Cardiovascular performance in Egyptian obese children and adolescents Soheir S. RezkAllah^a, Gehan M. Abd-El Maksoud^b, Bassant H. El-Refaey^c

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Background/purpose

Overweight and obesity during childhood and adolescence have been identified as a major health problem. In Egypt, increasing rates of obesity in children and adolescents are an emerging concern and have a great impact on the healthcare system. The aim of the study was to compare between nonobese and obese children and adolescents with respect to maximum oxygen consumption, resting heart rate, systolic blood pressure (BP), diastolic BP, pulse pressure, and mean arterial pressure (MAP).

Participants

A total of 360 children and adolescents, aged 7–18 years (the children were aged 7–12 years and adolescents were aged >12-18 years), participated in this study. **Methods**

BMI was computed for all participants to categorize them into two groups: the nonobese group, which included normal-weight participants, and the obese group, which included overweight and obese participants. One-mile endurance run test was completed by all participants. Maximal oxygen consumption was predicted using a specific equation. Heart rate, systolic BP, diastolic BP, pulse pressure, and MAP were assessed at rest. Age differences were investigated in both groups.

Results

Significant differences in all measured variables were found between nonobese and obese groups (P<0.05). There was significant interaction effect between group and age on all measured variables (P<0.05).

Conclusion

Childhood and adolescence obesity affects cardiovascular performance. It decreases aerobic fitness and increases resting BP, heart rate, pulse pressure, and MAP. Adolescents are affected more than children.

Keywords:

adolescents, aerobic fitness, cardiovascular performance, children, obesity

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Introduction

Obesity is an independent risk factor for cardiovascular disease (CVD). The last two decades of the previous century have witnessed dramatic increase in healthcare costs because of obesity and related issues among children and adolescents [1,2]. During the past 3 decades, childhood and adolescent obesity (defined as age-specific and sex-specific BMI at \geq 95th percentile) has increased threefold to sixfold, with the rate of increase dependent on age, sex, and ethnicity [3].

Worldwide, children are becoming overweight and obese at progressively younger ages [4]. In 2014, the global number of overweight children under the age of 5 years was estimated to be over 42 million, with 31 million of them living in developing countries [5]. In developed countries, the prevalence of overweight and obesity in children and adolescents increased by a magnitude of two to five times in the last quarter of the 20th century [6,7]. In almost all the developed countries, the prevalence has been increasing in a similar way in children and adolescents. At least 20 million children under the age of 5 years were overweight in 2005 [5].

Egyptians are considered the fattest among African populations, with nearly 70 percent of the country's adult population being overweight or obese. It is also ranked 14th in the world with respect to obesity, according to the WHO statistics issued for the year 2010 [8]. The high prevalence of overweight and obese children and adolescents in Egypt signals a very alarming trend as seen in the comparison between the two national surveys of 2004 and 2001 [9].

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In Egypt, the prevalence of overweight and obesity among primary school children was estimated in Port Said and Alexandria, and a relatively high prevalence was found [7,10].

Overweight and obese children and adolescents have a high probability of becoming overweight or obese as adults [11,12]. Obesity in childhood, even without the presence of overt complications, has been shown to be a significant predictor of future morbidity and mortality in adulthood [13].

Maximum oxygen uptake (VO₂ max) is generally considered the best measure of cardiorespiratory fitness [14]. VO₂ max reflects the physical fitness of a person [15]. Moreover, although VO₂ max has a major genetic component, it is greatly influenced by the participants activity level, thereby serving also to assess activity levels [14]. This has meant that the determination of VO₂ max is a routine procedure in research projects to assess the cardiovascular capacity of the participants. Obese children had lower relative VO₂ max compared with nonobese children [16].

Several studies have investigated the relationship between adiposity in children and the cardiovascular capacity. Sorof *et al.* [17] have found that blood pressure (BP) increases with BMI in children. Obese children are three times more likely to have hypertension compared with nonobese children [18]. Obese children who have high BP were at increased risk for developing serious cardiac problems [19]. Resting pulse rate is positively associated with BP and adiposity [20]. Mean arterial pressure (MAP) was found to be elevated in obese and overweight school children [21]. However, there is lack of literature regarding the interaction effect between body weight and age on the measured variables in children and adolescents.

This study was conducted to examine the main and interaction effects of obesity and age regarding VO_2 max, resting heart rate (RHR), systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP), and MAP.

Methods

Study design

An observational, cross-sectional study was approved by the ethical committee of the Faculty of Physical Therapy, Cairo University. The independent variables of this study were weight (obese and nonobese) and age (children and adolescents), whereas the dependent variables were VO₂ max, RHR, SBP, DBP, PP, and MAP.

Participants

The study population comprised school and college students from grade 1 primary school to first grade high school education, aged 7–18 years [22]. School students were selected from nine public schools in Giza and college students were selected from three faculties in Cairo University, which agreed to share our work. They were chosen from the Cairo University zone to enable the 1-mile endurance run at Cairo University Courts. Totally, 500 participants were screened, from whom 360 students were selected and they continued the study. In each institute, 30 participants (15 nonobese and 15 overweight or obese) were selected. All the participants were required to bring a consent form written and signed by their parents before their participation in the study.

Eligible participants were categorized into the nonobese group, which included 180 normal-weight participants, and the obese group, which included 180 overweight and obese participants. Eligibility criteria for participants were being normal, overweight, or obese, as defined by WHO 2006 [5]. The nonobese group included normal-weight (fifth to <85% percentile) students. The obese group included overweight (85th to <95th percentile) and obese (\geq 95th percentile) students. Exclusion criteria included being underweight (<5%) or extremely obese (BMI \geq 120% of the 95th percentile of BMI-for-age or BMI \geq 35.0 kg/m²), having syndromal obesity, having a chronic illness, taking medications associated with weight change, and inability to complete the presidential 1-mile run test.

Measurements

Assessments were made by the research team members. Height was measured in centimeters and weight in kilograms for computation of BMI. BMI-for-age percentiles were computed from the growth charts of WHO 2006 [5].

Aerobic fitness (maximum oxygen consumption)

Evaluation of aerobic fitness was performed by calculation of VO₂ max. VO₂ max was predicted from the 1-mile endurance run time using age, sex, and BMI-specific equation: VO₂ max (ml/kg/min)= $(0.21\times(age\timessex \text{ code}))-(0.84\times\text{BMI})-(8.41\times\text{mile time})$ + $(01.34\times\text{mile time}\times\text{mile time})+(108.94)$, where age is in years, sex code is 0 for female participants and 1 for male participants, BMI is in kg/m², and mile time is in minutes [23].

Resting heart rate, systolic blood pressure, diastolic blood pressure, pulse pressure, and mean arterial pressure

RPR, SBP, and DBP were assessed three times (with 1min re-filling period) in the right arm at the level of the heart. Assessments of BP were made after 5 min of quiet seated rest using Omron HEM 7120 E IntelliSense Automatic Blood Pressure Monitors (Model: M2 Basic (HEM-7120-E), Serial No: 20140804386VG, OMRON Healthcare Co., Ltd. Koyoto, Japan, Made in Vietnam) and appropriately sized cuffs. When errors or disparate readings occurred, BP assessment was repeated by auscultation (sphygmomanometer and stethoscope). PP was computed as SBP minus DBP. MAP was computed as the sum of DBP and one-third PP. Measurements were taken in a quiet environment and confidentiality was maintained [24].

Data analyses

All statistical analyses were performed using the statistical package for the social sciences program, version 20, for Windows (SPSS; SPSS Inc., Chicago, Illinois, USA). Before the final analysis, data were screened for normality assumption and for the presence of extreme scores. This was done as a prerequisite for parametric calculation of the analysis of difference and analysis of relationship measures. Descriptive analysis using histograms with a normal distribution curve showed that the data were normally distributed and did not breach the parametric assumption for the measured dependent variables. Additionally, testing for the homogeneity of covariance using Box's test revealed that there was no significant difference (P>0.05). Box and whiskers plots of the tested variables after removal of the outliers were determined. Normality test of data using the Shapiro-Wilk test was used for testing the normality of data for all dependent variables. All these findings allowed the researchers to conduct parametric analysis.

Two-way multivariate analysis of variance was used to compare the variables of interest in different groups and age and also to determine the interaction effect between both independent variables (age and weight) on all dependant variables. The α level was set at 0.05.

Results

A total of 500 participants were evaluated and 360 were included in the final data analysis. They were divided into two groups: the nonobese group and the obese group, the latter including overweight and obese participants. Each group included 180 participants. The mean age of the nonobese group was 14.67 years and that of the obese group was 14.39 years. The nonobese group included 44.4% boys and 55.5% girls, whereas the obese group included 37.2% boys and 62.7% girls. In the nonobese group 38.8% were children and 61.1% were adolescents, whereas in the obese group 50% were children and 50% were adolescents. Pearson's χ^2 -test showed no significant difference in sex between groups (*P*=0.671), nor any significant difference in age category between groups (*P*=0.245) (Table 1).

Main effect of weight without regard to age

Considering the effect of weight on all dependent variables, two-way multivariate analysis of variance revealed that there was a significant difference between the two groups for all dependent variables (F=0.107, P=0.0001). Table 2 shows the mean±SD and multiple pairwise comparisons (post-hoc tests) for all dependent variables between the two groups. Multiple pairwise comparison tests revealed that there was significant reduction in VO₂ max in the obese group compared with the nonobese group (P<0.05), and a significant increase in RHR, SBP, DBP, PP, and MAP (P<0.05) in the obese group.

Interaction effect between weight and age

Statistical analysis revealed that there were significant differences in age effect (F=187.206, P=0.0001) and

Table 1 Frequency distribution of sex and age betwee	een		
nonobese and obese groups			

	Nonobese group (<i>n</i> =180) [<i>n</i> (%)]				
Sex					
Boys	80 (44.44)	67 (37.22)	0.671		
Girls	100 (55.56)	113 (62.78)			
Age stage					
Children	70 (38.9)	90 (50)	0.245		
Adolescents	110 (61.1)	90 (50)			
BMI %	30.88 (24.9)	93.22 (6.32)			

P<0.05, significant.

Table 2 Descriptive statistics and multiple pairwise
comparison tests (post-hoc tests) for maximum oxygen
consumption, resting heart rate, systolic blood pressure,
diastolic blood pressure, pulse pressure, and mean arterial
pressure in the tested groups without regard to age effect

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Parameters	Nonobese group (<i>n</i> =180)	Obese group (<i>n</i> =180)	P value
VO ₂ max (ml/kg/min)	53.009±11.2	44.88±1.87	0.0001*
RHR (bpm)	99.11±12.89	122.67±4.76	0.0001*
SBP (mmHg)	107.11±16.61	129.5±5.27	0.0001*
DBP (mmHg)	77.33±5.29	88.17±5.29	0.0001*
PP (mmHg)	30.28±12.25	49.17±5.22	0.0001*
MAP (mmHg)	87.42±10.56	96.71±10.56	0.0001*

DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; RHR, resting heart rate; SBP, systolic blood pressure; VO₂ max, maximal oxygen consumption. *Significant at α <0.05.

interaction effect (F=74.562, P=0.0001), which indicates that the effect of the tested group (first independent variable) on the dependant variables was influenced by the age effect (second independent variable) and between-participant effect (F=324.077, P=0.0001). Table 3 presents the mean±SD and multiple pairwise comparisons for all dependent variables in both groups at different ages.

With regard to the age effect, multiple pairwise comparison tests revealed that there was significant increase (P<0.05) in DBP and a significant reduction (P<0.05) in RHR, SBP, and PP in the children compared with that in adolescents in the obese group, whereas there was a nonsignificant difference (P>0.05) in VO₂ max and MAP in children compared with that in adolescents in the obese group. There was significant increase (P<0.05) in RHR and significant reduction (P<0.05) in VO₂ max, SBP, DBP, PP, and MAP in the children compared with the adolescents in the nonobese group.

With regard to weight effects, multiple pairwise comparison tests revealed that there was significant reduction in VO₂ max in the obese group (P<0.05) compared with that in the nonobese group among children and adolescents, whereas there was significant increase in RHR, SBP, PP, and MAP (P<0.05) in the obese group among both children and adolescents. Additionally, there was significant increase (P<0.05) in DBP in the obese group in children and a nonsignificant difference (P>0.05) between the two groups in adolescents.

Discussion

This study extends results from previously published studies of pediatric obesity by comparing aerobic fitness and cardiovascular performance between nonobese and obese children and adolescents. In addition, we evaluated the age-related differences of these variables between the two groups.

Aerobic fitness (maximum oxygen consumption)

The present study showed that there was a significant difference in VO2 max between nonobese and obese participants. The obese children and adolescents had lower VO_2 max compared with the nonobese group. This may be attributed to the poor performance in the 1mile run as excess weight reduces activity level. Generally speaking, being physically active is uncommon in Egyptian culture, and this contributes to a sedentary lifestyle and consequently to obesity [25]. Most studies on habitual physical activity in children have found that obese children are less active than their nonobese peers [26–28] and have poorer fundamental movement skills compared with their normal-weight counterparts [29]. Excessive body fat exerts an unfavorable effect on cardiac function, particularly during exhaustive exercise [30]. Loss of weight during weight reduction programs in the obese increases the VO₂ max because of withdrawal of fat-induced inhibitory action on oxygen utilization by the body musculature [31]. Elevated myocardial oxidative stress has been reported in patients with obesity. In obese individuals there is increase in type II muscle fibers and decrease in type I muscle fibers, which may have an important effect on reduced oxygen uptake [32]. The functional consequence of lower VO_2

Table 3 Descriptive statistics and multiple pairwise comparison tests (post-hoc tests) for maximum oxygen consumption, resting heart rate, systolic blood pressure, diastolic blood pressure, pulse pressure, and mean arterial pressure in the tested groups at different ages

Dependent variables	Nonobese group			Obese group		
	Adolescents	Children	Adole	escents	Children	
VO ₂ max	53.07±2.62	52.94±0.37	37.55	5±6.69	48.45±5.84	
RHR	95.87±15.20	105.42±0.91	124.66±5.27		120.66±3.10	
SBP	117.35±11.40	91.42±10.46	131.3	3±3.88	127.66±5.82	
DBP	78.66±0.47	74±9.12	79.64	1±7.12	82±2.17	
PP	36.64±10.08	17.42±3.83	52.66	6±4.05	45.66±3.70	
MAP	92.51±8.11	79.8±9.41	96.21±1.23		97.21±3.37	
Age effect (adolescents ve	s. children)					
Dependent variables	VO ₂ max	RHR	SBP	DBP	PP	MAP
Nonobese	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*
Obese	0.831	0.001*	0.0001*	0.0001*	0.0001*	0.256
Weight effect (nonobese v	vs. obese)					
Dependent variables	VO ₂ max	RHR	SBP	DBP	PP	MAP
Adolescent	0.0001*	0.0001*	0.0001*	0.196	0.0001*	0.0001*
Children	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*	0.0001*

DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; RHR, resting heart rate; SBP, systolic blood pressure; VO₂ max, maximal oxygen consumption. *P<0.05, significant at the α level.

max (ml/kg/min) among obese individuals than among nonobese individuals could be their poorer performance in weight-dependent activities. In running, walking, stair climbing, etc. they have more body mass to transport, which requires and costs energy [30]. The current results are supported by Shirur *et al.* [30], Chouhan *et al.* [33], and Berndtsson *et al.* [16]. They concluded that there was a significant reduction in VO₂ max with an increase in BMI.

In contrast to the nonobese group, no age-related differences were detected in obese children and adolescents. This result is inconsistent with that of Sunburg [34], who found that VO₂ max/kg was uninfluenced by age in normal-weight boys. However, the result is consistent with that of Berndtsson *et al.* [16], who reported that no age-related differences were found in relative VO₂ max in the obese group, in contrast to the reference group, which saw age-related differences in VO₂ max.

Resting heart rate

The results of the present study showed that there was a significant difference in RHR between nonobese and obese groups, with a higher value obtained in obese participants. This could be sympathetic attributed to nervous system hyperactivity [35]. Additionally, positive a correlation between resting pulse rate and BP was found. The current results are consistent with those of Sorof et al. [36], who reported that a higher heart rate was observed in obese compared with nonobese adolescents. Also, Norman et al. [37] showed that RHR was significantly greater in overweight than in nonoverweight adolescents.

There was a significant difference in RHR between children and adolescents in the nonobese and obese groups. Pulse rate was higher in children compared with that in adolescents in the nonobese group, whereas it was higher in adolescents than in children in the obese group. This suggests that the adiposity in adolescence increases the risk for CVD more than in childhood. This result comes in agreement with that of Silvetti *et al.* [38], who reported that with increasing age there is a progressive and significant decrease in heart rate.

Blood pressure

A strong relationship has been established between obesity and elevated BP for both adults and children, which synergistically increases cardiovascular risk [39]. The association between BP and CVD indicators such as body size or adiposity is not yet established for children, but the association with body weight or BMI is strong [40].

The results of the present study revealed that there was a significant difference in SBP and DBP between the nonobese and obese groups. The SBP and DBP were higher in obese participants than in nonobese participants. This may be attributed in part to arterial stiffness, which increases MAP [41]. Chronic inflammation is also often cited as a key etiologic factor in the development of hypertension [41-43]. Higher SBP levels may reflect the progressive stiffening of the arterial wall, changes in the vascular structure, and the development of atherosclerosis [44]. The adipose tissue-derived hormone leptin has been implicated in mediating obesity-induced increases in BP [45]. Many studies [46,47] support current results. They showed that at the start of turning overweight, the effect of adiposity on BP increases fourfold.

There was a significant difference in SBP between children and adolescents in the nonobese and obese groups. SBP was higher in adolescents than in children in the nonobese and obese groups. DBP differed significantly between children and adolescents in each group and between groups during childhood only. It was higher in adolescents than in children in the nonobese group and higher in children than in adolescents in the obese group. This agrees with the findings of Tu *et al.* [46], who stated that the risk for elevated BP is increased in youth aged 10 years or older with BMI-for-age percentile ≥ 85 th.

Pulse pressure

It has been reported to be a more reliable marker of CHD than either SBP or DBP in the adult population [48]. The results of the current study showed that there was a significant difference in PP between nonobese and obese groups, with PP higher in obese participants than in nonobese participants. This could be attributed to elevated SBP and increased arterial stiffness, which increases the MAP [44]. Change in PP for a given stroke volume is directly related to vascular stiffness, which could be either functional or structural in nature [48]. The results of the current study come in line with those of Chandramohan et al. [49], who found that there is a significant independent association between high PP and wide waist circumference. They observed a significantly higher mean PP in the obese, as well as in those with high waist circumference (WC) and high BP.

There was a significant difference in PP between children and adolescents in the nonobese and obese

groups. PP values were higher in adolescents than in children in both groups. This indicates that PP increases with age in normal-weight children and adolescents; it increases also with age due to adiposity as the SBP increases with age in obese children and adolescents.

Mean arterial pressure

The results of the current study revealed that there was a significant difference in MAP between nonobese and obese groups. MAP was observed to be higher in obese participants compared with that in nonobese participants. This is consistent with the study of Mittal *et al.* [50], who found that physical inactivity related to obesity leads to increased MAP. Hvidt [51] also found that there was a significant difference in MAP between obese and normal-weight children and adolescents.

Regarding age, MAP differed significantly between nonobese and obese children and adolescents, with higher values seen in obese participants. It increased significantly in adolescents more than in children in the nonobese group only. This indicates that MAP increases with age in normal-weight young people, but it does not change with age in relation to obesity. Clark *et al.* [24] reported that MAP increases from elementary to middle to high school, which supports the present results.

Thus, it can be concluded that obesity in children and adolescents lowers aerobic fitness and negatively affects cardiovascular performance, which differs with age and is greater in adolescents than in children.

Limitations

Since this study was confined to a single geographical area (Giza), extrapolation of the results to other areas requires validation by larger studies. Another limitation is the grouping of overweight and obese participants into a single group to enhance recruitment. It is recommended to study each category separately.

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Conflicts of interest

There are no conflicts of interest.

References

- Wang G, Dietz WH. Economic burden of obesity in youths 2. Aged 6 to 17 years: 1979–1999. Pediatrics 2002; 109:E81–E81.
- 2 Dietz WH. Health consequences of obesity and youth: childhood predictors of adult disease. Pediatrics 1998; 101:518–525.
- 3 Wang Y, Beydoun M. The obesity epidemic in the United States, gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta regression analysis. Epidemiol Rev 2007; 29:6–28.
- 4 Lobstein T, Baur L, Uauy R. Obesity in children and young people: a crisis in public health. Obes Rev 2004; 5(Suppl 1):4–85.
- 5 WHO. Obesity and overweight, WHO Fact Sheet No. 311. Geneva: World Health Organization; 2006.
- 6 World Health Organization (WHO): Global Health Observatory (GHO) Repository. 2014. Available at: http://apps.who.int/gho/data/node.main (Accessed 6 October 2015)
- 7 Lissau I. Overweight and obesity epidemic among children. Answer from European countries. Int J Obes Relat Metab Disord 2004; 28(Suppl 28): S10–S15.
- 8 WHO Statistical Information System (WHOSIS). World Health Statistics 2010. Medical /Public Health. pp. 117–124.
- 9 Shaheen FM, Hathout M, Tawfik AA. National survey of obesity in Egypt (Final Report). Egypt: National Nutrition Institute; 2004.
- 10 El-Shafie AM, Hogran HH, Dohein AM. Prevalence of obesity in primary school children living in Alexandria Governorate. Menoufia Med J 2014; 27:529–532.
- 11 Lawlor D, Smith G, O'Callaghan M. Epidemiologic evidence for the fetal over-nutrition hypothesis: findings from the mater-university study of pregnancy and its outcomes. Am J Epidemiol 2007; 165:418–424.
- 12 Singh AS, Mulder C, Twisk JWR, Twisk C, van Mechelen W, Chinapaw MJM. Tracking of childhood overweight into adulthood: a systematic review of the literature. Obes Rev 2008; 9:474–488.
- 13 Baker JL, Olsen LW, Sørensen TI. Childhood body mass index and the risk of coronary heart disease in adulthood. N Engl J Med 2007; 357:2329–2337.
- 14 Umesh KP, Joshi AS. Comparison of VO₂max in obese and non-obese young Indian population. Indian J Physiol Pharmacol 2011; 55:188–192.
- 15 Dagan SS, Segev S, Novikov I, Dankner R. Waist circumference vs. body mass index in association with cardiorespiratory fitness in healthy men and women: a cross sectional analysis of 403 subjects. Nutr J 2013; 12:12.
- 16 Berndtsson G, Mattsson E, Marcus C, Larsson UE. Age and gender differences in VO₂max in Swedish obese children and adolescents. Acta Paediatr 2007; 96:567–571.
- **17** Sorof JM, Lai D, Turner J, Poffenbarger T, Portman RJ. Overweight, ethnicity, and the prevalence of hypertension in school-aged children. Pediatrics 2004; 113:475–482.
- 18 Wiegand S, Dannemann A, Krude H, Grüters A. Impaired glucose tolerance and type 2 diabetes mellitus: a new field for pediatrics in Europe. Int J Obes 2005; 29:S136.
- 19 Hanevold C, Waller J, Daniels S, Portman R, Sorof J, International Pediatric Hypertension Association. The effects of obesity, gender, and ethnic group on left ventricular hypertrophy and geometry in hypertensive children: a collaborative study of the International Pediatric Hypertension Association. Pediatrics 2004; 113:328.
- **20** Rabbia F, Grosso T, Cat GG, Conterno A, de Vito B, Mulatero P, *et al.* Assessing resting heart rate in adolescents: determinants and correlates. J Hum Hypertens 2002; 16:327–332.
- 21 Ogunleye AA, Sandercock GR, Voss C, Eisenmann JC, Reed K. Prevalence of elevated mean arterial pressure and how fitness moderates its association with BMI in youth. Public Health Nutr 2013; 16:2046–2054.
- 22 Knoppert D, Reed M, Benavides S, Totton J, Hoff D, Moffett B, et al. Paediatric age categories to be used in differentiating between listing on a model essential medicines list for children [position paper]. 20 April 2007
- 23 Plowman SA, Meredith MD. Fitnessgram/activitygram reference guide. 4th ed. Dallas, TX: The Cooper Institute; 2013.
- 24 Clark BR, White ML, Royer NK, Burlis TL, DuPont NC, Wallendorf M, Racette SB. Obesity and aerobic fitness among urban public school students in elementary, middle, and high school. PLoS One 2015; 10: e0138175.
- 25 Barsoum G, Ramadan AM, Roushdy R, Rashed A, Hosny O. Survey of young people of Egypt, Final report, Population Council, Wesrt Asia and North Africa Office, Population Council, January 2011.
- 26 Hills AP, King NA, Armstrong TP. The contribution of physical activity and sedentary behaviors to the growth and development of children and

adolescents: implications for overweight and obesity. Sports Med 2007; 37:533-545.

- 27 Trost SG, Kerr LM, Ward DS, Pate RR. Physical activity and determinants of physical activity in obese and non-obese children. Int J Obes Relat Metab Disord 2001; 25:822–829.
- 28 Planinsec J, Matejek C. Differences in physical activity between non overweight, overweight and obese children. Coll Antropol 2004; 28: 747–754.
- 29 Okely AD, Booth ML, Chey T. Relationships between body composition and fundamental movement skills among children and adolescents. Res Q Exerc Sport 2004; 75:238–247.
- 30 Shirur SY, Rajeshwari L, Swathi HN. Effect of increased adiposity on cardiorespiratory fitness of young Indian individuals. Int J Biomed Res; 2014; 5:662–664.
- 31 Chatterjee S, Chatterjee P, Bandyopadhyay A. Cardiorespiratory fitness of obese boys. Indian J Physiol Pharmacol 2005; 49:353–357.
- 32 Prabhu S, Padmanabha BV, Doddamani BR. Correlation between obesity and cardiorespiratory fitness. Int J Med Sci Public Health 2013; 2: 300–304.
- 33 Chouhan S, Trigotra S, Dashora LS, Mangat EK. An assessment of cardiorespiratory fitness in normal weight, overweight and obese young adults. Int J Basic Appl Physiol 2014; 3:24–29.
- **34** Sunberg S. Maximal oxygen uptake in relation to age in blind and normal boys and girls. Acta Paediatr Scand 1982; 71:603–608.
- **35** Sorof J, Daniels S. Obesity hypertension in children a problem of epidemic proportions. Brief review. Hypertension 2002; 40:441–447.
- 36 Sorof JM, Poffenbarger T, Franco K, Bernard L, Portman RJ. Isolated systolic hypertension, obesity, and hyperkinetic hemodynamic states in children. J Pediatr 2002; 140:660–666.
- 37 Norman AC, Drinkard B, McDuffie JR, Ghorbani S, Yanoff LB, Yanovski JA. Influence of excess adiposity on exercise fitness and performance in overweight children and adolescents. Pediatrics 2005; 115:e690–e696.
- 38 Silvetti MS, Drago F, Ragonese P. Heart rate variability in healthy children and adolescents is partially related to age and gender. Int J Cardiol 2001; 81:169–174.

- 39 Rosner B, Prineas R, Daniels SR, Loggie J. Blood pressure differences between blacks and whites in relation to body size among US children and adolescents. Am J Epidemiol 2000; 151:1007–1019.
- 40 Cole TJ, Flegal KM, Nicholls D, Jackson AA, Schild HH. Body mass index cut offs to define thinness in children and adolescents: international survey. BMJ 2007; 335:194.
- 41 Dart AM, Kingwell BA. Pulse pressure a review of mechanisms and clinical relevance. J Am Coll Cardiol 2001; 37:975–984.
- 42 Rizvi AA. Hypertension, obesity, and inflammation: the complex designs of a deadly trio. Metab Syndr Relat Disord 2010; 8:287–294.
- 43 Vaziri ND, Rodriguez-Iturbe B. Mechanisms of disease: oxidative stress and inflammation in the pathogenesis of hypertension. Nat Clin Pract Nephrol 2006; 2:582–593.
- 44 Weber T. Low-grade systemic inflammation, arterial structure and function, and hypertension. Am J Hypertens 2010; 23:346.
- 45 Carethers M, Blanchette PL. Pathophysiology of hypertension. Clin Geriatr Med 1989; 5:657–674.
- 46 Tu W, Eckert GJ, DiMeglio LA, Yu Z, Jung J, Pratt JH. Intensified effect of adiposity on blood pressure in overweight and obese children. Hypertension 2011; 58:818–824.
- 47 Raj M, Sundaram KR, Paul M, Sudhakar A, Kumar RK. Body mass index trend and its association with blood pressure distribution in children. J Hum Hypertens 2010; 24:652–658.
- 48 Safar ME. Systolic blood pressure, pulse pressure and arterial stiffness as cardiovascular risk factors. Curr Opin Nephrol Hypertens 2001; 10: 257–261.
- 49 Chandramohan G, Kalantat-Zadih K, Kermah D, Go SCM, Vaziri ND, Norris VKC. Relationship between obesity and pulse pressure in children: results of the National Health and Nutrition Survey (NHANES) 1988–1994. J Am Soc Hypertens 2012; 6:277–283.
- 50 Mittal M, Arora M, Bachhel R, Kaur N, Sidhu RA. Physical activity, indices of obesity and mean arterial blood pressure: does place of living matters? Rural vs. urban. JCDR 2011; 5:1038–1042.
- 51 Hvidt KN. Blood pressure and arterial stiffness in obese children and adolescents. Dan Med J 2015; 62:1–22.