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Metabolically Healthy Obese (MHO) in Adults and Adolescents: Where We Are

Gilles Plourde*

Associate Professor at the Faculty of Health Sciences, University of Ottawa, Canada

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***Corresponding author:** Gilles Plourde, Associate Professor Department of Clinical Pharmacology and Physiology, Faculty of Medicine, University of Montreal, Montreal, Quebec, Canada, Email: gilles.plourde@hc-sc.gc.ca; drgplourde@gmail.com

Abstract

A unique subset of obese individuals who appear to be protected from the development of metabolic disturbances has been identified in clinical practice and is termed metabolically healthy but obese (MHO). The true prevalence of the MHO phenotype varies widely from approximately 6% to75% of obese adults and from 6–36% in obese adolescents. Currently, there are no clear accepted criteria on the definition of MHO. The strong effect of pubertal status on metabolic health cannot be excluded in obese adolescents. To clarify this definition we need the collaboration of the scientific community. Numerous possible mechanisms underlying MHO have been suggested, including adipose tissue distribution and inflammation. The current evidence cannot confirm that MHO subjects are permanently protected from the risk of obesity-associated metabolic complications. This transition might be minimized by appropriate lifestyle habits. No standard practice guidelines for the prevention and treatment of MHO can be proposed to clinicians. A decent attitude would be to regularly monitor CVD risk factors in obese adult and adolescent MHO patients especially elevated triglycerides, glycaemia, HOMA and C-reactive protein as well as low HDL-C. A special surveillance should be applied to prevent any increase in waist circumference (WC) as the MHO phenotype may be maintained by promoting lower WC and by the prevention of any further weight gain. Identifying obese patients with this protective profile could help the medical community determine which part of the obese patients need to be only periodically observed and which need to have early therapeutic interventions.

Keywords: Metabolically healthy obese (MHO); Metabolically unhealthy obese (MUHO); Type 2 diabetes mellitus; Cardiovascular disease; Dyslipidemia; Impaired glucose tolerance; Metabolic syndrome; CVD risk factors; Insulin resistance; Treatment; Clinical practice.

Abbreviations: T2DM: Type 2 Diabetes Mellitus; CVD: Cardiovascular Disease; MHO: Metabolically Healthy Obese; MUHO: Metabolically Unhealthy Obese; OR: Odds Ratio; BMI: Body Mass Index; MVPA: Moderate-Vigorous Physical Activity; HDL-C: High Density Lipoprotein Cholesterol Levels

Introduction

Obesity is associated with a reduced life expectancy, mostly because obese individuals are at an increased risk of Type 2 Diabetes Mellitus (T2DM), Cardiovascular Disease (CVD), and several types of cancer. However, there is a subset of healthy obese individuals, i.e., a subset known as Metabolically Healthy Obese (MHO) that are not at a higher risk of mortality and morbidity than their Metabolically Unhealthy Obese counterparts (MUHO) [1-3]. MHO accounts for a substantial proportion of the obese adult population and this proportion may vary depending on the criteria used to define MHO. Currently, there are no universally accepted criteria to define MHO but the definition needs that the patient be obese and lack the obesity-associated metabolic complications mentioned above. MHO individuals display less visceral adipose tissue, smaller adipocytes, and a reduced inflammatory profile relative to MUHO. In adults, few years ago, Plourde G and Karelis A have been able to provide an interesting definition of MHO [1]. This definition is now used internationally under the term of (PK definition; Plourde and Karelis definition) and is used to determine the prevalence of MHO vs the prevalence of MUHO in adult German populations [4]. As in adults, identifying obese adolescent patients with this potential protective profile could help the medical community determines which part of the obese adolescent and adult populations need to be only periodically observed and which need to have early therapeutic interventions [2]. In adolescent, it is even more complicated considering that previous studies have demonstrated that being obese in this age group increase the risk of having obesity and the obesity-associated complications at later age [5]. As just stated, the main difficulty in estimating the actual prevalence of MHO is the lack of consensus pertaining to its definition. For example, it was demonstrated that nearly half (55.2%) of obese subjects were MHO using the National Cholesterol Edu¬cation Program Adult Treatment Panel III (NCEP ATP III) criteria to define metabolic syndrome [6]. Recently, Rey-Lopez et al. performed a systemic re¬view on the prevalence of MHO based on the frequency of different variables used among studies and they reported that the MHO prevalence ranged from 6% to 75% [7]. They also mentioned that this prevalence may also vary according to sev¬eral socio-demographic factors such as gender, age, and race/ethnicity [7]. By stratifying the analysis ac¬cording to gender and age, these authors were able to reveal that the MHO prevalence was higher in women and younger-

aged in¬dividuals [7]. Considering the marked heterogeneity of MHO definitions and the varied prevalence described in the literature, it is clear that a common MHO definition still need to be established.

A second major concern in the identification of the real prevalence of MHO is the fact that a subject's health status can switch from MHO to MUHO and vice versa with time [1]. For example, Soriguer et al. showed that 30% to 40% of individuals with MHO converted to a MUHO status after 6 years of follow-up [8]. Because there is an accumulation of evidence suggesting that MHO is not a static condition, attention should be focused on the variables that predict metabolic deteri¬oration to MUHO in individuals with MHO [1]. According to a study conducted in Spain by Schroder H et al. the factors that predict the transition from MHO to MUHO were an increase in BMI and abdominal obesity measured by the waist circumference (WC), or by the waist-to-hip ratio [9]. On the other hand, the incorporation of a healthy lifestyle, in¬cluding a healthy diet, moderate to high level of physical activity, no smoking, or smoking cessation, helped prevent the tran¬sition from MHO to MUHO [9]. Hwang YC et al. demonstrated that nearly two-thirds of Japanese Americans with MHO de¬veloped MUHO over 10 years, and a higher conversion to MUHO was associated with greater visceral abdominal fat, female gender, higher fasting insulin levels, and lower baseline of High Density Lipoprotein Cholesterol Levels (HDL-C) [10]. Taken together, the main characteristics suggested to preserve metabolic health in individuals with MHO include a healthier lifestyle, greater incretin response to meals, less abdominal fat distribution, less visceral and ectopic fat accumulation, lower levels of inflammation, and greater insulin sen¬sitivity [1-3, 7-10]. Therefore, an adequate surveillance of these factors in MHO individuals may prevent the progression to an MUHO phenotype. Another research from Primeau V et al. also suggests that although MHO individuals display a favorable metabolic profile; this does not necessarily translate into a decrease in mortality [11]. However, a recent meta-analysis by Zheng R et al. found that MHO individuals were not at an increased risk of all-cause mortality but were at an increased risk of CVD events [12]. While other studies found that relative to the MUHO subjects, MHO individuals were at a lower risk of T2DM but not of CVD [13]. On the other hand, MHO patients are certainly at a higher risk of CVD compared to metabolically healthy non-obese individuals (MHNO) [1, 13-15]. According to Badoud F et al. the relatively low risk of CVD disease among people with MHO relative to MUHO has been attributed to differences in white adipose tissue function between the two groups [16].

The prevention and treatment of obesity is a relevant medical, socioeconomic and public health issues and the interventions are not always successful [1-3]. Notably, different independent studies have shown that individuals with MHO may not be able to significantly reduce their obesity-related CVD and metabolic risk using anti-obesity treatment strategies [1]. However, the same public health message remains for obese patients to maintain an appropriate lifestyle that contains an adequate diet and regular physical activity [1-3]. The review made by Plourde G and Prudhomme D might be very useful for the physicians to guide their MHO patients achieve and maintain their weight-loss [17]. The current mini review aimed to present current issues regarding MHO including the suggested mechanisms that might explain MHO and its definition in adult and adolescent populations [1-2].

Which Mechanisms Might Explain MHO?

Although the exact mechanism responsible for the MHO phenotype is still unclear, some possible mecha¬nisms have been suggested in both human and animal studies [18-19]. They include the maintenance of insulin sensitivity, the presence of specific fat distribution with low visceral and ectopic fat accumulation including low liver and skeletal mus¬cle fat storage compared with subcutaneous fat depots [18-19]. The mechanisms also include a normal adipose tissue function defined by a lower im¬mune cell infiltration into adipose tissue, a normal adi¬pokine secretion patterns, and finally a high level of physical activi¬ty, and fitness [18-19]. According to Bluher M, obesity is a multifactorial disorder that is influenced by the interplay between genetic, behavioral, lifestyle, and environmental factors, including fetal program¬ming, the control of appetite and energy expenditure, and the availability of nutritional food [20]. These factors and their interactions lead to an expan¬sion in fat mass due to an increase in the mean of fat cell volume (cell hypertrophy) and in the number of adipocytes (cell hyperplasia) mentioned Spalding KL et al. [21]. In most obese individuals, the adipocyte storage capacity may be exceeded and lipids may accumulate ectopically in visceral fat depots, liver, muscle, and β-cells, where¬as in MHO subcutaneous adipose tissue has the intrinsic ability to expand, leading to preserved insulin sensitivity [21]. However, the genetic and environmental factors involved in the fat expandability are still unknown.

During a positive energy balance, due to an excessive energy intake and/or a sedentary lifestyle or low energy expenditure, subcutaneous adipose tissue expands and accumulates lipids in the form of triglycerides [3,21]. If this positive energy balance is prolonged, a point is eventually reached where subcutaneous adipose tissue cannot further expand and energy surplus no longer can be safely stored mentioned Spalding KL et al. [21]. Once the limit on storage capacity has been exceeded, the dietary lipids start spilling and accumulate ectopically in other organs such as the omentum, the liver, the pancreas and the muscles, forming lipid byproducts that are toxic to the cells. Unfortunately, there is currently no clinically useful screening method to predict which obese individuals will develop metabolic derangements, especially T2DM and CVD [3,21]. Esser N et al. have proposed that inflammation in adipose tissue is an¬other key factor that explains the metabolic alterations associated with obesity [22]. However, studies comparing the inflammatory status among individuals with MHO have yielded conflicting results [23-26].

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For exam¬ple, Phillips and Perry demonstrated that individuals with MHO presented with a more favorable inflamma¬tory status than their MUHO counter¬parts [23]. According to most definitions, MHO presents with lower concentrations of complement component 3, C-reactive protein, tumor necrosis factor α, interleukin 6, and plasminogen activator inhibitor-1; higher adiponectin levels; and reduced white blood cell count compared to their MUHO counterparts [23-26].

Logistic regression analysis identified greater likelihood of MHO among individuals with lower levels of complement component 3 (odds ratios [ORs], 2–3.5), IL-6 (ORs, 1.7–2.9), plasminogen activator inhibitor-1 (ORs, 1.7–2.9), and white blood cells (ORs, 2.1–2.5) and higher adiponectin concentrations (ORs, 2.6–4.0). The authors of this study concluded that the favorable inflammatory status is positively associated with metabolic health in obese and non-obese individuals [23]. In contrast, a study by Gomez-Ambrosi J et al. on a West¬ern population that used the same MHO definition showed that circulating concentrations of pro-inflam¬matory factors, including CRP, were increased in both the MHO and MUHO groups [25]. Although these conflicting findings may be explained by various differences between studies including in the ethnicities, age groups, low numbers of subjects, limited inflam¬matory profiling, and/or the use of different metabolic health criteria to define MHO; a better understanding of the association between MHO and inflammation is therefore necessary to increase our comprehension of this relation [26].

Identifying MHO Individuals in Adults

Defining the MHO phenotype is an important aspect for studying the mechanisms by which fat accumulation in obese subjects causes or contributes to the obesity-associated metabolic complications and/or CVD risk factors [1,3]. As discussed above and in our previous review [1], another complication has been found in defining MHO; the cutoff values for each pa¬rameter, including insulin resistance and inflammato¬ry markers, have not been clearly established [1,3]. Adults defined as MHO are generally characterized for the absence of the metabolic abnormalities such as those mentioned earlier, they also present with lower visceral, hepatic, muscle fat accumulation and gene expression-encoding markers of adipose cell differentiation [1,3]. However, it is important to note that MHO individuals may also have multiple intermediate metabolic risk factors that may signal an increased risk for T2DM and CVD later in life [1]. Plourde G and Karelis A first believed that the definition of adult's MHO include a WC of ≥80 cm for women and ≥94 cm for men that should be used to identify adults MHO subjects instead of a BMI of ≥30 kg/m2. These authors then suggested the following metabolic markers with their cut-points: glycemia ≥ 5.6 mmol/l, HDL-C ≥1.3 mmol/l for women and ≥ 1.03 mmol/l for men, triglycerides <1.7mmol/l, and blood pressure <120/80 mm Hg [1]. The proposed choice of these clinical markers was based from the criteria for the identification of metabolic syndrome in adults from the International Diabetes Federation (IDF) [27]. Plourde G and Karelis A proposed that adults MHO individuals may be defined (PK definition) when all four of the metabolic markers are met [1]. PK seeks to apply a strict method because their goal was to identify a "true" MHO population which could be different from a non-metabolic syndrome population [1]. According to Truthmaan J et al. the PK criteria, which define MHO by the fulfilment of all proposed criteria, may be the more appropriate definition to determine true MHO [4].

Potential definition of MHO in Adolescents

As in adults, knowing MHO in adolescent patients may help attributing more importance to the treatment of MUHO and prevention to MHO. There is no reason to believe that the mechanisms responsible for developing MHO and MUHO in the adolescent populations would be much different from those discussed above for adults and will not be repeated here. However, there are important concepts specific to the adolescent population that is relevant to discuss. The study by Prince RL et al. was designed to determine the proportions of adolescent with obesity classified as MHO as well as to examine the anthropometric, and the lifestyle predictors that could be associated with MHO [28]. It was, first, observed that about 20% to 33% of adolescent with obesity were at relatively low risk for CVD despite possessing a high level of body fat. Second, that several different adiposity-, diet-, and physical activity-related variables independently predicted MHO status. Finally, the predictors of MHO status varied depending on which classification system we used to determine individuals being MHO or MUHO.

In adolescents such as in adults, the documented prevalence of MHO also varied considerably with proportions ranging from 6–36% in various countries depending on the definition used to determine MHO in adolescents [29-32] and/or the strong effect of pubertal status on metabolic health in obese adolescent [33]. The diversity of the risk factors and their thresholds applied to determine CVD risk may contribute to the inter-study variability in the prevalence of MHO in adolescent patients as observed in adults. For the adolescent, it is recommended that we use the existing criteria for adults [1]. However, at this point, we still do not know the number of these metabolic biomarkers is necessary to correctly define MHO and to clarify this situation we need the collaboration of the scientific community [34]. Old data have demonstrated that being obese at a young age and for a longer period of time is associated with a high risk of T2DM and CVD risk factors later in life which complicated further the definition of MHO in adolescent population [5]. However, this reinforces the importance of weight management, or at a minimum, the prevention of further weight gain in adolescent. Currently, the best predictors of MHO is WC (Odds Ratio [OR], 0.82; 95% confidence interval [CI], 0.77–0.88; P < 0.001) and

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Body Mass Index (BMI) standard deviation score (OR, 0.24; 95% CI, 0.15–0.39; P < 0.001), respectively [28].

Weigensberg MJ et al. found that pediatric patients who consumed a high level of dietary fat had higher levels of IR and acute insulin response to glucose compared with their peers who consumed less fat, but this was exclusive to African Americans (not Caucasians) [34]. This observation do not allow us to speculate on the amount of dietary fat that is responsible for the increased in IR; however, at least in this study, the impact of dietary fat on MHO was modest in comparison with the role of central body fatness [34]. Moderate-Vigorous Physical Activity (MVPA) emerged as an important predictor of MHO [34]. Data demonstrate that doing MVPA may have a clinically meaningful impact on the CVD risks status of adolescent with obesity [34]. This finding is particularly important given how difficult it is for adolescents as well as in adults with obesity to lose and maintain their weight loss over time [17]. From a practical point of view, those individuals classified as having higher CVD risks could be prioritized for care sooner or identified to receive more aggressive therapies than their lower-risk peers [1]. For instance, MHO adolescent may benefit from interventions that help them to maintain their current weight, whereas MUHO may benefit from more intensive health care's to promote weight loss [1].The ethnic homogeneity of the population indicates that there is a need to examine whether MHO varied across ethnicities. The data related to sexual maturation needs also to be collected since sexual maturation influenced body weight and fat distribution differently in female versus male during the adolescence [5]. Since determining the sexual maturation in regular clinical practice is highly complicated Reinehr T et al. [33]. Some authors have found that puberty and age are highly correlated with one another, and age can therefore represent a reasonable approximation of maturity [35-36].

Conclusion

Accumulating evidence suggests that, although the risk of all-cause mortality and CVD events might be higher in people with MHO compared with metabolically healthy people of a normal weight, the risk is substantially lower than in individuals with MUHO [1]. As seen, the prevalence of MHO varies according to the population and definition used in both adults and adolescent patients. There is current¬ly no consensus criterion for the definition of MHO in both adults and adolescent populations, which render difficult the development of clinical practice guidelines for both surveillance and treatment of MHO [1]. Numerous possible mechanisms underlying MHO have been suggested, including adipose tissue distribution and inflammation [1,3]. However, the prognostic value of MHO still needs to be debated. Also, adult and adolescent MHO individuals may be considered as metabolically healthy they can also present other obesity-related comorbidities such as sleep apnea, knee osteoarthritis, poorer body image and many others comorbidities [1,3]. Therefore, studies on comorbidities associated with obesity other than metabolic or CVD risk factors are necessary to assess more accurately whether MHO individuals are really "healthy." On the basis of the above evidence or until future studies can state otherwise, a prudent attitude would be to regularly monitor CVD risk factors in obese adult and adolescent MHO patients (especially elevated triglycerides, glycaemia, HOMA and C-reactive protein as well as low HDL-C), in order to detect as early as possible a negative evolution of their cardio-metabolic profile as well as their other obesity-related comorbidities [1]. In particular, a special surveillance should be applied to prevent any increase in WC as it was previously concluded that the MHO phenotype may be maintained by promoting lower WC. Therefore, every patient with obesity should be motivated to loss weight especially considering that moderate weight loss (5% to 10%) is often sufficient to lower the risk of adverse outcomes [17]. However, how much weight needs to be lost to transform MUHO to MHO status is still unknown. Obviously, this transition might be supported by appropriate lifestyle habits including an adequate diet, and regular physical activity that affect CVD risk, independent of a loss in body fat [17].

References

1. Plourde G, Karelis AD (2014) Current issues in the identification and treatment of metabolically healthy but obese individuals. Nutr Metab Cardiovasc Dis 24: 455-459.
2. Plourde G (2017) Is there a definition of Metabolically Healthy Obese Pediatric Patients? Endocrinol Diabetes Metab J S1(110): 1-3.
3. Jung CH, Lee WJ, Song K (2017) Metabolically healthy obesity: a friend or foe? Korean J Intern Med 32: 611-621.
4. Truthmann J, Mensink GB, Bosy Westphal A (2016) Metabolic Health in Relation to Body Size: Changes in Prevalence over Time between 1997-99 and 2008-11 in Germany. PLoS One 11: e0167159.
5. Plourde G (2002) Impact of obesity on glucose and lipid profiles in adolescents at different age groups in relation to adulthood. BMC Family Practice 3: 18.
6. (2001) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 285: 2486-2497.
7. Rey Lopez JP, de Rezende LF, Pastor Valero M, Tess BH (2014) The prevalence of metabolically healthy obesity: a systematic review and critical evaluation of the definitions used. Obes Rev 15: 781-790.
8. Soriguer F, Gutierrez Repiso C, Rubio Martin E (2013) Metabolically healthy but obese, a matter of time? Findings from the prospective Pizarra study. J Clin Endocrinol Metab 98: 2318-2325.
9. Schroder H, Ramos R, Baena Diez JM (2014) Determinants of the transition from a cardiometabolic normal to abnormal overweight obese phenotype in a Spanish population. Eur J Nutr 53: 1345-1353.
10. Hwang YC, Hayashi T, Fujimoto WY (2015) Visceral abdominal fat accumulation predicts the conversion of metabolically healthy obese subjects to an unhealthy phenotype. Int J Obes (Lond) 39: 1365-1370.
11. Primeau V, Coderre L, Karelis, AD, Brochu M (2010) Characterizing the profile of obese patients who are metabolically healthy. International Journal of Obesity 35: 971-981.
12. Zheng R, Zhou D, Zhu Y (2016) The long- term prognosis of cardiovascular disease and all-cause mortality for metabolically

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healthy obesity: a systematic review and meta-analysis. Journal of Epidemiology and Community Health 70: 1024-1031.

13. Phillips CM (2013) metabolically healthy obesity: Definitions, determinants and clinical implications. Reviews in Endocrine and Metabolic Disorders. 14: 219-227.

14. Roberson LL, Aneni EC, Maziak WA (2014) Beyond BMI: The "Metabolically healthy obese" phenotype and its association with clinical/subclinical cardiovascular disease and all cause mortality a systematic review. BMC Public Health 14: 14.

15. Fan J, Song, Yiqing YC, Yu HR, Zhang W (2013) Combined effect of obesity and cardio-metabolic abnormality on the risk of cardiovascular disease: A meta-analysis of prospective cohort studies. International Journal of Cardiology 168: 4761- 4768.

16. Badoud F, Perreault M, Zulyniak MA, Mutch DM (2014) Molecular insights into the role of white adipose tissue in metabolically unhealthy normal weight and metabolically healthy obese individuals. FASEB Journal 29: 748-758.

17. Plourde G, Prud homme D (2012) Managing Obesity in Adults in Primary Care. CMAJ 184: 1039-1044.

18. Stefan N, Haring HU, Hu FB, Schulze MB (2013) Metabolically healthy obesity: epidemiology, mechanisms, and clinical implications. Lancet Diabetes Endocrinol 1: 152-162.

19. Bluher M (2014) Are metabolically healthy obese individuals really healthy? Eur J Endocrinol 171: R209-R219.

20. Bluher M (2010) The distinction of metabolically 'healthy' from 'unhealthy' obese individuals. Curr Opin Lipidol 21:38-43.

21. Spalding KL, Arner E, Westermark PO (2008) Dynamics of fat cell turnover in humans. Nature 453: 783-787.

22. Esser N, L homme, De Roover A (2013) Obesity phenotype is related to NLRP3 inflammasome activity and immunological profile of visceral adipose tissue. Diabetologia 56: 2487-2497.

23. Phillips CM, Perry IJ (2013) Does inflammation determine metabolic health status in obese and nonobese adults? J Clin Endocrinol Metab 98: E1610-E1619.

24. Karelis AD, Faraj M, Bastard JP (2005) The metabolically healthy but obese individual presents a favorable inflammation profile. J Clin Endocrinol Metab 90: 4145- 4150.

25. Gomez Ambrosi J, Catalan V, Rodriguez A (2014) Increased cardiometabolic risk factors and inflammation in adipose tissue in obese subjects classified as metabolically healthy. Diabetes Care 37: 2813-2821.

26. Wildman RP, Kaplan R, Manson JE (2011) Body size phenotypes and inflammation in the Women's Health Initiative Observational Study. Obesity (Silver Spring) 19: 1482-1491.

27. Alberti KG, Zimmet P, Shaw J (2006) Metabolic syndrome a new worldwide definition. A Consensus Statement from the International Diabetes Federation. Diabet Med 23: 469-480.

28. Prince RL, Kook JL, Ambler KA, Dhaliwal J, Ball GD (2014) Predictors of metabolically healthy obesity in children. Diabetes Care 37: 1462-1468.

29. Bokor S, Frelut ML, Vania A (2008) Prevalence of metabolic syndrome in European obese children. Int J Pediatr Obes 3(Suppl 2): 3-8.

30. Shaibi GQ, Goran MI (2008) Examining metabolic syndrome definitions in overweight Hispanic youth: a focus on insulin resistance. J Pediatr 152: 171-176.

31. Cruz ML, Weigensberg MJ, Huang TTK (2004) The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. J Clin Endocrinol Metab 89: 108-113.

32. Ball GD, Mc Cargar LJ (2003) Childhood obesity in Canada: a review of prevalence estimates and risk factors for cardiovascular diseases and type 2 diabetes. Can J Appl Physiol 28: 117-140.

33. Reinehr T, Wolters B, Knop C, Lass N, Holl RW (2015) Strong effect of pubertal status on metabolic health in obese children: a longitudinal study. J Clin Endocrinol Metab 100: 301-308.

34. Weigensberg MJ, Ball GDC, Shaibi GQ (2005) Dietary fat intake and insulin resistance in black and white children. Obes Res 13: 1630-1637.

35. Mc Gavock JM, Torrance BD, McGuire KA (2009) Cardiorespiratory fitness and the risk of overweight in youth: the Healthy Hearts Longitudinal Study of Cardiometabolic Health. Obesity (Silver Spring) 17: 1802-1807.

36. van Vliet M, von Rosenstiel IA, Schindhelm RK (2009)Identifying the metabolic syndrome in obese children and adolescents: do age and definition matter? Curr Clin Pharmacol 4: 233-238.

DOI: 10.19080/OAJGGM.2018.04.555634

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