The Association between Parental Body Mass Index and the Risk of Adolescent Obesity

by Mustakim Mustakim

Submission date: 11-Jun-2024 11:43AM (UTC+0700)
Submission ID: 2400159584
File name: Manuscript_Mustakim_Parental_FINAL.docx (164.26K)
Word count: 3632
Character count: 20672

The Association between Parental Body Mass Index and the Risk of Adolescent Obesity

ABSTRACT

This study aimed to examine the prevalence of adolescent obesity in urban areas and the potential association with parental body mass index (BMI). A cross-sectional design was used and participants were selected from institutions of higher education in Jakarta City. Anthropometric data were obtained by trained volunteers while other variables were acquired through a self-administered questionnaire completed using a Google online form. Among the 420 questionnaires distributed, 370 (88%) were retrieved and completed. Data were analyzed by bivariate and multivariate analysis using chi-square and logistic regression adjusted by lifestyle covariates. The results showed that 59.65% and 63.16% of participants had father or mother who was overweight and obese. The tendency to acquire obesity was higher when father was obese compared to mother (OR=1.42; 95% CI=0.92-2.19). A correlation was found between parental BMI and overweight/obesity in adolescent, with father being a stronger predictor than mother. Although there was no significant association, this study underscored the significance of adolescent obesity and overweight as a public health issue, suggesting the need for urgent preventive measures.

Keywords: Overweight; Obesity; Adolescent; Parental BMI

Conflict of Interest

The author declares no conflict of interest.

INTRODUCTION

The incidence of obesity during childhood is extremely high and continues to rise in low- and middle-income countries (LMICs)[1]. This condition is concerning given that childhood obesity is associated with a host of adverse short- and long-term health outcomes, including

reduced quality of life [2], a higher possibility of seeking medical attention [3], and increased financial expenses for both individuals and society.

Obesity or overweight is defined as an abnormal or excessive fat buildup that poses a health risk. Individuals with body mass index (BMI) over 25 are classified as overweight, while those with values above 30 are obese. The Worldwide Burden Obesity of Illness report from 2017 showed that the problem has escalated to epidemic proportions, with over 4 million deaths annually attributable to overweight or obesity. Globally, the percentage of children and adolescent aged 5-19 who are overweight or obese grew by more than four times, from 4% to 18%, between 1975 and 2016 [4,5].

In general, obesity in childhood is associated with a variety of adverse effects issues, as well as an increased chance of acquiring disorders at a young age. According to studies, children and adolescent who are fat will most possibly remain obese in adulthood [6]. Obesity data in Indonesia shows that 13.5% of adults aged 18 years and above are overweight, while 28.7% are obese (BMI> 25) based on the 2016 National Health Survey. The rate increased to 20.7% (BMI> 27) and 33.5% at BMI> 25 [7].

Adolescent obesity is a multifactorial chronic disease influenced by biological, behavioral, and environmental factors [8]. The occurrence in children and adolescent can affect immediate health, educational attainment, mental health, and quality of life. Furthermore, childhood obesity is associated with problems in the life course and continues in adolescence and maturity [9]. A higher risk of several health consequences, including metabolic illnesses including elevated fasting glucose, impaired glucose tolerance, type 2 diabetes mellitus (T2DM), metabolic syndrome, and fatty liver disease, is associated with an increased BMI, particularly in adolescence [10,11].

Parental weight has been shown as an important predictor of obesity development in children and adolescent. Although several studies reported the association with parental

obesity, the underlying mechanisms were poorly understood. A study from Iranian on 6-18-year-old children reported that obesity in children was significantly associated with parental BMI [12]. A meta-analysis of 23 studies also found a significant association between children and parental BMI/obesity [13]. Among children and adolescent, obesity is caused by multiple factors including sleep duration, sedentary lifestyle, skipping breakfast, physical activity, and others [14].

A systematic review and meta-analysis of prospective cohort studies showed that sleep duration was significantly associated with obesity[15]. The incidence of obesity was higher among those who experienced a short duration of sleep. Other studies on 8,718 children or adolescent aged 16 years in the USA reported that sleep duration was significantly associated with obesity [16]. Skipping or changing breakfast routines has been connected to childhood obesity in numerous studies [17]. Furthermore, daily breakfast may reduce childhood obesity by 34%, according to a systematic review and meta-analysis [18]. Another systematic study found that missing breakfast might be a straightforward way to gauge the risk of becoming overweight or developing metabolic diseases [19]. An energy imbalance from unhealthy eating patterns and insufficient physical activity poses the risk of obesity.[20,21] More precisely, a strong correlation exists between the onset of obesity and sedentary behaviors, including increasing video gaming, television watching, and computer screen time.[22,23].

Obesity is caused by a variety of factors, and only a few studies have examined the connection between obesity in parents and children. Therefore, this study aimed to investigate the association between overweight and obesity in children and adolescent living in urban settings as well as parental BMI.

MATERIAL AND METHODS

This survey was performed in urban and suburban areas in Jakarta, with participants selected from Institutions of higher education by cluster random sampling method. An equal number of institutions were selected from each area to ensure even representation. The total sample size was calculated as 420 participants of which 88% completed and returned the survey. Institutions with the greatest number of populations were allocated higher slots for participants. Subsequently, the samples were selected randomly using data provided by the institutions.

Trained volunteers conducted the physical examination for height and weight using calibrated instruments to measure BMI of participants. These volunteers were health students selected from institutions of higher education in Jakarta area. Training was carried out to measure anthropometric indices according to standard protocols. The volunteers collect the data from participants by gathering in designated areas in the institution.

Standing height was recorded without shoes to the nearest 0.1 cm, while weight was measured with the subject in light clothing to the nearest 0.1 kg. BMI was calculated as weight (Kg) divided by height in square meters (m^2). The categories were defined according to World Health Organization (WHO) reference for different ages and groups [13] including underweight (BMI <18.5), normal weight (18.5<BMI<24.9), overweight (BMI >= 25-29.9), and obese (BMI >=30). Age and sex information were also collected, including behavior comprising sleep duration, breakfast habits, stress level, physical activity, and sedentary lifestyle. Sleep duration was defined as the total hours of sleep during weekdays and weekends, while breakfast habits referred to how many times participants skipped breakfast in a week. Stress levels were measured by questionnaire, physical activity was also assessed by IPAQ, while sedentary lifestyle was defined as the total of sedentary activity in a week measured using questionnaire.

This study was approved by the Institutional Review Board or Health Research Ethics Committee of the Faculty of Medicine and Health, University of Muhammadiyah Jakarta 071/PE/KE/FKK-UMJ/IX/2020.

Statistical Analyses

All analyses were conducted using the survey analysis method in SAS software, with categorical data presented as numbers and percentages. The weight status of adolescent was analyzed as an ordinal outcome variable. Parental weight status was categorized into three group categories including underweight, normal, and overweight or obese, which was investigated as an ordinal response variable. Pearson chi-square statistic was used to determine the association between weight status and the characteristics of participants.

Logistic regressions were applied to the odds ratios (ORs) and 95% confidence interval (CIs) of parental BMI status, then adjusted for potential confounders. Model 1 was adjusted by age and sex, Model 2 by breakfast habit and sleep duration, Model 3 by physical activity, and Model 4 by the condition of participants (stress level). All the tests were two-sided, and the significance level was 0.05.

RESULT

In total, 370 participants (84.6% girls) completed the survey, with the mean and SD age being 19.91±1.03 years. For the overweight or obesity category, 59.65% and 63.16% of participants had father or mother who was overweight and obese. About 42.11% of adolescent with obesity had 3-5 times/week of breakfast habit, 54.39% had less physical activity, 64.91% had average sleep duration, while 45.61% had a sedentary lifestyle and experienced stress, respectively. The primary and demographic characteristics of participants are presented in Table 1.

Table 2 shows the multivariate regression models for the association between obesity and parental BMI in adolescent, adjusted for potential confounders including age, sex, breakfast habit, sleep duration, physical activity, sedentary lifestyle, and stress level. In logistic regression, the risk of parental BMI was associated with adolescent obesity but the result was not significant. When adjusted by covariates, the association remained unchanged, but the odds ratio increased. In the final model, the odds ratio of adolescent obesity in father's BMI was higher than mother (OR=1.42; 95% CI=0.92-2.19 and OR=1.02, 95% CI=0.67-1.56).

DISCUSSION

This study found that parental BMI did not influence adolescent obesity but an increased risk was associated with father's BMI compared to mother's. In other words, an adolescent is at higher risk of obesity when father is overweight or obese compared to mother.

The results were in contrast to a previous study on the Iranian population stating that children or adolescent obesity was significantly associated with parental obesity in both boys and girls, after adjusting for confounders [24]. Other studies from rural north China and Mashhad, Iran, also showed that obese parents were more likely to have overweight or obese children compared to normal-weight parents [25,26]. The differences in the results could be attributed to several factors such as covariates, race, total number of participants, and others.

Based on the results, childhood or adolescent obesity was not significantly associated with parental obesity after adjusting lifestyle confounders. However, a correlation has been established between higher parental and childhood BMI [27]. This association is probably related to the direct intrauterine effect of mother BMI, a shared environment and genetics, or a mix of these variables. In terms of shared genetics, one or both parents may pass on genetic variations that raise BMI of children. Shared environmental factors, including lifestyle choices or diet, can also lead to a higher BMI in children and parents. Maternal obesity is particularly caused

by an intrauterine environment that programs the metabolic processes of a fetus, raising the chance of childhood obesity. Although evidence pointing to a possible causative intrauterine mechanism for maternal obesity is growing, certain studies indicate that maternal BMI has more significant impacts than paternal [9].

Another mechanism that plays an essential role in parents and children obesity is epigenetics. For example, DNA methylation is the epigenetic mechanism showing how inherited childhood or adulthood-related genes interact with environmental variables to cause the development of obesity[28]. Epigenetic changes that potentially affect obesity-related hormones such as leptin, insulin, and ghrelin are dynamically regulated by nutritional and metabolic status [29]. Leptin plays a role in reducing food intake and decreasing body weight [30].

Basic, translational, and clinical studies on childhood and teenage obesity still have many knowledge gaps [9]. For example, adopted children and parents have a paradoxically weak BMI association. Although genetic loci associated with obesity have been identified, the effect size is modest. The mechanisms of genetic, epigenetic, environmental, and social factors underlying the association between parental with childhood and adolescent obesity remain unclear [31].

Based on the results, the possibility of being overweight or obese in children/adolescent was affected not only by parental or genetic factors but also lifestyle habits. The rapid rise in adolescent obesity rates is primarily due to the significant changes in lifestyles imbibed over the years. People spend significant time sitting down and immobile due to the job demand and dietary habits, while also consuming calorie-rich meals[32]. Furthermore, obesity was more common in children and adolescent who slept for short periods. A previous study on 8,718 16-year-old adolescent in the USA showed a strong correlation between obesity and sleep duration. A comprehensive review and meta-analysis found that eating breakfast every day could prevent adolescent obesity by 34% [18]. Obesity is defined as an energy imbalance caused by poor eating

habits and inadequate exercise. [20,21] More specifically, there is a strong connection between the incidence of obesity and sedentary activities, such as increased computer screen time, video gaming, and television viewing [22,23].

The psychological factor is also a significant causative element for the occurrence of obesity. According to a recent study, there is a connection between obesity and psychological stress [33] associated explicitly with dietary patterns, including eating more high-calorie foods, or less low-calorie meals. Stress can negatively affect weight and health status by increasing body adiposity [34]. Furthermore, it may influence negative behaviors in children related to the body and weight, resulting in bulimia, anorexia nervosa, or binge eating disorder caused by an overwhelming concern for regulating weight. Binge eating disorder is particularly challenging to detect in young patients [35]. Children who suffer from eating disorders may experience weight gain and obesity.

The results from this study cannot be generalized for the entire urban population of Jakarta because only a small part was sampled. However, this study could serve as baseline data for further investigations regarding parental BMI and obesity. Another limitation was the use of a cross-sectional design, underscoring the need for a prospective study. In addition, self-reporting lifestyle variables as well as parental weight and height may result in potential bias.

CONCLUSION AND RECOMMENDATION

In conclusion, overweight and obesity in children or adolescent appeared to be rising at a startling rate attributed to both environmental and genetic factors. Although there was a correlation between parental BMI and overweight/obesity in adolescent, father's BMI proved to be a stronger predictor than mother's. This study underscored the significance of childhood obesity and overweight as a public health issue, suggesting the need for urgent preventive

measures. Individuals with the intention of having children should be educated to prevent childhood obesity in the future.

ACKNOWLEDGMENT

The author is grateful to the Dean of the Faculty of Public Health, Universitas Muhammadiyah Jakarta, and the Rector of Universitas Muhammadiyah Jakarta for funding this study.

REFERENCE

1. Popkin, B. M., Corvalan, C. & Grummer-Strawn, L. M. Dynamics of the double burden of malnutrition and the changing nutrition reality. *The Lancet* vol. 395 65–74 Preprint at https://doi.org/10.1016/S0140-6736(19)32497-3 (2020).
2. Buttitta, M., Iliescu, C., Rousseau, A. & Guerrien, A. Quality of life in overweight and obese children and Adolescent: a literature review. *Quality of life research : an international journal of quality of life aspects of treatment, care and rehabilitation* vol. 23 1117–1139 Preprint at https://doi.org/10.1007/s11136-013-0568-5 (2014).
3. Hayes, A. *et al*. Early childhood obesity: Association with healthcare expenditure in Australia. *Obesity* **24**, 1752–1758 (2016).
4. National Heart Lung and Blood Institute. Overweight and Obesity - What Are Overweight and Obesity? | NHLBI, NIH. 1–2 (2022).
5. WHO. Obesity and overweight. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight (2021).
6. WHO. No Title. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight (2021).

7. Kementerian Kesehatan RI. Epidemi Obesitas. *Jurnal Kesehatan* 1–8 Preprint at http://www.p2ptm.kemkes.go.id/dokumen-ptm/factsheet-obesitas-kit-informasi-obesitas (2018).
8. Mead, E. *et al*. Diet, physical activity and behavioural interventions for the treatment of overweight or obese children from the age of 6 to 11 years. *Cochrane Database of Systematic Reviews* vol. 2017 Preprint at https://doi.org/10.1002/14651858.CD012651 (2017).
9. Lister, N. B. *et al*. Child and adolescent obesity. *Nat Rev Dis Primers* **9**, (2023).
10. Cioana, M. *et al*. The Prevalence of Obesity among Children with Type 2 Diabetes: A Systematic Review and Meta-analysis. *JAMA Netw Open* **5**, E2247186 (2022).
11. Daniels, S. R. *et al*. Overweight in children and Adolescent: Pathophysiology, consequences, prevention, and treatment. *Circulation* vol. 111 1999–2012 Preprint at https://doi.org/10.1161/01.CIR.0000161369.71722.10 (2005).
12. Bahreynian, M. *et al*. Association between Obesity and Parental Weight Status in Children and Adolescent. *J Clin Res Pediatr Endocrinol* **9**, 111–117 (2017).
13. Lee, J. S., Jin, M. H. & Lee, H. J. Global relationship between parent and child obesity: a systematic review and meta-analysis. *Clin Exp Pediatr* **65**, 35–46 (2022).
14. Lister, N. B. *et al*. Child and adolescent obesity. *Nat Rev Dis Primers* **9**, (2023).
15. Li, L., Zhang, S., Huang, Y. & Chen, K. Sleep duration and obesity in children: A systematic review and meta-analysis of prospective cohort studies. *J Paediatr Child Health* **53**, 378–385 (2017).
16. Sluggett, L., Wagner, S. L. & Harris, R. L. Sleep Duration and Obesity in Children and Adolescent. *Can J Diabetes* **43**, 146–152 (2019).

17. Blondin, S. A., Anzman-Frasca, S., Djang, H. C. & Economos, C. D. Breakfast consumption and adiposity among children and Adolescent: an updated review of the literature. *Pediatr Obes* **11**, 333–348 (2016).

18. Poorolajal, J., Sahraei, F., Mohamdadi, Y., Doosti-Irani, A. & Moradi, L. Behavioral factors influencing childhood obesity: a systematic review and meta-analysis. *Obes Res Clin Pract* **14**, 109–118 (2020).

19. Monzani, A. *et al*. A Systematic Review of the Association of Skipping Breakfast with Weight and Cardiometabolic Risk Factors in Children and Adolescent. What Should We Better Investigate in the Future? *Nutrients* **11**, 387 (2019).

20. Paeratakul, S., Ferdinand, D. P., Champagne, C. M., Ryan, D. H. & Bray, G. A. Fast-food consumption among US adults and children: Dietary and nutrient intake profile. *J Am Diet Assoc* **103**, 1332–1338 (2003).

21. Hernández, B. *et al*. Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico City. *Int J Obes* **23**, 845–854 (1999).

22. Andersen, R. E., Crespo, C. J., Bartlett, S. J., Cheskin, L. J. & Pratt, M. Relationship of Physical Activity and Television Watching With Body Weight and Level of Fatness Among Children. *JAMA* **279**, 938 (1998).

23. Vicente-Rodríguez, G. *et al*. Television watching, videogames, and excess of body fat in Spanish Adolescent: The AVENA study. *Nutrition* **24**, 654–662 (2008).

24. Bahreynian, M. *et al*. Association between obesity and parental weight status in children and Adolescent. *JCRPE Journal of Clinical Research in Pediatric Endocrinology* **9**, 111–117 (2017).

25. Shafaghi, K. *et al*. Parental body mass index is associated with adolescent overweight and obesity in Mashhad, Iran. *Asia Pac J Clin Nutr* **23**, 225–231 (2014).

26. Jiang, M. han, Yang, Y., Guo, X. fan & Sun, Y. xian. Association between child and adolescent obesity and parental weight status: A cross-sectional study from rural north China. *Journal of International Medical Research* **41**, 1326–1332 (2013).

27. Kivimäki, M. *et al*. Substantial intergenerational increases in body mass index are not explained by the fetal overnutrition hypothesis: the Cardiovascular Risk in Young Finns Study. *Am J Clin Nutr* **86**, 1509–1514 (2007).

28. Herrera, B. M., Keildson, S. & Lindgren, C. M. Genetics and epigenetics of obesity. *Maturitas* **69**, 41–49 (2011).

29. Bouret, S., Levin, B. E. & Ozanne, S. E. Gene-Environment Interactions Controlling Energy and Glucose Homeostasis and the Developmental Origins of Obesity. *Physiol Rev* **95**, 47–82 (2015).

30. Halaas, J. L. *et al*. Physiological response to long-term peripheral and central leptin infusion in lean and obese mice. *Proceedings of the National Academy of Sciences* **94**, 8878–8883 (1997).

31. Albuquerque, D., Nóbrega, C., Manco, L. & Padez, C. The contribution of genetics and environment to obesity. *Br Med Bull* **123**, 159–173 (2017).

32. Panahi, S. & Tremblay, A. Sedentariness and Health: Is Sedentary Behavior More Than Just Physical Inactivity? *Front Public Health* **6**, (2018).

33. Miller, A. L. & Lumeng, J. C. Pathways of Association from Stress to Obesity in Early Childhood. *Obesity* **26**, 1117–1124 (2018).

34. Tryon, M. S., Carter, C. S., DeCant, R. & Laugero, K. D. Chronic stress exposure may affect the brain's response to high calorie food cues and predispose to obesogenic eating habits. *Physiol Behav* **120**, 233–242 (2013).

35. Latzer, Y. & Stein, D. A review of the psychological and familial perspectives of childhood obesity. *J Eat Disord* **1**, 7 (2013).

Table 1. Study sample characteristics.

Characteristics	Underweight (n=80)	Normal (n=233)	Overweight or Obesity (n=57)	*p-value*
Father's BMI				0.0918
Underweight	55 (68.75)	144 (61.80)	22 (28.60)	
Normal	4 (5.00)	3 (1.29)	1 (1.75)	
Overweight or Obesity	21 (26.25)	86 (36.91)	34 (59.65)	
Mother's BMI				0.9581
Underweight	54 (67.50)	118 (50.64)	20 (35.09)	
Normal	4 (5.00)	1 (0.43)	1 (1.75)	
Overweight or Obesity	22 (27.50)	114 (48.93)	36 (63.16)	
Sex				0.0246
Female	64 (80.00)	199 (85.41)	40 (70.18)	
Male	16 (20.00)	34 (14.59)	17 (29.82)	
Age				0.4092
Less than equal 19	30 (37.50)	71 (30.47)	21 (36.84)	
More than 19	50 (62.50)	162 (69.53)	36 (63.16)	
Breakfast habit				0.2124
Less than equal 2 times/week	11 (13.75)	55 (23.61)	15 (26.32)	
3-5 times/week	31 (38.75)	91 (39.06)	24 (42.11)	
More than 6 times/week	38 (47.50)	87 (37.34)	18 (31.58)	
Physical activity				0.5419
Less active	43 (53.75)	112 (48.07)	31 (54.39)	
Active	121 (51.93)	121 (51.93)	26 (45.61)	
Sleep duration				0.4625
Less than equal 6 hours	23 (28.75)	70 (30.04)	11 (19.30)	
7-8 hours	46 (57.50)	123 (52.79)	37 (64.91)	
More than 8 hours	11 (13.75)	40 (17.17)	9 (15.79)	
Sedentary lifestyle				0.7611
No	40 (50.00)	114 (48.93)	31 (54.39)	
Yes	40 (50.00)	119 (51.07)	26 (45.61)	
Stress				0.6523

No	44 (55.00)	116 (49.79)	31 (54.39)
Yes	36 (45.00)	117 (50.21)	26 (45.61)

Significant at p<0.05

Table 2. Adjusted odd ratios of parental BMI with overweight and obesity in Adolescent.

	Crude	Model 1	Model 2	Model 3	Model 4
	OR 95% CI	**OR 95% CI**	**OR 95% CI**	**OR 95% CI**	**OR 95% CI**
Father's BMI					
Normal	Ref	Ref	Ref	Ref	Ref
Underweight	2.24 (0.59-8.42)	2.15 (0.57-8.13)	2.29 (0.60-8.69)	2.33 (0.61-8.81)	2.36 (0.62-8.97)
Overweight or obesity	1.44 (0.94-2.19)	1.37 (0.89-2.10)	1.37 (0.89-2.10)	1.41 (0.91-2.17)	1.42 (0.92-2.19)
Mother's BMI					
Normal	Ref	Ref	Ref	Ref	Ref
Underweight	3.13 (0.69-14.07)	3.07 (0.68-13.95)	2.95 (0.65-13.37)	3.21 (0.70-14.74)	3.25 (0.71-14.96)
Overweight or obesity	0.99 (0.65-1.50)	1.00 (0.66-1.53)	1.00 (0.66-1.53)	1.02 (0.67-1.56)	1.02 (0.67-1.56)

Model 1 adjusted by age and sex

Model 2 adjusted by model 2+breakfast habit and sleep duration

Model 3 adjusted by model 2+physical activity and sedentary lifestyle

Model 4 adjusted by model 3+stress

The Association between Parental Body Mass Index and the Risk of Adolescent Obesity

ORIGINALITY REPORT

27%	25%	16%	18%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	www.jcrpe.org Internet Source	9%
2	Submitted to Glasgow Caledonian University Student Paper	2%
3	listens.online Internet Source	2%
4	www.mdpi.com Internet Source	2%
5	koreascience.or.kr Internet Source	1%
6	Submitted to Republic of the Maldives Student Paper	1%
7	Submitted to University of Bedfordshire Student Paper	1%
8	irep.iium.edu.my Internet Source	1%
9	Andi Eka Yunianto, Rosyanne Kushargina, Tria Astika Endah Permatasari. "Body Fat	1%

	Composition Related to Fatty Snack Habit Consumption of Office Workers in Urban Area", Open Access Macedonian Journal of Medical Sciences, 2022 Publication	
10	www.science.gov Internet Source	1 %
11	Epidemiology of Obesity in Children and Adolescents, 2011. Publication	1 %
12	Submitted to University of Cincinnati Student Paper	1 %
13	1library.org Internet Source	1 %
14	garuda.kemdikbud.go.id Internet Source	1 %
15	www.spandidos-publications.com Internet Source	1 %
16	Submitted to University of Birmingham Student Paper	<1 %
17	Submitted to University of Ulster Student Paper	<1 %
18	Submitted to Utah Education Network Student Paper	<1 %
19	f1000research.com Internet Source	

		<1 %
20	www.researchgate.net Internet Source	<1 %
21	link.springer.com Internet Source	<1 %
22	res.mdpi.com Internet Source	<1 %
23	core.ac.uk Internet Source	<1 %
24	docplayer.net Internet Source	<1 %
25	en.wikipedia.org Internet Source	<1 %
26	sci-hub.se Internet Source	<1 %
27	Han Hyuk Lim. "Sleep duration independently influences metabolic body size phenotype in children and adolescents: a population-based study", Sleep Medicine, 2018 Publication	<1 %
28	Natalie B. Lister, Louise A. Baur, Janine F. Felix, Andrew J. Hill et al. "Child and adolescent obesity", Nature Reviews Disease Primers, 2023 Publication	<1 %

Exclude quotes	On	Exclude matches	Off
Exclude bibliography	On		