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Letters

Tracking Body Mass Index From Childhood to Adulthood for Subclinical Cardiovascular Diseases at Adulthood

Subclinical cardiovascular diseases (CVDs), including arterial stiffness measured with carotid-femoral pulse wave velocity (cfPWV), carotid atherosclerosis measured with carotid artery intima-media thickness (cIMT), and left ventricular hypertrophy measured with left ventricular mass index (LVMI), may serve as the surrogate indexes of CVD (mainly coronary heart diseases and stroke). Previous studies have examined the relationship between childhood body mass index (BMI) and adult CVD risk, as well as subclinical CVD (1,2). However, the effect of BMI change from childhood to adulthood has not been clearly established. Moreover, to our knowledge, no study has attempted to investigate this association from childhood in Asian population. Thus, in the present study, we aimed to investigate the joint effect of overweight status at both childhood and adulthood on long-term subclinical CVD in a Chinese population.

The Beijing Blood Pressure Cohort study was a school-based prospective study that started in 1987, and the characteristics of this study have been described elsewhere (3,4). The average follow-up duration was 22.9 ± 0.6 years. The current analysis was restricted to 1,225 individuals who had baseline information at childhood and experienced the examination at adulthood in 2011. Childhood overweight (including obesity) was defined as a BMI ≥ the sex- and age-specific 85th percentile in the baseline population, and adult overweight (including obesity) was defined as a BMI ≥24 kg/m^2. We dichotomized markers of subclinical CVD, including cfPWV, cIMT, and LVMI, into values equal or above the upper quartile, and logistic regression model was used to examine the joint effect of overweight status in childhood and adulthood on subclinical CVD in adulthood.

Subjects who were overweight in childhood were more likely to be overweight in adulthood than those who were not overweight in childhood (88.1% vs. 45.8%; $p < 0.001$). As is shown in **Table 1**, subjects who kept their overweight status from childhood to adulthood had the highest incidence rates of high cfPWV (36.2%), high cIMT (42.3%), and high LVMI (50.3%) among the BMI category groups. Compared with subjects who were not overweight at both childhood and adulthood, those who were overweight from childhood to adulthood had the highest risk of subclinical CVD, especially for high cIMT (odds ratio [OR]: 3.73, 95% confidence interval [CI]: 2.37 to 5.85) and high LVMI (OR: 9.58, 95% CI: 5.92 to 15.49), followed by those who were not overweight in childhood but were overweight in adulthood for high cIMT (OR: 2.92, 95% CI: 2.04 to 4.18) and high LVMI (OR: 5.96, 95% CI: 4.01 to 8.86), with full adjustments of sex, age, length of follow-up, blood pressure (BP) at both childhood and adulthood, use of antihypertensive medications, and other factors at adulthood (type 2 diabetes, dyslipidemia, smoking, drinking, and physical activity). Interestingly, subjects who were overweight in childhood but changed to nonoverweight status in adulthood had a low risk of high cIMT (OR: 1.95, 95% CI: 0.68 to 5.53) and high LVMI (OR: 2.43, 95% CI: 0.85 to 6.95). In addition, the joint effect of overweight in childhood and adulthood on high cfPWV was statistically significant after adjustment for sex, age, and length of follow-up. However, the effect disappeared after further adjustment for BP in childhood and adulthood and other factors in adulthood, suggesting that the effect of BMI on long-term risk of high cfPWV was mediated through BP and other factors.

In our study, we found that the effect of childhood overweight status on long-term high cIMT and high LVMI was independent of BP and other factors, whereas the effect on high cfPWV was dependent on BP and other factors. In addition, if an overweight child became an adult with healthy weight, the risk of subclinical CVD became lower. Consistent with our findings, the pooled data from 4 child cohort studies by Juonala et al. (5) reported that subjects with overweight in childhood but became nonobese in adulthood had similar cardiovascular risk profiles, including type 2 diabetes, hypertension, dyslipidemia, and carotid artery atherosclerosis, when

TABLE 1 ORs (95% CIs) of Subclinical Cardiovascular Diseases in Adulthood According to BMI Status in Childhood and Adulthood

Subclinical Cardiovascular Diseases at Adulthood	Adulthood Nonoverweight		Adulthood Overweight	
	Childhood Nonoverweight (n = 564)	Childhood Overweight (n = 22)	Childhood Nonoverweight (n = 476)	Childhood Overweight (n = 163)
High cfPWV				
Incidence	17.7 (100/564)	22.7 (5/22)	29.2 (139/476)	36.2 (59/163)
Model 1	Reference	1.46 (0.52-2.61)	1.92 (1.42-2.61)*	2.67 (1.78-4.02)*
Model 2	Reference	1.40 (0.49-4.02)	1.00 (0.69-1.45)	1.02 (0.62-1.68)
High cIMT				
Incidence	12.2 (69/564)	22.7 (5/22)	33.6 (160/476)	42.3 (69/163)
Model 1	Reference	2.11 (0.75-5.90)	3.63 (2.65-4.98)*	5.27 (3.53-7.87)*
Model 2	Reference	1.95 (0.68-5.53)	2.92 (2.04-4.18)*	3.73 (2.37-5.85)*
High LVMI				
Incidence	9.0 (51/564)	22.7 (5/22)	35.1 (167/476)	50.3 (82/163)
Model 1	Reference	2.90 (0.99-8.30)	5.44 (3.85-7.67)*	10.18 (6.69-15.51)*
Model 2	Reference	2.43 (0.85-6.95)	5.96 (4.01-8.86)*	9.58 (5.92-15.49)*

Values are % (n/N) or odds ratio (95% confidence interval). *OR different from 1, $p < 0.001$. Model 1: adjusted for sex, age, and length of follow-up; Model 2: additionally adjusted for childhood systolic and diastolic blood pressure, adult systolic and diastolic blood pressure, use of antihypertensive medications, and other factors at adulthood (type 2 diabetes, dyslipidemia, smoking, drinking, and physical activity).

BMI = body mass index; cfPWV = carotid-femoral pulse wave velocity; cIMT = carotid artery intima-media thickness; LVMI = left ventricular mass index.

compared with subjects who had healthy weight at both childhood and adulthood. These findings suggest that the effect of childhood overweight on long-term CVD risk could be attenuated by adopting weight loss in adulthood. Yet, early prevention of childhood overweight should not be ignored, as BMI strongly tracks from childhood to adulthood.

There are some limitations to this study. First, although the present study consisted of more than 1,000 participants, the sample size in the subgroups might be insufficient. Second, many risk factors (diabetes, dyslipidemia, smoking, drinking, and physical activity) in childhood were not measured; thus, we cannot rule out the confounding effects of those factors on our observed association. Third, BMI was used as a measure of overweight status, which reflects both fat and fat-free mass. Thus, it might be inaccurate to assess the true obesity status.

In conclusion, childhood overweight status increased the risk of long-term subclinical CVD in combination with overweight status in adulthood. Most importantly, subclinical CVD could be mostly attenuated if overweight or obese children achieve normal weight at adulthood. This study enhances the importance of early prevention and life-course control and treatment of overweight and obesity.

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