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Concordance of haemoglobin A1c, blood pressure and C-reactive protein between children and their parents in Chinese households

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Summary

Background: China has the world's highest diabetes prevalence, which along with hypertension and inflammation continues to grow particularly among children. Little is known about the strength of the association of these cardiometabolic risk factors between parents and their children; thus, the potential of household-based strategies to reduce risk is unknown.

Objectives: The objective of the study is to examine the parent–child association for haemoglobin A1c (HbA1c), blood pressure (BP) and C-reactive protein (CRP) in a large, geographically diverse Chinese sample.

Methods: In 940 parent–child pairs (children aged 7–17 years) who participated in the 2009 China Health and Nutrition Survey, we measured each individual's HbA1c and CRP using fasting blood and BP. We used sex-specific random-effects linear regression to examine the parent–child association for these risk factors, accounting for within-family clustering.

Results: Child's HbA1c was positively associated with parental HbA1c. Beta coefficients ranged from 0.06 (95% CI 0.03–0.12) for father–daughter to 0.43 (95% CI 0.28–0.58) for mother–son pairs. We also detected a positive mother–daughter association for BP and positive father–child associations for CRP.

Conclusion: The statistically significant parent–child association for HbA1c, BP and CRP in Chinese families suggests that household-based interventions could be useful for confronting the high rates of diabetes, hypertension and inflammation in China.

Keywords: Cardiometabolic risk factors, China, household structure, parent–offspring association, urbanization.

Introduction

The prevalence of cardiometabolic disease (CMD) risk factors (e.g. obesity, diabetes and hypertension) has increased dramatically over the past two decades in China, with a faster increase in children relative to adults (1,2). Children share genetic and environmental factors, and health behaviours with parents (3), which could underlie clustering in CMD risk factors in the household. Parental obesity has been associated with childhood obesity (4). Correlations of fasting insulin (5) and blood pressure (BP) (6) between children and their parents were also observed in Western countries. To our knowledge, no study has determined the parent–child associations for haemoglobin A1c (HbA1c) and BP in a large, geographically diverse Chinese sample.

Much of the literature in China has focused on the role of grandparents rather than parents. Partially, this is because three-generation (grandparents, parents and children) households are common in China. Li *et al.* (7) found that living with grandparents was positively associated with childhood obesity, because grandparents are more likely to indulge their grandchildren with modern, unhealthy food and screen-based sedentary behaviours, especially in single-child households (7). Nevertheless, to our knowledge, no study has examined whether living with a grandparent is associated with children's obesity-related CMD risk factors.

Furthermore, whether the parent–child concordance in these risk factors differs when grandparents live in the household has been unaddressed in the literature. Understanding these questions will help identify children at high risk of CMD.

To address these gaps, we used data from children and parents enrolled in the China Health and Nutrition Survey (CHNS) in 2009 to determine the parent–child associations for HbA1c and BP, because diabetes and hypertension have been growing particularly fast in Chinese children (1). Given existing evidence in the impact of inflammation on the development of CMD risk factors (8), we also examined the parent–child association for C-reactive protein (CRP). We further tested whether household structure (i.e. living with grandparents or not and having siblings or not) relates to children's HbA1c, BP and CRP or modifies the parent–child association for these factors.

Methods

CHNS

The CHNS collected health data in 228 communities in nine diverse provinces throughout China (North: Heilongjiang, Liaoning; Central: Shandong, Henan, Jiangsu; and South: Hunan, Hubei, Guangxi, Guizhou) in eight survey rounds from 1989 to 2009. We used questionnaires to collect sociodemographic, anthropometric and health information. The 2009 survey collected fasting blood for the first time. Using a multistage, random cluster design, a stratified probability sample was used to select counties and cities stratified by income using State Statistical Office definitions (9). Communities and households were then selected from these strata. The CHNS cohort initially mirrored national age–sex–education profiles (10), and these initial households were followed over time. Details on the survey procedures are described elsewhere (11). The study was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill, the China-Japan Friendship Hospital, Ministry of Health and the National Institute for Nutrition and Health, Chinese Center for Disease Control and Prevention. Subjects gave informed consent for participation.

Analysis sample

We used data from all children and adolescents aged 7–17 years in the 2009 CHNS, when biomarker measurements were collected from school-aged children who were living at home ($n = 1111$). We excluded individuals who were missing all HbA1c, BP and CRP data ($n = 65$), living with neither of their parents ($n = 28$), missing both parents' HbA1c, BP and CRP data ($n = 76$) or missing any covariates ($n = 76$). We also excluded the parent–offspring pairs if parents were taking diabetes ($n = 2$) or hypertension medication ($n = 37$). For CRP, we excluded subjects who had CRP $> 10\,mg\,L^{-1}$ ($n = 22$) because it indicates current infection (12). Our final analytic sample has a total of 940 parent–child pairs, including 598 mother–child and 525 father–child pairs for HbA1c, 810 mother–child and 735 father–child pairs for BP and 577 mother–child and 493 father–child pairs for CRP. Excluded children were slightly older, had lower household income and were more likely to live in the South region and in rural areas than the analytic sample. There were no statistically significant differences in sex, HbA1c, systolic (SBP) or diastolic BP (DBP) *z*-scores, CRP or prevalence of overweight/obesity in those included and excluded from the analytic sample.

Measures

Blood samples were collected by venipuncture following overnight fasting. Laboratory analysis methods are described in detail elsewhere (13). While we use continuous HbA1c in our central analyses, elevated HbA1c was defined in secondary analyses using HbA1c $\geq$ 5.7% for both children and adults as recommended by American Diabetes Association for prediabetes and diabetes (14). We combined prediabetes and diabetes as elevated HbA1c because diabetes (HbA1c $\geq$ 6.5%) is rare in children and adolescents (1% in our study sample). We defined parental HbA1c status using the same cut-point for consistency.

Trained physicians measured BP in triplicate, and the mean was calculated. Children's SBP and DBP *z*-scores were calculated based on the age-, sex- and height-specific BP percentile algorithm for children using the US Centers for Disease Control and Prevention 2000 growth curve reference (15).We used continuous BP measures in central analyses, although in secondary analyses, elevated BP in children was defined as an average SBP *z*-score or DBP *z*-score $\geq$ 90th percentile of the algorithm or SBP $\geq$ 120 mmHg or DBP $\geq$ 80 mmHg, as recommended by National Institutes of Health for prehypertension and hypertension in children (16). We combined prehypertension and hypertension as elevated BP because hypertension (SBP *z*-score or DBP *z*-score $\geq$ 95th percentile of the algorithm) is uncommon in children and adolescents (7% in our study sample). We defined parental BP status using the same cut-point for consistency (17).

We measured high-sensitivity CRP via the immunoturbidimetric method (13). The intra-assay and inter-assay coefficients of variation were <6.0% and <7.0% respectively. We used continuous CRP measures in central analyses, and elevated CRP was defined as CRP 1–10 mg L^{-1} in children and 3–10 mg L^{-1} in adults in secondary analyses (12,18).

Trained staff measured child's height (without shoes), weight (in light clothing) and waist circumference (midway between the lower rib margin and the iliac crest). We classified pediatric weight status (normal/overweight/obese) using age- and sex-specific reference data from the International Obesity Task Force (19).

We collected each individual's dietary intake using three consecutive 24-h dietary recalls at the individual level and a food inventory at the household level (20). We used a seven-day physical activity (PA) recall across a variety of domains to collect each individual's participation and time spent in different types of PA and calculated PA using hours spent in each activity multiplied by metabolic equivalents for that activity (21).

Covariates

Covariates included in the analyses were child's age and sex, parental age, household structure (two-/three-generation; one/more than one child), household income (tertiles), geographical region (North/Central/South), highest parental education (none or primary/middle school/high school/technical, college or higher), household residence (urban/rural), parental smoking (current smoker/non-smoker) and for sensitivity analysis child's weight status. We also controlled for child's total energy intake (kcal d^{-1}, quartiles), sodium intake (mg d^{-1}, quartiles, BP models only) and PA (metabolic equivalents-h $week^{-1}$, quartiles) as risk factors of child's HbA1c, BP and CRP.

Statistical analyses

All analyses were conducted using Stata 14.0 (Stata Corporation, College Station, TX, USA). In descriptive analyses, we examined demographic characteristics of our analytic sample using the chi-squared test. We also tested differences in the predicted mean levels of HbA1c, BP and CRP in children by household structure using random-effects linear regression, accounting for within-household clustering and adjusting for child's age and sex. Then, using random-effects linear regression, we compared the predicted mean levels of clinical and anthropometric variables among children whose parents had elevated vs. normal HbA1c, BP or CRP, adjusting for household sociodemographics, child's energy intake and PA in all models and sodium intake in BP models only. To maximize sample size, we performed analyses separately for mother–child and father–child pairs. In the mother–child model, mother's elevated HbA1c, BP or CRP status was defined as elevated status for mothers, and elevated, or normal, or unknown status for fathers. A similar definition was used in the father–child model.

Last, we used sex-specific random-effects linear regression models to estimate the parent–offspring associations for HbA1c, BP and CRP, adjusting for the same set of covariates. To examine whether the associations differed by child's or parental age, household structure or other household sociodemographics, we tested effect measure modification of these factors using the Wald test.

Sensitivity analyses

We ran three sets of sensitivity tests. First, we ran logistic regression models to examine the odds ratios (ORs) and 95% confidence intervals (CIs) of elevated HbA1c, elevated BP or elevated CRP in children whose parents had elevated levels of these factors compared with children whose parents had normal levels. Second, we additionally adjusted for child's weight status in the random-effects linear regression models. Third, we tested the associations between overweight/obesity with elevated HbA1c, BP and CRP in children.

Results

Among the 867 mother–child and 779 father–child pairs, the mean ages of children, mothers and fathers were 12.1, 38.5 and 39.8 years respectively (Table 1). A majority of the children lived in three-generation households (65.7%) and had no siblings (73.5%). Mean HbA1c was 5.3% in children, with slightly higher values in only children (5.3%) vs. children living with siblings (5.2%, $p < 0.05$). Mean SBP *z*-scores, DBP *z*-scores and CRP in children were −0.5, 0.4 and 0.7 mg L^{-1}, respectively, and did not differ by household structure.

Boys and girls whose parents had elevated HbA1c had higher HbA1c (Table 2). For example, mean levels of HbA1c was 5.61 (95% CI = 5.46–5.76) and 5.26 (95% CI = 5.19–5.34) in boys whose mother had elevated vs. normal HbA1c respectively. For BP, daughters of mothers with elevated BP had higher SBP (mean = −0.24) and DBP (mean = 0.52) *z*-scores compared with those of mothers with normal BP (SBP = -0.63, DBP = 0.32). However, similar patterns were not observed for father–daughter pairs or for boys. Children whose fathers had elevated

Table 1 Characteristics of the analytic sample

No. of children	940
No. of mother-child pairs	867
No. of father-child pairs	779
Child's age, year (mean ± SD)	12.1 ± 2.9
Maternal age, year (mean ± SD)	38.5 ± 4.7
Paternal age, year (mean ± SD)	39.8 ± 5.0
Child's gender, % male	56.1
Highest parental education, %	
None/primary school	5.9
Middle school	16.4
High school	61.9
College, technical or higher	15.8
Annual household income, yuan (mean ± SD)*	40787 ± 43522
Household residence, % urban	24.9
Number of generation, % three-generation†	65.7
Number of children, % one child	73.5
Geographical region, %‡	
North	15.8
Central	31.4
South	52.8
Child's HbA1c, %	
Overall mean, mean ± SD	5.3 ± 0.5
Predicted mean (95% CI) by no. of generation in the household¶	
Two-generation	5.3 (5.3, 5.4)
Three-generation	5.3 (5.2, 5.4)
Predicted mean (95% CI) by no. of children in the household¶**	
More than one children	5.2 (5.1, 5.3)
One child	5.3 (5.3, 5.4)
Maternal HbA1c, % (mean ± SD)	5.3 ± 0.5
Paternal HbA1c, % (mean ± SD)	5.5 ± 0.9
Child's systolic, diastolic BP *z*-scores respectively	
Overall mean, mean ± SD	−0.5 ± 1.1, 0.4 ± 0.8
Predicted mean (95% CI) by no. of generation in the household¶	
Two-generation	−0.56 (−0.68, −0.43), 0.37 (0.28, 0.45)
Three-generation	−0.48 (−0.58, −0.38), 0.44 (0.37, 0.51)
Predicted mean (95% CI) by no. of children in the household¶	
More than one children	−0.62 (−0.78, −0.45), 0.32 (0.20, 0.44)
One child	−0.48 (−0.57, −0.40), 0.44 (0.38, 0.50)
Maternal systolic, diastolic BP, mmHg (mean ± SD)	113.6 ± 13.5, 75.4 ± 9.3
Paternal systolic, diastolic BP, mmHg (mean ± SD)	120.0 ± 12.4, 80.7 ± 9.6
Child's CRP, mg L^{-1}	
Overall mean, mean ± SD	0.7 ± 1.5
Predicted mean (95% CI) by no. of generation in the household¶	

Table 1 (Continued)

Two-generation	0.6 (0.4, 0.8)
Three-generation	0.8 (0.6, 0.9)
Predicted mean (95% CI) by no. of children in the household¶	
More than one children	0.7 (0.5, 0.9)
One child	0.7 (0.6, 0.8)
Maternal CRP, mg L^{-1} (mean ± SD)	1.2 ± 1.6
Paternal CRP, mg L^{-1} (mean ± SD)	1.5 ± 1.8

*Total household income inflated to 2011.
†Three-generation: children, parents and grandparents.
‡North: Heilongjiang, Liaoning; Central: Shandong, Henan, Jiangsu; South: Hunan, Hubei, Guangxi, Guizhou.
¶Mean values are predicted using mixed-effects linear regression models controlling for child's age and sex.
**Statistically significant difference between groups at $p < 0.05$.

CRP also had higher CRP compared with children whose fathers had normal CRP (1.4 mg L^{-1} vs. 0.8 mg L^{-1} in boys and 0.9 mg L^{-1} vs. 0.5 mg L^{-1} in girls, $p < 0.05$). There were no statistically significant differences in obesity measures [prevalence of overweight/obesity, waist circumference, waist-to-height ratio {waist circumference divided by height}] in children whose parents had elevated vs. normal HbA1c or BP, whereas boys were more likely to be overweight/obese if their fathers had elevated CRP.

Random-effects linear regression showed positive parent–child associations for HbA1c (Table S1 in the Supporting Information). Beta coefficients ranged from 0.06 (95% CI 0.03–0.12) for father–daughter pairs to 0.43 (95% CI 0.28–0.58) for mother–son pairs. The positive association was consistent with logistic regression models (Table S2), which shows increased odds of having elevated HbA1c comparing children of parents with elevated vs. normal HbA1c. ORs ranged from 3.91 (95% CI 1.77–8.67) for father–daughter pairs to 7.88 (95% CI 3.36–18.47) for mother–daughter pairs. For BP, linear regression showed positive associations between girls' SBP and DBP *z*-scores with their mothers' SBP and DBP respectively (Table S1). We also found a positive father–son association for CRP (beta coefficient = 0.15, 95% CI = 0.03–0.27; OR = 2.06, 95% CI = 1.05–4.04). There were no significant effect measure modifications by child's age, parental age, household structure or any of the household sociodemographic variables at $p < 0.1$ level.

To test whether findings remained once we accounted for childhood obesity, we further adjusted for child's weight status in the random-effects linear regression models in sensitivity analysis, which did not substantially change our results for HbA1c and BP, but the father–son association for CRP was attenuated (Table S3). Overweight/obesity was positively associated with BP in boys and with CRP in both boys and girls, but was not associated with HbA1c (Table S4).

Discussion

Our study suggests positive associations between children and their parents for HbA1c, between girls and their mothers for BP and between children and their fathers for CRP. Additionally, being an only child was associated with higher HbA1c.

Studies of CMD risk factors in childhood are of potential importance not only because of the increasing prevalence of these risk factors among children (1,2), but also because they track into adulthood (22). Our study shows significant positive parent–child associations for HbA1c. This result is consistent with previous studies on the parent–child association for insulin resistance or diabetes in both Western and Asian populations (6,23). Sinaiko *et al.* found correlation for fasting insulin between mothers and their adolescent children in a US population (23), whereas Park *et al.* observed parent–child correlation in fasting glucose in Korean adolescents (6).

For BP, We found a positive association for mother–daughter but not father–daughter or parent–son pairs. The current literature on the concordance between children's and parental BP is inconsistent. Some studies showed positive parent–child correlations for BP (6), whereas others did not (24). Despite the inconsistency, our observed association between girls' and their mothers' BP is supported by existing literature, which suggested that maternal history of hypertension was associated with greater offspring hypertension risk than paternal history (25).

We observed a strong parent–child association for HbA1c in both boys and girls. This is likely because children share genes, living environment and health behaviours, especially diet, with their parents (3).

Table 2 Predicted mean levels of clinical and anthropometric measures in children of parents with normal or elevated HbA1c, BP and CRP*

	Maternal HbA1c status			Paternal HbA1c status		
	Normal (<5.7%)	Elevated (≥5.7%)	*P* value	Normal (<5.7%)	Elevated (≥5.7%)	*P* value
Boys						
n	281	66		219	95	
Age, year†	12.1 ± 0.2	11.9 ± 0.3	0.47	12.2 ± 0.2	12.4 ± 0.3	0.61
HbA1c, %‡	5.26 ± 0.04	5.61 ± 0.08	<0.001	5.25 ± 0.05	5.52 ± 0.07	0.003
Height, cm‡	148.3 ± 0.4	147.8 ± 0.9	0.60	149.4 ± 0.5	148.7 ± 0.8	0.48
Weight, kg‡	40.4 ± 0.4	40.8 ± 0.9	0.94	41.4 ± 0.5	41.2 ± 0.8	0.86
Overweight/obesity, %‡§	13.1	13.6	0.90	13.1	13.6	0.91
Waist circumference, cm‡	64.0 ± 0.5	65.9 ± 1.0	0.10	64.7 ± 0.5	64.6 ± 0.8	0.98
Waist-to-height ratio‡¶	0.43 ± 0.003	0.46 ± 0.007	0.054	0.43 ± 0.003	0.44 ± 0.005	0.25
Girls						
n	219	58		156	76	
Age, year	12.1 ± 0.2	12.3 ± 0.3	0.61	12.3 ± 0.2	12.2 ± 0.3	0.76
HbA1c, %‡	5.22 ± 0.03	5.46 ± 0.05	<0.001	5.19 ± 0.03	5.47 ± 0.05	<0.001
Height, cm‡	146.2 ± 0.5	143.9 ± 1.0	0.046	145.5 ± 0.6	145.1 ± 0.9	0.63
Weight, kg‡	39.1 ± 0.5	37.6 ± 1.0	0.17	38.6 ± 0.6	39.3 ± 0.8	0.52
Overweight/obesity, %‡§	11.3	8.3	0.44	8.2	13.1	0.41
Waist circumference, cm‡	62.3 ± 0.5	61.2 ± 1.0	0.29	62.6 ± 0.6	61.9 ± 0.8	0.50
Waist-to-height ratio‡¶	0.43 ± 0.003	0.42 ± 0.006	0.71	0.43 ± 0.004	0.43 ± 0.005	0.77

	Maternal BP status			Paternal BP status		
	Normal (<120/80 mmHg)	Elevated (≥120/80 mmHg)	*P* value	Normal (<120/80 mmHg)	Elevated (≥120/80 mmHg)	*P* value
Boys						
n	254	197		144	275	
Age, year	12.0 ± 0.2	12.3 ± 0.2	0.39	12.3 ± 0.2	12.3 ± 0.2	0.99
SBP *z*-score‡	−0.53 ± 0.07	−0.59 ± 0.08	0.61	−0.59 ± 0.09	−0.59 ± 0.06	0.63
DBP *z*-score‡	0.42 ± 0.05	0.42 ± 0.05	0.91	0.39 ± 0.06	0.43 ± 0.04	0.61
Height, cm‡	148.0 ± 0.5	148.9 ± 0.5	0.25	149.3 ± 0.6	148.9 ± 0.5	0.59
Weight, kg‡	40.5 ± 0.5	41.2 ± 0.6	0.32	41.3 ± 0.7	41.6 ± 0.5	0.73
Overweight/obesity, %‡§	11.6	17.3	0.10	12.1	14.6	0.47
Waist circumference, cm‡	64.2 ± 0.5	65.6 ± 0.6	0.09	64.8 ± 0.7	65.2 ± 0.5	0.67
Waist-to-height ratio‡¶	0.43 ± 0.004	0.44 ± 0.004	0.19	0.43 ± 0.005	0.44 ± 0.003	0.63
Sodium intake, mg d^{-1}‡	4218 ± 193	3950 ± 219	0.36	4309 ± 207	3756 ± 155	0.02
Girls						
n	208	157		113	208	
Age, year	11.7 ± 0.2	12.7 ± 0.2	<0.001	11.9 ± 0.3	12.4 ± 0.2	0.11
SBP *z*-score‡	−0.63 ± 0.08	−0.24 ± 0.09	0.002	−0.39 ± 0.10	−0.48 ± 0.08	0.49
DBP *z*-score‡	0.32 ± 0.06	0.52 ± 0.07	0.03	0.35 ± 0.08	0.47 ± 0.06	0.24
Height, cm‡	145.3 ± 0.5	145.5 ± 0.6	0.88	144.6 ± 0.8	145.7 ± 0.6	0.28
Weight, kg‡	38.1 ± 0.5	38.9 ± 0.6	0.35	37.8 ± 0.7	39.1 ± 0.5	0.14
Overweight/obesity, %‡§	8.3	13.2	0.20	7.1	12.4	0.15
Waist circumference, cm‡	61.9 ± 0.5	62.2 ± 0.6	0.77	62.1 ± 0.7	62.3 ± 0.5	0.77
Waist-to-height ratio‡¶	0.43 ± 0.003	0.43 ± 0.004	0.63	0.43 ± 0.005	0.43 ± 0.003	0.94
Sodium intake, mg d^{-1}‡	3348 ± 132	3558 ± 150	0.29	3324 ± 175	3510 ± 126	0.40

Table 2 (Continued)

	Maternal CRP status			Paternal CRP status		
	Normal (<3 mg L^{-1})	Elevated (3–10 mg L^{-1})	*P* value	Normal (<3 mg L^{-1})	Elevated (3–10 mg L^{-1})	*P* value
Boys						
n	373	61		325	56	
Age, year	11.9 ± 0.2	13.0 ± 0.4	0.01	12.2 ± 0.2	12.6 ± 0.4	0.99
CRP, mg L^{-1}‡	0.8 ± 0.2	0.9 ± 0.5	0.57	0.8 ± 0.1	1.4 ± 0.3	0.02
Height, cm‡	148.0 ± 0.4	148.3 ± 1.1	0.83	148.4 ± 0.4	151.5 ± 1.0	0.01
Weight, kg‡	40.2 ± 0.4	40.5 ± 1.1	0.79	40.8 ± 0.5	44.7 ± 1.1	0.001
Overweight/obesity, %‡§	13.1	10.0	0.54	11.5	28.1	0.01
Waist circumference, cm‡	64.4 ± 0.5	64.4 ± 1.2	1.00	64.4 ± 0.7	66.7 ± 0.5	0.08
Waist-to-height ratio‡¶	0.44 ± 0.003	0.43 ± 0.008	0.74	0.43 ± 0.003	0.44 ± 0.008	0.50
Girls						
n	290	47		222	59	
Age, year	12.0 ± 0.2	12.8 ± 0.4	0.07	12.2 ± 0.2	12.2 ± 0.4	0.97
CRP, mg L^{-1}‡	0.6 ± 0.1	0.5 ± 0.2	0.84	0.5 ± 0.1	0.9 ± 0.2	0.03
Height, cm‡	145.7 ± 0.5	147.5 ± 1.1	0.17	145.2 ± 0.5	146.1 ± 1.1	0.46
Weight, kg‡	38.5 ± 0.4	41.2 ± 1.1	0.03	38.4 ± 0.5	40.5 ± 1.0	0.14
Overweight/obesity, %‡§	9.8	12.2	0.63	9.4	16.0	0.29
Waist circumference, cm‡	61.9 ± 0.4	63.2 ± 1.1	0.77	61.7 ± 0.5	65.4 ± 1.0	0.77
Waist-to-height ratio‡¶	0.42 ± 0.003	0.43 ± 0.007	0.67	0.42 ± 0.003	0.45 ± 0.006	0.94

*We conducted separate analyses for mother–child and father–child pairs.
†Data are means ± S.E. for all such values.
‡Adjusted for child's age, household income, geographical region, household residence, child's total energy intake, child's total physical activity, parental smoking and parental education using mixed-effects linear regression or logistic regression models.
§Overweight/obese: ≥85th percentile of the age- and sex-specific reference data from the International Obesity Task Force.
¶Waist-to-height ratio is calculated as waist circumference (cm) divided by height (cm).

Previous research has shown a strong correlation between children's and parental diet in China (26). Despite different study designs, studies in Western countries have found medium to weak parent–offspring associations for diet, as shown in a meta-analysis (27). The difference in the strength of association is possibly because of shared dishes in Chinese vs. Western families, which typically have separate plates. The strong parent–offspring correlation for diet likely has contributed to the strong parent–offspring associations for HbA1c in our study. Contrarily, we only observed parent–child associations for BP in mother-daughter pairs. This is possibly because BP has a weaker genetic aetiology compared with diabetes-related outcomes (28). Therefore, BP is likely more influenced by other factors including diet and obesity. In our study, children whose parents had elevated BP did not have higher sodium consumption compared with children whose parents had normal BP, which may underlie the observed similarity in BP in offspring of parents with elevated vs. normal BP. Further, we found a significant positive association between overweight/obesity and elevated BP in boys but not girls, as shown in Table S4. Thus, child's weight status may play a comparatively stronger role in BP in boys compared with the influence of parental BP status.

Children whose fathers had elevated vs. normal CRP also had higher CRP, and boys whose fathers had elevated CRP were also more likely to be overweight/obese, suggesting that childhood obesity might have played a role in the father–child association for CRP. We did not find mother–child associations for CRP possibly because of the lack of association between maternal CRP and child's weight status. Given existing evidence in the impact of inflammation on the development of diabetes (8), we hypothesize that the association between child's and paternal CRP might be related to the father–child association for HbA1c. We did not find differences in obesity measures among children of parents with elevated or normal HbA1c or BP. Further, additionally adjusting for child's weight status in regression models did not alter the parent–child associations for HbA1c or BP. These findings are supported by previous research (23), which indicated that elevated HbA1c and BP during childhood cannot be directly attributed to greater adiposity among these children, perhaps reflecting shared genetic, environmental, behavioural contributors and potentially other unmeasured factors. However, the mechanisms underlying the association between children's and parental HbA1c and BP require further study.

One-child households and three-generation households are more common in China than in Western countries. Evidence shows that living with their grandparents and/or being an only child are associated with a higher risk of childhood obesity (7). In this study we examined whether having no siblings or living with grandparents was associated with higher HbA1c, BP or CRP in children. Our findings suggest no differences in BP or CRP by household structure, whereas only children had higher HbA1c compared with children living with siblings.

Our study has several limitations. First, we chose cross-sectional study design because HbA1c was only measured in 2009. Second, we calculated child's BP *z*-scores using the US reference because no age-, sex- and height-specific reference for Chinese children is available. Third, as our study fills a gap in the literature by documenting parent–child associations in CMD risk factors, we did not explicitly investigate mechanisms underlying these associations. Thus, we cannot decompose how much of the association resulted from genetic predisposition vs. shared familial environment. Fourth, we were unable to examine whether child's pubertal status played a role in these associations as we did not have data on pubertal markers in children. While menopausal status could theoretically play a role, the vast majority of mothers in our study (96%) were below the mean age of menopause (49 years) in China (29). Nonetheless, we tested associations among younger vs. older children, as well as among children with younger vs. older mothers, and found minimal differences in association. Last, self-reported diet and PA data may be subject to recall bias.

Despite the limitations, our study has several notable strengths including the use of a regionally diverse sample from a national survey. Further, instead of collecting children's family history of diabetes and hypertension, we measured each parent–child pair's HbA1c, BP, CRP and ascertained elevated risk, which is otherwise largely undiagnosed in this population. Moreover, we defined children's elevated BP based on the age-, sex- and height-specific BP percentile algorithm for children, which further incorporates height compared with child BP references that are only age- and sex-specific (30). Because of the high correlation between children's height and BP, our approach more appropriately classified children's BP status (30).

In conclusion, our findings demonstrate general positive parent–child associations for HbA1c, BP and CRP, with variation across markers and by sex of the child and sex of the parent. Our study provides further evidence in identifying children at high risk of CMD, and the clustering of risk factors suggests that household-based interventions might be an approach worthy of attention.

Conflict of Interest Statement

No conflict of interest was declared.

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Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's web-site:

Table S1. Random-effects linear regression analysis of the relationships between parents and children's HbA1c, BP and CRP*.

Table S2. Multivariable logistic regression analysis of parent–child associations for elevated HbA1c, BP and CRP*.

Table S3. Random-effects linear regression analysis of the relationships between parents and children's HbA1c, BP and CRP, additionally adjusting for child's weight status*.

Table S4. Odds ratios (95% confidence intervals) of elevated HbA1c, BP and CRP* according to overweight/obese status† in children.