combined fashion to represent all ages of children due to the limited number of studies. We did not group the results based on

children's age, since age was considered indefinite and may have overlapped. For example, children <7 years were often grouped together, as they may experience adiposity rebound (AR). However, universal agreement on age at AR occurrence had yet to be done; AR may happened at ages 3-7 years, 5-7 years, or even above 7 years.^{9,10}

The pooled size of control, exposed (individuals with childhood obesity), positive and negative outcome groups, or the OR, were tabulated and analyzed by forest plot using *Review Manager 5.3* software for *Mac*. To estimate the effect of individual studies against each study's size or precision and to assess publication bias, we used funnel plots, also generated by *Review Manager 5.3* software for *Mac*.

Results

The study search and selection summary is depicted in accordance to the four-phase *PRISMA Statements'* flow diagram in **Figure 1**, which was divided into four main steps: identification, screening, assessment of eligibility, and inclusion. Initially, database searches identified 218 records [from PubMed (37), Scopus (36), PLOS (2), Cochrane (2), Science Direct (71), Clinical Key (70), ProQuest (0), and Wiley (0)]. Additional records were identified from a manual search (5), similar article suggestions (8), and bibliographies from other studies not identified in electronic searches (n=6). Thus, 237 total records were retrieved. Duplicates (7) were removed; 196 records were further

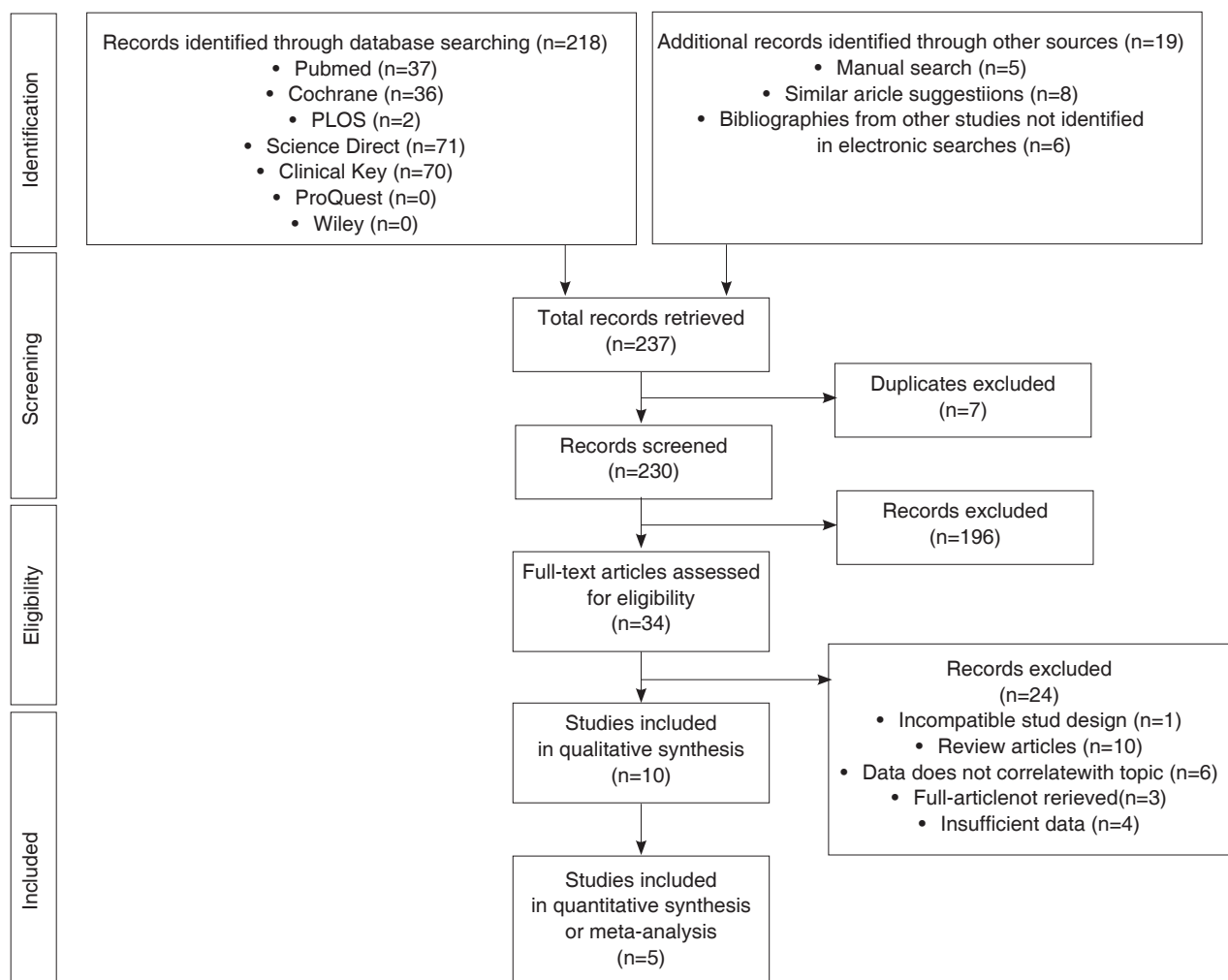


Figure 1. Study selection process

excluded, as they were irrelevant to topic or objectives of this review. One study had an incompatible study design, 10 studies were review articles, 5 studies did not correlate with topic, full-text articles of 3 studies cannot be found, and 4 studies did not have sufficient data. Hence, 10 studies were included in the qualitative synthesis.

The risk of bias and quality assessment for each study using the *Newcastle-Ottawa Scale* is depicted in **Table 2**. All included studies had the minimum score of 8/10, indicating low risk of bias and high validity. The most apparent bias from the individual studies was from outcome section number 3, as 5 studies did not adequately follow up on a number of subjects. An adequate follow-up was considered to be complete follow-up, no loss, or less than 20% loss. Nevertheless, according to the NOS-Cohort scoring system, none of these studies were graded as poor quality. Studies by Hypponen et al.,¹⁴ Mamun et al.,⁹ and Morrison et al.¹⁹ were the only three graded as fair quality, with the rest being of good quality.

The following 5 studies either had a follow-up rate less than 80% or included no description of the follow-up process. Erickson et al.¹⁰ and Forsen et al.¹¹ did not define follow up; Hypponen et al.¹⁴ had 74.7%

follow up; Mamun et al.⁹ had only 36.5%; and Power et al.¹⁵ had a 52.7% follow up. The lower follow-up rates may have arisen from the extensive study length, which could result in loss of interest, migration, or death of subjects. The most unbiased studies were by Hou et al.¹⁶ and Liang et al.,¹⁷ as a V was given to all categories.

Table 3 portrays the characteristics of the 10 studies, with a total of 73,533 participants included in this review. All studies used a cohort study design, either prospective or retrospective. Five studies were included in the meta-analysis. The definitions of childhood obesity and adult type 2 diabetes still varied, but since these definitions were only used alongside the accepted definitions and acted as an ancillary, the studies were still appropriate for meta-analysis. The other childhood obesity definition was *China's Working Group on Obesity (WGOC)* growth chart in studies by Hou et al.¹⁶ and Liang et al.¹⁷ Other definitions of type 2 diabetes in adults were current use of blood-glucose lowering agents by Liang et al.¹⁷ and Power et al.,¹⁵ and HbA1c in Hou et al.¹⁶ and Power et al.¹⁵

The locations of the 10 studies were fairly scattered, from Asia, Europe, Oceania, America, and the Middle East. As such, the studies represented the global population. The age at which obesity was

Publications	Representativeness	Selection of non-intervention cohort	Ascertainment of intervention	Demonstration of outcome interest was not present at start of study	Age, sex, marital status	Additional factors	Assessment of outcome	Ascertainment of intervention	Adequacy of follow up of cohorts
Erickson JG, et al. ¹⁰ (2015)	V	V	V	V	V	X	V	V	X
Forsen T, et al. ¹¹ (2000)	V	V	V	V	V	X	V	V	X
Hou D, et al. ¹⁶ (2016)	V	V	V	V	V	V	V	V	V
Hypponen E, et al. ¹⁴ (2003)	V	V	V	V	V	V	X	V	X
Lawlor DA, et al. ¹³ (2006)	V	V	V	V	V	V	X	V	V
Liang Y, et al. ¹⁷ (2015)	V	V	V	V	V	V	V	V	V
Mamun AA, et al. ⁹ (2020)	V	V	V	V	V	V	X	V	X
Morrison JA, et al. ¹⁹ (2010)	X	V	V	X	V	V	V	V	V
Power C, et al. ¹⁵ (2011)	V	V	V	X	V	V	V	V	X
Tirosh A, et al. ¹² (2010)	X	V	V	V	V	V	V	V	V

Table 2. Risk of bias and quality assessment using the NOS-Cohort scale

[X=compliance to the criteria of lower risk of bias, red Y=the paper did not satisfy the criteria of the scale.

Studies had low risk of bias according to the scale as all studies had the minimum score of 8/10]

Table 3. Characteristics of included studies

Author and publication year	Study design	Cut-off of childhood obesity	Cut-off of adult diabetes	Study location	Method of statistical analysis	Sample size at follow up	Child age, years	Adult age, years	Study included in meta-analysis	Outcome P<0.05; (95%CI)
Ericksson et al. ¹⁰ (2015)	Prospective cohort	BMI ≥ 16.8 for male; BMI ≥ 17.0 for female	Fasting glucose plasma > 7.0 mmol/L; 2-hr plasma glucose >11.1 mmol/L; HbA1c concentration >6.5%	Helsinki	Multivariate linear regression analysis	1,630	11	62	No	1.32 (1.03 to 1.68)
Forsen et al. ¹¹ (2000)	Prospective cohort	BMI >17.4 for male and female	Diagnosis by physician	Helsinki	Multivariate logistic regression	7,086	6-16	31-73	No	1.35 (0.70 to 2.62)
Hou D et al. ¹⁶ (2016)	Prospective cohort	BMI ≥ 95th percentile (2000 CDC Growth Chart) for age 6; BMI ≥ 95th percentile (WGOC 2005) for age 7-18	Fasting glucose plasma ≥ 7.0 mmol/L; 2-hr plasma glucose ≥ 11.1 mmol/L; HbA1c concentration ≥ 6.5%	Beijing, Taiwan	Chi-square test, multivariate linear regression analysis	1,225	6-18	29-41	Yes	2.96 (1.73 to 5.06)
Hypponen et al. ¹⁴ (2003)	Prospective cohort	BMI ≥ +3SD Z Score in BMI for age per sex chart	Self-report diabetes clinical diagnosis	England, Scotland, Wales	Univariate and multivariate logistic regression analysis	8,699	7,11,16	41	Yes	5.02 (2.89 to 8.70)
Lawlor et al. ¹³ (2006)	Prospective cohort	BMI ≥ +3SD Z Score in BMI for age per sex chart	Self-report diabetes clinical diagnosis	Scotland	Multivariate logistic regression analysis	5,793	4-5	46-50	No	1.22
Liang et al. ¹⁷ (2015)	Prospective cohort	BMI ≥ 95th percentile (2000 CDC Growth Chart) for age 6; BMI ≥ 95th percentile (WGOC 2005) for age 7-18	Fasting glucose plasma >7.0 mmol/L; 2-hr plasma glucose >11.1 mmol/L; current use of blood glucose-lowering agents	Beijing, Taiwan	ANOVA, Chi-square test, multivariate logistic and linear regression analysis	1,209	6-18	30-42	Yes	3.54 (2.14 to 5.85)
Al Mamun et al. ⁹ (2009)	Prospective cohort	BMI ≥ 19.30 for male; BMI ≥ 19.17 for female	Self-report diabetes clinical diagnosis	Brisbane, Australia	Multivariate logistic regression analysis	2,639	5	21	No	2.6
Morrison et al. ¹⁹ (2010)	Prospective cohort	BMI ≥ 95th percentile (CDC Growth Chart 2000)	Fasting glucose plasma >7.0 mmol/L	Cincinnati	Fischer's exact test	1,843	10-12	22-30	Yes	5.49 (2.56 to 11.78)

Table 3. Characteristics of included studies (continued)

Author and publication year	Study design	Cut-off of childhood obesity	Cut-off of adult diabetes	Study location	Method of statistical analysis	Sample size at follow up	Child age, years	Adult age, years	Study included in meta-analysis	Outcome P<0.05; (95%CI)
Power et al. ¹⁵ (2011)	Prospective cohort	BMI ≥ 95 th percentile (2000 CDC Growth Chart)	Self-reports of physician diagnosis; current use of blood glucose-lowering agents; HbA1c ≥ 7	England, Scotland, Wales	Multivariate logistic and linear regression analysis	5,735	7, 11, 16	45	Yes	3.61 (1.54 to 8.43)
Tirosh et al. ¹² (2010)	Retro-spective cohort	BMI ≥ 91 th percentile (2000 CDC Growth Chart)	Fasting glucose plasma ≥ 7.0 mmol/L and by physician	Israel	Cox proportional hazards analysis, multivariate linear regression analysis	37,674	16-18	60-68	No	2.95 (2.33 to 3.75)

Comprehensive summary of all included studies in this systematic review after thorough selection based on inclusion and exclusion criteria. Characteristics covered all relevant information necessary for a systematic review.

measured varied among studies, however, 7-16 years was the most common (6 studies). The outcomes of the studies also varied. Lawlor *et al.*¹³ had the lowest OR in this review, which was 1.22, while the OR by Morrison *et al.*¹⁹ was 5.49.

The forest plot for the association between childhood diabetes for all ages based on BMI and type 2 diabetes in adulthood is shown in **Figure 2**. Our meta-analysis revealed a positive and significant association from the 5 studies (OR 3.89; 95%CI 2.97-5.09; P<0.00001). Heterogeneity with I² statistics was 0% across the cohort used (P=0.6). As such, the review was considered homogenous as seen by the consistent findings across the studies: obese BMI in childhood increases the risk of diabetes in adulthood, the similarity in obesity and diabetes cut-offs, and the fairly equal distribution of age at baseline. Moreover, the funnel plot assessment of included studies showed a symmetrical appearance, which was evidence of the homogeneity of this review and a rather low publication bias (**Figure 3**).

The summary of bias risk in this review was done based on the Cochrane Handbook four-level assessment. Potential bias could result from authors' assumptions in accepted definitions used for childhood obesity and adult type 2 diabetes, such as the self-report of a type 2 DM physician diagnosis.

In this funnel plot, the standard error (SE) of the ln odds ratio (OR) was plotted against the OR for association between childhood obesity based on BMI and type 2 diabetes in adulthood. The funnel plot shows a symmetric distribution, hence, no publication bias was suggested.

Discussion

This systematic review and meta-analysis revealed that childhood BMI, particularly BMI > +3SD Z-score, is associated with the occurrence of adult-onset diabetes. The forest plot showed that children with obesity are 3.89 times more likely to become diabetic in adulthood (OR 3.89; 95%CI 2.97 to 5.09). The variability between studies was I²=0% (P=0.60), signifying that the studies were not heterogeneous, i.e., study results were highly similar. The P value for this meta-analysis was P<0.00001, indicating a valid and significant result. In all the included studies, childhood

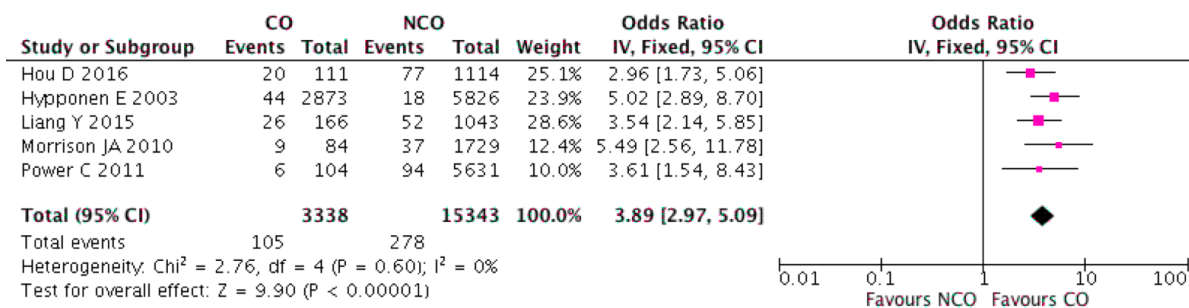


Figure 2. Forest plot for the association between childhood diabetes for all ages based on BMI and type 2 diabetes in adulthood. CO=childhood obesity, NCO=no childhood obesity.

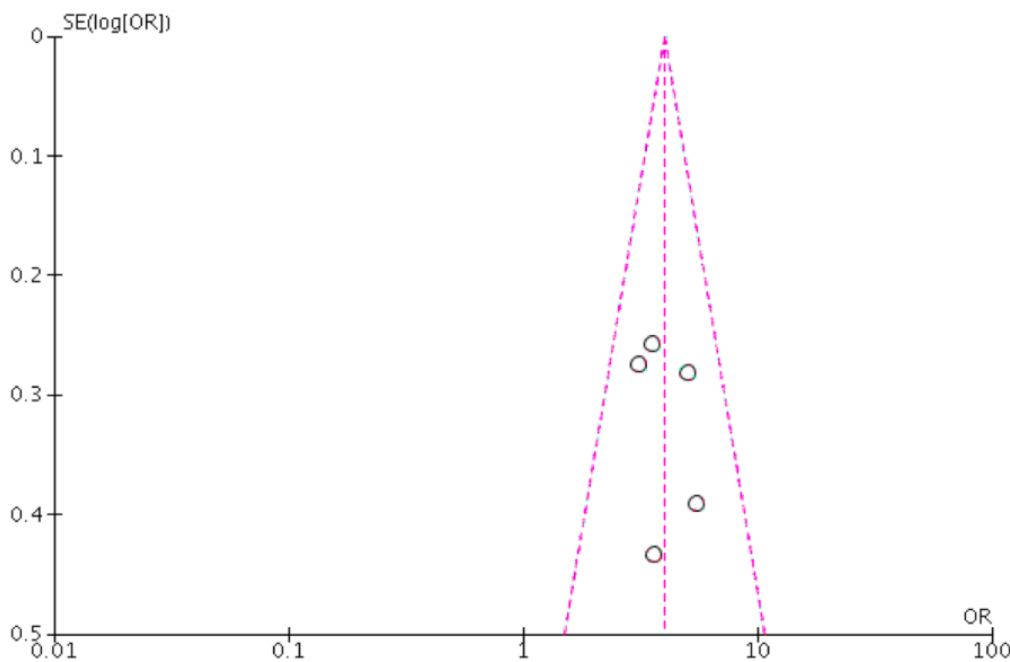


Figure 3. Funnel plot for the association between childhood obesity for all ages based on BMI and type 2 diabetes in adulthood.

obesity was a risk factor for adult-onset diabetes. The ORs of the studies ranged from 2.87 to 5.09, all of which are higher than 1.

The study with the highest weight was by Liang *et al.*,¹⁷ with 28.6%. This study, along with the one by Hou *et al.*¹⁶ which had the second highest weight of 25.1%, suggested that childhood obesity promotes adulthood obesity due to early adiposity rebound. Early adiposity rebound results in an increase in body fat composition and weight gain, causing the individual to enter the obesity cycle. In the obesity cycle, as the individual gains weight, he becomes less physically active, due to the difficulty of performing

physical activity. As obesity progresses, performing physical activity becomes excruciating and exhausting, increasing sleep and eating frequency due to fatigue. As it progresses further, it becomes harder to reduce one’s BMI, which along with social pressure, could lead to mental stress and depression.¹⁶⁻¹⁸ This vicious cycle also applies to childhood obesity.¹⁹ Increased cortisol due to stress results in increased appetite, aggravating the condition. Early onset of this cycle increases the risk of adult obesity, which itself is a major risk factor of adult diabetes.^{16,17} As both of these studies also had the least of risk of bias with scores of 10/10, this explanation is plausible.

The study by Hypponen *et al.*,¹⁴ which had the third highest weight, suggested that high numbers of adipocyte tissues would also result in sustained insulin increment, as insulin functions to inhibit adipose tissue breakdown. Prolonged high insulin concentration would result in insulin resistance. Obese children may have greater-than-normal adipocytes, causing a sustained high insulin condition. Such a mechanism may explain how childhood obesity leads to adult-onset diabetes, as it requires a certain amount of time for insulin resistance progression to reach a diabetic state.¹⁴

The results of this review revealed that risk factors of diabetes are not limited to adulthood. Health conditions during childhood, in this case childhood obesity, can be a determinant for diabetes in adulthood. Thus, early screening for diabetes and pediatric health guidelines could be modified accordingly. Childhood obesity should be added as a risk factor evaluated on early diabetes screening, while pediatric health guidelines should also include this information in order to prevent and reduce childhood obesity. In addition, public awareness needs to be raised, so that parents sense the urgency to reduce their children's obesity.

The strengths of this study included usage of structural guidelines, low risk of bias in included studies, large cohort population, representation from various countries, symmetrical funnel plot, no study heterogeneity, and high study specificity. This review was made based on *PRISMA Statement* to ensure the completion and comprehensiveness of the study, using *NOS-Cohort* to assess risk of bias of the included studies, and *Cochrane Handbook* to assess risk of bias across this review. All of the included studies had the minimum scale of 8/10 and a minimum of 1,000 participants to ensure the quality of each study. Eight countries were included in this review, originating from Europe, Asia, America, Oceania, and the Middle East. Funnel plot data was symmetrical, meaning that the included studies were highly homogenous.

The limitations of this systematic review and meta-analysis were few included studies, bias from accepted definitions, and lifestyle changes. Out of the 230 original studies retrieved, only a total of 10 studies were used in the qualitative review, and 5 studies in the quantitative review. Though there was no heterogeneity and high specificity, the final OR

from this review would be more representative for worldwide use if more studies were included in the forest plot. Though BMI is believed to follow normal distribution, bias can occur. Definition of self-reported diagnosis by physician for adulthood diabetes was accepted, but is also subject to bias. Third, as cohort studies needed for this review require a long follow-up period, and our review includes studies published from the year 2000, the cohort samples were children from the late 20th century instead of the 2000s. The follow-up period ranged from 12 years up to 52 years. As obesity is highly correlated with lifestyle, modern-day children may differ from those of the cohort.

In conclusion, this systematic review and meta-analysis show that individuals with childhood obesity has higher risk of adult-onset diabetes. The most likely causes are early adiposity rebound and/or early-onset insulin resistance from childhood obesity. The findings of this study are applicable to the field of public health, both as a screening method and prevention of adult diabetes.

As childhood obesity was determined by BMI-for-age, further research regarding other measurements to evaluate obesity and other body compositions could also be done to discover a more sensitive and specific obesity measurement and to analyze other variables during childhood that are related to adult-onset diabetes. As BMI-for-age is not the only parameter to evaluate childhood obesity, a study on other markers' specificity and sensitivity towards adult-onset diabetes is crucial to increase treatment effectivity. Other body compositions' effect on adult-onset diabetes should also be evaluated as obesity is a broad term used for general changes to the body composition. It is also essential to provide a more detailed and precise guideline for early screening and prevention of adult-onset diabetes. Age stratification and area-specific study should also be done in order to have a more detailed causality for each age group and certain geographic areas.

Conflicts of Interest

None declared.

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