INTRA-FAMILIAL CONCORDANCE IN LIFESTYLE BEHAVIORS AND CARDIOMETABOLIC RISK IN RAPIDLY MODERNIZING CHINA

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ABSTRACT

Fei Dong: Intra-Familial Concordance in Lifestyle Behaviors and Cardiometabolic Risk in Rapidly Modernizing China
(Under the direction of Penny Gordon-Larsen)

China has been experiencing rapid changes in lifestyle behaviors and dramatic increases in cardiometabolic disease (CMD) risk factors in both adults and children in the past two decades. Lifestyle behaviors are important contributors to cardiometabolic health. Children share lifestyle behaviors, genes, and home environments with parents, which could underlie clustering in CMD risk in households. However, the extent to which children share behaviors and CMD risk factors with each of their parents has not been studied in a geographically-diverse Chinese population undergoing rapid urbanization.

We capitalized on 18-year (1991-2009) longitudinal data from the China Health and Nutrition Survey (>3,000 households with children aged 7-17y), with measured CMD risk factors, diet, physical activity, and sociodemographics. Using random-effects regression, we investigated parent-offspring associations in CMD risk factors (waist-to-height ratio, hemoglobin A1c, blood pressure, and C-reactive protein) and lifestyle behaviors (animal-source foods, away-from-home eating, snacking, screen time, and leisure-time sports). We additionally examined the associations between these behaviors and risk factors in children and their parents, and determined whether household structure (presence of grandparents and presence of siblings in the household) was associated with children's behaviors or risk factors.

We found positive associations in lifestyle behaviors and CMD risk factors between children and their parents. However, the magnitude of parent-offspring associations for behaviors declined over time. We also found faster increases in away-from-home eating and snacking in children than their parents. Compared to children who lived with siblings in the household, only children consumed more animal-source foods, away-from-home foods, snacks, and had higher HbA1c, after adjusting for age, sex, household income, and urbanicity. CMD risk factors in children and their parents were negatively associated with the consumption of fruit and vegetable snacks and positively associated with screen time, with difference in associations between children and their parents for some risk factors.

Promoting healthy diet and decreasing screen time are commonly-used intervention strategies to reduce CMD risk. However, given the observed intergenerational differences in behavior changes and behavior-risk associations, generation-specific intervention strategies may be needed. Further, only-children households should specifically be considered for interventions targeting children's behaviors and CMD risk in this population.

To my parents,
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LIST OF ABBREVIATIONS

BP	Blood pressure
CHNS	China Health and Nutrition Survey
CI	Confidence interval
CMD	Cardiometabolic disease
CRP	C-reactive protein
HbA1c	Hemoglobin A1c
MET-hours	Metabolic equivalent-hours
OR	Odds ratio
PA	Physical activity
WHtR	Waist-to-height ratio

CHAPTER 1. INTRODUCTION

Background

The household context has been an important target for intervening on behaviors that influence cardiometabolic disease (CMD), which has increased across the globe among both adults and children, although with faster increases in children. [1, 2] We know from several studies that CMD risk factors (e.g. obesity, diabetes, hypertension, inflammation) cluster in families between children and their parents in Western countries. [3-9] However, we don't know the extent to which CMD risk factors cluster in urbanizing Chinese households with burgeoning CMD risk. Such clustering is likely due to shared genes, home environments, as well as diet and physical activity (PA) behaviors between children and their parents. [10-19] Determining the strength of associations for CMD risk factors between children and their parents will provide a better understanding of childhood CMD risk and help further identify children at high risk. In addition, lifestyle behaviors such as diet and PA are important contributors to CMD risk factors. [20, 21]Studying how these behaviors are shared between children and their parents will provide guidance for family-based interventions that target both parents and children, which have shown to be more effective than targeting children only on reducing children's CMD risk. [22] To our knowledge, these important questions have not been addressed in a large, geographically-diverse Chinese population, which has been experiencing rapid urbanization with burgeoning CMD rates.

Besides the influence of parents, the presence of grandparents and siblings may also play a role in children's CMD risk status and health behaviors, possibly because Chinese grandparents

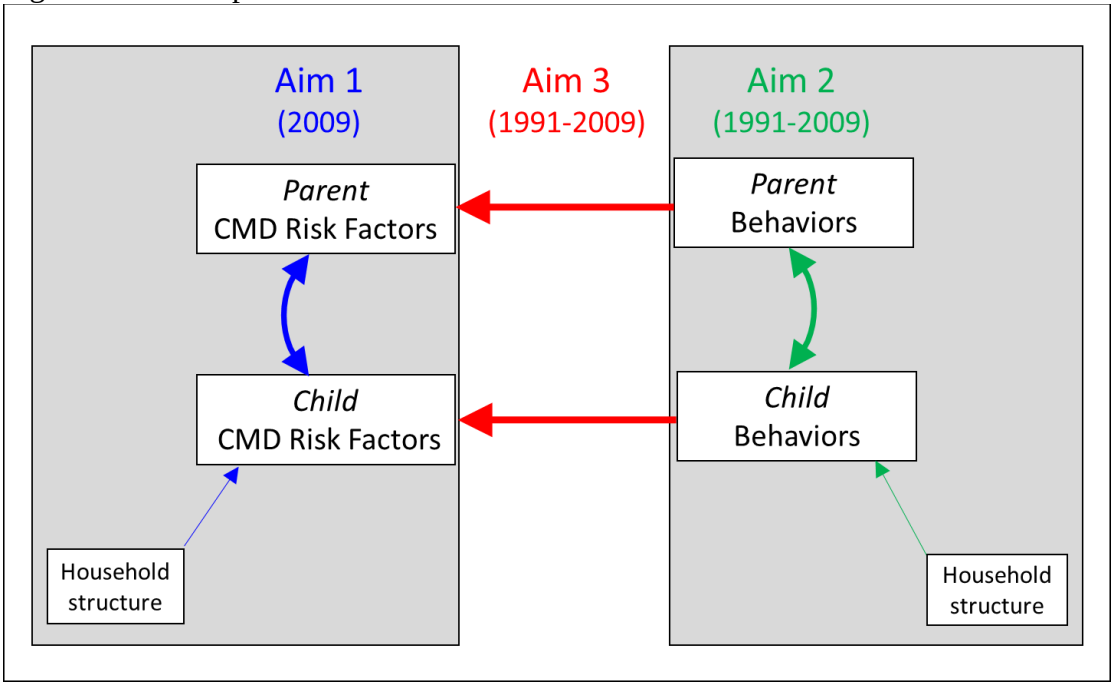
are more likely to indulge their grandchildren with modern, unhealthy foods and screen-based sedentary behaviors, especially in single-child households. [23-25] Nonetheless, to our knowledge, no research has examined how household structure (i.e. living with grandparents or not, having siblings or not) relates to children's CMD risk factors, diet, and PA behaviors in a large Chinese population-based study. Understanding this question will help identify children at high CMD risk and guide behavioral intervention efforts to reduce the risk.

Furthermore, although the associations between dietary and PA behaviors with CMD risk factors have been well-established, as shown in reviews and meta-analyses, [20, 26] as well as diet and PA guidelines and recommendations, [27, 28] few studies have examined and compared the behavior-risk associations among children versus their parents. [29] Previous research has suggested potential disparities in the behavior-risk associations in children versus adults, [2] possibly because of the difference in behavior patterns between children and adults, and potential age-related biological differences in disease etiologies across generations.

To sum up, the literature is lacking how children's CMD risk factors and lifestyle behaviors associate with those of their parents or household structure in a large Chinese population. The difference in the behavior-risk factor association between children and adults is also understudied. Therefore, we aimed to explicitly answer these questions by studying CMD risk factors and lifestyle behaviors in the household context. Our conceptual framework (Figure 1.1) included behaviors and risk factors in two generations and incorporated the household structure. Aims 1 and 2 addressed the associations between children's CMD risk factors (Aim 1) and lifestyle behaviors (Aim 2) with those of their parents, and how household structure was related to children's CMD risk factors (Aim 1) and lifestyle behaviors (Aim 2). Aim 3 tested

how lifestyle behaviors (Aim 2) predicted CMD risk factors (Aim 1) in children and their parents.

Figure 1.1. Conceptual model



We used 18-year longitudinal data (1991, 1993, 1997, 2000, 2004, 2006, 2009) on diet, PA, anthropometrics, and blood pressure (BP), and cross-sectional data on biomarkers (2009) from the China Health and Nutrition Survey (CHNS, over 3,000 households with children aged 7-17y). CHNS has detailed behavioral, anthropometric, demographic, and socioeconomic data collected at 7 waves from 1991 to 2009, and biomarkers measured using fasting blood samples in 2009. Using these unique data, we sought to 1) evaluate the associations between child and parent CMD risk factors including elevated hemoglobin A1c (HbA1c), elevated blood pressure (BP), and high C-reactive protein (CRP), and examine child CMD risk factors by household structure; 2) study changes in urbanization-related diet, screen time, and PA behaviors from 1991-2009 in children compared to their parents, examine whether child diet, screen time, and PA behaviors associated with those of their parents, or differed by household structure; and 3)

determine the longitudinal associations between lagged urbanization-related diet, screen time, and PA behaviors with high waist-to-height ratio (WHtR), elevated BP, elevated HbA1c, and high CRP at the next survey among children and their parents from 1991 to 2009, and test the difference in behavior-risk associations between children and their parents.

Research Aims

The primary goal of this dissertation was to determine the parent-offspring associations for CMD risk factors and lifestyle behaviors, and examine how these lifestyle behaviors associate with CMD risk factors in children compared to their parents living in the same household. We also aimed to test whether household structure is associated with children's CMD risk factors and lifestyle behaviors.

Aim 1: Determine the associations between children's and parents' HbA1c, BP, and CRP in 2009.

1a: Evaluate HbA1c, BP, and CRP among children aged 7-17 years by household structure (i.e. living with grandparents or not, having siblings or not).

1b: Using random-effects regression modeling, determine the parent-child associations in HbA1c, BP, and CRP; determine whether this association differs by children's age and sex, and by household structure.

We hypothesize higher HbA1c, BP, and CRP among only children and among children who live with grandparents in the same household. We also hypothesize positive associations between children's and parents' HbA1c, BP, and CRP, and different magnitude of associations by children's age and sex, and by household structure.

Aim 2: Determine the associations between children's diet, screen time, and PA behaviors with those of their parents from 1991 to 2009.

2a: Examine changes in urbanization-related lifestyle behaviors (animal-source foods consumption, away-from-home eating, snacking, screen time, and leisure-time sports) over time in children, mothers, and fathers.

2b: Using random-effects regression modeling, examine the parent-offspring associations in diet, screen time, and PA behaviors over time. Identify factors related to the concordance in these behaviors between children and their parents (child's age and sex, survey year, urbanicity, household income, geographic region).

2c: Examine children's diet, screen time, and PA behaviors by household structure (i.e. living with grandparents or not, having siblings or not)

We hypothesize differential changes in lifestyle behaviors between children and their parents over time, overall concordance in behaviors between children and their parents, and decreasing concordance over time with urbanization. We also hypothesize less healthy lifestyle behaviors in only children and children who live with grandparents in the same household.

Aim 3: Using longitudinal models, estimate whether lagged urbanization-related lifestyle behaviors (away-from-home eating, snacking, screen time, and leisure-time sports) are associated with high WHtR, elevated HbA1c, elevated BP, and high CRP at the next survey among children and their parents from 1991 to 2009, and examine whether the magnitude of associations differs between children and parents. We hypothesize positive associations between lagged away-from-home-eating, snacking, and screen time with CMD risk factors at the next survey, and negative associations between lagged leisure-time sports with CMD risk factors at the next survey. We expect that the magnitude of behavior-CMD risk factor associations will

differ between generations because of distinct behavior patterns or age-related biological differences in disease etiology between children and adults.

CHAPTER 2. LITERATURE REVIEW

Prevalence of CMD risk factors in China and its public health burden

CMD has been increasing rapidly and become a global burden. China is one of the countries with the most rapid increase in obesity and other CMD risk factors, [30] and has the world's highest diabetes prevalence, [31] causing a heavy public health burden. [1, 32] It is estimated that diabetic patients spent 16% of their annual income on the disease. [33] Besides the increasing economic burden, it is particularly essential to study factors associated with CMD risk in this population because 1) China has the largest population in the world. With the highest diabetes prevalence, the absolute number of individuals affected by diabetes is larger than any other country in the world. In 2015, it is estimated that 9.7% and 25.5% adults were diabetic and hypertensive in China, respectively. [34] This comes to 130 million and 340 million adult diabetes and hypertension patients in this one country, respectively. 2) Asian populations have a relatively higher proportion of body fat at a lower BMI compared to Western populations, especially abdominal fat, and develop CMD risk factors at lower BMI and younger ages. Investigating factors associated with the development of CMD risk factors in China not only contributes to better understanding of this issue in Asian populations, but is also a particularly important part of global prevention efforts.

Concordance of CMD risk factors between children and their parents

The prevalence of CMD risk factors in China has been increasing not only in adults, but also in children. [1] Previous research also shows that obesity has been increasing faster in children than in adults in the Chinese population. [2] Given that childhood CMD risk factors

track into adulthood, [35-37] the increasing prevalence of childhood CMD risk factors will further add to the public health burden in China in the next decade when children with high CMD risk enter adulthood. Therefore, studying factors related to childhood CMD risk factors is of primary importance in this population.

Parents play a particularly important role in the CMD risk status of their children due to shared genetic and environmental influences on CMD risk factors between children and their parents. Studying and documenting familial aggregation of CMD risk factors will not only provide better understanding on the development of CMD risk factor during childhood, which could assist in identifying children at high risk, but will also inform interventions to target both the parent and child to enhance treatment effectiveness. It has been suggested that family-based weight control intervention is more effective and cost-efficient compared with treating the child and parent separately. [22]The concordance of CMD risk factors between children and their parents have been reported in previous studies. [3-7, 9, 38-41] Parental obesity has been associated with elevated risk of childhood obesity in Germany, France, Australia, US, and China. [3-7] Besides obesity, Sinaiko et al. [9] found a correlation for fasting insulin between mothers and their adolescent children in a U.S. population, whereas Park et al. [39] observed parent-child correlation in both fasting insulin and fasting glucose in Korean adolescents. The current literature on the concordance between children's and parents' BP is inconsistent. Some studies showed that children's BP was positively correlated with their parents' BP in the Netherland, Korea, Canada, and China, [38-41] whereas two studies in US populations did not find any correlation. [9, 42] As a risk factor for diabetes and metabolic syndrome, CRP has also been shown to be correlated between children and their parents in a US population. [43]Nonetheless, no previous study has determined the strength of parent-child associations for CMD risk factors

in a large, geographically-diverse Chinese population with a dramatic surge of metabolic abnormalities.

Nutrition transition and behavioral changes in Chinese children and adults

Urbanization and rapid socioeconomic growth have contributed to dramatic shifts in dietary and PA behaviors in China. Traditional Chinese diets are dominated by cereals and vegetables with few animal-source foods, whereas in the past two decades, China has been experiencing a transition from traditional to westernized diets high in oil and animal-source foods. [44] There has also been a fast change in eating behaviors, such as increasing away-from-home eating and snacking in this population. [45] Percent total energy from foods prepared away-from-home in China increased from 7% in 1991 to 16% in 2011 among 2-18y old children, and from 7% in 1991 to 18% in 2011 among 19-59y old adults. [45] The percentage of snacking in Chinese adults and children increased from 15% in 1991 to 67% in 2011 among 2-18y old children, and from 9% in 1991 to 52% in 2011 among 19-59y old adults. [45]

On the contrary, there have been large declines in total PA, together with increases in sedentary behaviors over the past 20 years in China. [46-48] Average weekly PA among Chinese adults fell by 32% from 1991 to 2006, with the largest fall for occupational activity [47, 49] From 1997 to 2006, urban Chinese boys aged 13-18y increased their screen time from 0.5 hours to 1.7 hours. [46]

While substantial behavioral changes have been observed in China, it is unclear whether the rate of these changes is the same for children versus adults in this population. Evaluating and comparing behavioral disparities across generations will provide insights into whether behavioral changes in children mirror those of their parents and whether children and adults respond to urbanization differently in rapidly modernizing China. Understanding these questions will help

elucidate why children have been experiencing a faster increase in obesity than adults in this country. [2]Findings will inform intervention efforts by policy makers to develop effective generation-specific behavioral change strategies in this developing population. Few studies, however, have examined generational differences in lifestyle behavioral changes in countries experiencing behavior transitions. A discrepancy between offspring and maternal changes in obesogenic diet in response to urbanization has been reported in a Filipino population, indicating a more obesogenic diet in offspring than their mothers. [50] To our knowledge, no such study has been done in China, which has the largest population in the world and is undergoing rapid modernization.

Behavioral concordance between children and their parents and family-based interventions to reduce CMD risk

While interventions on reducing CMD risk have been targeting children predominantly, Epstein et al. [22] found that family-based behavioral intervention targeted both the parent and the child was more effective comparing to targeting the child alone for reducing childhood obesity in the long term. This finding highlights the benefits of directing the interventions at relationships rather than solely at individuals. To conduct household-based interventions, advanced understanding on how parental behaviors are associated with child behaviors is needed. Studies have shown positive correlations between parents' and children's dietary intakes in both Western and Chinese populations, [10-15] focusing on macronutrients, certain food groups, and overall dietary patterns. Less is known about the concordance of eating behaviors such as away-from-home eating and snacking in parent-child pairs.

For PA, positive associations between parent and child participation in vigorous activity and inactivity have also been observed in various populations. [16-19] Children who had

physically active parents were more likely to participate in sports relative to children with inactive parents in France, Finland, Sweden, and Australia [16, 19, 51, 52]; higher parental TV watching was associated with higher TV watching in children in the UK. [53]

A number of mechanisms may explain the observed parent-child associations for health behaviors. First, it is possible that parents act as role models for children, and children adopt their parent's behavioral habits and attitudes. Cheung et al. and Lau et al. found parental role modeling to be related to higher child PA participation in China. [54, 55] Second, shared household environment may have influenced both children's and adults' behaviors in similar ways. For example, studies have found positive associations between accessibility of facilities in the neighborhood (e.g., open space, recreational center, park) and participation in PA in both adults and children. [56, 57] Third, studies have shown that genetic predisposition may also contribute to the familial concordance of preference for food and exercise. [51, 58]

Past research on the parent-offspring association for diet, sedentary behaviors, and PA, however, is mostly cross-sectional. These studies were unable to examine the change in magnitude of associations over time, especially in modernizing populations such as China. Findings will reveal whether urbanization influences the familial aggregation of lifestyle behaviors. Given numerous food commercials targeting children as their main consumers, together with the increasing pocket money associated with growing household income, children are more likely to purchase meals or snacks on their own outside home, resulting in less resemblance in diet between children and their parents with urbanization. Testing this hypothesis will provide a better understanding of factors influencing child behaviors in a modernizing society. Findings could inform more effective intervention strategies in countries facing lifestyle transitions to reduce CMD risk in pediatric populations.

The role of household structure in children's health behaviors and CMD risk factors

One-child households and three-generation (grandparents, parents, and children) households are more common in China than in Western countries. Due to their past experience of food shortage and deprivation, and a belief that heavier children are healthier and children who eat more will grow taller, grandparents are more likely to indulge their grandchildren with large food portions, modern and unhealthy food, and screen-based sedentary behaviors, especially in single-child households. [23-25] A previous study among Japanese children also found an association between the presence of grandparents and physical inactivity. [59] Additionally, the "One-Child Policy" implemented in 1979 resulted in a high proportion of single-child households. These only children often receive more attention and the best care in the household than children living with siblings. [60]

Unhealthy diet behaviors and physical inactivity may have contributed to the higher risk of overweight/obesity among children living in only-child and/or three-generation households relative to those who have siblings and live in two-generation households, as observed in previous studies. [61-64] For example, Li et al. found that living with grandparents was positively associated with childhood obesity in China [24]. Other studies show that only children were more likely to be overweight compared to children with siblings in Europe and Japan. [62, 63] Intervention strategies may focus on improving dietary habits and promoting PA to enhance the ability to improve health outcomes in children in such household situations.

To our knowledge, no study has examined whether household structure is associated with children's obesity-related CMD risk factors (e.g., diabetes, hypertension, inflammation) in China. Nor has any research examined how household structure relates to children's diet, screen time,

and PA behaviors in a large Chinese population-based study. Understanding these factors will help identify children at high risk of CMD and guide intervention efforts for behavioral changes.

Are urbanization-related lifestyle behaviors associated with CMD risk factors?

1. Dietary behaviors and CMD risk factors

Diet is an important contributor to CMD risk factors. [20] Studies have shown that a westernized diet including high consumption of high-fat animal-source foods is associated with the increasing prevalence of obesity. [65, 66] China is not only experiencing a transition from traditional to westernized diet, but is also having a urbanization-related change in eating behaviors, such as increasing away-from-home eating and snacking. [45] Foods prepared away-from-home are generally high in energy density, saturated fat, trans fat, added sugar, and sodium, and low in fiber, [67] which have been associated with weight gain and adverse cardiometabolic profiles in both children [29] and adults [68, 69] in the US and Spain.

Current evidence for the association between snacking and health status is mixed. [70-74] Fisher et al. [72] and Howarth et al. [73] found positive relationships between snacking and obesity among US children and adults, respectively, whereas other studies found inverse [74] or null [70] associations among US children. Few studies examined the association between snacking and other CMD risk factors. Mekary et al. [75] found that snacking was longitudinally positively related to type 2 diabetes in US men. However, these studies did not differentiate types of snacks consumed, which could be a reason for the mixed evidence. Phillips et al. only found a significant association between BMI z-score with soda consumption among US adolescents, but not with total snacks. [76] Snacking is becoming common in China with modernization, especially in more urbanized areas. [77]The dominant snack in China is fruit, [77, 78] which may be partly responsible for the inverse association between snacking and BMI

among Chinese overweight children, due to the health benefits of high fiber content in fruits. [78] Contrarily, snacks in Western populations are mainly candy, sweetened beverages, and salty snacks, [79] which were found to be associated with weight gain in the US population. [72, 73]

2. Sedentary time, PA, and CMD risk factors

Previous studies of screen time or sedentary behaviors have suggested their relationships with obesity in adults [80-82] and children [83, 84] in various countries such as the US, Canada, France, Australia, and China. Earlier research also found positive associations between screen time with hypertension [85] and high CRP [86] among US children. A number of theories have been proposed to explain these associations. First, screen-based activities, especially TV watching, involve low energy expenditure, even compared to other sedentary behaviors such as reading and doing homework, thus reduce total energy expenditure. [87, 88] Second, TV watching is often accompanied by increased food and snack intakes, [89] which may result in higher caloric consumption. Third, sedentary behaviors may have direct influences on metabolic health through reducing insulin sensitivity and increasing triglycerides levels, [90, 91] in addition to the indirect effect on body weight or other health behaviors.

On the other hand, epidemiological studies have demonstrated health benefits of moderate-intensity PA in both adults and children, in reducing obesity, diabetes, and hypertension. [92, 93] Additionally, a previous review also found a long-term anti-inflammatory response of exercise in children. [94] However, it is suggested that leisure-time sports alone may not be sufficient to prevent obesity, because the amount of PA required for obesity prevention is difficult to achieve through leisure-time sports only, especially with increasing sedentary time. [46, 95] In fact, studies have found stronger associations between body weight with sedentary time versus leisure-time PA among US and Australian adults. [96, 97] Focus may need to be

placed on promoting an active lifestyle in general rather than emphasizing on leisure-time PA only. [98]

Many of the studies examining the behavior-risk associations are cross-sectional and do not allow temporality between health behaviors and CMD risk factors, which may take years to develop. [83] Previous studies have reported different results examining the association between sedentary time and CMD risk factors cross-sectionally versus longitudinally, suggesting a "delayed effect" of sedentary time on CMD risk factors. [83]For example, Davison et al. did not find a cross-sectional association between non-Hispanic white girls' TV viewing and BMI, but longer TV time at age 7 was related to higher probability of being overweight at age 11. [99]Similarly, baseline sedentary time did not predict insulin resistance after 1-year follow-up in UK adults, [100]but was significantly and positively associated with insulin resistance after 5.6 years of follow-up. [101]Therefore, a longitudinal study design with a "latency period" between health behaviors and CMD risk factors is preferred to examine the behavior-risk associations.

Do behavior-CMD risk factor associations differ between children and adults?

Although obesity and other CMD risk factors have been increasing in both adults and children globally, research has revealed differences in prevalence and trends of obesity comparing the adults and children populations. [2]In China, the prevalence of overweight and obesity was 2.7 times in adults aged ≥18y as in children aged 10-17y in 1991, but the annual relative change in the prevalence of overweight and obesity was 10.4% in children relative to 8.6% in adults from 1991 to 2004. [2]These data suggest that the gap of obesity prevalence between adults and children is closing and childhood obesity is accelerating. With the increasing obesity epidemic in children, the prevalence of type 2 diabetes and hypertension, which were once

considered adult diseases, has also been increasing in pediatric populations around the globe. [102-104]

The difference in increasing rates of obesity and other CMD risk factors in children versus adults are hypothesized to be due to a few reasons. [2]First, children and adults may respond to the changing environment differently, characterized by disparities in change rates or patterns in health behaviors across generations. Studying and comparing the changes in adult versus child behaviors over time can provide more insights into this hypothesis. Second, there could be age-related biological differences between children and adults that cause different responses to the same environment and health behaviors. An examination of the association between eating and PA behaviors with CMD risk factors among adults versus children will improve understanding of this hypothesis. Although dietary and PA correlates of CMD risk factors are well-documented in adults and children, [105-107] few studies have examined or compared the behavior-risk association among children versus adults. [29] The household-level data in CHNS provides a unique opportunity to examine how health behaviors are related to CMD risk factors in children and their parents differently, controlling for shared socioeconomic and living environment.

Summary

To better inform family-based behavioral intervention efforts to reduce the CMD epidemic in Chinese households, especially in children, improved understanding of the associations between parental with children's CMD risk factors as well as diet and activity behaviors, and how household structure (i.e. number of child and generations of family member in the households) relates to child behaviors and CMD risk factors is needed. Examination into how these behaviors predict CMD risk factors in children versus their parents is also warranted.

In this dissertation, we used geographically–diverse longitudinal CHNS data to address these questions. First, we determined the association between parent and child CMD risk factors. Second, we compared the longitudinal changes in diet and activity behaviors in children and their parents, and examined the relationship between parental with children's diet and activity behaviors over time. Third, we investigated how these behaviors longitudinally predict CMD risk factors in children and their parents. We additionally tested whether children's behaviors and CMD risk factors differed by household structure.

CHAPTER 3. CONCORDANCE OF HEMOGLOBIN A1C, BLOOD PRESSURE, AND C-REACTIVE PROTEIN BETWEEN CHILDREN AND THEIR PARENTS IN CHINESE HOUSEHOLDS[1]

Overview

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. We aimed to examine the parent-child association for hemoglobin A1c (HbA1c), blood pressure (BP), and C-reactive protein (CRP) in a large, geographically-diverse Chinese sample.

In 940 parent-child pairs (children aged 7-17y) 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.

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.

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.

Introduction

The prevalence of cardiometabolic disease (CMD) risk factors (e.g., obesity, diabetes, 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 behaviors with parents, [12] which could underlie clustering in CMD risk factors in the household. Parental obesity has been associated with childhood obesity. [108] Correlations of fasting insulin [109] and blood pressure (BP) [39] between children and their parents were also observed in Western countries. To our knowledge, no study has determined the parent-child associations for hemoglobin 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. 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 behaviors, especially in single-child households. [24] 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 on the impact of inflammation on the development of CMD risk factors, [110] 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, 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; South: Hunan, Hubei, Guangxi, Guizhou) in eight survey rounds from 1989-2009. 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. [111] Communities and households were then selected from these strata. The CHNS cohort initially mirrored national age-sex-education profiles [112] and these initial households were followed over time. Details on the survey procedures are described elsewhere. [113] 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 7-17y 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>10mg/L (n=22) because it indicates current infection. [114] 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, 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. [32] While we use continuous HbA1c in our central analyses, elevated HbA1c was defined in secondary analyses using HbA1c≥5.7% for both children and adults as recommended by American Diabetes Association for prediabetes and diabetes. [115] We combined prediabetes and diabetes as elevated HbA1c because diabetes (HbA1c≥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 CDC 2000 growth curve reference. [116] 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≥90th percentile of the algorithm, or SBP≥120mmHg or DBP≥80mmHg, 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≥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. [117]

We measured high-sensitivity CRP via the immunoturbidimetric method 13. The intra- 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 1-10mg/L in children and 3-10mg/L in adults in secondary analyses. [114, 118]

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. [119]

We collected each individual's dietary intake using three consecutive 24-hour dietary recalls at the individual level and a food inventory at the household level. [120] 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 (METs) for that activity. [121]

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), geographic 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/nonsmoker), and for sensitivity analysis child's weight status. We also controlled for child's total energy intake (kcal/d, quartiles), sodium intake

(mg/d, quartiles, BP models only), and PA (MET-hrs/wk, 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 versus 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 to 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 12.1y, 38.5y, and 39.8y, respectively (Table 3.1). 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%) versus 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 0.7mg/L, respectively, and did not differ by household structure.

Boys and girls whose parents had elevated HbA1c had higher HbA1c (Table 3.2). 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 versus 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 to 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 CRP also had higher CRP compared to children whose fathers had normal CRP (1.4mg/L versus 0.8 mg/L in boys and 0.9mg/L versus 0.5mg/L 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 versus 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 (Supplemental Table 3.1). 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 (Supplemental Table 3.2), which shows increased odds of having elevated HbA1c comparing children of parents with elevated versus 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 (Supplemental Table 3.1). 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 analyses, which did not substantially change our results for HbA1c and BP, but the father-son association for CRP was attenuated (Supplemental Table 3.3). Overweight/obesity was positively associated with BP in boys and with CRP in both boys and girls, but was not associated with HbA1c (Supplemental Table 3.4).

Discussion

Our study suggests positive associations between children and their parents for HbA1c, between girls and their mothers for BP, as well as 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. [37] 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. [9, 39] Sinaiko et al. found a correlation for fasting insulin between mothers and their adolescent children in a U.S. population, [9] whereas Park et al. observed parent-child correlation in fasting glucose in Korean adolescents. [39]

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. [42] 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. [8]

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 behaviors, especially diet, with their parents. [12] Previous research has shown a strong correlation between children's and parental diet in China. [11] Despite different study designs, studies in Western countries have found medium to weak parent-offspring associations for diet, as shown in a meta-analysis. [13] The difference in the strength of association is possibly due to shared dishes in Chinese versus 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 etiology compared to diabetes-related outcomes. [122] 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 to

children whose parents had normal BP, which may underlie the observed similarity in BP in offspring of parents with elevated versus 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 to the influence of parental BP status.

Children whose fathers had elevated versus 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 due to the lack of association between maternal CRP and child's weight status. Given existing evidence on the impact of inflammation on the development of diabetes, [110] 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, [9] which indicated that elevated HbA1c and BP during childhood cannot be directly attributed to greater adiposity among these children, perhaps reflecting shared genetic, environmental, behavioral 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. [24] 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 to 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 versus 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 (49y) in China. [123] Nonetheless, we tested associations among younger versus older children, as well as among children with younger versus 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 to child BP references that are only age- and sex-specific. [124] Due to 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.

Tables and figures

Table 3.1. Characteristics of the analytic sample

No. of children	940
No. of mother-child pairs	867
No. of father-child pairs	779
Child's age, y (mean ± S.D.)	12.1±2.9
Maternal age, y (mean ± S.D.)	38.5±4.7
Paternal age, y (mean ± S.D.)	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 ± S.D.)*	40787±43522
Household residence, % urban	24.9
Number of generation, % three-generation†	65.7
Number of children, % one child	73.5
Geographic region, %‡	
North	15.8
Central	31.4
South	52.8
Child's HbA1c, %	
Overall mean, mean ± S.D.	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 ± S.D.)	5.3±0.5
Paternal HbA1c, % (mean ± S.D.)	5.5±0.9
Child's systolic, diastolic BP z-scores, respectively	
Overall mean, mean ± S.D.	-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 ± S.D.)	113.6±13.5, 75.4±9.3
Paternal systolic, diastolic BP, mmHg (mean ± S.D.)	120.0±12.4, 80.7±9.6
Child's CRP, mg/L	
Overall mean, mean ± S.D.	0.7±1.5

Predicted mean (95% CI) by no. of generation in the household¶	
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 (mean ± S.D.)	1.2±1.6
Paternal CRP, mg/L (mean ± S.D.)	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.
** indicates statistically significant difference between groups at $p<0.05$.

Table 3.2. Predicted mean levels of clinical and anthropometric measures in children of parents with normal or elevated HbA1c, BP, and CRP*

	Maternal HbA1c status		P value	Paternal HbA1c status		P value
	Normal (<5.7%)	Elevated (≥5.7%)		Normal (<5.7%)	Elevated (≥5.7%)	
Boys						
n	281	66		219	95	
Age, y[†]	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, y	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		P value	Paternal BP status		P value
	Normal (<120/80mmHg)	Elevated (≥120/80mmHg)		Normal (<120/80mmHg)	Elevated (≥120/80mmHg)	
Boys						
n	254	197		144	275	
Age, y	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‡	4218±193	3950±219	0.36	4309±207	3756±155	0.02
Girls						
n	208	157		113	208	
Age, y	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‡	3348±132	3558±150	0.29	3324±175	3510±126	0.40

	Maternal CRP status		P value	Paternal CRP status		P value
	Normal (<3mg/L)	Elevated (3-10mg/L)		Normal (<3mg/L)	Elevated (3-10mg/L)	
Boys						
n	373	61		325	56	
Age, y	11.9±0.2	13.0±0.4	0.01	12.2±0.2	12.6±0.4	0.99
CRP, mg/L‡	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, y	12.0±0.2	12.8±0.4	0.07	12.2±0.2	12.2±0.4	0.97

CRP, mg/L ‡	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, geographic 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).

Supplemental Table 3.1. Random-effects linear regression analysis of the relationships between parents' and children's HbA1c, BP, and CRP*

	Maternal HbA1c (%)		Paternal HbA1c (%)	
	Beta	95% CI	Beta	95% CI
HbA1c (%)				
Boys (n=364)	**0.43**	0.28, 0.58	**0.18**	0.08, 0.28
Girls (n=299)	**0.25**	0.16, 0.34	**0.06**	0.03, 0.12
	Maternal BP (mmHg)		Paternal BP (mmHg)	
	Beta	95% CI	Beta	95% CI
BP z-score				
Boys (n=496)				
SBP z-score	0.007	-0.001, 0.016	0.004	-0.004, 0.013
DBP z-score	**0.010**	0.002, 0.018	0.003	-0.005, 0.011
Girls (n=398)				
SBP z-score	**0.020**	0.011, 0.028	0.004	-0.006, 0.014
DBP z-score	**0.016**	0.007, 0.025	**0.010**	0.005, 0.019
	Maternal CRP (mg/L)		Paternal CRP (mg/L)	
	Beta	95% CI	Beta	95% CI
CRP (mg/L)				
Boys (n=364)	0.06	-0.05,0.16	**0.15**	0.03, 0.27
Girls (n=299)	0.02	-0.08, 1.11	0.04	-0.03, 0.11

Bold estimates are statistically significant at $p<0.05$ level.

*We conducted separate regression models for mother-child and father-child pairs. Results are shown in beta coefficients and 95% confidence intervals (CIs). Models adjusted for child's age, household income (tertiles), parental education (none or primary/middle school/high school/technical, college or higher), parental smoking (current smoker/nonsmoker), household residence (urban/rural), geographic region (North/Central/South), number of generation in households (2-/3-generation), number of child in households (1/>1) , child's total sodium intake (mg/d, quartiles, in BP models only), child's total energy intake (kcal/d, quartiles), and child's physical activity (MET-hrs/wk, quartiles).

Supplemental Table 3.2. Multivariable logistic regression analysis of parent-child associations for elevated HbA1c, BP, and CRP*

	Maternal elevated HbA1c†		Paternal elevated HbA1c†	
	OR	95% CI	OR	95% CI
Elevated HbA1c†				
Boys (n=364)	**5.47**	2.50, 11.96	**5.11**	2.18, 11.96
Girls (n=299)	**7.87**	3.18, 19.48	**4.33**	1.83, 10.23
	Maternal elevated BP†		Paternal elevated BP†	
	OR	95% CI	OR	95% CI
Elevated BP†				
Boys (n=496)	0.81	0.48, 1.37	0.99	0.55, 1.79
Girls (n=398)	1.63	0.89, 2.97	1.26	0.66, 2.41
	Maternal elevated CRP†		Paternal elevated CRP†	
	OR	95% CI	OR	95% CI
Elevated CRP†				
Boys (n=364)	1.30	0.63, 2.72	**2.06**	1.05, 4.04
Girls (n=299)	1.44	0.63, 3.29	2.00	0.91, 4.38

Bold estimates are statistically significant at $p<0.05$ level.

*We conducted separate logistic regression models for mother-child and father-child pairs. Results are shown in odds ratios (ORs) and 95% confidence intervals (CIs). Models adjusted for child's age, household income (tertiles), parental education (none or primary/middle school/high school/technical, college or higher), parental smoking (current smoker/nonsmoker), household residence (urban/rural), geographic region (North/Central/South), child's total sodium intake (mg/d, quartiles, BP models only), number of generation in households (2-/3-generation), number of child in households (1/>1), child's total energy intake (kcal/d, quartiles) and child's physical activity (MET-hrs/wk, quartiles).

†Elevated HbA1c: HbA1c≥5.7%. Elevated BP: SBP z-score or DBP z-score ≥90^{th} percentile of the age-, sex-, and height- specific BP percentile or BP≥120/80mmHg for children using the US CDC 2000 growth curve reference; BP≥120/80mmHg for adults. Elevated CRP: 1-10 mg/L for children and 3-10 mg/L for adults.

Supplemental Table 3.3. Random-effects linear regression analysis of the relationships between parents' and children's HbA1c, BP, and CRP, additionally adjusting for child's weight status*

	Maternal HbA1c (%)		Paternal HbA1c (%)	
	Beta	95% CI	Beta	95% CI
HbA1c (%)				
Boys (n=364)	**0.46**	0.30, 0.62	**0.18**	0.08, 0.28
Girls (n=299)	**0.24**	0.16, 0.33	**0.06**	0.01, 0.11
	Maternal BP (mmHg)		Paternal BP (mmHg)	
	Beta	95% CI	Beta	95% CI
BP (z-score)				
Boys (n=496)				
SBP z-score	0.007	-0.001, 0.015	0.003	-0.005, 0.012
DBP z-score	**0.010**	0.002, 0.017	0.002	-0.006, 0.010
Girls (n=398)				
SBP z-score	**0.019**	0.010, 0.028	0.005	-0.005, 0.014
DBP z-score	**0.016**	0.007, 0.025	**0.010**	0.001, 0.019
	Maternal CRP (mg/L)		Paternal CRP (mg/L)	
	Beta	95% CI	Beta	95% CI
CRP (mg/L)				
Boys (n=364)	0.07	-0.04,0.17	**0.12**	0.001, 0.25
Girls (n=299)	0.01	-0.08, 0.11	0.04	-0.04, 0.11

Bold estimates are statistically significant at $p<0.05$ level.

*We conducted separate regression models for mother-child and father-child pairs. Results are shown in beta coefficients and 95% confidence intervals (CIs). Models adjusted for child's age, parental education, household income (tertiles), parental education (none or primary/middle school/high school/technical, college or higher), parental smoking (current smoker/nonsmoker), household residence (urban/rural), geographic region (North/Central/South), child's total sodium intake (mg/d, quartiles, BP models only), number of generation in households (2-/3-generation), number of child in households (1/>1), child's total energy intake (kcal/d, quartiles), child's physical activity (MET-hrs/wk, quartiles), and child's weight status (normal weight/overweight/obese using the International Obesity Task Force reference).

Supplemental Table 3.4. Odds ratios (95% confidence intervals) of elevated HbA1c, BP, and CRP* according to overweight/obese status† in children

	Elevated HbA1c		Elevated BP		Elevated CRP	
	Normal weight	Overweight/ obese	Normal weight	Overweight/ obese	Normal weight	Overweight/ obese
		Boys				
No. of cases (%)	59 (17.6)	7 (15.9)	66 (15.2)	18 (26.5)	98 (31.4)	24 (58.5)
Multivariable model‡	1.00 (ref.)	0.75 (0.28, 1.97)	1.00 (ref.)	2.36 (1.29, 4.34)	1.00 (ref.)	3.45 (1.74, 6.89)
		Girls				
No. of cases (%)	41 (14.2)	7 (22.6)	61 (19.6)	13 (31.7)	68 (21.5)	13 (38.2)
Multivariable model‡	1.00 (ref.)	1.27 (0.44, 3.67)	1.00 (ref.)	1.96 (0.95, 4.02)	1.00 (ref.)	3.00 (1.28, 7.01)

Bold estimates are statistically significant at $p<0.05$ level.

*Elevated HbA1c: HbA1c≥5.7%. Elevated BP: SBP z-score or DBP z-score ≥90^{th} of the age-, sex-, and height-specific BP percentile or BP≥120/80mmHg for children using the US CDC 2000 growth curve reference; BP≥120/80mmHg for adults. Elevated CRP: 1-10 mg/L for children and 3-10 mg/L for adults.

†Overweight/obese: ≥85^{th} percentile of the age- and sex-specific reference data from the International Obesity Task Force.

‡Adjusted for child's age, parental education, household income (tertiles), parental education (none or primary/middle school/high school/technical, college or higher), parental smoking (current smoker/nonsmoker), household residence (urban/rural), geographic region (North/Central/South), child's total sodium intake (mg/d, quartiles, BP models only), child's total energy intake (kcal/d, quartiles), and child's physical activity (MET-hrs/wk, quartiles)

ENDNOTES

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CHAPTER 4. PARENT-CHILD ASSOCIATIONS FOR CHANGES IN DIET, SCREEN TIME, AND PHYSICAL ACTIVITY ACROSS TWO DECADES IN MODERNIZING CHINA: CHINA HEALTH AND NUTRITION SURVEY 1991-2009

Overview

While the household context is important for lifestyle interventions to reduce cardiometabolic disease, few studies have examined parent-child associations for diet and physical activity (PA) changes over time in a rapidly urbanizing country with burgeoning cardiometabolic disease rates. We aimed to investigate changes in diet, screen time, and PA behaviors over time in children and their parents living in the same household, and examine the parent-child association for these behaviors.

We studied dietary, screen time, and PA behaviors in 5,201 parent-child pairs (children aged 7-17y) using longitudinal data from the China Health and Nutrition Survey (1991, 1993, 1997, 2000, 2004, 2006, and 2009). We collected three-day 24-hour recall diet data to generate percentages of energy from animal-source foods, away-from-home eating, and snacking from 1991-2009, which are known urbanization-related behaviors. We used a seven-day PA recall to collect screen time (hours/week) and leisure-time sports participation (yes/no) since 2004. We examined the changes in children's and parents' behaviors over time using random-effects negative binomial regression for diet and screen time, and random-effects logistic regression for leisure-time sports. We then regressed each of the behaviors of offspring on each of their parents' same behaviors to examine the parent-child association, using the same set of models.

We observed increases in energy from animal-source foods, eating away-from-home, and snacking, as well as screen time and leisure-time sports in parents and children over time, with different rates of change between children and their parents for some behaviors. We found positive parent-child associations for diet, screen time, and PA. When parental intakes increased by 10% energy from each dietary behavior, children's increase in intakes ranged from 0.86% to 1.27% total energy for animal-source foods, 0.23% to 0.42% for away-from-home eating, and 3.21% to 7.92% for snacking. Children were also more likely to participate in leisure-time sports if their parents participated in leisure-time sports.

Our findings support household-based health behavior interventions targeting both children and their parents. However, generation-specific intervention strategies may be needed for children and adults, especially for dietary behaviors, which changed differentially in children versus parents in this rapidly modernizing population.

Introduction

The prevalence of cardiometabolic disease risk factors (e.g., obesity, type 2 diabetes, hypertension) has been burgeoning over the past two decades in China among both adults and children, with a faster increase in children. [1, 2] Dietary and physical activity (PA) behaviors are important contributors to cardiometabolic health. [20, 21] Thus, understanding factors that influence these behaviors can be essential for designing successful interventions. Family indisputably plays an important role in shaping health-related behaviors of the household unit. Children's diet and PA behaviors have been shown to be associated with those of their parents. [10-13, 125-127]This is possibly because parents may share meals and participate in PA with their offspring and can serve as role models and influence children's preferences and attitudes towards diet and PA. However, in the face of environmental change, such as that seen in China

with modernization, rates of change in childhood eating behaviors have been shown to differ from those of adults. [50]

Past research on the parent-offspring association for diet and PA, however, is mostly cross-sectional. These studies were unable to examine the change of diet and PA behaviors over time, especially in rapidly modernizing populations such as China. Evaluating and comparing changes in diet and PA behaviors in children and their parents is essential to understand whether urbanization influences children and adults differently and to track whether changes in children mirror those of their parents. Although a few longitudinal studies have investigated the parent-child association for diet and PA behaviors, these studies have focused on high income countries [12] or only examined the mother-child association. [11, 125] Besides the influence of parents, the presence of grandparents and siblings may also play a role in children's health behaviors, possibly because grandparents are more likely to indulge their grandchildren with modern, unhealthy foods and screen-based sedentary behaviors, especially in single-child households. [23-25] Nonetheless, to our knowledge, no research has examined how household structure (i.e. living with grandparents or not, having siblings or not) relates to children's diet and PA behaviors in large Chinese population-based studies.

To address these gaps, we used longitudinal data from children and their parents enrolled in the China Health and Nutrition Survey (CHNS) from 1991 to 2009 to study changes in urbanization-related diet, screen time, and PA behaviors over time in children compared to their parents. We also examined whether children's diet, screen time, and PA behaviors associated with those of their parents, and whether these behaviors in children differed by household structure.

Methods

CHNS. The CHNS is a household-based longitudinal cohort study with ongoing data collection in 228 communities across nine provinces throughout China (North: Heilongjiang, Liaoning; Central: Shandong, Henan, Jiangsu; South: Hunan, Hubei, Guangxi, Guizhou) in nine survey rounds from 1989-2011. 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. [111] Communities and households were then selected from these strata. We used questionnaires to collect demographic, socioeconomic, behavioral, and health information from each household member. The CHNS cohort initially mirrored national age-sex-education profiles [112, 128, 129] and the provinces in the CHNS sample constituted 44% of China's population in 2009 (according to 2009 census). More details on the survey procedures are described previously. [130] 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 longitudinal data from 1991, 1993, 1997, 2000, 2004, 2006, and 2009 when data on parents and children were collected. Eligible subjects were parent-child pairs with children aged 7-17y at any exam period who had at least two waves of diet, screen time, and PA data (n=5,287). Due to the age restriction, participants aged in and out of our sample at different years during the 18-year follow up, thus at each year there was a mixture of different groups of individuals followed for 2-4 surveys instead of one group of participants who were followed over time. We excluded parent-child pairs with missing covariates (n=86). Our final analytic sample included 5,201 unique parent-child pairs with data across the study period

(5,151 mother-child and 5,091 father-child pairs; 4,979 households had all three members) with an average of 2.4 visits for diet behaviors; 2.3 visits for screen time and PA behaviors. Compared to the excluded parent-child pairs, children included in the analytic sample were more likely to have lower household income, to live in less urbanized areas, to have parents with lower education level, and to fit the age criteria in earlier survey years (Supplemental Table 1). Included children also had lower mean values for animal source-foods, away-from home eating, and screen time relative to excluded children.

Diet. Our diet data were derived from three consecutive 24-hour dietary recalls at the individual level and a food inventory at the household level occurring during the same 3-day period, randomly starting from Monday to Sunday. All foods available in the household were measured on a daily basis for the food inventory. For the 24-hour recalls, trained interviewers recorded types and amounts of foods, types of meal, and places of food preparation of all food items consumed by each household member. For children younger than 10y, mothers or mother substitutes were asked to recall children's dietary intake. The energy content of foods was based on a Chinese food composition table. [131] In this study we focused on three dietary behaviors: animal-source food intake, away-from-home food consumption, and snacking. Animal-source foods included meats and meat products, eggs and egg products, fish and seafood, milk and dairy products. We defined away-from-home foods as foods prepared away from home (no matter if they were consumed at or away from home) and snacks were reported as foods consumed outside the three main meals (breakfast, lunch and dinner).

Physical activity (PA). Our PA data were derived from seven-day PA recalls across a variety of domains. Children and parents were asked about their participation and time spent in different types of sedentary behavior and PA. Parents or primary caregivers completed or

assisted with completing the surveys for children under 10y. In this study we examined screen time (hours/week) and leisure-time sports participation (yes/no). Screen time referred to time spent on TV/videotape watching, video games, and computer usage. Leisure-time sports included gymnastics, dancing, track and field sports, swimming, ball sports (e.g. basketball, tennis), and other sports (e.g., martial arts, tai chi). We focused on children's leisure-time sports outside school only (including before or after school for those in school and any activity for those not in school) for the purpose of studying the parent-child correlation, since in-school sports participation is mostly influenced by school instead of parents. Due to the low participation rate in leisure-time sports in both parents and children in all survey years, we dichotomized the variable into any versus no participation. Since data on screen time in adults were first collected in 2004 and the leisure time sports survey changed in 2004, we restricted our analyses for these behaviors to 2004, 2006, and 2009 only.

Covariates. We collected participants' age (y) at baseline (year when the child aged into the 7-17y range), sex, number of children in the household (1/>1), generation of family members in the household (2-generations: parents and children; 3-generations: grandparents, parents, and children), household income (inflated to 2011, tertiles), geographic region (North/Central/South), year of study entry, and highest parental education (none or primary/middle school/high school/technical, college or higher). We also used the CHNS multicomponent urbanicity scale comprised of 12 urban environment domains representing infrastructure, economic, and social service. The scale has high reliability and validity. [132] The scale ranges from 0-120 with a higher score reflecting higher urbanicity, which was categorized into year-specific tertiles.

Statistical analysis. We conducted all analyses using Stata 14.0 (Stata Corp, College Station, TX, USA), and used a p-value < 0.05 as our significance level. In the descriptive

analysis, we examined demographic characteristics of our analytic sample and tested differences of these characteristics over time using the chi-squared test (categorical variables) and one-way ANOVA (continuous variables).

For statistical models, we conducted random-effects negative binomial regression for our three dietary behaviors and screen time, with the random intercept for individuals. Three dietary variables are presented as the percentage of total energy (% energy) from these foods. Screen time is presented as hours per week. We used negative binomial models because the distribution of these variables was skewed and over-dispersed. We ran separate models for each behavior. For leisure-time sports, we used random-effects logistic regression to determine the change in the probability of participation in sports over time, accounting for repeated measures.

First, we examined the change in diet, screen time, and PA behaviors in children and their parents over time. Main exposures included year and dummy variables indicating household members (child, mother, father). To test whether the change in behaviors differed between children and their parents, we examined interactions between year and household members using the Wald test. We also tested if children's change in behaviors differed by child's baseline age and sex. Models controlled for sex and baseline age, time-varying household income and urbanicity, geographic region, and year of study entry. Then, we predicted adjusted mean values or adjusted probability of each behavior at each year by household member, with the year-household member interaction included in the models where the interaction was statistically significant.

Because participants aged in and out of our sample at different times during the 18-year follow up, at each wave there was a mixture of different groups of individuals surveyed for 2-4 time points, instead of one identical group of participants who were followed over time. To test

whether changes in behaviors were similar for an identical group of participants followed over time, we tested such differences in sensitivity analysis. Due to the age restriction of our sample, the maximum number of surveys a parent-child pair could complete was four. Therefore, in this sensitivity analysis we re-ran our dietary models among two separate groups of individuals who completed all four surveys. The first group of identical individuals was followed from 1991 to 2000 (n=1,392), whereas the second was followed from 2000 to 2009 (n=768). For screen time and PA, we re-ran our analyses among one group of individuals who completed all three surveys from 2004 to 2009 (n=1,725).

Next, to determine the association between children's behaviors with parental behaviors, we regressed each behavior of offspring on their parents' same behavior, controlling for the same set of potential confounders. We ran separate models for mother-child and father-child pairs. For animal-source foods, away-from-home eating, and snacking, we predicted the change of children's daily intake (% energy) when parental intake increased by 10% total energy. For screen time, we predicted children's change in screen time (hours/week) when parental screen time increased by one hour per week. We also estimated the odds ratios (OR) of leisure-time sports participation in children based on parental participation status. To examine whether the associations differed across years, we tested for interactions between parental behaviors with year. To further explore factors modifying the association, we examined the modification of association by sociodemographic factors including child's sex and baseline age, household income, urbanicity, and geographic region. We predicted the associations with the interaction terms included in the models where the interactions were statistically significant.

Last, we examined whether children's diet, screen time, and PA varied by household structure after adjusting for potential confounders. Main exposures were year and household

structure variables (number of children in the household, generation of family members in the household). Then, we predicted adjusted mean values or adjusted probability of each behavior across all years by household structure using the models.

Results

We observed increases in parental education level, household income, and urbanicity from 1991 to 2009 ($p<0.05$; Table 1). The proportions of three-generation households and one-child households also increased over time.

Model-adjusted predictions showed increasing percentages of energy from animal-source foods, away-from-home eating, and snacking in both children and their parents from 1991 to 2009 (Figure 4.1; beta coefficients shown in Supplemental Table 4.2). Compared to their parents, children consumed less energy from animal-source foods and away-from-home eating, but more energy from snacks. We detected statistically significant differences in the rate of change in behaviors for children compared to their parents for away-from-home eating and snacking ($p<0.05$), indicating a faster increase of these behaviors in children versus adults in later years. Screen time and leisure-time sports participation increased in both children and their parents over time. Children spent less time on screen-based activities and were more likely to participate in leisure-time sports compared to their parents. Children's screen time increased faster than their parents from 2004-2006 and slower than their parents from 2006-2009. We saw no differences in rates of change in the probability of leisure-time sports participation over time between children and their parents. Nor did we observe differences in change in these behaviors over time by child's sex or baseline age.

Sensitivity analysis showed similar patterns of changes in animal-source foods and snacking among two groups of identical individuals followed from 1991 to 2000 and from 2000 to 2009, respectively (Supplemental Figure 4.1), compared to our central analysis. For eating away-from-home among individuals followed from 1991 to 2000 we observed faster increase among children than their parents, but the same rate for children and their parents from 2000 to 2009. For screen time, the rate of change did not differ across household members. There was no statistically significant change in the probability of leisure-time sports participation from 2004 to 2009 for all household members.

We found positive associations between children's behaviors with those of their parents (Table 4.2). There was statistically significant effect measure modification by year for all behaviors except for leisure-time sports and by urbanicity for animal-source foods, away-from-home eating, and screen time, so results are presented by year for all behaviors and by urbanicity where the interaction was statistically significant. We also detected statistically significant modification by geographic region and by household income for some behaviors. Due to the inclusion of multiple interactions in our models, we present the parent-child association by year and urbanicity at the medium income level in the Central region in Table 4.2. We saw no evidence of modification of the parent-child association in diet, screen time, and PA by sex or by child's baseline age.

Children's dietary behaviors were positively associated with those of their parents, although in general the magnitude of associations declined over time. For example, at the medium urbanicity level, with 10% increase in energy from animal-source foods in mothers, children's intake increased by 1.25% (95% CI: 1.16-1.35) in 1991 and by 0.90% (95% CI: 0.80, 1.00) in 2009. For away-from-home eating, when maternal intake increased by 10%, children's

intake increased by 0.41% (95% CI: 0.38, 0.43) in 1991 and by 0.25% (95% CI: 0.22, 0.27) in 2009. For snacking, with 10% increase in maternal intake, children's intake increased by 6.44% (95% CI: 3.46, 9.43) in 1991 and by 3.44% (95% CI: 1.73, 5.36) in 2009. For animal-source foods in both mother-child and father-child pairs, and for away-from-home eating in mother-child pairs, the parent-offspring associations were modified by urbanicity, as shown in Table 2. The associations were weaker with higher urbanicity levels. For instance, with 10% increase in paternal intake of animal-source foods in 1991, children's intake increased by 0.90% (95% CI: 0.79, 1.01) in high urbanicity areas, compared to by 1.44% (95% CI: 1.33, 1.56) in low urbanicity areas.

Similarly, children's screen time and PA were positively associated with those of their parents. For example, at medium urbanicity, with one hour/week increase in mother's screen time, children's screen time increased by 0.02 hour/week (95% CI: 0.02, 0.03) in 2004 and by 0.01 hour/week (95% CI: 0.00, 0.02) in 2009. Compared to children whose parents did not participate in leisure-time sports, children whose parents participated were more likely to participate in leisure-time sports [OR: 1.97, 95% confidence interval (CI): 1.23-3.18 for mother-child pairs; OR: 2.80, 95% CI: 1.74, 4.50 for father-child pairs], with no difference across years.

In North and South regions, the magnitude of parent-offspring associations for these behaviors also declined over time, similar to the Central region (data not shown). In Supplemental Table 4.3, we show the parent-child association by household income and geographic region at year 2000 for all dietary behaviors and at year 2004 for screen time. We found no interaction for leisure-time sports by any covariates; therefore leisure-time sports is not included in Supplemental Table 3.

Children's diet and PA behaviors varied significantly by household structure (Table 4.3). Compared to children living with siblings, children without siblings consumed higher percentages of energy from all three categories. Only children were also more likely to participate in leisure-time sports than children living with siblings. Compared to children in households without grandparents, those living with grandparents consumed slightly less away-from-home foods. .

Discussion

This longitudinal study provides insights into diet, screen time, and PA dynamics among children and their parents in rapidly modernizing China. Our findings suggest that over the follow-up, changes in behaviors over time differed between children and their parents. Despite the difference, overall we found positive parent-offspring associations for diet, screen time, and PA, although the magnitude of associations varied across behaviors. Higher intakes of animal-source food, away-from-home food, and snacks in parents were associated with higher intakes of these foods in their children. Children also spent more time on screen-based activities when their parents had higher screen time, and children were more likely to participate in leisure-time sports if their parents engaged in leisure-time sports. On the other hand, the magnitude of parent-offspring associations for these behaviors weakened over time for some but not all behaviors, suggesting differences in the face of the changing environment in China. We also observed statistically significant differences in diet and PA behaviors in children by household structure. Children with no siblings had higher intakes of animal-sources food, away-from-home food, and snacks compared to children who shared households with siblings.

We found statistically significant parent-child differences for the rate of changes in some, but not all, diet, screen time, and PA behaviors. Particularly, the increase in away-from-home

eating and snacking was greater in children than in their parents in later years. Others have found some of these behavioral changes are linked with obesity. [133] A similar difference between offspring and maternal changes in obesogenic diet in response to urbanization was reported in a Filipino population. [50] This is possibly because children adjust more quickly to environmental changes than adults who are reluctant to accept changes. [134] Numerous food commercials targeting children as well as the increasing availability, accessibility, and affordability of meals and snacks at school may have also contributed to more rapid increase in intakes of away-from-home food and snacks in children than in adults. This greater change in diet behaviors in children might partly explain the faster increase of obesity in children compared to adults in the past two decades. [2]

Although similar diet, screen time, and PA behaviors between children and their parents seem to be apparent, Wang et al have shown inconsistent parent-offspring relationships for diet in their meta-analysis. [13] The inconsistencies across studies may be due to differences in the study population, study time, diet and PA assessment tools, and analytical methods. [13] In our study, we found positive parent-offspring associations for diet, screen time, and PA behaviors. While previous studies mostly focused on intakes of certain food groups or nutrients when studying the parent-child resemblance in diet, [10, 12, 135] we found positive parent-offspring associations for eating away-from-home and snacking. The magnitude of associations differed across behaviors but was stronger for snacking and leisure-time sports than other behaviors. A number of mechanisms may explain the observed parent-child associations for health behaviors. First, it is possible that parents act as role models for children, and children adopt their parent's behavioral habits and attitudes. Second, shared household environment may have influenced both children's and adults' behaviors in similar ways. For example, studies have found positive

associations between accessibility of facilities in the neighborhood (e.g., open space, recreational center, park) and participation in physical activity in both adults and children. [56, 57] Third, studies have shown that genetic predisposition may also contribute to the familial concordance of diet and PA behaviors. [51, 58]

Despite the observed parent-offspring association for diet behaviors and screen time, the magnitude of these associations decreased over time, indicating possible differential influences of urbanization-related environmental changes in adults versus children. This is supported by our observed differential magnitude of parent-offspring associations for some of the behaviors by urbanicity, which indicates weaker associations in higher urbanicity areas. Increasing pocket money associated with the growing household income over time might have also led to less concordance in behaviors between children and their parents, particularly in diet, since children with extra pocket money are more likely to purchase meals or snacks on their own outside home.

In addition to the influence of parents, we found that children's diet and PA behaviors also differed by household structure. This was particularly clear in the case of one-child family structure, which was associated with more modernized dietary behaviors in children relative to households with more than one child, independent of urbanicity and income levels of the household. The "One-Child Policy" implemented in 1979 resulted in a high proportion of single-child households. These only children often receive more attention and the best care in the household than children living with siblings. [60] As a compensation of their past experience of food shortage and deprivation, parents tend to indulge their only children with modern foods. The presence of grandparents in the household has also been associated with less healthy dietary habits in children, including larger portions of meals and higher consumption of unhealthy snacks. [23-25] This is due to grandparents' desire to over-indulge their grandchildren and a

belief that heavier children are healthier and children who eat more will grow taller. A previous study among Japanese children found an association between the presence of grandparents and physical inactivity. [59] In our research, children living with a grandparent had a lower probability of engaging in leisure-time sports, although the difference was not statistically significant. Unhealthy diet behaviors and physical inactivity may have contributed to the higher risk of overweight/obesity among children living in only-child and/or three-generation households relative to those who have siblings and live in two-generation households, as observed in previous studies. [61-64] Intervention strategies may focus on improving dietary habits and promoting PA to enhance the ability of improving health outcomes in children in such household situations.

Our study has some limitations. First, self-reported diet, screen time, and PA data may be subject to recall and social desirability biases, although the CHNS data are based on highly detailed recall methods, including analysis of household-specific recipes, weighing and measuring of condiments. Previous research on selected components of the diet (energy, protein, sodium, monosodium glutamate) suggest strong validity, [120, 136-139] and the PA components have been found to be highly predictive of incident obesity and weight gain in adults. [49, 140-142] Second, loss to follow-up could have caused selection bias since we restricted to parent-child pairs who had at least two waves of diet, screen time, and PA data within the age range. We could have slightly underestimated the predicted values of some of the behaviors due to lower intakes of animal-sources food and away-from-home food, and lower levels of screen time in the included compared to the excluded samples. Third, for younger children who were under 10y, parents who reported their own diet, screen time, and PA and those of their children could have resulted in potential same-source bias and high parent-child correlation, especially for diet.

However, we conducted sensitivity analyses to test whether our finding were robust to whether the child's survey was aided by their parents (aged 7-10y only, 22% total observations) and found similar pattern of changes and magnitude of estimated associations (not presented). Fourth, the lack of sampling weights in CHNS does not allow predicting population-relevant estimates of these behaviors. Finally, we cannot imply causation from these associations as it is impossible to unravel within-household dynamics.

In spite of its limitations, our study has several notable strengths. First, CHNS is one of the only population-based, longitudinal studies with diet and PA data collected at the household level over 20 years of rapid environmental change. Capitalizing on this unique dataset, we were able to determine and compare changes in diet, screen time, and PA in children and their parents during two decades in this rapidly modernizing country. Studying changes in health behaviors helps us understand the rapidly increasing prevalence of cardiometabolic risk factors in China in the past twenty years, whereas comparing the difference in changes between children and their parents provides insights into why urbanization-related increase of obesity is faster in children relative to adults. [2] Second, we collected detailed dietary behaviors from each member of the household, allowing us to examine additional diet behaviors besides nutrients or food intakes commonly assessed in previous studies. Third, using random-effects negative binomial regression models instead of simple correlation analysis, we were able to account for repeated measures over time within individuals.

In summary, our findings suggest that changes in diet, screen time, and PA behaviors over the past two decades differed between children and parents in urbanizing China. Children's behaviors were positively associated with parental behaviors, although the magnitude of the associations declined over time. Our work supports household-based versus individual-based

health behavior interventions for both parents and children in promoting healthy dietary habits and increasing PA. However, generation-specific intervention strategies may be needed for children versus adults, especially for dietary behaviors, in household-based interventions due to the difference in changes of behaviors between generations with rapid environmental change. Household structure should also be considered for interventions targeting children's behaviors in this population.

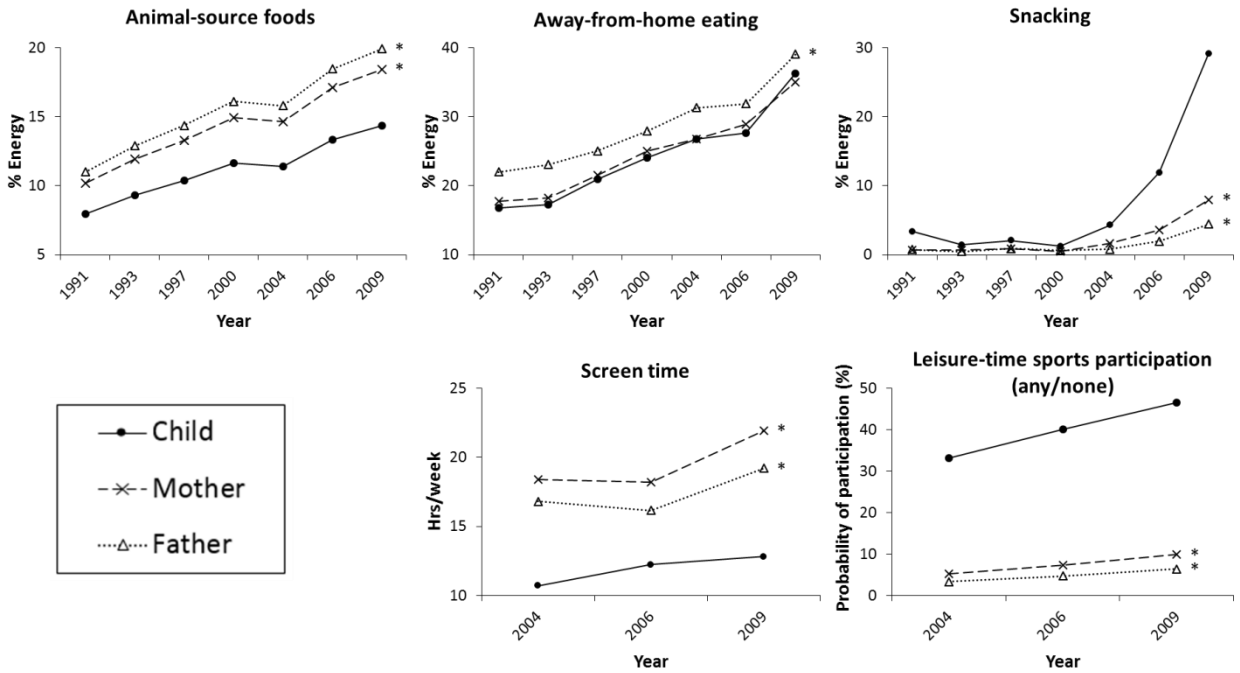
Tables and Figures

Table 4.1. Characteristics of analytic sample over time, China Health and Nutrition Survey 1991-2009

	1991	2000	2009
No. of parent-child pairs	2257	2638	772
Child's age, y (mean ± SD)*	12.6±3.2	13.0±2.9	14.2±2.3
Mother's age, y (mean ± SD)*	38.5±5.4	39.1±5.5	40.3±4.4
Father's age, y (mean ± SD)*	40.4±6.0	40.5±5.8	41.8±5.0
Child's gender, % male	51.4	52.7	53.2
Highest parental education, %*			
None/primary school	18.8	8.2	9.2
Middle school	28.4	18.5	22.8
High school	49.1	67.0	58.9
College, technical or higher	3.8	5.8	9.1
Number of generation, % three-generation†*	26.1	33.1	57.9
Number of children, % one child*	47.2	45.9	72.4
Annual household income, 1000 yuan (mean ± SD)‡*	11.4±8.2	19.2±19.8	40.0±40.0
Urbanicity (mean ± SD)§*	42.4±15.2	54.2±16.9	61.6±18.4

*Statistically different across years at the $p<0.05$ level using one-way ANOVA (continuous variables) or chi-squared test (categorical variables).
†Three-generation: children, parents and grandparents (versus two-generation: children and parents).
‡Total household income inflated to 2011.
§Urbanicity defined by a multicomponent urbanicity scale ranging from 0-120 [132].

Figure 4.1. Predicted mean or probability of diet, screen time and PA among children, mothers, and fathers[a]



[a]Separate random-effects negative binomial regression models for each behavior predicted adjusted mean values of animal-source foods, away-from-home eating, snacking, and screen time; random-effects logistic regression model predicted adjusted probability of leisure-time sports participation. All models controlled for baseline age (y), household income (tertiles), urbanicity (tertiles), geographic region (North/Central/South), and year of study entry. The rate of changes in away-from-home eating, snacking, and screen time was different across household members (p for interaction<0.05). PA, physical activity.

*p<0.05 comparing the mean dietary, screen time, and PA measures (across all years) between the starred parent and the child (the reference).

Table 4.2. Predicted parent-offspring associations for diet (% energy), screen time (hours/week), and leisure-time sports (any/none)[a]

	1991	1993	1997	2000	2004	2006	2009	P-interaction with year
	Beta (95% CI)	Beta (95% CI)	Beta (95% CI)	Beta (95% CI)	Beta (95% CI)	Beta (95% CI)	Beta (95% CI)	
Animal-source foods, % energy[b]								
Mother (n=4264)								
Low urbanicity	1.57 (1.47, 1.66)	1.48 (1.38, 1.57)	1.43 (1.34, 1.51)	1.27 (1.19, 1.34)	1.31 (1.24, 1.39)	1.23 (1.15, 1.31)	1.21 (1.11, 1.31)	
Med urbanicity	1.25 (1.16, 1.35)	1.16 (1.07, 1.25)	1.11 (1.03, 1.19)	0.95 (0.88, 1.02)	1.00 (0.93, 1.07)	0.92 (0.84, 1.00)	0.90 (0.80, 1.00)	<0.001
High urbanicity	0.91 (0.82, 1.01)	0.82 (0.74, 0.91)	0.77 (0.70, 0.85)	0.62 (0.55, 0.68)	0.66 (0.59, 0.73)	0.58 (0.51, 0.66)	0.56 (0.46, 0.66)	
Father (n=3906)								
Low urbanicity	1.44 (1.33, 1.56)	1.36 (1.25, 1.47)	1.30 (1.20, 1.39)	1.19 (1.10, 1.28)	1.21 (1.13, 1.30)	1.16 (1.07, 1.25)	1.12 (1.01, 1.23)	
Med urbanicity	1.21 (1.10, 1.32)	1.13 (1.02, 1.23)	1.06 (0.97, 1.16)	0.96 (0.88, 1.04)	0.98 (0.90, 1.06)	0.93 (0.84, 1.02)	0.89 (0.78, 1.00)	<0.001
High urbanicity	0.90 (0.79, 1.01)	0.82 (0.71, 0.92)	0.75 (0.66, 0.84)	0.65 (0.57, 0.73)	0.67 (0.59, 0.74)	0.61 (0.53, 0.70)	0.58 (0.47, 0.69)	
Away-from-home eating, % energy[b]								
Mother (n=4280)								
Low urbanicity	0.45 (0.42, 0.48)	0.43 (0.41, 0.46)	0.41 (0.38, 0.43)	0.35 (0.32, 0.37)	0.33 (0.31, 0.36)	0.35 (0.32, 0.38)	0.29 (0.26, 0.32)	
Med urbanicity	0.41 (0.38, 0.43)	0.39 (0.37, 0.42)	0.37 (0.34, 0.39)	0.31 (0.28, 0.33)	0.29 (0.27, 0.31)	0.31 (0.28, 0.34)	0.25 (0.22, 0.27)	<0.001
High urbanicity	0.33 (0.30, 0.36)	0.31 (0.29, 0.34)	0.29 (0.26, 0.31)	0.23 (0.21, 0.25)	0.21 (0.19, 0.24)	0.23 (0.21, 0.26)	0.17 (0.14, 0.20)	
Father (n=3917)	0.28 (0.25, 0.30)	0.28 (0.26, 0.30)	0.27 (0.25, 0.29)	0.25 (0.23, 0.27)	0.24 (0.22, 0.26)	0.26 (0.24, 0.29)	0.23 (0.20, 0.26)	0.002
Snacking, % energy[b]								
Mother (n=4276)	6.44 (3.46, 9.43)	7.92 (4.89, 10.95)	7.50 (4.79, 10.22)	5.60 (3.50, 7.70)	7.91 (5.86, 9.95)	6.09 (4.34, 7.84)	3.55 (1.73, 5.36)	<0.001
Father (n=3918)	4.97 (3.46, 6.48)	7.68 (5.75, 9.60)	5.72 (4.16, 7.28)	4.21 (2.68, 5.74)	7.38 (4.97, 9.78)	4.91 (3.14, 6.68)	3.21 (1.57, 4.85)	<0.001
Screen time, hrs/wk[c]								
Mother (n=845)								
Low urbanicity	-	-	-	-	0.03 (0.02, 0.04)	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)	

Med urbanicity	-	-	-	-	0.02 (0.02, 0.03)	0.01 (0.00, 0.02)	0.01 (0.00, 0.02)	<0.001
High urbanicity	-	-	-	-	0.03 (0.02, 0.04)	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)	
Father (n=742)	-	-	-	-	0.02 (0.01, 0.02)	0.01 (0.01, 0.02)	0.01 (-0.00, 0.01)	0.009
Any leisure-time sports participation, %[d]								
Mother (n=817)	-	-	-	-	1.97 (1.23, 3.18)	1.97 (1.23, 3.18)	1.97 (1.23, 3.18)	0.320
Father (n=735)	-	-	-	-	2.80 (1.74,4.50)	2.80 (1.74,4.50)	2.80 (1.74,4.50)	0.996

[a]Table shows coefficients at medium income in the Central region, and by urbanicity where there was statistically significant interaction by urbanicity. Separate random-effects negative binomial regression models for each behavior predicted beta coefficients for animal-source foods, away-from-home eating, snacking, and screen time; random-effects logistic regression model predicted odds ratios (OR) for leisure-time sports participation. All models controlled for child's baseline age (y) and sex, household income (tertiles), urbanicity (tertiles), geographic region (North/Central/South), year of study entry, and highest parental education (none or primary/middle school/high school/technical, college or higher).

[b]Beta coefficients for animal-source foods, away-from-home eating, and snacking indicate the change of child's daily intake in percentage of total energy with mother's or father's intake increased by 10% total energy.

[c]Beta coefficients for screen time indicate the change of child's screen time in hours/week with mother's or father's screen time increased by one hour per week.

[d]Parent-child association presented as ORs of participation in children based on parental participation status.

Table 4.3. Predicted mean/probability (standard error) of diet, screen time, and PA in children by household structure[a]

	Has siblings	Only child	*P*	No grandparents	Has grandparents	*P*
Animal-source foods, % energy	12.0 (0.3)	13.4 (0.3)	<0.001	12.8 (0.3)	12.7 (0.3)	0.82
Away-from-home eating, % energy	21.1 (0.3)	25.0 (0.3)	<0.001	23.8 (0.3)	22.1 (0.3)	<0.001
Snacking, % energy	3.2 (0.7)	6.0 (1.2)	<0.001	5.1 (1.1)	6.0 (1.3)	0.21
Screen time, hrs/wk	13.3 (0.4)	14.1 (0.3)	0.11	14.2 (0.4)	13.5 (0.3)	0.12
Any leisure-time sports participation, %	26.4 (1.8)	32.9 (1.3)	0.01	32.5 (1.6)	29.1 (1.4)	0.13

[a]Separate random-effects negative binomial regression models for each behavior predicted adjusted mean values of animal-source foods, away-from-home eating, snacking, and screen time; random-effects logistic regression model predicted adjusted probability of leisure-time sports participation. All models controlled for child's age (y) and sex, household income (tertiles), urbanicity (tertiles), geographic region (North/Central/South), year of study entry, and highest parental education (none or primary/middle school/high school/technical, college or higher). PA, physical activity.

Supplemental Table 4.1. Characteristics of children included and excluded in the analytic sample, CHNS 1991-2009[a]

	Included	Excluded	P-value
No. of subject	5201	86	
Age, y (mean ± SD)	10.2±2.5	10.3±2.4	0.62
Gender, % male	52.0	57.0	0.06
Annual household income, 1000 yuan (mean ±SD)†	15.2±18.4	20.2±22.2	<0.001
Urbanicity (mean ± SD)‡	47.9±17.7	56.4±19.7	<0.001
Geographic region, %			0.01
North	15.2	10.1	
Central	31.4	30.7	
South	53.4	59.3	
Highest parental education, %			<0.001
None/primary school	10.3	8.4	
Middle school	21.7	11.7	
High school	61.8	69.2	
College, technical or higher	6.3	10.8	
Animal-source food, % energy (mean ± SD)	10.1±9.9	13.1±10.6	<0.001
Away-from-home eating, % energy (mean ± SD)	20.5±17.0	28.3±22.1	<0.001
Snacking, % energy (mean ± SD)	1.0±3.6	1.3±4.2	0.22
Screen time, hours/week (mean ± SD)	8.5±7.3	11.4±8.1	<0.001
Any leisure-time sports participation, %	19.4	24.0	0.10
Year of study entry, %			<0.001
1991	43.4	2.8	
1993	9.5	1.3	
1997	22.5	7.0	
2000	12.1	9.5	
2004	8.9	41.0	
2006	3.7	38.4	

[a]Difference tested using one-way ANOVA (continuous variables) or chi-squared test (categorical variables).
†Total household income inflated to 2011.
‡Urbanicity defined by a multicomponent urbanicity scale ranging from 0-120. [132]
CHNS: China Health and Nutrition Survey

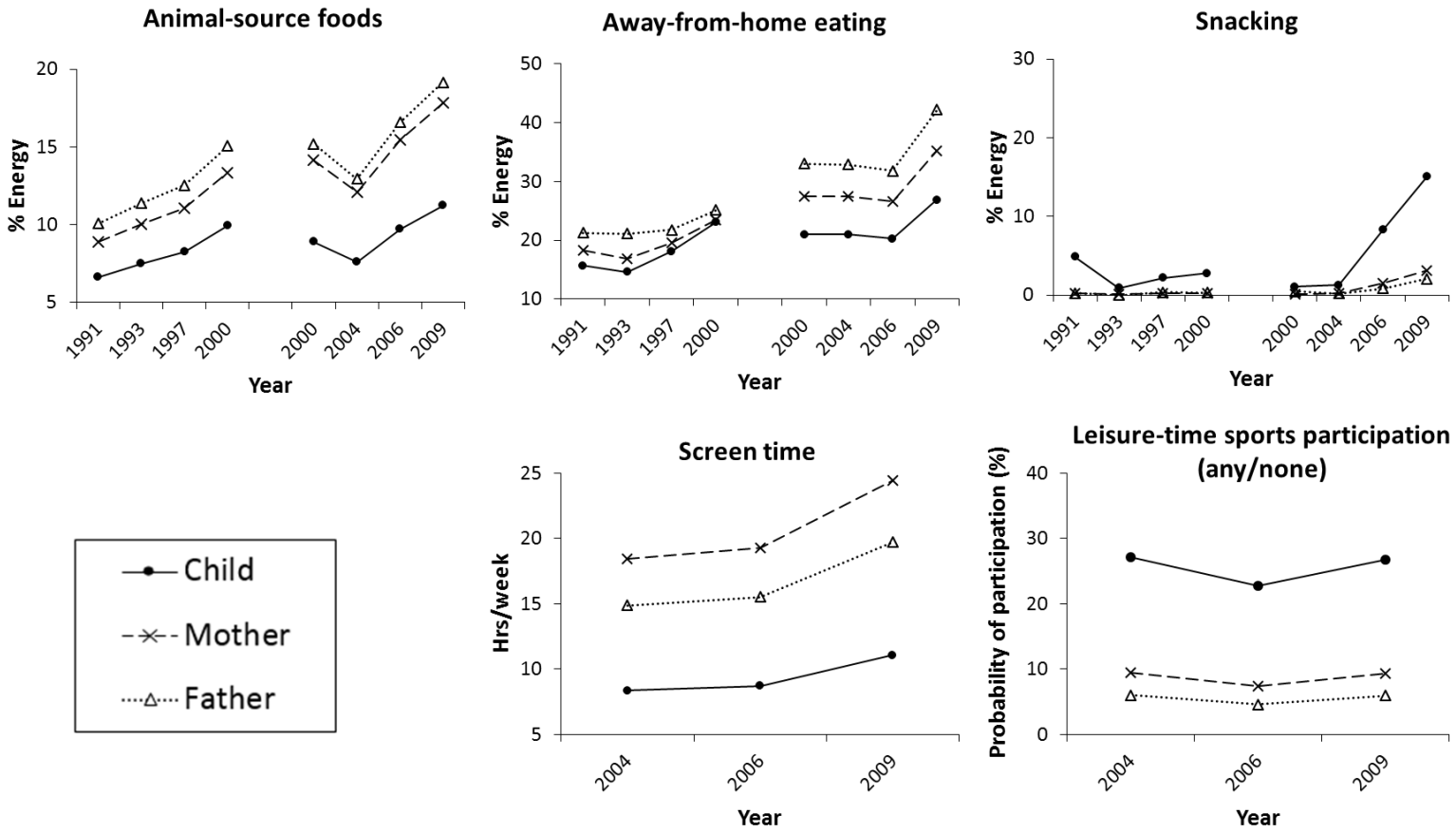
Supplemental Table 4.2. Beta coefficients/odds ratios (OR) of diet, screen time, and PA among children, mothers, and fathers[a]

	Animal-source foods, % energy		Away-from-home eating, % energy		Snacking, % energy		Screen time, hrs/wk		Any leisure-time sports participation, %	
	Beta	95% CI	Beta	95% CI	Beta	95% CI	Beta	95% CI	OR	95% CI
1991		ref.		ref.		ref.				
1993	0.06	-0.01,0.13	-0.05	-0.11,0.00	-0.95	-1.38,-0.52				
1997	0.25	0.18,0.33	0.25	0.18,0.31	-0.67	-1.12,-0.22				
2000	0.50	0.42,0.58	0.38	0.31,0.45	-1.36	-1.85,-0.88				
2004	0.57	0.48,0.66	0.47	0.39,0.54	-1.58	-2.15,-1.01		ref.		ref.
2006	0.76	0.66,0.87	0.41	0.32,0.50	0.37	-0.21,0.95	0.18	0.10,0.27	0.43	0.21,0.64
2009	1.06	0.94,1.17	0.78	0.68,0.88	1.27	0.62,1.92	0.28	0.18,0.38	0.81	0.56,1.06
Child		ref.		ref.		ref.		ref.		ref.
Mother	0.25	0.13,0.37	0.05	-0.04,0.14	-1.6	-2.25,-0.96	0.54	0.36,0.72	-2.84	-3.83,-1.86
Father	0.33	0.20,0.45	0.27	0.17,0.36	-1.53	-2.21,-0.85	0.45	0.26,0.64	-3.41	-4.45,-2.37
Mother X 1991	-	-		ref.		ref.	-	-	-	-
Mother X 1993	-	-	0	-0.07,0.07	0.81	0.30,1.31	-	-	-	-
Mother X 1997	-	-	-0.03	-0.10,0.04	0.76	0.24,1.28	-	-	-	-
Mother X 2000	-	-	-0.02	-0.09,0.06	0.73	0.20,1.27	-	-	-	-
Mother X 2004	-	-	-0.05	-0.13,0.03	0.66	0.12,1.21		ref.	-	-
Mother X 2006	-	-	-0.01	-0.09,0.08	0.40	-0.15,0.95	-0.14	-0.24,-0.04	-	-
Mother X 2009	-	-	-0.09	-0.19,0.01	0.30	-0.32,0.93	0	-0.11,0.11	-	-
Father X 1991	-	-		ref.		ref.	-	-	-	-
Father X 1993	-	-	0.02	-0.05,0.09	0.40	-0.13,0.93	-	-	-	-
Father X 1997	-	-	-0.09	-0.16,-0.02	0.70	0.16,1.24	-	-	-	-
Father X 2000	-	-	-0.12	-0.20,-0.05	0.92	0.37,1.47	-	-	-	-
Father X 2004	-	-	-0.11	-0.19,-0.03	-0.14	-0.72,0.45		ref.	-	-
Father X 2006	-	-	-0.12	-0.21,-0.04	-0.28	-0.86,0.31	-0.17	-0.28,-0.07	-	-
Father X 2009	-	-	-0.19	-0.30,-0.09	-0.36	-1.02,0.30	-0.04	-0.16,0.07	-	-
Med. Urban X 1991		ref.		ref.		ref.	-	-	-	-
Med. Urban X 1993	0.14	0.04,0.23	0.18	0.11,0.24	-0.26	-0.78,0.27	-	-	-	-

Med. Urban X 1997	0.13	0.04,0.23	0	-0.07,0.08	0.11	-0.42,0.64	-	-	-	-
Med. Urban X 2000	0.03	-0.07,0.14	0.12	0.04,0.19	0.27	-0.28,0.82	-	-	-	-
Med. Urban X 2004	-0.06	-0.17,0.05	0.17	0.09,0.25	1.96	1.37,2.55		ref.	-	-
Med. Urban X 2006	0.03	-0.09,0.15	0.27	0.18,0.36	1.23	0.65,1.80	-0.1	-0.20,0.00	-	-
Med. Urban X 2009	-0.28	-0.42,-0.14	0.14	0.04,0.25	1.22	0.58,1.86	-0.19	-0.30,-0.08	-	-
High Urban X 1991		ref.		ref.		ref.	-	-	-	-
High Urban X 1993	0.11	0.01,0.20	0.06	-0.01,0.13	0.29	-0.23,0.81	-	-	-	-
High Urban X 1997	-0.06	-0.16,0.05	-0.07	-0.14,0.01	0.29	-0.25,0.83	-	-	-	-
High Urban X 2000	-0.26	-0.36,-0.15	-0.15	-0.23,-0.07	0.53	-0.02,1.09	-	-	-	-
High Urban X 2004	-0.38	-0.50,-0.26	-0.14	-0.23,-0.06	2.15	1.55,2.75		ref.	-	-
High Urban X 2006	-0.54	-0.66,-0.42	-0.01	-0.10,0.08	1.02	0.44,1.59	-0.05	-0.15,0.06	-	-
High Urban X 2009	-0.78	-0.93,-0.64	-0.15	-0.25,-0.04	1.02	0.37,1.67	-0.11	-0.22,0.01	-	-

[a]Separate random-effects negative binomial regression models for each behavior of animal-source foods, away-from-home eating, snacking, and screen time; random-effects logistic regression models for leisure-time sports participation. All models controlled for age (y), household income (tertiles), urbanicity (tertiles), geographic region **(North/Central/South), and year of study entry.** PA, physical activity.

Supplemental Figure 4.1. Complete-case analysis: predicted mean (or probability of) diet, screen time, and PA over time



[a]We ran complete-case dietary analyses among two separate groups of individuals who completed all four surveys from 1991-2000 (n=1392), and from 2000-2009 (n=768), respectively, to compare the change of dietary behaviors over time with results from mixed groups of individuals in our main analysis who completed 2-4 surveys (Figure 1). For screen time and PA, we ran complete-case analyses among one group of individuals who completed all three surveys from 2004-2009 (n=1725), to compare the change of screen time and PA over time with results from mixed groups of individuals in our main analysis who completed 2-3 surveys. Separate random-effects negative binomial regression models for each behavior predicted adjusted mean values of animal-source foods, away-from-home eating, snacking, and screen time; random-effects logistic regression model predicted adjusted probability of leisure-time sports participation. All models controlled for baseline age (y), household income (tertiles), urbanicity (tertiles), geographic region (North/Central/South), and year of study entry. The rate of changes in away-from-home eating and snacking was different across household members (p for interaction<0.05**)**. PA, physical activity.

Supplemental Table 4.3. Predicted parent-offspring associations for diet, screen time, and PA by household income and geographic region[a]

	Animal-source food (% energy)[b]		Away-from-home food (% energy)[b]		Snacking (% energy)[b]		Screen time (hours/week)[c]	
	Mother Beta (95% CI)	Father Beta (95% CI)	Mother Beta (95% CI)	Father Beta (95% CI)	Mother Beta (95% CI)	Father Beta (95% CI)	Mother Beta (95% CI)	Father Beta (95% CI)
Household income[d]								
Low	1.12 (1.04,1.20)	1.13 (1.04,1.22)	0.34 (0.31,0.36)	0.28 (0.26,0.31)	n/a	n/a	n/a	n/a
Medium	0.97 (0.89,1.05)	0.98 (0.89,1.07)	0.32 (0.29,0.34)	0.26 (0.24,0.28)	n/a	n/a	n/a	n/a
High	0.86 (0.78,0.93)	0.84 (0.75,0.93)	0.30 (0.28,0.33)	0.24 (0.21,0.26)	n/a	n/a	n/a	n/a
Geographic region[e]								
North	0.91 (0.83,0.99)	0.89 (0.80,0.98)	0.31 (0.28,0.33)	0.24 (0.21,0.26)	3.85 (1.97,5.72)	3.13 (1.57,4.70)	0.00 (-0.01,0.01)	0.00 (-0.01,0.01)
Central	0.97 (0.89,1.05)	0.98 (0.89,1.07)	0.32 (0.29,0.34)	0.26 (0.24,0.28)	5.60 (3.50,7.70)	4.21 (2.68,5.74)	0.01 (0.00,0.02)	0.01 (0.01,0.02)
South	0.76 (0.69,0.84)	0.81 (0.72,0.89)	0.27 (0.25,0.30)	0.21 (0.19,0.23)	5.65 (3.63,7.66)	5.20 (3.75,6.65)	0.01 (0.00,0.02)	0.02 (0.01,0.02)

[a]Table shows predicted associations in year 2000 due to statistically significant modification by year at $p<0.05$. Year 2000 was chosen as the mid-point of year 1991 to 2009. Predicted beta coefficients and 95% confidence intervals (CI) were estimated using separate random-effects negative binomial regression models for each behavior. n/a indicates no modification by household income (p for interaction>0.05); coefficients are the same across all household income levels in 2000 and are shown in Table 2 in the main text. All models controlled for child's baseline age (y) and sex, household income (tertiles), urbanicity (tertiles), geographic region (North/Central/South), year of study entry, and highest parental education (none or primary/middle school/high school/technical, college or higher). PA, physical activity.

[b]Beta coefficients for animal-source foods, away-from-home eating, and snacking indicate the change of child's daily intake in percentage of total energy when mother's or father's intake increased by 10% total energy.

[c]Beta coefficients for screen time indicate the change of child's screen time in hours when mother's or father's screen time increased by one hour per week.

[d]Coefficients for household income were predicted for the Central region due to interactions between income with the behaviors, and between region with the behaviors.

[e]Coefficients for region were predicted at the medium income level due to interactions between income with the behaviors, and between region with the behaviors.

CHAPTER 5. LONGITUDINAL ASSOCIATIONS OF DIET, SCREEN TIME, AND PHYSICAL ACTIVITY BEHAVIORS WITH CARDIOMETABOLIC RISK FACTORS AMONG CHINESE CHILDREN AND THEIR PARENTS

Overview

Understanding intergenerational differences in associations of urbanization-related lifestyle behaviors with cardiometabolic disease (CMD) risk factors in children and their parents is important in rapidly urbanizing China.

We tested the intergenerational differences in longitudinal associations of away-from-home eating, snacking, screen time, and leisure-time sports with high waist-to-height ratio (WHtR), elevated blood pressure (BP), elevated hemoglobin A1c (HbA1c), and elevated C-reactive protein (CRP) among Chinese children and their parents.

We studied 5,180 children (aged 7-17y) and their parents participated in China Health and Nutrition Survey (1991-2009) with measured WHtR, BP, HbA1c, and CRP. We collected three-day 24-hour dietary recall to derive away-from-home eating (non-consumer, <1meal/day, and ≥1meals/day) and consumption of fruit/vegetable snacks (any/none) and other snacks (any/none). We used a seven-day physical activity recall to collect screen time (≤1hr/day, 1-2hrs/day, and >2hrs/day) and leisure-time sports (any/none). Random-effects logistic regression was used to examine the associations of lagged behaviors with CMD risk factors.

We detected intergenerational differences in associations between lagged behaviors and risk factors ($P_{interaction}<0.1$). Generation-specific models showed that lagged away-from-home eating ≥1 (versus no) meals/day was negatively associated with parents' high WHtR (OR=0.68,

95% CI=0.53, 0.88) but positively associated with children's high WHtR (OR=1.47, 95% CI=1.02, 2.13) at follow-up. Lagged fruit/vegetable snack consumption was negatively related to parents' high WHtR, parents' elevated BP, and children's high WHtR at follow-up.. Lagged screen time was positively associated with parents' high WHtR and children's high WHtR, elevated BP, and high CRP at follow-up. The associations between lagged behaviors and risk factors did not differ across years.

CMD risk factors were negatively associated with fruit/vegetable snack consumption and positively associated with screen time in both generations. Away-from-home eating was associated with higher WHtR in children but lower WHtR in parents. Generation-specific intervention strategies may be needed for behavioral changes to reduce CMD risk.

Introduction

China has the world's highest diabetes prevalence, [31] which along with obesity, hypertension, and inflammation, has continued to grow in the past two decades, imposing a heavy burden on its health care system, [1, 32] While cardiometabolic disease (CMD) and its associated risk factors have been increasing in both adults and children, previous research has documented a faster increase rate of overweight in children compared to adults in China. [2] Intergenerational studies in the household context provide a unique opportunity to investigate this disparity in risk between children and adults. Such studies allow comparison of risk across generations experiencing the same environmental changes while controlling for common household sociodemographics. Understanding this intergenerational difference in risk will provide insights into differential response to urbanization varies across generations, which is essential for designing effective household-based interventions to lower the risk across generations in urbanizing China.

Dietary and physical activity (PA) behaviors are important contributors to these CMD risk factors. [20, 21] Studies have shown that westernized diet, including increased consumption of high-fat animal-source foods, is associated with the increasing prevalence of obesity. [65, 66] China is not only experiencing a transition from traditional to westernized diets, but is also having a dramatic change in eating behaviors, such as increasing away-from-home food consumption and snacking. [45] Percent total energy from foods prepared away-from-home in China increased from 7% in 1991 to 16% in 2011 among 2-18y old children, and from 7% in 1991 to 18% in 2011 among 19-59y old adults. [45] Away-from-home eating has been positively associated with overweight [29, 68] and higher insulin levels [29] in US and Spanish populations. In addition, the percentage of snacking in Chinese adults and children increased from 15% in 1991 to 67% in 2011 among 2-18y old children, and from 9% in 1991 to 52% in 2011 among 19-59y old adults. [45] The dominant snack in China is fruit, [77, 78] which may be partly responsible for the inverse association between snacking and BMI among Chinese overweight children. [78] Contrarily, snacks in Western populations are mainly candy, sweetened beverages, and salty snacks, [79, 143, 144] which were found to be associated with weight gain in the US population. [72, 73] No previous study has examined how these dietary behaviors associate with various CMD risk factors in large Chinese samples, nor compared whether the associations differ between children and adults. Understanding these questions will guide intervention strategies to reduce CMD risk across generations in this urbanizing country with rapid behavioral change.

Another possible driver of the increasing CMD risk factors is large declines in PA, together with increases in sedentary behaviors over the past 20 years in China. [46-48] Several studies have found positive associations of sedentary behavior with CMD risk factors such as

obesity, type 2 diabetes, and hypertension. [81, 95, 145] However, some evidence suggests that children may compensate sedentary behaviors with more leisure-time sports. [83]

Although a number of studies have examined the associations between these health behaviors and CMD risk factors, few studies have examined differences in behavior-risk associations across generations. [29] Examination of intergenerational differences will provide a better understanding of the disparity in the increased rate of risk in children versus adults, which will inform more effective intervention efforts to reduce risk in each generation. The household setting in China provides a unique opportunity to examine whether urbanization-related changes affect generations differentially and whether this relationship associates with different health outcomes once shared socioeconomic and living environment is controlled. Studying this question in a longitudinal setting provides temporality between behaviors and risk factors, which will help us to better understand the differences in associations of behaviors in earlier years with risk factors in later years and whether this association varies in child versus adults.

We aimed to determine the longitudinal associations of away-from-home eating, snacking, screen time, and leisure-time sports with increasingly prevalent CMD risk factors including high waist-to-height ratio (WHtR), elevated blood pressure (BP), elevated hemoglobin A1c (HbA1c), and high C-reactive protein (CRP) among school-aged Chinese children and their parents from 1991 to 2009. We also sought to test whether the associations between these behaviors and CMD risk factors differed in children relative to their parents.

Methods

CHNS. The CHNS is a household-based longitudinal cohort study with ongoing data collection in nine provinces across China (North: Heilongjiang, Liaoning; Central: Shandong, Henan, Jiangsu; South: Hunan, Hubei, Guangxi, Guizhou) in nine survey rounds from 1989-

2011. Using a multistage, random cluster design, we used a stratified probability sample to select counties and cities stratified by income levels using State Statistical Office definitions. [111] We then selected communities and households from these strata. The CHNS cohort initially mirrored national age-sex-education profiles, [112, 128, 129] and the provinces in the CHNS sample constituted 44% of China's population in 2009 (according to 2009 census). More details on survey procedures are described elsewhere. [146] 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 longitudinal data from 1991, 1993, 1997, 2000, 2004, 2006, and 2009 when diet, PA, BP, and anthropometric data on parents and children were collected. Fasting blood samples were first collected in 2009. Eligible households were those with children aged 7-17y at any wave; children and at least one parent completed at least two surveys (n=3,526 households). We excluded households if children or both parents were missing all risk factor measures (n=30 households), or if children or both parents were diagnosed with hypertension or diabetes (n=25 households). We further excluded households with missing covariates (n=23 households). For CRP, we excluded subjects who had CRP>10mg/L (n=50 subjects) because it indicates current infection. [114] Our final analytic sample included 5,180 children, 3,388 mothers, and 3,235 fathers. The included households were slightly more likely to have higher household income and to live in more urbanized areas. Compared to the excluded participants, children included in the analytic sample were younger, whereas their parents were older. There was no statistically significant difference in the prevalence of the CMD risk factors between included and excluded individuals.

Diet. We used three consecutive 24-hour dietary recalls at the individual level and a food inventory at the household level occurring during the same 3-day period to collect diet data, randomly starting from Monday to Sunday. For 24-hour recalls, trained interviewers recorded types and amounts of food, types of meal, and places of food preparation of all food items consumed by each household member. For children under 10y, mothers or a mother substitute recalled the children's diet intake. All foods available in the household were measured daily for the food inventory. This method has been validated relative to doubly labeled water (r^2 men: 0.56; women: 0.60) for energy [147] and urine for sodium (r^2: 0.58), potassium (r^2: 0.59), and MSG (r^2: 0.82). [148] In this study, we focused on away-from-home eating and snacking. We defined away-from-home eating as consuming foods prepared away-from-home (no matter if they were consumed at or away from home), which was categorized into non-consumer, <1 meal/day, and ≥1 meals/day. Snacks were reported as foods consumed outside the three main meals (breakfast, lunch, and dinner), further separated into fruit/vegetable snacks and other snacks, and categorized into any versus no consumption for both types.

Physical activity (PA). Our PA data were derived from seven-day PA recalls across a variety of domains. Children and parents were asked about their participation and time spent in different types of sedentary behaviors and PA. We calculated PA using hours spent in each activity multiplied by metabolic equivalents (METs) for that activity and defined total PA as the sum of MET-hours for all activities. [149, 150] Parents or primary caregivers completed the surveys for children under 10y. In this study, we examined screen time and leisure-time sports participation. Screen time referred to time spent on TV/videotape watching, video games, and computer usage. We categorized individuals into ≤1 hr/day, 1-2 hrs/day, and >2 hrs/day. Leisure-time sports included gymnastics, dancing, track and field sports, swimming, ball sports (e.g.

basketball, tennis), and other sports (e.g., martial arts, tai chi). We focused on children's leisure-time sports outside school (including before or after school for those in school and any activity for those not in school) because we were interested in children's discretionary PA. Due to the low participation rate in leisure-time sports, we dichotomized the variable into any versus none. Since data on screen hours in adults were first collected in 2004 and the leisure-time sports survey changed in 2004, we restricted our PA analyses to 2004, 2006, and 2009 only.

Anthropometric measures. At each survey from 1991 to 2009, trained staff measured height (without shoes) and waist circumference (midway between the lower rib margin and the iliac crest, first collected in 1993). WHtR is calculated as waist circumference (cm) divided by height (cm). We classified high WHtR using WHtR≥0.5. [151]

BP. Trained physicians measured BP in triplicate at each visit from 1991 to 2009 and these values were averaged. Children's systolic BP (SBP) and diastolic BP (DBP) z-scores were calculated based on the age-, sex-, and height-specific BP percentile algorithm for children using the US CDC 2000 growth curve reference. [116] We defined elevated BP in children as an SBP z-score or DBP z-score≥90th percentile, or SBP≥120mmHg or DBP≥80mmHg, as recommended by National Institutes of Health for prehypertension and hypertension in children. [152] We combined prehypertension and hypertension as elevated BP because hypertension (SBP z-score or DBP z-score≥95th percentile of the algorithm) is uncommon in children and adolescents (7% in our study sample). We defined parental elevated BP as prehypertension and hypertension using the same cut-point (SBP≥120mmHg or DBP≥80mmHg) for consistency. [117]

Biomarkers. Blood samples were collected by venipuncture following overnight fasting in 2009. Laboratory analysis methods are described in detail elsewhere. [32] We defined elevated HbA1c using HbA1c≥5.7% (recommended by American Diabetes Association for

prediabetes and diabetes). [115] We combined prediabetes and diabetes as elevated HbA1c because diabetes (HbA1c≥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.

We measured high-sensitivity CRP via the immunoturbidimetric method. We defined elevated CRP as CRP 1-10mg/L in children and 3-10mg/L in adults. [114, 118]

Covariates. At each survey, we collected participants' age (y), sex, household income (inflated to 2011, tertiles), geographic region (North/Central/South), year of study entry, highest parental education (none or primary/middle school/high school/technical, college or higher), smoking (yes/no), total energy intake (kcal/d), total fat intake (% total energy), total fruit and vegetable intake (% total energy), sodium intake (mg/d, BP models only), and total PA (MET-hrs/wk). We also used the CHNS multicomponent urbanicity scale comprised of 12 urban environment domains representing infrastructure, economic, and social service. The scale has high reliability and validity, [132] ranging from 0-120 with a higher score indicating higher urbanicity, which was categorized into year-specific tertiles.

Statistical Analysis. We conducted all analyses using Stata 14.0 (Stata Corp, College Station, TX, USA), and used a p-value <0.05 as our significance level. In descriptive analyses, we examined baseline characteristics of children and their parents by diet, screen time, and PA behavior categories and tested differences of these characteristics across behavior categories using the chi-squared test (categorical variables) and one-way ANOVA (continuous variables).

To establish temporality between behaviors and risk factors (i.e., latency period), we lagged behaviors by the period of time between surveys of analysis (*t-1*). We conducted random-effects logistic regression models to examine the association of lagged diet, screen time, and PA with high WHtR and elevated BP at follow-up, with random intercepts for repeated measures

within individuals and clustering within households. Since biomarkers were only collected in 2009, we ran logistic regression models to examine the association of diet, screen time, and PA in 2006 with elevated HbA1c and elevated CRP in 2009. We ran separate models for diet, screen time, and PA. All dietary variables were included in the diet models; both screen time and leisure-time sports were included in the PA models. To test whether the associations differed between child and parent generations, we first included both generations in the same models with interaction terms between behaviors and a generation variable (children/parents). Since the interaction terms were statistically significant ($p<0.1$) for away-from-home eating, consumption of non-fruit/vegetable snacks, and leisure-time sports, we stratified all models by generation for consistency and conducted generation-specific analyses. We ran two sets of models. The first set adjusted for covariates mentioned above and we additionally adjusted for snacking in PA models, because evidence suggests an association between TV watching and snacking. [89] The second set of models additionally adjusted for WHtR in models with elevated BP, HbA1c, and CRP outcomes to test whether the behavior-risk factor associations were independent of central obesity, because previous research suggests that some behaviors, such as sedentary behavior, may have a direct influence on metabolic health in addition to the indirect effect of body weight. We present two sets of model results (fully-adjusted models with and without WHtR adjustment). In addition, we tested whether the behavior-risk associations varied by survey year, age of children or parents, sex, urbanicity, or year of study entry using the Wald test.

Results

At baseline, individuals who consumed away-from-home foods or any type of snacks had higher household income and/or lived in more urban areas, compared to individuals who did not consume any away-from-home foods or snacks (Table 5.1). A greater proportion of individuals

who participated in leisure-time sports lived in more urban areas. Adults who consumed any (versus none) away-from-home foods generally had lower total energy consumption and lower total PA. Those who spent >2 (versus ≤ 1**)** hrs/day screen time and who participated in any (versus none) leisure-time sports also had lower total PA. Children who spent >2 hrs/day screen time and who participated in leisure-time sports had higher total PA. Demographic and behavioral characteristics of parents and children in 2000 and 2009 are shown in Supplemental Table 5.1. The prevalence of CMD risk factors in parents and children from 1993 to 2009 is shown in Supplemental Table 5.2.

Associations between lagged dietary behaviors and CMD risk factors at follow-up

The associations between eating-away-from home and high WHtR, as well as between consumption of non-fruit/vegetable snacks and elevated HbA1c differed between children and their parents (p for interaction<0.1), thus we ran generation-specific models for all dietary analyses for consistency. We detected statistically significant effect measure modification (p<0.1) by parental age group (20-39y; ≥40y) for the association between lagged away-from-home eating and high WHtR at follow-up, so the results are presented separately by age groups for this association (Table 5.2). The associations between lagged dietary behaviors and CMD risk factors at follow-up did not differ by survey year or other covariates. Younger parents (20-39y) who had one or more (versus no) away-from-home meals per day in the previous survey were less likely to have high WHtR at follow-up (OR=0.68, 95% CI=0.53, 0.88), whereas children who had one or more (versus no) away-from-home meals per day in the previous survey were more likely to have high WHtR at follow-up (OR=1.47, 95% CI=1.02, 2.13). For both parents and children, fruit and vegetable snackers were less likely to have high WHtR at follow-up compared to non-snackers (OR=0.76, 95% CI=0.59, 0.97 for parents; OR=0.57, 95%

CI=0.33, 0.99 for children). Parents who consumed fruit and vegetable snacks (versus non-snackers) in the previous survey also had lower odds of having elevated BP at follow-up (OR=0.75, 95% CI=0.61, 0.91) in fully-adjusted model with WHtR adjustment.

Associations of lagged screen time and PA with CMD risk factors at follow-up

The associations of leisure-time sports with elevated HbA1c and high CRP differed between children and their parents (p for interaction<0.1), thus we ran generation-specific models for all screen time and PA analyses for consistency. We found statistically significant effect measure modification by parental age group (20-39y; ≥40y) for the association between lagged screen time and high WHtR at follow-up, so the results are presented separately by parental age group for this association (Table 5.3). The associations between lagged screen time and PA with CMD risk factors at follow-up did not differ by other covariates. We found that for older parents (≥40y) and children, those who were in the highest lagged screen time category (>2 hrs/day) versus the lowest (<1 hr/day) were more likely to have high WHtR at follow-up (OR=2.55, 95% CI=1.56, 4.19 for older parents; OR=1.64, 95% CI=1.03, 2.63 for children). Children who spent >2 (versus <1) hrs/day in the previous survey were also more likely to have elevated BP at follow-up (OR=1.51, 95% CI=1.04, 2.21) in fully-adjusted model with WHtR adjustment. In addition, lagged screen time of 1-2 hrs/day was associated with higher odds of high CRP at follow-up (OR=3.44, 95% CI=1.32, 8.99) in children but no significant association was found for screen time >2 hrs/day. On the contrary, children who participated (versus did not) in leisure-time sports in the previous survey had lower odds of high CRP at follow-up (OR=0.24, 95% CI=0.11, 0.53). Lagged leisure-time sports participation was not associated with any of the risk factors at follow-up among parents.

Comparing fully-adjusted models that did and did not adjust for WHtR, associations between lagged behaviors and risk factors at follow-up were attenuated after adjusting for WHtR for most models. In particular, parents who consumed non-fruit and vegetable snacks in the previous survey were more likely to have elevated HbA1c at follow-up (OR=2.63, 95% CI=1.00, 6.88) before adjusting for WHtR. However, after WHtR adjustment, the association was no longer statistically significant (OR=2.41, 95% CI=0.95, 6.14), suggesting that the association between non-fruit and vegetable snack consumption and elevated HbA1c in parents was not independent of central obesity.

Discussion

We found that lagged away-from-home eating, fruit and vegetable snacking, screen time, and leisure-time sports were associated with various CMD risk factors at follow-up in Chinese children and their parents. The magnitude and direction of association varied by behaviors and by risk factors, and differed between children and their parents. In particular, the association between lagged away-from-home eating and WHtR at follow-up was negative in parents but positive in children. Lagged consumption of fruit and vegetable snacks was negatively related to WHtR at follow-up in both children and their parents, and was negatively associated with BP at follow-up in parents. Lagged screen time was positively associated with WHtR at follow-up in both children and their parents. In addition, in children we observed positive associations between lagged screen time with BP and CRP at follow-up, and a negative association between lagged leisure-time sports with CRP at follow-up.

Lagged dietary behaviors and CMD risk factors at follow-up

Our findings suggest a positive association between frequency of eating away-from-home and obesity among Chinese children, which is consistent with findings from a previous study

conducted among US adolescents. [29] Foods prepared away-from-home are generally high in energy density, saturated fat, trans fat, added sugar, and sodium, and low in fiber, [67] which have been associated with weight gain and adverse cardiometabolic profile in both children [29] and adults [68, 69] in the US and Spain. In our study, lagged away-from-home eating was inversely associated with WHtR in parents but was positively related to WHtR in children at follow-up. This inconsistency is possibly due to the difference in types of away-from-home foods consumed by parents versus their children in our study or overall differences in diet choices between two generations. One study on the consumption of processed foods in China suggested higher consumption of processed foods in children versus adults, and a positive association between processed food consumption and overweight in children but not adults, suggesting less healthy food choices in children. [133] Differences in food choices between children and adults may reflect generational disparities in response to urbanization.

Current evidence for the direction of association between snacking and health status is mixed. [71-73, 76, 153] Higher snacking and frequent eating have been related to lower waist circumference and less % body fat in US children and adolescents, [154, 155] suggesting that frequent eating without increasing total energy intake may stimulate certain hormonal signals that increase satiety and suppress appetite. [156, 157] In contrast, Fisher et al. [72] and Howarth et al. [73] found positive relationships between snacking and obesity among US children and adults, respectively. Few studies examined the association between snacking and other CMD risk factors. Mekary et al. [75] found that snacking was longitudinally positively related to type 2 diabetes in US men, but the association was attenuated after BMI adjustment. However, these studies did not differentiate types of snacks, which could be a reason for the mixed evidence. Phillips et al. only found a significant association between BMI z-score with soda consumption

among US adolescents, but not with total snacks. [158] In our study population, fruits were the most common snack. We observed that after adjusting for total energy intake, fruit and vegetable consumed as snacks were inversely associated with WHtR and BP in children, as well as WHtR in parents at follow-up. Fruit and vegetable are high in dietary fiber, which can provide health benefits for both children and adults. [159] On the contrary, high amount of refined carbohydrate and sugar in snacks other than fruit and vegetable may associate with type 2 diabetes. [75] Concordant with a previous study examining the snacking-diabetes association among US men, [75] our observed association between total energy-adjusted non-fruit/vegetable snack consumption and elevated HbA1c in parents was attenuated and no longer statistically significant after adjusting for WHtR, suggesting a potential role of obesity in the snacking-diabetes association in adults. The null association between non-fruit/vegetable snacks and HbA1c in children might be due to different types of snacks consumed by children and adults in our population or the difference in nutrient metabolism between children and adults, which are worth further investigation.

Lagged screen time, PA, and CMD risk factors at follow-up

Our results are consistent with previous studies of screen time or sedentary behaviors in relation to obesity in adults [80-82] and children [83, 84] in various countries such as the US, Canada, France, Australia, and China. Additionally, we found similar findings compared to earlier research suggesting positive associations between screen time with hypertension [85] and high CRP [86] among US children. A number of theories have been proposed to explain these associations. First, screen-based activities, especially TV watching, involve low energy expenditure, even compared to other sedentary behaviors such as reading and doing homework, and thus reduce total energy expenditure. [87, 88] Second, TV watching is often accompanied by

increased food and snack intakes, [89] which may result in higher caloric consumption. Third, sedentary behaviors may have direct influence on metabolic health through reducing insulin sensitivity and increasing triglycerides levels, [90, 91] in addition to the indirect effect of body weight or other health behaviors. The positive associations between screen time with high WHtR, elevated BP, and high CRP in children observed in our study emphasize the importance of intervention efforts on reducing the increasing sedentary behaviors in children. On the other hand, we did not observe any associations between screen time and CMD risk factors besides high WHtR among adults. The lack of association is supported by a recent review of prospective studies by Proper et al. , [145] which concluded insufficient evidence for a longitudinal relationship between sedentary behaviors and CVD risk factors among adults.

We found that lagged leisure-time sports participation was associated with lower odds of high CRP at follow-up in children, independent of WHtR. The inverse association is supported by a previous review, [94] which found a "long-term anti-inflammatory" response of exercise. However, besides inflammation, lagged participation in leisure-time sports was not associated with any other CMD risk factor at follow-up in our study in either parents or their children, inconsistent with previous epidemiological evidence for the health benefits of moderate-intensity PA. [28] This is possibly because screen time and leisure-time sports were positively associated in our study. After mutually adjusting for these two behaviors in our models, high WHtR at follow-up was positively associated with lagged screen time in parents and their children independent of leisure-time sports, whereas no association was found for leisure-time sports. This finding is supported by earlier studies which reported stronger association between body weight with sedentary time versus leisure-time PA participation among US and Australian adults [96, 97]. In particular, Bauman et al. [98] suggested that in China, leisure-time sports alone may

be not sufficient to prevent obesity, because the amount of PA required for obesity prevention is difficult to achieve through leisure-time sports only, especially with increasing sedentary time. [46, 95] Focus should be placed on promoting an active lifestyle in general rather than emphasizing on leisure-time PA only. [98]

Our study has several limitations. First, HbA1c and CRP were only measured at one time point in 2009, thus sample sizes for the models for these risk factors were limited, especially in children, compared to the larger samples for repeated measurements of WHtR and BP. 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, we were unable to test if child's pubertal status has played a role in these associations because we did not have data on pubertal markers in children. Nonetheless, we tested for differences in associations among younger versus older children and found minimal differences in the behavior-risk association. Similarly, menopausal status could have played a role in the behavior-risk association in mothers, but the vast majority of mothers in our study (95%) were below the average age of menopause (49y) in China. [123] Last, self-reported behavior data may be subject to recall bias, although the CHNS data are based on highly detailed recall methods, including analysis of household-specific recipes and weighing of condiments. Previous research on selected components of the diet (energy, protein, sodium, monosodium glutamate) suggest strong validity, [160] and the PA components have been found to be highly predictive of incident obesity and weight gain in adults. [49, 140-142]

In spite of its limitations, our study has several notable strengths including the use of a regionally diverse sample from a national survey with detailed diet and PA data collected at the household level over 20 years. Data collected from each member of the household allowed us to

examine and compare the behavior-risk associations among children and their parents. Further, we measured each individual's HbA1c and BP, 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 to child BP references that are only age- and sex-specific. [124] Due to the high correlation between children's height and BP, our approach more appropriately classified children's BP status. [124] Last, repeated measures of health behaviors, WHtR, and BP allowed us to longitudinally examine behaviors in relation to risk factors. Using lagged behaviors we were able to establish a latency period between health behaviors and risk factors, although lagged and current behaviors are likely to be correlated within individuals. Several previous studies have reported longitudinal but not cross-sectional associations of sedentary time with body fat among non-Hispanic white girls [99] and with insulin resistance in Caucasian adults. [100, 101] The difference in results from longitudinal versus cross-sectional studies highlights the need for including a latency period between health behaviors and risk factors.

In conclusion, we found that CMD risk factors in children and their parents were negatively associated with the consumption of fruit/vegetable snacks, and positively associated with screen time. Away-from-home eating was associated with higher WHtR in children but lower WHtR in parents. These differences may be relate to different food choices between child and parent generations in this population, likely reflecting generational differences in response to urbanization. Generation-specific intervention strategies may be needed for behavioral changes to reduce CMD risk in this population.

Tables and figures

Table 5.1. Characteristics of analytic sample at baseline according to dietary, screen time, and PA behaviors (CHNS 1991 for diet, CHNS 2004 for screen time and PA)

	Away-from-home eating				Snacking – fruit & vegetable			Snacking – other snacks		
	Non-consumer	<1 meal/d	≥1 meal/d	*P*	Non-snacker	Snacker	*P*	Non-snacker	Snacker	*P*
Parents										
n	1713	196	518		2560	161		2607	114	
Age, y*	39.8 (6.5)	38.9 (5.9)	39.1 (5.9)	0.01	39.5 (6.3)	38.9 (5.9)	0.10	39.5 (6.3)	39.9 (5.5)	0.78
Gender, % male	42.6	50.6	57.3	<0.001	47.2	41.6	0.17	46.8	48.3	0.76
Total energy intake, kcal/d*	2763 (673)	2690 (629)	2649 (659)	0.001	2733 (666)	2633 (618)	0.06	2723 (665)	2811 (618)	0.17
% energy from fat, %*	18.4 (9.7)	23.3 (9.2)	26.7 (9.9)	<0.001	20.6 (10.2)	25.0 (10.6)	<0.001	20.8 (10.2)	23.6 (10.4)	0.004
% energy from FV, %*	2.4 (1.5)	2.3 (1.4)	2.3 (1.6)	0.31	2.3 (1.4)	2.9 (1.9	<0.001	2.3 (1.5)	3.3 (2.1)	<0.001
Total PA, MET-hrs/d*	316 (184)	272 (170)	226 (158)	<0.001	293 (180)	271 (181)	0.13	291 (180)	296 (180)	0.76
Household income, 1000 yuan/yr†‡*	34.8 (43.5)	37.3 (28.6)	48.3 (47.2)	0.002	37.6 (45.0)	46.9 (37.9)	0.01	39.1 (45.6)	45.1 (34.9)	0.11
Urbanicity†§*	54.8 (15.0)	61.1 (19.3)	71.1 (18.3)	<0.001	59.8 (18.1)	69.0 (18.7)	<0.001	60.8 (18.0)	68.1 (19.8)	<0.001
Geographic region, %†										
North	9.0	35.8	17.1		8.7	33.3		17.5	16.7	
Central	17.6	18.4	40.3	<0.001	25.9	30.4	<0.001	26.0	31.0	0.45
South	73.4	45.9	42.6		65.4	36.2		56.5	52.3	
Children										
n	1260	301	301		1690	167		1713	144	
Age, y*	11.6 (2.4)	11.2 (2.5)	11.6 (2.5)	0.053	11.6 (2.5)	11.0 (2.4)	0.002	11.6 (2.5)	10.8 (2.4)	<0.001
Gender, % male	50.6	53.5	51.2	0.66	50.9	52.1	0.77	50.8	53.5	0.54
Total energy intake, kcal/d*	2191 (634)	2138 (586)	2165 (553)	0.38	2187 (623)	2090 (512)	0.05	2175 (609)	2220 (671)	0.40
% energy from fat, %*	18.4 (9.9)	24.3 (9.4)	25.6 (9.8)	<0.001	20.1 (10.2)	24.7 (9.8)	<0.001	20.2 (10.1)	24.7 (11.1)	<0.001
% energy from	2.5	2.7	2.5	0.49	2.3	4.9	<0.001	2.5	3.5	<0.001

FV, %*	(1.7)	(2.1)	(1.9)		(1.5)	(2.7)	(1.7)	(2.6)
Total PA, MET-hrs/d*	-	-	-		-	-	-	-

	Screen time				Leisure-time sports participation		
	≤1 hr/d	1-2 hrs/d	>2 hrs/d	*P*	None	Any	*P*
Parents							
n	657	890	919		2426	132	
Age, y*	41.7 (6.8)	40.4 (6.1)	39.9 (5.9)	<0.001	40.6 (6.3)	40.3 (5.7)	0.71
Gender, % male	44.0	46.2	49.6	0.08	46.6	59.9	0.003
Total energy intake, kcal/d*	2216 (627)	2254 (627)	2290 (645)	0.07	2250 (635)	2403 (634)	0.01
% energy from fat, %*	24.9 (10.8)	26.9 (10.3)	28.9 (11.0)	<0.001	26.8 (10.8)	30.9 (11.2)	<0.001
% energy from FV, %*	2.8 (1.9)	2.9 (2.1)	2.9 (2.2)	0.65	3.0 (2.5)	3.2 (2.5)	0.28
Total PA, MET-hrs/d*	322 (238)	284 (225)	236 (212)	<0.001	278 (229)	190 (151)	<0.001
Household income, 1000 yuan/yr†‡*	23.0 (21.2)	22.6 (19.1)	22.1 (22.5)	0.80	22.4 (21.6)	23.6 (20.8)	0.35
Urbanicity†§*	57.9 (19.7)	58.5 (19.3)	60.4 (18.6)	0.15	57.8 (19.3)	64.0 (19.2)	<0.001
Geographic region, %†							
North	26.7	19.7	26.3		23.3	22.2	
Central	29.7	30.2	20.2	0.001	26.2	26.9	0.90
South	43.6	50.1	53.5		50.5	50.9	
Children							
n	472	407	391		850	379	
Age, y*	13.0 (2.9)	12.2 (2.9)	12.9 (3.2)	<0.001	12.1 (3.0)	13.2 (2.7)	<0.001
Gender, % male	46.0	51.1	60.1	<0.001	45.4	66.5	<0.001
Total energy intake, kcal/d*	1883 (617)	1861 (587)	1932 (678)	0.27	1784 (561)	2081 (683)	<0.001
% energy from fat, %*	27.4 (11.1)	27.7 (10.6)	27.8 (11.0)	0.86	27.4 (11.1)	28.5 (10.1)	0.10
% energy from FV, %*	2.9 (2.4)	3.0 (2.1)	3.0 (2.5)	0.76	3.1 (2.9)	2.9 (2.2)	0.10
Total PA, MET-hrs/d*	48.2 (60.3)	47.3 (51.3)	70.1 (95.0)	<0.001	29.4 (40.0)	93.1 (71.1)	<0.001

*Data are mean (S.D.).
†These are household-level variables that are identical for parents and children so are not repeated below for children.
‡Total household income inflated to 2011.
§Urbanicity defined by a multicomponent urbanicity scale ranging from 0-120 [132].
-: Child physical activity was not measured in 1991.
Abbreviations: CHNS, China Health and Nutrition Survey; PA, physical activity; FV, fruits and vegetables

Table 5.2. Odds ratios (95% confidence intervals) of cardiometabolic risk factors among parents and children according to categories of eating-away-from home and snacking in the *previous* survey year. China Health and Nutrition Survey 1991-2009*

	Away-from-home eating							**Snacking - fruit & vegetable**				**Snacking - other snacks**			
	None	<1 meal/d			≥1 meal/d			Non-snacker	Snacker			Non-snacker	Snacker		
		OR	95% CI	*P*	OR	95% CI	*P*		OR	95% CI	*P*		OR	95% CI	*P*
Parents															
High WHtR† (n=5579)															
Fully-adjusted model‡								Ref.	**0.76**	**0.59,0.97**	**0.03**	Ref.	0.70	0.49,1.01	0.06
18-39y	Ref.	0.82	0.63,1.07	0.14	**0.68**	**0.53,0.88**	**0.003**								
≥40y	Ref.	1.20	0.96,1.50	0.10	0.92	0.74,1.16	0.50								
Elevated BP† (n=5577)															
Fully-adjusted model without WHtR‡	Ref.	1.07	0.93,1.22	0.33	0.9	0.79,1.03	0.12	Ref.	**0.71**	**0.58,0.87**	**0.002**	Ref.	1.07	0.82,1.40	0.63
Fully-adjusted model with WHtR§	Ref.	1.06	0.93,1.22	0.39	0.94	0.82,1.08	0.41	Ref.	**0.75**	**0.61,0.91**	**0.004**	Ref.	1.12	0.85,1.47	0.43
Elevated HbA1c† (n=942)															
Fully-adjusted model without WHtR‡	Ref.	0.76	0.43,1.37	0.36	1.12	0.67,1.85	0.67	Ref.	1.00	0.58,1.73	0.99	Ref.	**2.63**	**1.00,6.88**	**0.049**
Fully-adjusted model with WHtR§	Ref.	0.71	0.41,1.26	0.25	1.09	0.67,1.78	0.74	Ref.	1.13	0.67,1.90	0.66	Ref.	2.41	0.95,6.14	0.06
High CRP† (n=925)															
Fully-adjusted model without WHtR‡	Ref.	1.16	0.70,1.91	0.57	0.94	0.59,1.50	0.81	Ref.	1.04	0.65,1.68	0.86	Ref.	0.98	0.38,2.52	0.97
Fully-adjusted model with WHtR§	Ref.	0.94	0.56,1.59	0.82	0.87	0.55,1.40	0.58	Ref.	1.23	0.75,2.02	0.41	Ref.	0.88	0.31,2.46	0.80
Children															
High WHtR† (n=3829)															
Fully-adjusted model	Ref.	1.07	0.72,1.60	0.74	**1.47**	**1.02,2.13**	**0.01**	Ref.	**0.57**	**0.33,0.99**	**0.048**	Ref.	1.19	0.68,2.10	0.54
Elevated BP† (n=3451)															
Fully-adjusted model without WHtR‡	Ref.	1.07	0.87,1.33	0.51	0.99	0.80,1.22	0.89	Ref.	1.05	0.79,1.39	0.76	Ref.	1.01	0.73,1.40	0.94
Fully-adjusted model with WHtR§	Ref.	1.06	0.85,1.32	0.63	1.04	0.84,1.30	0.72	Ref.	1.13	0.86,1.49	0.39	Ref.	1.07	0.78,1.45	0.69
Elevated HbA1c† (n=264)															
Fully-adjusted	Ref.	0.80	0.26,2.40	0.69	1.27	0.45,3.58	0.66	Ref.	1.78	0.62,5.13	0.29	Ref.	0.74	0.27,2.05	0.57

model without WHtR‡															
Fully-adjusted model with WHtR§	Ref.	0.89	0.29,2.70	0.84	1.27	0.44,3.62	0.66	Ref.	1.76	0.60,5.12	0.30	Ref.	0.72	0.25,2.03	0.53
High CRP† (n=236)															
Fully-adjusted model without WHtR‡	Ref.	0.66	0.29,1.49	0.32	0.49	0.23,1.06	0.07	Ref.	0.85	0.41,1.77	0.66	Ref.	0.91	0.40,2.06	0.82
Fully-adjusted model with WHtR§	Ref.	0.50	0.20,1.26	0.14	0.48	0.21,1.10	0.08	Ref.	0.96	0.44,2.08	0.92	Ref.	1.05	0.43,2.58	0.91

*We conducted separate random-effects logistic models for parents and children, with random intercept for individuals.

†High WHtR: WHtR≥0.5. Elevated BP: SBP z-score or DBP z-score ≥90th percentile of the age-, sex-, and height- specific BP percentile or BP≥120/80mmHg for children using the US CDC 2000 growth curve reference; BP≥120/80mmHg for adults. Elevated HbA1c: HbA1c≥5.7%. Elevated CRP: 1-10 mg/L for children and 3-10 mg/L for adults.

‡Models adjusted for age (y), sex, household income (tertiles), geographic region (North/Central/South), year of study entry, highest parental education (none or primary/middle school/high school/technical, college or higher), smoking (yes/no), total energy intake (kcal/d, quartiles), total fat intake (% total energy, quartiles), total fruits and vegetables intake (% total energy, quartiles), sodium intake (mg/d, quartiles, BP models only), and total PA (MET-hrs/wk, quartiles).

§Additionally adjusted WHtR.

Abbreviations: WHtR, waist-to-height ratio; BP, blood pressure; HbA1c, hemoglobin A1c; CRP, C-reactive protein.

Table 5.3. Odds ratios (95% confidence intervals) of cardiometabolic risk factors among parents and children according to categories of screen time and leisure-time sports participation in the *previous* survey year. China Health and Nutrition Survey 2004-2009*

	Screen time							Leisure-time sports participation			
	≤1 hr/d	1-2 hrs/d			>2 hrs/d			None	Any		
		OR	95% CI	*P*	OR	95% CI	*P*		OR	95% CI	*P*
Parents											
High WHtR† (n=1900)											
Fully-adjusted model‡								Ref.	1.59	0.85,2.98	0.09
18-39y	Ref.	1.12	0.67,1.88	0.67	1.09	0.64, 1.84	0.76				
≥40y	Ref.	**2.13**	**1.34,3.39**	**0.001**	**2.55**	**1.56, 4.19**	**<0.001**				
Elevated BP† (n=1899)											
Fully-adjusted model without WHtR‡	Ref.	1.21	0.94,1.57	0.14	1.2	0.92,1.57	0.17	Ref.	0.88	0.57,1.38	0.59
Fully-adjusted model with WHtR §	Ref.	1.17	0.90,1.51	0.24	1.15	0.88,1.50	0.31	Ref.	0.81	0.51,1.27	0.36
Elevated HbA1c† (n=898)											
Fully-adjusted model without WHtR‡	Ref.	0.82	0.49,1.36	0.44	0.99	0.58,1.70	0.98	Ref.	1.16	0.49,2.71	0.74
Fully-adjusted model with WHtR §	Ref.	0.81	0.49,1.34	0.41	1.00	0.59,1.68	0.99	Ref.	1.19	0.51,2.79	0.69
High CRP† (n=881)											
Fully-adjusted model without WHtR‡	Ref.	1.52	0.94,2.46	0.09	1.03	0.61,1.75	0.91	Ref.	1.38	0.67,2.83	0.38
Fully-adjusted model with WHtR §	Ref.	1.45	0.89,2.38	0.14	1.05	0.61,1.81	0.85	Ref.	1.50	0.70,3.19	0.30
Children											
High WHtR† (n=1321)											
Fully-adjusted model‡	Ref.	**1.63**	**1.07,2.49**	**0.02**	**1.64**	**1.03,2.63**	**0.04**	Ref.	1.33	0.91,1.96	0.15
Elevated BP† (n=1154)											
Fully-adjusted model without WHtR‡	Ref.	1.08	0.77,1.50	0.67	**1.56**	**1.06,2.29**	**0.02**	Ref.	1.00	0.74,1.36	0.99
Fully-adjusted model with WHtR §	Ref.	1.01	0.72,1.39	0.98	**1.51**	**1.04,2.21**	**0.03**	Ref.	1.03	0.76,1.38	0.87
Elevated HbA1c† (n=234)											
Fully-adjusted model without WHtR‡	Ref.	1.34	0.47,3.81	0.58	1.47	0.47,4.57	0.51	Ref.	0.67	0.25,1.80	0.43
Fully-adjusted model with WHtR §	Ref.	1.10	0.37,3.20	0.87	1.29	0.41,4.02	0.66	Ref.	0.71	0.26,1.92	0.50
High CRP† (n=210)											
Fully-adjusted model without WHtR‡	Ref.	**2.56**	**1.07,6.15**	**0.04**	1.76	0.70,4.41	0.23	Ref.	**0.33**	**0.16,0.69**	**0.003**
Fully-adjusted model with WHtR §	Ref.	**3.44**	**1.32,8.99**	**0.01**	2.15	0.76,6.06	0.15	Ref.	**0.24**	**0.11,0.53**	**<0.001**

*We conducted separate random-effects logistic models for parents and children, with random intercept for individuals.

†High WHtR: WHtR≥0.5. Elevated BP: SBP z-score or DBP z-score ≥90th percentile of the age-, sex-, and height- specific BP percentile or BP≥120/80mmHg for children using the US CDC 2000 growth curve reference; BP≥120/80mmHg for adults. Elevated HbA1c: HbA1c≥5.7%. Elevated CRP: 1-10 mg/L for children and 3-10 mg/L for adults.

‡Models adjusted for age (y), sex, household income (tertiles), geographic region (North/Central/South), year of study entry, highest parental education (none or primary/middle school/high school/technical, college or higher), smoking (yes/no), total energy intake (kcal/d, quartiles), total fat intake (% total energy, quartiles), total fruits and vegetables intake (% total energy, quartiles), sodium intake (mg/d, quartiles, BP models only), and total PA (MET-hrs/wk, quartiles).

§Additionally adjusted WHtR.
Abbreviations: WHtR, waist-to-height ratio; BP, blood pressure; HbA1c, hemoglobin A1c; CRP, C-reactive protein.

Supplemental Table 5.1. Characteristics of analytic sample according to dietary and physical activity behaviors, China Health and Nutrition Survey 2000 and 2009

	Away-from-home eating				Snacking - fruits & vegetables			Snacking - other snacks		
	None	<1 meal/d	≥1 meal/d	*P*	Non-snacker	Snacker	*P*	Non-snacker	Snacker	*P*
Year 2000										
Parents										
n	1418	820	740		2797	177		2855	119	
Age, y *	40.2 (6.2)	39.9 (6.2)	39.6 (5.7)	0.06	40.0 (6.1)	39.6 (5.6)	0.47	39.9 (6.0)	41.1 (6.7)	0.03
Gender, % male	43.5	46.3	52.8	<0.001	46.8	44.1	0.49	46.4	51.3	0.30
Total energy intake, kcal/d*	2319 (633)	2326 (612)	2321 (640)	0.97	2328 (625)	2225 (679)	0.04	2322 (629)	2322 (637)	1.00
% energy from fat, %*	24.8 (10.9)	26.6 (10.4)	32.1 (9.9)	<0.001	27.3 (10.7)	27.6 (13.0)	0.87	27.4 (10.8)	26.1 (10.8)	0.52
% energy from FV, %*	3.3 (3.7)	3.4 (4.5)	3.0 (3.2)	0.56	3.0 (3.8)	5.5 (4.1)	<0.001	3.2 (3.9)	3.1 (2.4)	0.92
Total PA, MET-hrs/d*	356 (240)	317 (228)	211 (158)	<0.001	311 (228)	277 (196)	0.05	308 (227)	328 (216)	0.35
Household income, 1000 yuan/yr*†‡	16.3 (12.7)	19.9 (16.9)	22.9 (20.9)	<0.001	18.4 (15.9)	25.2 (21.1)	<0.001	18.5 (16.2)	24.1 (17.3)	<0.001
Urbanicity*†§	48.9 (14.5)	55.8 (16.9)	65.5 (16.0)	<0.001	54.3 (16.7)	62.1 (18.9)	<0.001	54.4 (16.8)	61.7 (18.1)	<0.001
Geographic region, %†										
North	21.0	24.2	16.3		18.9	43.8		20.2	26.3	
Central	26.1	26.9	42.7	<0.001	30.8	26.6	<0.001	29.6	45.8	<0.001
South	52.9	48.9	41.1		50.3	29.7		50.3	28.0	
Children										
n	991	495	497		1851	128		1861	118	
Age, y*	12.5 (2.8)	12.3 (2.7)	12.8 (2.7)	0.03	12.6 (2.7)	12.5 (2.9)	0.79	12.6 (2.7)	12.1 (2.8)	0.049
Gender, % male	54.5	52.7	52.7	0.73	53.4	56.3	0.64	54.0	48.3	0.23
Total energy intake, kcal/d*	1930 (588)	1910 (592)	1980 (593)	0.15	1939 (583)	1916 (690)	0.66	1939 (594)	1926 (532)	0.82
% energy from fat, %*	25.3 (11.3)	28.4 (11.0)	31.6 (10.5)	<0.001	28.5 (11.3)	28.7 (11.4)	0.47	27.5 (11.2)	29.7 (12.2)	0.23
% energy from FV, %*	3.1 (3.4)	3.8 (5.7)	3.0 (3.2)	0.17	3.0 (4.0)	5.3 (4.8)	<0.001	3.2 (4.0)	4.3 (5.5)	0.09

Total PA, MET-hrs/d*	44.5 (66.6)	40.8 (58.2)	39.2 (42.6)	0.22	42.3 (60.4)	41.5 (41.3)	0.89	42.8 (60.8)	33.4 (25.8)	0.10
Year 2009										
Parents										
n	522	270	540		913	414		1204	123	
Age, y*	41.6 (5.4)	40.4 (4.7)	40.6 (4.7)	<0.001	41.2 (5.2)	40.4 (4.6)	0.01	41.0 (5.0)	40.8 (4.8)	0.77
Gender, % male	46.4	42.2	53.5	0.01	50.9	43.2	0.01	49.0	43.9	0.28
Total energy intake, kcal/d*	2202 (598)	2109 (556)	2207 (650)	0.07	2224 (634)	2107 (553)	0.001	2176 (609)	2298 (641)	0.04
% energy from fat, %*	27.1 (10.7)	30.5 (10.9)	31.2 (10.4)	<0.001	28.8 (11.0)	30.7 (10.1)	0.01	29.1 (10.8)	33.4 (9.4)	<0.001
% energy from FV, %*	3.3 (2.3)	3.8 (2.7)	3.8 (3.2)	0.01	2.7 (2.1)	5.4 (3.3)	<0.001	3.4 (2.7)	5.0 (3.4)	<0.001
Total PA, MET-hrs/d*	303 (220)	300 (252)	236 (195)	<0.001	282 (210)	262 (241)	0.12	281 (221)	229 (203)	0.01
Household income, 1000 yuan/yr*†‡	34.8 (43.5)	37.3 (28.6)	48.3 (47.2)	0.002	37.6 (45.0)	46.9 (37.9)	0.01	39.1 (45.6)	45.1 (34.9)	0.11
Urbanicity*†§	54.8 (15.0)	61.1 (19.3)	71.1 (18.3)	<0.001	59.8 (18.1)	69.0 (18.7)	<0.001	60.8 (18.0)	68.1 (19.8)	<0.001
Geographic region, %†										
North	9.0	35.8	17.1		8.7	33.3		17.5	16.7	
Central	17.6	18.4	40.3	<0.001	25.9	30.4	<0.001	26.0	31.0	0.45
South	73.4	45.9	42.6		65.4	36.2		56.5	52.3	
Children										
n	233	109	258		390	207		423	174	
Age, y*	13.2 (2.2)	13.7 (2.0)	13.5 (2.0)	0.03	13.4 (2.1)	13.5 (2.0)	0.63	13.5 (2.1)	13.2 (2.0)	0.06
Gender, % male	57.9	50.5	57.8	0.37	60.3	49.8	0.01	57.7	54.0	0.41
Total energy intake, kcal/d*	1727 (562)	1889 (512)	1856 (624)	0.02	1787 (618)	1864 (513)	0.13	1786 (569)	1881 (617)	0.07
% energy from fat, %*	27.4 (10.1)	30.7 (10.3)	30.8 (10.8)	0.001	28.4 (10.9)	31.5 (9.7)	0.001	28.3 (10.8)	32.5 (9.4)	<0.001
% energy from FV, %*	3.4 (2.9)	4.1 (2.8)	3.7 (2.9)	0.11	2.7 (2.0)	5.5 (3.4)	<0.001	3.4 (2.7)	4.3 (3.2)	<0.001
Total PA, MET-hrs/d*	63.4 (77.0)	58.9 (64.4)	56.3 (73.9)	0.57	63.8 (79.8)	51.6 (59.6)	0.054	58.9 (75.6)	61.2 (68.8)	0.72

*Data are mean (S.D.).

†Shown for parents only because they are measured at the household level and are identical for parents and children.
‡Total household income inflated to 2011.
§Urbanicity defined by a multicomponent urbanicity scale ranging from 0-120 [132].
Abbreviations: FV, fruits and vegetables; PA, physical activity.

Supplemental Table 2. Prevalence of cardiometabolic disease risk factors* of analytic sample, China Health and Nutrition Survey 1993-2009

	1993	1997	2000	2004	2006	2009	*P*
Parents							
n	3582	3344	3624	3216	2975	1814	
High WHtR	27.9	33.1	42.1	44.4	45.0	47.5	<0.001
Elevated BP	44.9	50.1	53.8	55.8	55.0	56.7	<0.001
Elevated HbA1c	-	-	-	-	-	26.1	-
Elevated CRP	-	-	-	-	-	17.2	-
Children							
n	2736	2610	2638	2039	1740	984	
High WHtR	6.0	5.8	6.3	8.2	8.0	9.7	0.002
Elevated BP	11.4	13.1	15.8	19.4	13.8	18.3	<0.001
Elevated HbA1c	-	-	-	-	-	15.0	-
Elevated CRP	-	-	-	-	-	34.1	-

*High WHtR: WHtR≥0.5. Elevated BP: SBP z-score or DBP z-score ≥90th percentile of the age-, sex-, and height- specific BP percentile or BP≥120/80mmHg for children using the US CDC 2000 growth curve reference; BP≥120/80mmHg for adults.
Elevated HbA1c: HbA1c≥5.7%. Elevated CRP: 1-10 mg/L for children and 3-10 mg/L for adults.
-: HbA1c and CRP were only measured in 2009
Abbreviations: WHtR, waist-to-height ratio; BP, blood pressure; HbA1c, hemoglobin A1c; CRP, C-reactive protein

CHAPTER 6. SYNTHESIS

Overview of findings

In this research, we sought to examine the associations between parent and child CMD risk factors as well as diet and activity behaviors, whether household structure (i.e. number of child and generation of family member in the households) relates to child CMD risk factors and behaviors, and how these behaviors predict CMD risk factors in children versus their parents. To address these questions, we first cross-sectionally determined the parent-child associations for HbA1c, BP, and CRP, and tested whether household structure (i.e. living with grandparents or not, having siblings or not) related to children's HbA1c, BP, and CRP in Chinese households in 2009. Second, using longitudinal data collected over the 1991-2009 period, we studied changes in urbanization-related diet, screen time, and PA behaviors over time in children compared to their parents living in the same household. We also examined whether children's diet, screen time, and PA behaviors associated with those of their parents, and whether these behaviors in children differed by household structure. Finally, we longitudinally determined the associations between lagged diet, screen time, and PA with high WHtR, elevated BP, elevated HbA1c, and high CRP at the next survey among Chinese children and their parents from 1991 to 2009. We also tested the intergenerational difference in associations between these behaviors and risk factors in children versus their parents.

We used data from CHNS, one of the only household-based, longitudinal studies with data collected from each household member in over 3000 two-generation and over 1000 three-generation households. We have repeated detailed measurements of diet, PA, and anthropometric

data from 1991 to 2009, and biomarker data collected in 2009 using fasting blood. Capitalizing on this unique dataset, we were able to study health behaviors and CMD risk factors at the household level in the context of rapid environmental change. We determined the extent to which child behaviors and CMD risk factors associated with those of their parents over time, investigated the difference in child behaviors and CMD risk factors by household structure, and examined the association between behaviors and CMD risk factors in children and their parents. Answering these questions is important as it improves understanding on familial factors associated with child CMD risk in fast-urbanizing China with surging CMD risk in children. Findings can inform household-based behavioral intervention in identifying individuals at high CMD risk and designing efficacious strategies to reduce CMD in the households in this modernizing population. Below we present a brief review of our findings, limitations and strengths of our research, its significance and public health impact, as well as future directions.

Do children share CMD risk factors, as well as diet, screen time, and PA behaviors with their parents?

To understand the resemblance in CMD risk factors and lifestyle behaviors between children and their parents, we examined the relationship between children's CMD risk factors and lifestyle behaviors with those of their parents. First, using random-effects linear regression and logistic regression models, we determined how children's HbA1c, BP, and CRP correlated with those of their parents. We found positive associations between children and their parents for HbA1c, between girls and their mothers for BP, as well as between children and their fathers for CRP.

Next, we ran random-effects negative binomial regression and random-effects logistic regression models to examine the associations between children's with parental animal-source

food consumption, away-from-home eating, snacking, screen time, and leisure-time sports over time, and tested whether the magnitude of associations differed across years or by household sociodemographics. We found that children's behaviors were positively associated with those of their parents. Higher intakes of animal-source food, away-from-home food, and snacks in parents were associated with higher intakes of these foods in their children. Children also spent more time on screen-based activities when their parents had higher screen time, and children were more likely to participate in leisure-time sports if their parents engaged in leisure-time sports. Nonetheless, overall the magnitude of associations decreased over time, and was weaker in more urbanized areas, suggesting weakened parent-offspring associations in behaviors with urbanization in China over the past two decades.

Overall, the positive parent-offspring associations for CMD risk factors and health behaviors indicate that family environment, especially parents, plays an important role in child lifestyle behaviors and CMD health status. Although we cannot draw causal inferences from our research, our findings emphasize that intervention efforts should target family environment when designing behavior intervention strategies to reduce child CMD risk due to the observed parent-offspring concordance in behaviors and risk.

Do child CMD risk factors or lifestyle behaviors differ by household structure?

Besides the association with parents, children's behaviors and CMD risk may also differ by household structure because Chinese grandparents are more likely to indulge their grandchildren with modern, unhealthy foods and screen-based sedentary behaviors, especially in single-child households. [23-25] To investigate whether household structure was related to child CMD risk factors or lifestyle behaviors, we explicitly tested child risk factors and behaviors by household structure, adjusting for child age and sex, as well as household socioeconomic factors

such as income and urbanicity. We found that being an only child was associated with higher HbA1c compared to living with siblings in the household, whereas living with grandparents was not associated with child HbA1c, BP, or CRP status. Children's behaviors also differed by the presence of siblings. Compared to children living with siblings, only children consumed more animal-source foods, away-from-home foods, and snacks. On the contrary, living with a grandparent was associated with slightly less away-from-home eating.

Overall, our findings on the association between household structure with child CMD risk factors and health behaviors indicate differences in child risk and behaviors by the number of child and the number of family member generation in the household. Children without siblings may be at greater risk of developing CMD risk factors due to their relatively more modernized eating habits, compared to children living with siblings in the household. Only-children households should specifically be considered for interventions targeting children's lifestyle behaviors and CMD risk in this population.

Do behavioral changes or behavior-CMD risk factor associations differ in children and their parents?

Previous research has found a faster increase rate of obesity in children compared to adults in China, suggesting different change rates in behaviors between children and adults in response to urbanization, and potential age-related biological differences in disease etiology across generations. [2]To examine whether there were intergenerational differences in urbanization-related behavior changes or behavior-risk associations, we addressed these questions in our research using longitudinal models. First, using random-effects negative binomial regression and random-effects logistic regression models, we examined and compared the changes in selected urbanization-related diet, screen time, and PA behaviors over time in

children versus their parents. We found increases in animal-source food intake, away-from-home eating, snacking, screen time, and the probability of leisure-time sports participation in both child and parent generations from 1991 to 2009. However, the rate of changes in behaviors differed between children and their parents. Particularly, the increase in away-from-home eating and snacking was greater in children than in their parents in later years. This disparity suggests that children and adults might have responded to urbanization differently. Faster increases in away-from-home eating and snacking in children relative to their parents may be at least partly contributable to the greater rate of the increasing obesity epidemic in children versus adults in China. [2]

Next, we determined the associations between lagged diet, screen time, and PA behaviors with the probability of high WHtR, elevated BP, elevated HbA1c, and high CRP at the next survey in children and their parents, using generation-specific random-effects logistic regression models. Overall, CMD risk factors were negatively associated with lagged consumption of fruit and vegetable snacks and leisure-time sports, and positively associated with lagged screen time in both children and adults. Yet, away-from-home eating was negatively associated with parental high WHtR but positively associated with childhood high WHtR. The opposite direction of association may be due to distinct behavioral patterns between children and adults, such as difference in types of away-from-home foods consumed by parents versus their children. Another possible reason for the discrepancy in the association between children and adults could be age-related biological differences in disease etiology. [2]

Overall, we observed generational disparities in the rate of changes in lifestyle behaviors as well as the behavior-CMD risk factor associations between child and parent generations. Intervention should take these disparities into account when designing behavior change strategies

and consider generation-specific strategies to reduce CMD risk across generations in fast modernizing societies.

Limitations

Our study has several limitations. The first is related to the diet, screen time, and PA measurements. Diet was measured during a short 3-day period, thus is limited in estimating usual intake of periodically consumed food such as some types of snacks (e.g. sugar-sweetened beverages). Therefore, snacking might have been underestimated in our study. Further, due to shared dishes in Chinese households, we allocated proportions of foods consumed at the household level to each individual based on the proportion of a dish one person consumed. Thus the measurement of some foods might not be accurate at the individual level, such as animal-source foods, because one household member might have consumed more of one component than another in a mixed-component dish. For activity measurements, since data on screen time in adults were first collected in 2004 and the leisure-time sports survey changed in 2004, we were only able to compare these two behaviors in children and adults in 2004, 2006, and 2009. In addition, self-reported diet, screen time, and PA data may be subject to recall and social desirability biases, although the CHNS data are based on highly detailed recall methods, including analysis of household-specific recipes, weighing and measuring of condiments. Previous research on selected components of the diet (energy, protein, sodium, monosodium glutamate) suggests strong validity, [120, 136-139] and the PA components have been found to be highly predictive of incident obesity and weight gain in adults. [49, 140-142] Finally, for younger children who were under 10y, parents who reported their own diet, screen time, and PA and those of their children could have resulted in potential same-source bias and high parent-child correlation, especially for diet. However, we conducted sensitivity analyses to test whether

our finding was robust to whether the child's survey was aided by their parents (aged 7-10y only, 22% total observations) and found similar results.

Second, we were unable to determine how unobserved heterogeneity might have biased our results. For example, genetic predisposition has been found to partly contribute to the intra-familial concordance of CMD risk factors, [122, 161] diet, [58, 162, 163] and PA behaviors. [51, 164] However, since we do not have genetic data, we are unable to address to what extent the genetic components influenced our observed parent-child concordance in CMD risk factors and lifestyle behaviors.

Third, loss to follow-up could have caused selection bias since we restricted to parent-child pairs who had at least two waves of diet, screen time, or PA data within the 7-17y age range of children. We could have slightly underestimated the predicted values of some behaviors due to the difference in behaviors in the included compared to the excluded samples.

Fourth, we were unable to test if child's pubertal status has played a role in the parent-offspring associations for CMD risk factor, or the behavior-risk associations in children, because we did not have data on pubertal markers in children. Nonetheless, we tested these associations among younger versus older children and found minimal differences in associations. Similarly, menopausal status could theoretically play a role, but the vast majority of mothers in our study (95%) were below the mean age of menopause (49y) in China. [123]

Fifth, we focused on urbanization-related diet, screen time, and PA behaviors in this dissertation, thus some behaviors were more salient in urban (versus rural) areas, such as snacking. However, we did not directly compare the difference in behaviors by urbanization level in this dissertation because we focused on illustrating the concordance of behaviors between children and their parents, which differed by urbanization level. In addition, we did not

address the cohort effects of these lifestyle behaviors and CMD risk factors, which have been observed in urbanizing China. [11, 165]

Other limitations include: 1) we calculated child's BP z-scores using the US reference because no age-, sex- and height-specific reference for Chinese children is available; 2) we cannot imply causation from the parent-offspring associations as it is impossible to unravel within-household dynamics; and 3) the lack of sampling weights in CHNS does not allow predicting population-relevant estimates of the behaviors and CMD risk factors.

Strengths

Despite the limitations, our study has several key strengths. First, our data source CHNS is one of the only population-based, longitudinal studies with diet, PA, anthropometric data collected repeatedly at the household level over 20 years of rapid environmental change. In particular, the combination of three 24-hour recalls at the individual level and the food inventory at the household level provides very detailed and precise dietary measures; our PA data captures a wide range of sedentary behaviors and activity behaviors. Additionally, data collected at the household level allow us to examine how household environment (i.e. parental behavior and health status, household structure) relates to children's lifestyle behaviors and health. As household indisputably plays an important role in children's health, the rich household data in CHNS made our research possible in informing intervention efforts in the household context. Further, trained staff measured anthropometry and BP at each survey and collected fasting blood samples in 2009 from each member of the household. This minimizes potential sources of biases of the CMD risk factors, as self-reported height, weight, and waist circumference are often biased due to social desirability, and there is a high prevalence of undiagnosed CMD risk factors in this population.

Another important strength of our study is the definition of children's elevated BP based on the age-, sex-, and height-specific BP percentile algorithm for children, which further incorporates height compared to child BP references that are only age- and sex-specific. [124] Due to the high correlation between children's height and BP, our approach more appropriately classified children's BP status compared to other commonly used child BP references that are only age- and sex-adjusted. [124]

Significance and public health impact

Our research has significant public health implications. First, our findings show significant and positive associations between children's CMD risk factors and lifestyle behaviors with those of their parents, emphasizing family-based intervention efforts to reduce CMD risk in Chinese households to improve efficiency. Second, we found differences in childhood CMD risk factors and lifestyle behaviors by household structure, which can be used to identify children at high risk of CMD and guide behavioral interventions to reduce risk. Third, we observed that CMD risk factors were negatively associated with consumption of fruit and vegetable snacks as well as leisure-time sports, and positively associated with screen time, suggesting potential benefits of promoting healthy food choices and an active lifestyle. Finally, our research demonstrated that changes in lifestyle behaviors in children differed from those in parents, and that the behavior-CMD risk factor association differed in children versus their parents, suggesting generation-specific intervention strategies to change behaviors in children and adults.

CMD risk factors and lifestyle behaviors cluster between children and their parents

Our research is the first to describe and document the parent-offspring associations in various CMD risk factors and lifestyle behaviors in regionally-diverse Chinese households. We found that children's CMD risk factors, as well as diet, screen time, and PA behaviors were

positively associated with those of their parents. This finding highlights the need for interventions to target not only the children, but also their parents to reduce CMD risk through behavioral changes due to the clustering of these risk factors and behaviors in the household. Behavioral changes in the parents may be even more critical since parental behaviors not only contribute to parental CMD risk, but might also influence children's behaviors, which in turn affect children's CMD risk.

Household structure is associated with children's CMD risk factors and lifestyle behaviors

In this dissertation, we studied children's CMD risk factor and health behaviors in the household context. Besides the potential influence of parents, household structure could also associate with children's risk and behaviors, as suggested by previous research. [23-25] To our knowledge, no study has examined whether household structure is associated with children's obesity-related CMD risk factors in China. Nor has any research examined how household structure relates to children's diet, screen time, and PA behaviors in a large Chinese population-based study. To address these questions, we explicitly investigated whether children's lifestyle behaviors and CMD risk factors differed by the presence of siblings or the presence of grandparents. We found that compared to children who lived with siblings in the household, only children consumed more animal-source foods, away-from-home foods, snacks, and had higher HbA1c. These findings contribute to better understanding on how household structure related to childhood behaviors and health in the Chinese population where only-child and three-generation households are common. We suggest that intervention efforts pay specific attention to children in single-child households because they may be at greater risk of CMD.

Urbanization-related lifestyle behaviors are associated with CMD risk factors

Examining the association between urbanization-related lifestyle behaviors and CMD risk factors, our data provide evidence that away-from-home eating, snacking, screen time, and leisure-time sports were associated with various CMD risk factors in Chinese children and their parents. For example, high WHtR was negatively associated with consumption of fruit and vegetable snacks, and positively associated with screen time in both children and adults. Contrarily, consumption of snacks other than fruit and vegetable was related to higher probability of elevated HbA1c in adults. In addition, leisure-time sports participation was negatively associated with elevated CRP in children.

With urbanization, the Chinese population is shifting to a more Westernized lifestyle with increased snacking behavior and screen-based activities. [45-48] Based on our findings, interventions should focus on promoting healthy food choices when targeting dietary behaviors such as snacking among urbanizing populations with rapid behavior change. Decreasing screen time should also be emphasized along with encouraging leisure-time PA.

Intergenerational differences in behavioral changes and behavior-risk associations

Our study is the first to compare changes in urbanization-related lifestyle behaviors between children and their parents in a large Chinese sample. We found that the rate of changes in away-from-home eating and snacking over time was faster in children relative to their parents. This finding is consistent with a previous study, which reported a faster shift to an obesogenic diet in response to urbanization in Filipino offspring compared to their mothers. [50] In addition, we observed decreasing magnitude of parent-offspring associations over time with urbanization. Taken as a whole, these findings suggest that there might be a generational difference in behavioral changes in response to urbanization, leading to less concordance of lifestyle behaviors

between children and their parents with increasing urbanization. Younger generations may be more susceptible to behavioral changes compared to older generations. This finding provides important insights into why children have been experiencing a faster increase in obesity compared to adults in China.

Our research also addressed another understudied gap on CMD in the literature on how diet, screen time, and PA behaviors predict CMD risk factors distinctly in younger versus older generations, while controlling for shared genes and socioeconomic environment. We found different directions and magnitudes of the behavior-risk associations between children and their parents for some behaviors and risk factors, possibly due to distinctive behavioral patterns or age-related biological differences in disease etiology between children and adults.

Overall, our observed intergenerational differences in urbanization-related behavioral changes and behavior-CMD risk factor associations suggest that intervention efforts may need to carry out generation-specific strategies for behavioral changes in populations undergoing fast modernization. Our findings may also inform further studies to further investigate the discrepancy in behavioral patterns and the age-related biological differences in disease etiology between children and adults.

Future directions

While this study documented the parent-offspring associations for CMD risk factors, we did not explicitly investigate mechanisms underlying these associations. Thus, we cannot decompose how much of the associations resulted from genetic predisposition versus shared familial environment. Understanding the mechanisms may guide interventions to focus on modifiable factors with significant influences on the development of CMD risk factor in the Chinese population.

Further, we cannot imply causation from the parent-offspring associations for lifestyle behaviors in our study. Future studies are desired to investigate the within-household dynamics and study whether children's behaviors or attitudes may affect parental behaviors, or whether the parent-child association is mostly due to genetic components and/or shared environment. While the positive parent-offspring associations for lifestyle behaviors support household-based interventions, it would also be helpful to understand what other household-level factors (e.g., household socioeconomic status, living environment) have strong influences on children's behaviors for the purpose of designing more efficacious and successful interventions.

Moreover, while we used lagged behaviors to predict CMD risk factors at follow-up in the third aim, we found in our second aim that these behaviors have been changing with modernization in the past two decades. Therefore, future studies are needed to address how the change in behaviors predicts CMD risk factors. Studying this question will provide a better understanding of how urbanization contributes to the increased risk of CMD in this population.

Lastly, one limitation of our research is that fasting blood was first measured in 2009, thus we were not able to study incident elevated HbA1c and high CRP. It is important for future studies to examine how urbanization-related lifestyle behaviors predict incident CMD risk factors in this population.

These future directions will improve understanding of changes of lifestyle behaviors in relation to the dramatic increase in CMD risk factors in both children and adults in urbanizing China. With findings from our research and these proposed future studies, public health researchers will be able to make more informed decisions regarding identifying children at high CMD risk and conducting household-based interventions for behavioral changes to reduce this increasing burden in China.

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