European Journal of Clinical Nutrition (2016) **70,** 511–516
© 2016 Macmillan Publishers Limited All rights reserved 0954-3007/16
www.nature.com/ejcn

ORIGINAL ARTICLE

Transmission of energy-saving efficiency from obese parents to their offspring: the Korean National Health and Nutrition Examination Survey 2007–2011

Y Kim[1], H Kim[2] and Y-C Hong[3,4]

BACKGROUND/OBJECTIVES: Concerns of a growing obesity epidemic have increased since the association between obesity in parents and that in offspring was reported. However, the evidence regarding whether the energy-saving efficiency of obese parents is conveyed to their offspring and the duration of the expression of such transmitted efficiency is limited.
SUBJECTS/METHODS: We included 7647 matching sets of parent–offspring trios from South Korea. Multiple linear regression models were performed to estimate the energy-saving efficiency, as assessed by the associations between energy intake and obesity-related indices (waist-to-height ratio, waist circumference and body mass index ***z***-score), and to compare the energy-saving efficiency of offspring of obese and non-obese parents. All analyses were based on a complex sample design and were stratified by gender and age.
RESULTS: We identified a parental influence on obesity, that is, the more obese the parent, the higher the obesity-related indices of their offspring, in both genders and all age groups. The energy-saving efficiency of child offspring was highest when both parents were obese and lowest when both were non-obese; this difference was significant ($P < 0.05$) with regard to the energy-saving efficiency of all types of intake studied, except fat. However, the energy-saving efficiency of obese and non-obese parents did not differ when their offspring were adolescents and adults.
CONCLUSIONS: The critical window for transmission of energy-saving efficiency is limited to childhood. These findings suggest that children of obese parents should be more emphatically advised to maintain a balanced diet and to engage in regular physical activity.

European Journal of Clinical Nutrition (2016) **70,** 511–516; doi:10.1038/ejcn.2015.172; published online 7 October 2015

INTRODUCTION

Obesity, considered an important risk factor for type 2 diabetes, cardiovascular disease and hypertension, has increased in most industrialised and developing countries,[1–3] owing to the lifestyle changes in modern society (for example, high-calorie diets, a sedentary lifestyle and reduced physical activity). Concerns are growing of a greater obesity epidemic in the future, because obesity has been reported to be heritable or transferrable from parents to their offspring, and thus increasing the risk for childhood obesity.[4–6]

One unresolved issue regarding parental influence on the development of obesity is whether the 'energy-saving efficiency', which is the capability to store excess energy in the body owing to an energy imbalance between intake and expenditure,[7] is transmitted from parents to their offspring. Neel suggested the thrifty genotype hypothesis,[8,9] in which following natural selection, the genes of surviving hunter–gatherers would have a strong tendency to amass energy stores during times of abundance. The genes favouring survival during times of famine have, unfortunately, led to obesity after the rapid lifestyle changes associated with modern society; and this obesity may be transmitted to the next generation in times of abundance. Marked increases in obesity and type 2 diabetes in the populations of some Pacific islands exemplify this hypothesis, showing how rapid lifestyle changes amplify energy-saving efficiency and lead to health problems.[2,10] In the same context, Hales and Barker[11] suggested the thrifty phenotype hypothesis, where a defective intrauterine development due to maternal malnutrition is associated with type 2 diabetes, hypertension and coronary heart disease in middle age. The adverse effect of the programming in early life that leads to increased adiposity is most likely attributable to the thrifty phenotype hypothesis, but several reports have shown that the adverse effect on offspring was also attributable to maternal overnutrition during pregnancy.[12–16]

Despite the possible contribution of the transfer of energy-saving efficiency between generations, evidence of such transmission by obese parents and of the duration of the expression of such efficiency is limited. In this study, we assessed whether the energy-saving efficiency of the offspring of obese parents was maintained from childhood to adolescence and into adulthood.

MATERIALS AND METHODS

Study design

The Korean National Health and Nutrition Examination Survey (KNHANES) is a national project to assess the health and nutritional status of the

[1]Department of Pediatric Infectious Diseases, Institute of Tropical Medicine, Nagasaki University, Nagasaki, Japan; [2]Graduate School of Public Health, Seoul National University, Seoul, South Korea; [3]Institute of Environmental Medicine, Seoul National University College of Medicine, Seoul, South Korea and [4]Environmental Health Center, Seoul National University College of Medicine, Seoul, South Korea. Correspondence: Dr Y-C Hong, Institute of Environmental Medicine, Seoul National University College of Medicine, 103 Deahak-ro Jongro-gu, Chongro-Gu, Seoul 110-799, South Korea.
E-mail: ychong1@snu.ac.kr
Received 8 January 2015; revised 21 July 2015; accepted 3 August 2015; published online 7 October 2015

population of South Korea. The Korea Centers for Disease Control and Prevention (KCDC) of the Ministry of Health and Welfare collected the data from interviews and physical examinations performed at mobile examination centres and gathered information about nutritional status by making home visits. The KNHANES operates year round and examines representative samples of the Korean population selected by a complex sample design. Details of this sampling technique have been described elsewhere.[17,18]

We used the data of the KNHANES IV and V (2007–2011) in this study. Among the 42 347 participants, 84.3% of subjects completed all of the interviews, physical examinations and nutritional surveys (Figure 1). Offspring samples were included only if both paternal and maternal data were available. Offspring samples were excluded if they suffered from hyperlipidaemia, stroke, diabetes, cancer, renal failure and thyroid disease or if they showed weight changes of >10 kg over the last year. Finally, we obtained 7647 matching sets of parent–offspring trios, consisting of 4404 child ($\leqslant$ aged 12 years), 1555 adolescent (aged 13–18 years) and 1688 adult ($>$ aged 18 years) offspring.

Assessment of obesity indices

Three obesity indices were used for the offspring as health outcomes: waist-to-height ratio, waist circumference (cm) and the body mass index (BMI; kg/m^2) ***z***-score, based on evidence that these indices provide a better index of obesity compared with the BMI.[19–22] The BMI ***z***-score was standardised for gender and age group, with a mean of 0 and standard deviation of 1. We used the waist-to-height ratio in the main results, whereas the others were used in sensitivity analyses. All offspring samples (7647) were categorised into three groups according to the obesity status of their parents, which was defined as follows: 'both' if both parents were overweight or obese ($\geqslant$25 BMI), 'either' if only one parent was overweight or obese and 'none' if both parents were normal or underweight ($<$25 of BMI).

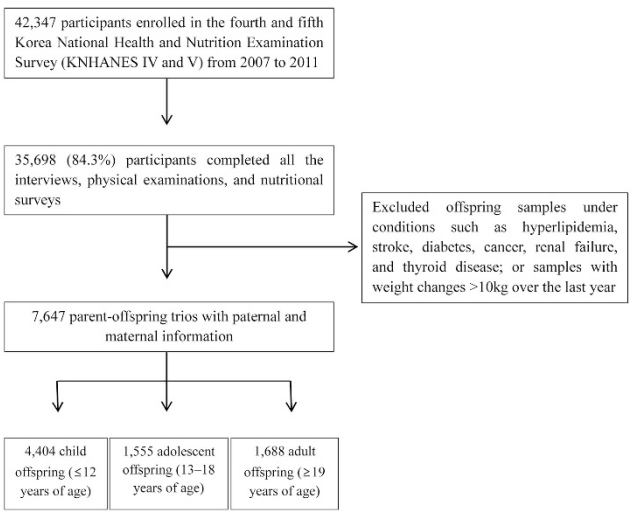

Figure 1. Flow diagram showing identification of study samples.

Energy intake and covariates

The KNHANES listed the daily intake of total energy (kcal; 1 kcal = 4.186 kJ), carbohydrates (g), fats (g) and proteins (g) for people aged 1 and older, and these data were gathered by trained dietary interviewers via a 24-h dietary recall method. Supplementary tools (that is, two-dimensional food containers and food models) were used to accurately measure portions. The dietary intake records were processed using a database of factors that convert food volume to weight, food composition and food ingredients for Koreans.

Gender, age, educational level and physical activity were considered as covariates. Educational level was classified as 'high school graduate or less' or 'college/university graduate or more'. Physical activity was also categorised into two groups: 'yes' for people who walked $\geqslant$ five times per week for over 30 min at a time, and 'no' for the others; these data were collected with a Korean version of the International Physical Activity questionnaire, which has been tested for validity and reliability.[23] Physical activity was measured only in adolescents and adults. Educational level was measured in all age groups, but it was used in the analysis of only the adults to avoid multicollinearity, because educational level and age are strongly correlated in children and adolescents owing to compulsory education.

Statistical analyses

Because the KNHANES adopted a complex sample design to select participants, our analyses, which were specialised to meet the demands of this design, were part of the PROC SURVEYSELECT procedure served by SAS version 9.3 (SAS Institute Inc., Cary, NC, USA). In addition, we calculated new weights for each sample to use the data over the combined 5 years.[18] As the actual sample size in 2007 was approximately half that in the other years, the 2007 samples were weighted more heavily compared with those for the other 4 years.

The finalised data were stratified into six sub-samples according to the age ($\leqslant$12 for children, 13–18 for adolescents and $>$18 for adults) and gender of the offspring. The univariate analyses used Wald's *F* test and the Rao–Scott chi-square test (applied to meet the demands of a complex sample design) to compare the characteristics (for example, gender or parental obesity status) of the offspring between groups (Tables 1 and 2). A multiple linear regression model (also applied to meet the needs of the

Table 1. Baseline characteristics of the study participants (offspring) according to age[a]

	Child ($\leqslant$ 12)			*Teen (13–18)*			*Adult ($>$18)*		
	Male	*Female*	P *value*[b]	*Male*	*Female*	P *value*[b]	*Male*	*Female*	P *value*[b]
No. of participants	2331	2073		800	755		798	890	
Age (years)	6.7 (3.4)	6.6 (3.4)	0.35	15.1 (1.7)	15.1 (1.7)	0.19	28.7 (7.9)	26.1 (5.8)	<0.001
BMI (kg/m^2)	17.6 (3.0)	16.9 (2.5)	<0.001	21.5 (3.7)	20.7 (3.4)	0.006	23.7 (3.5)	21.2 (3.2)	<0.001
Waist circumference (cm)	56.6 (10.5)	54.1 (8.6)	<0.001	73.6 (9.8)	68.5 (8.1)	<0.001	81.5 (9.5)	71.1 (8.2)	<0.001
Waist-to-height ratio	0.47 (0.06)	0.45 (0.06)	<0.001	0.43 (0.05)	0.43 (0.05)	0.94	0.47 (0.05)	0.44 (0.05)	<0.001
Total energy intake (kcal)	1648.4 (662.9)	1423.6 (566.5)	<0.001	2345.5 (915.3)	1852.9 (770.9)	<0.001	2419.3 (1099.9)	1721.2 (730.3)	<0.001
Carbohydrate intake (g)	265.6 (108.6)	231.0 (93.8)	<0.001	362.9 (138.2)	293.8 (120.7)	<0.001	350.5 (135.2)	260.7 (111.1)	<0.001
Fat intake (g)	39.5 (22.7)	34.0 (20.1)	<0.001	60.4 (37.5)	47.2 (30.9)	<0.001	58.6 (45.0)	44.5 (28.1)	<0.001
Protein intake (g)	58.2 (28.8)	49.9 (23.8)	<0.001	85.1 (40.8)	65.1 (32.4)	<0.001	91.0 (54.0)	64.0 (36.2)	<0.001
Physical activity (%)				56.5	53.2	0.10	51.4	49.7	0.05
Education (%)									
High school							60.7	41.3	<0.001
College/University							39.3	58.7	

[a]The summary statistics of continuous variables indicate the mean and standard deviation. [b]The statistical significance of the difference between males and females was examined by Wald's *F* test for continuous variables and the Rao–Scott chi-square test for categorical variables, applying a complex sample design.

© 2016 Macmillan Publishers Limited

Table 2. Obesity-related outcomes and energy intake by parental obesity status[a]

	Obesity status of parents[b]	*Male*				*Female*			
		None	*Either*	*Both*	P *value*[c]	*None*	*Either*	*Both*	P *value*[c]
Child (⩽12)	No. of participants	1093	1020	218		966	904	203	
	BMI (kg/m^2)	17.0 (2.6)	17.9 (3.1)	19.3 (3.7)	< 0.001	16.3 (2.1)	17.2 (2.6)	18.3 (3.0)	< 0.001
	Waist circumference (cm)	55.0 (9.5)	57.4 (10.8)	60.9 (12.6)	< 0.001	52.9 (7.7)	54.6 (8.8)	57.3 (10.5)	< 0.001
	Waist-to-height ratio	0.46 (0.05)	0.47 (0.06)	0.49 (0.06)	< 0.001	0.45 (0.06)	0.46 (0.06)	0.48 (0.06)	< 0.001
	Total energy intake (kcal)	1624.8 (651.4)	1670.1 (678.3)	1665.6 (646.5)	0.53	1423.0 (552.6)	1427.2 (589.0)	1410.7 (531.5)	0.69
	Carbohydrate intake (g)	262.5 (107.6)	268.6 (109.4)	266.4 (110.3)	0.74	231.8 (92.8)	229.8 (96.0)	232.5 (89.0)	0.91
	Fat intake (g)	38.7 (21.9)	40.3 (23.7)	40.2 (21.3)	0.51	33.8 (19.2)	34.6 (21.0)	32.5 (19.7)	0.51
	Protein intake (g)	56.9 (26.3)	59.4 (31.9)	59.1 (24.8)	0.28	49.7 (22.5)	50.5 (25.6)	48.8 (21.7)	0.49
Teen (13–18)	No. of participants	332	372	96		349	319	87	
	BMI (kg/m^2)	20.3 (3.1)	21.8 (3.6)	24.2 (4.2)	< 0.001	20.0 (3.2)	20.9 (3.1)	23.0 (3.8)	< 0.001
	Waist circumference (cm)	70.7 (8.4)	74.6 (9.7)	79.7 (10.9)	< 0.001	66.9 (7.9)	68.9 (7.2)	73.8 (9.8)	< 0.001
	Waist-to-height ratio	0.42 (0.05)	0.44 (0.05)	0.47 (0.06)	< 0.001	0.42 (0.05)	0.43 (0.04)	0.46 (0.06)	< 0.001
	Total energy intake (kcal)	2387.5 (943.0)	2360.2 (903.8)	2143.3 (841.9)	0.02	1833.1 (716.1)	1894.2 (854.6)	1781.1 (649.6)	0.43
	Carbohydrate intake (g)	366.6 (138.7)	368.8 (139.9)	327.2 (125.0)	0.03	293.8 (118.2)	296.6 (127.4)	283.4 (105.1)	0.62
	Fat intake (g)	61.9 (38.1)	60.4 (38.3)	54.8 (32.0)	0.01	45.8 (26.7)	49.6 (36.0)	44.3 (26.4)	0.13
	Protein intake (g)	89.1 (46.0)	83.3 (36.5)	78.4 (36.0)	0.01	63.8 (30.2)	66.5 (36.0)	65.3 (26.9)	0.88
Adult (> 18)	No. of participants	339	342	117		367	399	124	
	BMI (kg/m^2)	22.7 (3.0)	24.1 (3.4)	25.4 (4.0)	< 0.001	20.5 (2.7)	21.4 (3.5)	22.8 (3.1)	< 0.001
	Waist circumference (cm)	79.5 (8.7)	82.3 (9.4)	85.0 (10.9)	< 0.001	69.5 (7.4)	71.2 (8.6)	75.1 (7.8)	< 0.001
	Waist-to-height ratio	0.46 (0.05)	0.48 (0.06)	0.49 (0.06)	< 0.001	0.43 (0.05)	0.44 (0.05)	0.47 (0.05)	< 0.001
	Total energy intake (kcal)	2474.6 (1203.1)	2411.7 (1061.8)	2281.9 (868.3)	0.16	1750.9 (730.8)	1694.1 (722.5)	1720.8 (755.9)	0.55
	Carbohydrate intake (g)	354.2 (141.1)	353.1 (136.9)	332.5 (109.9)	0.13	265.6 (103.6)	256.1 (117.3)	261.3 (112.4)	0.50
	Fat intake (g)	58.7 (44.3)	59.5 (47.0)	55.8 (41.3)	0.66	45.0 (29.9)	44.0 (26.5)	44.4 (27.6)	0.86
	Protein intake (g)	94.8 (61.6)	89.2 (49.4)	85.3 (41.2)	0.25	64.3 (38.9)	63.2 (33.4)	66.0 (36.7)	0.56

Abbreviation: BMI, body mass index. [a]The summary statistics of continuous variables are means and standard deviations. [b]The obesity status of parents was defined as 'both' if both parents were overweight or obese (⩾25 of BMI), 'either' if one parent was overweight or obese, or 'none' if both parents were normal or underweight (< 25 of BMI). [c]Statistical significance according to parental obesity status was examined by Wald's *F* test, applying a complex sample design.

design) was used with an interaction term between parental obesity status and the energy intake of offspring, adjusted for the characteristics of the offspring (that is, age, survey year, educational level (adults only) and physical activity (adolescents and adults only)) to examine whether parental obesity status modified the association between waist-to-height ratio and energy intake. We estimated the association between waist-to-height ratio and energy intake according to parental obesity status (none, either and both) and tested the difference between the groups (Figure 2). The same regression models were also applied to analyses stratified by the age and gender of the offspring.

The relationship between waist-to-height ratio and energy intake was evaluated by the regression coefficient for the change in the waist-to-height ratio per unit increase in energy intake, which indicated the energy-saving efficiency. For the estimation, larger rather than single-unit increases in energy intake were used: 500 kcal for total energy, 100 g for carbohydrate, 20 g for fat and 30 g for protein.

Sensitivity analyses were performed to confirm whether our findings (the influence of parental obesity on the energy-saving efficiency of offspring) were consistent. First, a subset composed of those people who responded that their 24-h recall was based on their usual amount of food was created (88.1% of children, 74.5% of adolescents and 71.7% of adults). Second, overweight or obese people (⩾25 of BMI) who were more likely to underreport their energy intake were excluded (2.6% of children, 13.8% of adolescents and 21.3% of adults) because underreporting of energy intake in obese people has been well documented.[24,25] The cut-offs for being overweight or obese (⩾25 of BMI) in children and adolescents were based on the International Obesity Task Force.[26] Third, analyses used waist circumference and the BMI ***z***-score as the obesity indices instead of the waist-to-height ratio.

Code availability

The main code used in the statistical analyses in this study is available in the Supplementary information.

RESULTS

The demographic characteristics, obesity indices and energy intake of study participants (offspring) by age and gender are shown in Table 1. There were differences between genders in obesity indices (the waist-to-height ratio, waist circumference and BMI ***z***-score) and energy intake (total energy, carbohydrate, fat and protein) across the age groups, suggesting the need for analysis stratification by gender.

Table 2 shows changes in the distribution of the obesity indices and the energy intake in offspring according to the obesity status of the parents. The more obese the parents, the higher the obesity index scores of their offspring in all groups. However, except among male adolescents, energy intake levels were not associated with the parental obesity status.

Figure 2 presents the effects of parental obesity status on the association between the waist-to-height ratio and energy intake, which indicates the energy-saving efficiency, by age and gender after adjustment for confounding factors. In males, we found clear patterns showing that parental obesity status modified the association between waist-to-height ratio and the intake of all types of energy studied, except fat in the child group. The associations were highest when both parents were obese ('both' group), and they were lowest when both were non-obese ('none' group; reference); in addition, the difference between the groups was significant ($P < 0.05$). This suggests that the energy-saving efficiency of children, as assessed by the ratio of the change in the waist-to-height ratio per unit increase in energy intake, is influenced by parental obesity status. However, the clear patterns disappeared in adolescent and adult offspring. In females, the results for child and adult offspring were similar (less clear) to those in males but differed from those of adolescent males (Figure 2). The energy-saving efficiency of adolescent females was considerably higher when both parents were obese rather than from the 'none' or 'either' groups, although this difference was not significant. This suggests that the increased energy-saving efficiency due to parental obesity may be of longer duration in females than in males.

We found similar patterns in sensitivity analyses using waist circumference and the BMI ***z***-score instead of the waist-to-height ratio (Supplementary Figures S1 and S2) and using a different data

© 2016 Macmillan Publishers Limited

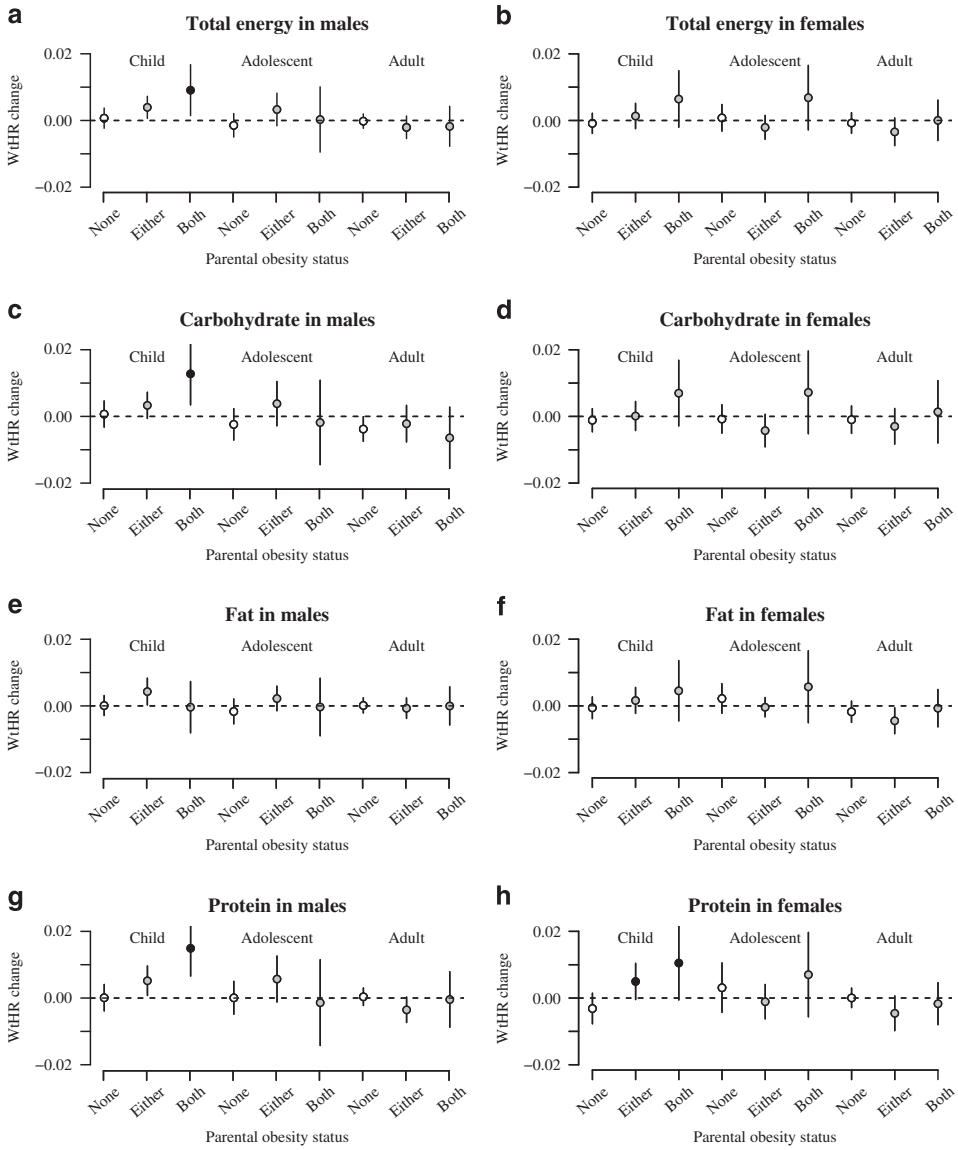

Figure 2. Waist-to-height ratio (WtHR) change estimates (and 95% confidence intervals) for a unit increase in energy intake (500 kcal for total energy, 100 g for carbohydrate, 20 g for fat and 30 g for protein) by parental obesity status, stratified by age and gender. (**a**–**h**) Stratified analysis by energy intake and gender: total energy in males (**a**); total energy in females (**b**); carbohydrate in males (**c**); carbohydrate in females (**d**); fat in males (**e**); fat in females (**f**); protein in males (**g**); protein in females (**h**). The white circles indicate the reference groups (none). The circles marked in black indicate that the energy-saving efficiency (i.e., the WtHR change per unit increase in intake for the reference group) differed significantly from that of the others (either or both) ($P < 0.05$). The circles are marked in grey if no such difference was observed. All effect estimates were adjusted for possible confounding factors.

subset (Supplementary Figures S3–S4). In particular, the results for carbohydrate and protein intake were consistent in male children and statistically significant ($P < 0.05$), particularly with regard to differences in the energy-saving efficiency between the 'none' and 'both' groups. The results for fat intake remained inconsistent in all groups.

DISCUSSION

The energy-saving efficiencies in the child offspring of obese parents were significantly higher than those of non-obese parents, whereas the energy-saving efficiency in adult offspring did not differ according to parental obesity status, regardless of gender and age. In addition, gender differences in adolescents were observed; the energy-saving efficiency associated with having obese parents seemed to be maintained in adolescent females but not in males.

Consistent with previous studies of the association between parent obesity and offspring obesity,[27–29] the more obese the parents, the more obese their offspring, regardless of the age of the offspring. However, the energy-saving efficiency of the offspring (the extent of being obese per unit increase of intake) of obese and non-obese parents significantly differed in the children but not in the adult offspring. This suggests that childhood is a key period for obesity attributable to high energy-saving efficiency transmitted by obese parents. Childhood obesity is associated with an increased risk of premature death[30] and cardiometabolic morbidity (for example, diabetes, hypertension and stroke).[31] A balanced diet should be emphasised, particularly for children of obese parents, to prevent them from amplifying the risk of childhood obesity.

© 2016 Macmillan Publishers Limited

The higher energy-saving efficiency in children of obese parents could be explained by genetic or epigenetic mechanisms and interactions between genetic and environmental factors. Obesity-susceptibility genes such as *FTO*, *MC4R* and *NPC1* are reported to be associated with childhood obesity.[4] Some studies have suggested that foetal hypernutrition caused by maternal diabetes or overnutrition during pregnancy induces childhood obesity.[12–15] Foetal hypernutrition is associated with an increased glucose delivery and may lead to increased insulin release and greater adipogenesis.[12] In a study in rats, pups delivered to high-fat-fed dams during pregnancy showed higher body weight (g), fat mass (g) and fat (%) than those delivered to dams fed a control diet.[16]

Genetic and environmental factors may interact to affect the obesity of offspring, and the factors that make the greatest contribution to obesity have yet to be identified. A recent Framingham Heart Study[32] using third-generation data aimed to assess the variation in a BMI-related genetic risk score over time. They reported that the association between parental obesity and offspring BMI was stronger in the younger generations (second–third) than in the older generations (first–second), but the genetic risk score did not significantly change over time. They concluded that the obesity of younger generations may be more attributable to a shared environment within the family rather than to genetic variation. In our study, the physical activity, which represented a proxy for a shared environment, of adolescents with two obese parents was lower (49.0%) than that of others (53.8% and 53.6%, respectively), although this difference was not significant ($P = 0.22$). This suggests that sharing an environment with obese parents may contribute (albeit not strongly in this study) to being obese and that this contribution may be independent from energy-saving efficiency. However, distinguishing genetic from environmental effects on the association between parent obesity and offspring obesity is more complex because of epigenetics (that is, the effects of environmental factors on gene function), which should be considered in gene–environment interactions.[12]

The energy-saving efficiency transmitted from parents was no longer present in the adult offspring of obese parents. This suggests that the transmission of high energy-saving efficiency was completed before adulthood and that the ability to amass fat in the adult offspring of obese parents may be weakened and more similar to the offspring of non-obese parents. Adaptation to the obesogenic environment might contribute to the attenuated energy-saving efficiency of adult offspring of obese parents, and several long-term observational studies have supported this explanation.[33,34] Cooper *et al.*[33] reported that a higher maternal BMI weakened the positive association between offspring BMI and risk factors for cardiovascular disease (for example, total cholesterol and triglycerides) in middle age. Another study measuring biomarkers of middle-aged subjects showed that total cholesterol levels and the prevalence of diabetes in the non-obese offspring of obese parents were lower than those in the (non-obese) offspring of non-obese parents.[34]

The results reflected an interesting pattern regarding gender: the higher energy-saving efficiency in the 'both' group (when both parents were obese) was maintained in female adolescents. Although this was not statistically significant, no such lingering effect was observed in male adolescents. The discrepancy between males and females may be based on physiological differences in the development of adiposity. An animal study[35] reported that the increased adiposity of rat adolescent offspring of mothers fed a diet rich in fat, sugar and salt in pregnancy and lactation was more pronounced in females than in males.

To our knowledge, this is the first study to demonstrate the modifying effects of parental obesity on the association between energy intake and obesity indices in offspring and to ascertain whether this phenomenon changes over time. The data set used, which covered parent–offspring trios over 5 years derived from population-based representative data, was a major strength of this study.

On the other hand, the study design and analysis have several limitations. First, because the KNHANES was conducted on the basis of a cross-sectional design, it was not possible to consider the variability of individual physical changes over time. To reduce error introduced by this variability, individuals who had undergone any rapid physical changes were excluded. Second, our findings represent those from one nation, and therefore, they may be limited in their application to other populations. Additional longitudinal studies in other countries are required. Third, the dietary information in this study was derived from 24-h dietary recalls, which made determining the usual food intake over a long time period difficult. However, the sensitivity analysis, which relied on a subset of people who responded that the 24-h recall represented their usual amount of dietary intake (81.7%), revealed similar findings. The 24-h recall method is also likely to be another source of bias because it may reflect a specific season of the survey date. However, the KNHANES operates year round; thus, any seasonal effects are most likely offset by the use of averaged data. Fourth, we adjusted for physical activity as a covariate in only adolescent and adult offspring owing to the lack of data for children. Fifth, our main findings were inconsistent with the fat-intake results such that it was unclear whether the obesity status of the parents modified the energy-saving efficiency from fat intake. This is probably because carbohydrates are the main energy source in the Korean population. Indeed, in this study, carbohydrates accounted for 64% of the total energy intake, compared with 22% for fat. Furthermore, the correlation between carbohydrate and fat intake was significantly negative ($r = -0.82$, $P < 0.001$), indicating that higher carbohydrate intake (which forms a major portion of the energy intake of Koreans) is correlated with reduced fat intake. This correlation between carbohydrate and fat intake may explain the nonsignificant effect of energy-saving efficiency. Finally, we were not able to consider the interplay between genes and the foetal or early postnatal environment, because genetic information and data on the perinatal environment were unavailable.

In this study, parental obesity was associated with a higher energy-saving efficiency in children and, possibly, in female adolescents. This suggests that the energy-saving efficiency from obese parents was transmitted in a limited manner to children and, possibly, to adolescent females, despite the strong parental influence on obesity across all ages. Therefore, it is important to provide personal and social support to help children of both genders and adolescent females of obese parents maintain a balanced diet and engage in regular physical activity.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

This study was supported by the Ministry of Environment as an Environmental Health Center Program. We thank the KNHANES 2007–2011 participants and investigators as well as the Korea Centers for Disease Control and Prevention for making the public data available.

REFERENCES

1 Finucane MM, Stevens GA, Cowan MJ, Danaei G, Lin JK, Paciorek CJ *et al.* National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. *Lancet* 2011; **377**: 557–567.
2 Ezzati M, Riboli E. Behavioral and dietary risk factors for noncommunicable diseases. *N Engl J Med* 2013; **369**: 954–964.

© 2016 Macmillan Publishers Limited

3 Caballero B. The global epidemic of obesity: an overview. *Epidemiol Rev* 2007; **29**: 1–5.
4 Garver WS, Newman SB, Gonzales-Pacheco DM, Castillo JJ, Jelinek D, Heidenreich RA *et al*. The genetics of childhood obesity and interaction with dietary macronutrients. *Genes Nutr* 2013; **8**: 271–287.
5 de Onis M, Blössner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. *Am J Clin Nutr* 2010; **92**: 1257–1264.
6 Gillman MW, Ludwig DS. How early should obesity prevention start? *N Engl J Med* 2013; **369**: 2173–2175.
7 Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? *Science* 2003; **299**: 853–855.
8 Neel JV. Diabetes mellitus: a "thrifty" genotype rendered detrimental by "progress"? *Am J Hum Genet* 1962; **14**: 353–362.
9 Stöger R. The thrifty epigenotype: an acquired and heritable predisposition for obesity and diabetes? *Bioessays* 2008; **30**: 156–166.
10 Diamond JM. Human evolution. Diabetes running wild. *Nature* 1992; **357**: 362–363.
11 Hales CN, Barker DJP. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia* 1992; **35**: 595–601.
12 Gluckman PD, Hanson MA. Developmental and epigenetic pathways to obesity: an evolutionary-developmental perspective. *Int J Obes* 2008; **32**: S62–S71.
13 Silverman BL, Cho NH, Rizzo TA, Metzger BE. Long-term effects of the intrauterine environment: The Northwestern University Diabetes in Pregnancy Center. *Diabetes Care* 1998; **21**: B142–B149.
14 Abenhaim HA, Kinch RA, Morin L, Benjamin A, Usher R. Effect of prepregnancy body mass index categories on obstetrical and neonatal outcomes. *Arch Gynecol Obstet* 2007; **275**: 39–43.
15 Taylor PD, Poston L. Developmental programming of obesity in mammals. *Exp Physiol* 2007; **92**: 287–298.
16 Guo F, Jen KL. High-fat feeding during pregnancy and lactation affects offspring metabolism in rats. *Physiol Behav* 1995; **57**: 681–686.
17 Kweon S, Kim Y, Jang MJ, Kim Y, Kim K, Choi S *et al*. Data resource profile: the Korea National Health and Nutrition Examination Survey (KNHANES). *Int J Epidemiol* 2014; **43**: 69–77.
18 Korean Centers for Disease Control and Prevention, Ministry of Health & Welfare. An instruction for the fifth Korea National Health and Nutrition Examination Survey. 2011. Available from https://knhanes.cdc.go.kr/knhanes/index.do (accessed 30 October 2014).
19 Savva SC, Tornaritis M, Savva ME, Kourides Y, Panagi A, Silikiotou N *et al*. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *Int J Obes Relat Metab Disord* 2000; **24**: 1453–1458.
20 Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. *Obes Rev* 2012; **13**: 275–286.
21 Heo M, Wylie-Rosett J, Pietrobelli A, Kabat GC, Rohan TE, Faith MS. US pediatric population-level associations of DXA-measured percentage of body fat with four BMI metrics with cutoffs. *Int J Obes* 2014; **38**: 60–68.
22 Inokuchi M, Matsuo N, Takayama JI, Hasegawa T. BMI ***z***-score is the optimal measure of annual adiposity change in elementary school children. *Ann Hum Biol* 2011; **38**: 747–751.
23 Kim SH, Kim TH, Hwang HJ. The relationship of physical activity (PA) and walking with sarcopenia in Korean males aged 60 years and older using the fourth Korean National Health and Nutrition Examination Survey (KNHANES IV-2, 3), 2008–2009. *Arch Gerontol Geriat* 2013; **56**: 472–477.
24 Forrestal SG. Energy intake misreporting among children and adolescents: a literature review. *Matern Child Nutr* 2011; **7**: 112–127.
25 Kye S, Kwon SO, Lee SY, Lee J, Kim BH, Suh HJ *et al*. Under-reporting of energy intake from 24- hour dietary recalls in the Korean National Health and Nutrition Examination Survey. *Osong Public Health Res Perspect* 2014; **5**: 85–91.
26 Cole TJ, Faith MS, Peitrobelli A, Heo M. What is the best measure of adiposity change in growing children: BMI, BMI %, BMI ***z***-score or BMI centile? *Eur J Cli Nutr* 2005; **59**: 419–425.
27 Lawlor DA, Lichtenstein P, Långström N. Association of maternal diabetes mellitus in pregnancy with offspring adiposity into early adulthood: sibling study in a prospective cohort of 280,866 men from 248,293 families. *Circulation* 2011; **123**: 258–265.
28 Patel R, Martin RM, Kramer MS, Oken E, Bogdanovich N, Matush L *et al*. Familial associations of adiposity: findings from a cross-sectional study of 12,181 parental-offspring trios from Belarus. *PLoS One* 2011; **6**: e14607.
29 Power C, Pouliou T, Li L, Cooper R, Hyppönen E. Parental and offspring adiposity associations: insights from the 1958 British birth cohort. *Ann Hum Biol* 2011; **38**: 390–399.
30 Franks PW, Hanson RL, Knowler WC, Sievers ML, Bennett PH, Looker HC. Childhood obesity, other cardiovascular risk factors, and premature death. *N Engl J Med* 2010; **362**: 485–493.
31 Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int J Obes* 2011; **35**: 891–898.
32 Fox CS, Pencina MJ, Heard-Costa NL, Shrader P, Jaquish C, O'Donnell CJ *et al*. Trends in the association of parental history of obesity over 60 years. *Obesity* 2014; **22**: 919–924.
33 Cooper R, Pinto Pereira SM, Power C, Hyppönen E. Parental obesity and risk factors for cardiovascular disease among their offspring in mid-life: findings from the 1958 British Birth Cohort Study. *Int J Obes* 2013; **37**: 1590–1596.
34 Lieb W, Pencina MJ, Lanier KJ, Tofler GH, Levy D, Fox CS *et al*. Association of parental obesity with concentrations of select systemic biomarkers in nonobese offspring: the Framingham Heart Study. *Diabetes* 2009; **58**: 134–137.
35 Bayol SA, Simbi BH, Bertrand JA, Stickland NC. Offspring from mothers fed a 'junk food' diet in pregnancy and lactation exhibit exacerbated adiposity that is more pronounced in females. *J Physiol* 2008; **586**: 3219–3230.

Supplementary Information accompanies this paper on European Journal of Clinical Nutrition website (http://www.nature.com/ejcn)

© 2016 Macmillan Publishers Limited