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Associations Between Parental and Child Overweight and Obesity

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A B S T R A C T

Results of the analysis showed that parents and children overweight/obesity were significantly correlated. The sample includes 318 pairs of mothers and children, and 336 pairs of fathers and children at the age 11.3±0.4 years in Trogir, Croatia. Child overweight and obesity were defined according to body mass index (BMI) 25 and 30 equivalents (kg/m^2). The prevalence of total overweight in girls was 25.6% and among boys was 20.5%. Mother's weight (p=0.003) and BMI (p=0.006) were greater in obese than in other groups of children. Overweight/obese children were more often found among overweight/obese mothers (p=0.009) and fathers (p=0.039). Correlation between overweight/obese children and their father (odds ratio 3.2, 95% CI 1.5–6.8) was stronger than between overweight/obese children and their mothers (odds ratio 2.2, 95% CI 1.2–3.9). Associations with mothers' and daughters' overweight/obesity were stronger (p=0.017) than mothers' and sons' (p=0.12). Correlations between children's BMI and fathers' BMI (r=0.265, p<0.0001) and between children's BMI and mothers' BMI (r=0.173, p=0.002) were significant. Children whose parents are overweight/obese look for greater attention in future preventive programme.

Key words: *overweight, obesity, body mass index, adolescence, parent, child*

Introduction

Obesity in childhood is a public health problem, whose importance has progressively increased in the last few years[1,2,3]. The increase in childhood obesity will, unchecked, accentuate the rise in early adult type 2 diabetes and cardiovascular disease[4]. Impaired glucose tolerance and type 2 diabetes are far more common in obese European children than previously thought[5]. Insulin-resistant syndrome is developing in growing numbers of obese children[6]. In grossly obese children, both insulin resistance and impaired insulin secretion contribute to the elevation of glycemia, and the degree of obesity is related to cardiovascular risk factors independently of insulin resistance[7].

In Croatia there were 11.2% overweight boys and 9.8% girls, and 5.7% obese boys and 5.4% girls among the children aged from 7 to 15[8]. Furthermore, in Croatia there were 41% overweight and 18% obese males and females aged between 18 and 65 years[9]. Longitudinal studies of children followed into young adulthood suggest that overweight children may become overweight adults, particularly if obesity is present in adolescence[10]. Identification of risk factors early in life may help target prevention toward high-risk children and allow healthy lifestyle to be established before the onset of obesity[11]. For both sexes, having an obese parent greatly increased the odds for youth obesity[12]. Several reports showed that parents provide both the genes and eating environment for their children and familial patterns of adiposity are the result of gene-environment interactions[13,14].

We examined associations between parental and child overweight and obesity in Trogir, Croatia. Trogir is a town in the south of Croatia, on the Adriatic sea, 20 kilometers near to Split, with 28,000 inhabitants. The city of Trogir is on the list of cultured heritage of the world and is under protection of the UNESCO because of its exceptionally historic monuments. The main sources of income for Trogir's inhabitants are tourism, shipbuilding, floriculture, fruit and vegetable cultivation. The hypothesis of this study was that parents and children overweight / obesity were significantly correlated.

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Subjects and Methods

The associations between parental and child overweight and obesity was investigated in Trogir, Croatia during the spring 2003. The participants were school children (N=487), borne in 1991 or 1992 in Trogir, who participated in the obligatory health examination at the age of 11 years. Anthropometric measurements, body height (cm) and body weight (kg) were obtained[15] with digital anthropometrics device (Secca 220, Hamburg, Germany) during the morning. Biases of the device were ±0.01 kg and ±5 mm. Highly trained medical staff collected all anthropometric data. Interobserver reliability was checked on a regular basis. Out of 487 children there 481 (98.8%) were measured (254 (52.8%) boys and 227 (47.2%) girls). The heights and weights of the parents were self-reported when their children were 11 years old. The validity of self-reports for height and weight was established by Epstain et al.[16], who showed that the correlation between actual and self-report is quite high (r=0.96). Data on body weight and height were obtained from 336 (68.9%) fathers and 318 (65.3%) mothers. Body mass index (BMI) was calculated as weight / height^2 (kg/m^2) in children and their parents. The adult cut off points were for overweight 25 (kg/m^2), and 30 (kg/m^2) for obesity[17]. We showed the prevalence of total overweight (overweight or obesity). Total overweight defined as BMI cut offs corresponding to a BMI of 25 (kg/m^2) at that age[18]. Also, according to international cut off points for body mass index for overweight and obesity by sex and age which defines body mass index of 25 (kg/m^2) and 30 (kg/m^2), we compared our incidence of overweight and obesity[18].

Household socio-economic status when the child was 11 years old was represented by a mother's and father's educational attainment (finished school years) and number of children in the family. Data related education was obtained from 88.9% fathers 68.9% mothers, and number of children in family was obtained from all 487 (100%) subjects.

There was not significant difference between cohort of study subjects and total population of the region in investigated anthropometric and socioeconomic characteristic, and this homogeneity is due to little and closed social environment. Also, because boys and girls at the age 11 did not significantly differ according BMI, they were processed as an unique group during information processing, despite their gender.

Because of lack of anthropometric data, from study were excluded 6 (1.2%) children, 169 (34.7%) mothers and 151 (31.1%) fathers. Finally, the study sample includes 318 pairs of mothers and their children, and 336 pairs of fathers and their children.

Statistical method

The data were showed in tabular and graphic way. Statistical methods used were χ^2 test, Student's t-test independent by groups, ANOVA to analyze quantitative variables by more than two groups and linear regression test, Pearson's correlation coefficient significance.

A p value of less than 0.05 was considered to be statistically significant.

Results

In Trogir, Croatia, observed population of children and their parents was homogeneous according analyzed anthropometric characteristics. There was statistically significant differences between boys and girls only in body height (t=2.2, p=0.03), and we could not demonstrate difference in body weight (t=0.19, p=0.84) and BMI (t=0.29, p=0.77) (Table 1).

We found 110 (22.9%) overweight/obese children, and 371 (77.1 %) normal weight children at the age11.3±0.4 years. In our population, among 110 overweight/obese children, there were 58 (52.7%) girls and 52 (47.3%) boys. The prevalence of total overweight in girls was 25.6 % and among boys was 20.5%. In the studied population there were 219(65.6%) overweight/obese fathers and 95 (42.9%) overweight/obese mothers.

Mother's weight (F=6, p=0.003) and BMI (F=5.2, p=0.006) were significantly greater in obese group (75. 6 kg and 26.1 kg/m^2) than in overweight (68.9 kg and 24.1 kg/m^2) and normal (67.5 kg and 23.8 kg/m^2) groups of children (Table 2). Likewise, obese and overweight children had heavier fathers then it is the normal weight (91.1 *vs.* 90.5 *vs.* 86.2 kg) (F=3.7, p=0.026). Mother's and father's actual height was not significantly different in the different children's weight status groups. Normal weight, overweight and obese children's groups were not statistically significant different according to father's BMI.

Our results show that children's BMI at the age of 11 depend on mother's and father's BMI (Figure 1 and 2).

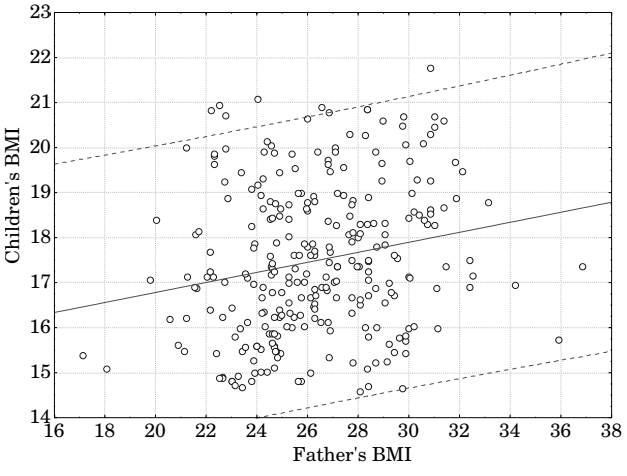

Fig. 1. Correlation between children's body mass index (BMI), (kg/m^2) and father's BMI (kg/m^2). Dependent variable – children's BMI, independent variable – father's BMI, y=12.4+0.22x, F=8.7, p=0.003, standard error of estimate = 1.6.

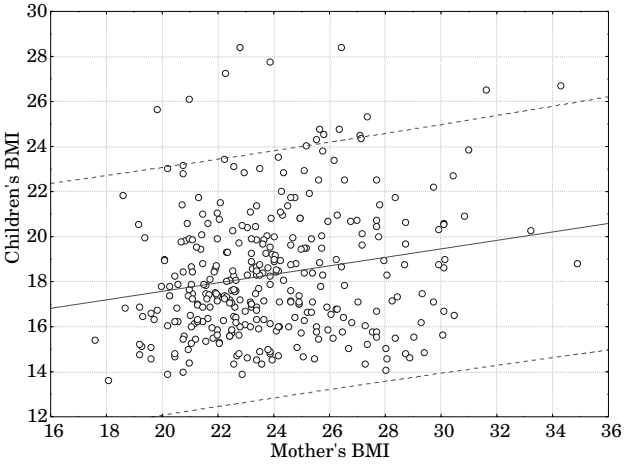

Fig. 2. The correlation between children's body mass index (BMI), (kg/m^2) and mother's BMI(kg/m^2). Dependent variable – children's BMI; Independent variable – mother's BMI, y=13.8+0.19x, F=13.2, p<0.0001, standard error of estimate = 2.8.

Correlations between children's BMI and father's BMI were statistically significant (r=0.265, p<0.0001). Also, correlations between mother's BMI and children's BMI were statistically significant (r=0.173, p=0.002). In Trogir, Croatia, correlation between overweight/obese children and their fathers (odds ratio 3.2, 95%, CI 1.5–6.8) was stronger than between overweight/obese children and their mothers (odds ratio 2.2, 95%, CI 1.2–3.9).

Overweight/obese children clustered in the group of overweight/obese mothers (χ^2=6.4, p=0.0018) and overweight/obese fathers (χ^2=9.1, p=0.003) (Table 3). In group of normal weight children there were 38.2% fathers and 72.9% mothers with BMI less then 25 kg/m^2, whereas there were 61.8% fathers and 27.1% mothers with BMI equal and greater then 25 kg/m^2. In the group of overweight/obese children there were even 83.9% overweight/obese fathers and only 16.1% fathers with BMI less then 25kg/m^2. There were 44.4% overweight/obese and 55.6% mothers with BMI less then 25 kg/m^2 in the group of overweight/obese children.

TABLE 1
ANTHROPOMETRICAL CHARACTERISTICS MEASURED AMONG CHILDREN AT THE AGE 11 AND THEIR PARENTS IN TROGIR CROATIA

	N	X±SD	Min–Max	95% CI
Girls' weight (kg)	227	42.7±9.4	27.5–81	41.7–43.8
Girls' height(cm)	227	151±7.2	131–173.5	150.7–152.4
Girls' BMI (kg/m^2)	227	18.5±3.2	13.6–34.8	18.1–18.6
Boys' weight (kg)	254	42.6±9.3	27–81	41.4–43.7
Boys' height(cm)	254	150.2±6.6	133–172	149.4–151.1
Boys' BMI (kg/m^2)	254	18.6±2.9	12.9–30.2	18.2–18.9
Children's weight (kg)	481	42.6±9.5	27–81	41.8–43.4
Children's height (cm)	481	151±6.9	131–173.5	150.4–151.5
Children's BMI (kg/m^2)	481	18.5±3.1	12.9–34.8	18.3–18.8
Mothers' weight (kg)	318	67.9±9.7	45–107	66.9–68.9
Mothers' height (cm)	318	168.5±5.7	152–185	167.9–169.1
Mothers' BMI (kg/m^2)	318	23.9±3.1	17.6–42.8	23.6–24.2
Fathers' weight (kg)	336	86.5±11.3	45–130	85.4–87.7
Fathers' height (cm)	336	180.6±6.9	162–203	179.8–181.4
Fathers' BMI (kg/m^2)	336	26.5±2.9	17.1–36.8	26.2–26.8

BMI – body mass index, Min – minimum, Max – maximum, 95% CI – 95% confidence interval

TABLE 2
CHILDREN'S WEIGHT STATUS AT THE AGE OF 11 AND ANTHROPOLOGICAL CHARACTERISTICS OF THEIR PARENTS IN THE TROGIR, CROATIA

	Normal weight*		Overweight*		Obesity*		p
	N	X±SD	N	X±SD	N	X±SD	
Mothers' weight (kg)	264	67.5 ± 9.1	38	68.9 ± 9.2	17	75.58 ± 14.0	0.003
Mothers' height (cm)	264	168.5 ± 5.6	37	168.8 ± 5.1	17	169.8 ± 6.5	0.65
Mothers' BMI (kg/m^2)	264	23.8 ± 2.8	37	24.1 ± 3.0	17	26.1 ± 3.7	0.006
Fathers' weight (kg)	280	86.2 ± 11.2	40	90.5 ± 11.6	17	91.1 ± 13.6	0.026
Fathers' height (cm)	280	180.6 ± 8.5	40	181.7 ± 5.8	16	180.6 ± 6.9	0.75
Fathers' BMI (kg/m^2)	280	26.6 ± 4.7	40	27.4 ± 2.9	16	28.0 ± 3.2	0.28

*normal weight, overweight and obesity according to Cole's BMI-25 and BMI-30-equivalents[18], BMI – body mass index

TABLE 3
ASSOCIATIONS BETWEEN PARENTAL AND CHILD OVERWEIGHT/OBESITY

Parents' BMI (kg/m²)		Childrens' BMI (kg/m²)		χ^2, p	Odds ratio (95% CI)
		<25 normal weight* N (%)	≥25 overweight/obesity* N (%)		
Fathers	<25 normal weight*	106 (92.2)	9 (7.8)	9.1, 0.003	3.2 (1.5–6.8)
	≥25 overweight/obesity*	172 (78.5)	47 (21.5)		
Mothers	<25 normal weight*	191 (86.4)	30 (13.6)	6.4, 0.018	2.2 (1.2–3.9)
	≥25 overweight/obesity*	71 (74.7)	24 (25.3)		

*normal weight, overweight /obesity according to Cole's BMI-25 and BMI-30-equivalents[18], BMI – body mass index, 95% CI – 95% confidence interval

TABLE 4
ASSOCIATIONS BETWEEN PARENTS'AND THEIR CHILDRENS' OVERWEIGHT/OBESITY

Parents' BMI (kg/m²)		Childrens' BMI (kg/m²)			
		<25 normal weight*		≥25 overweight/obesity*	
		Daughter	Son	Daughter	Son
Fathers	<25 normal weight*	67	39	4	5
	≥25 overweight/obesity*	102	70	25	22
Mothers	<25 normal weight*	113	78	15	15
	≥25 overweight/obesity*	56	15	14	10

*normal weight, overweight /obesity according to Cole's BMI-25 and BMI-30-equivalents[18], BMI – body mass index

TABLE 5
ASSOCIATIONS BETWEEN CHILDREN'S OVERWEIGHT/OBESITY AND MOTHER' AND FATHER'S EDUCATIONAL ATTAINMENT

Parents' education (years)		Childrens' BMI (kg/m²)		p
		<25 normal weight* N (%)	≥25 overweight/ obesity* N (%)	
Fathers	<8	42 (79.2)	11 (20.8)	
	8–12	247 (76)	78 (24)	0.82
	>12	51 (78.5)	14 (21.5)	
Mothers	<8	26 (72.2)	10 (27.8)	
	8–12	212 (76.8)	64 (23.2)	0.73
	>12	59 (78.7)	16 (21.3)	

*normal weight, overweight /obesity according to Cole's BMI-25 and BMI-30-equivalents[18], BMI – body mass index

Despite the fact that groups of overweight/obese children made according to gender were small, it could be observed that overweight/obese mothers rather had overweight/obese daughter's (χ^2=12.3, p=0.017) than overweight/obese sons (χ^2=13.2, p=0.12) (Table 4). Also, daughters' overweight/obesity had significant relationship with fathers' BMI ≥ 25 kg/m² (χ^2=7.8, p=0.02), but there was a weak relationship between fathers' and sons' overweight/obesity (χ^2=3.15, p=0.21).

In the observed population there were 108 (22.7%) children from one child family, 251 (51.5%) from two children family, 95 (19.5) from three children family and 33 (6.8%) from more than three children families. In Trogir, Croatia, overweight/obese children more often originated from families with less children in relation with counterparts with normal weight (1.98 *vs.* 2.2) (p=0.02).

In study population there were 9.9% fathers and 9.3% mothers with less then 8 years of finished school, 75.1% fathers and 71.3% mothers with 8–12 years of finished school and 15% fathers and 19.4% mothers with greater then 12 finished school years. Mothers' (χ^2=0.1, p>0.99) and fathers' (χ^2=0.7, p=0.95) educational attainment did not have influence on overweight/obesity of their children (Table 5).

Discussion

This study explored the associations between parental and child overweight/obesity. In our population, there were significant association between parental and their child overweight/obesity. The correlation between the degree of obesity in the parents and the weight of the child both in childhood and in adult life indicates that genetic factors influence the weight level throughout life[19]. As reported elsewhere, having parents, especially mothers, who were overweight or obese, may increase the risk of children being overweight or obese[20,21]. This study confirms more significant correlation between mothers' and daughters' overweight and obesity than between mothers' and their sons' overweight and obesity. The consistent obesity relationship between mother and offspring may indicate the key role of the mother in

primary obesity prevention[22]. Safer et al. explained that familial factors (biological and/or environmental) are affecting the development of childhood obesity[23]. The parent-child relationship of inactivity appeared to be stronger than that of vigorous activity, and parents who want to reduce their children's inactivity may have to pay attention to their own lifestyle[24]. Moussa et al. showed that family history of obesity, diet, physical activity and mother education are significant factors for development of obesity after adjusting for other confounding covariates[25]. As others have found, there is a clear relationship between mothers' and their children's overweight and obesity, but rarely with fathers' and their children's. But, in Trogir, Croatia, association between fathers' and their children overweight /obesity were stronger than in mothers and their children. Furthermore, we postulated the father's weight as possible link with heavier daughters rather than sons. These findings could obviously not have been exclusively explained with inheritance, but might be clarified with the patriarchal structure of Croatian family where the father, »pater familas«, is the central person with very high influence in forming child feeding practices and life style.

Results of our study confirmed familial overweight and obesity aggregation. Results from Davison et al. highlight the centrality of the family in the etiology of childhood overweight and the necessity of incorporating parents in the treatment of childhood overweight[26]. Children with at least one obese parent seemed to have higher BMI during childhood[27]. Lake et al. showed that subjects with two obese parents are fatter in childhood and also show a stronger pattern of tracking from childhood to adulthood [28]. Tracking of BMI was established from 6 onwards to 20 years was stronger for those subjects with both parents overweight compared with those with only one or neither parent overweight[29].

Socioeconomic status, as measured by household income, parents' educational level, occupation of family head, or other variables were positively correlated with children's weight[30]. Among children, age, race, income, and mother's BMI were significantly correlated with child BMI[31]. Furthermore, the most important factors influencing children's BMI values were being an only child and residence in small communities[32]. Overweight families of low SES have the highest risk of overweight and obese children[33]. Social and economical circumstances, in which these observed children groups were growing, were equalized to their mothers' and fathers' finished school years. Our study showed that the overweight/obesity occurred more often among children from families with less number than among children from families with more children, like study by Rasmussen et al.[34].

Overweight and obesity are increasing health problems in Croatia as well as in the western and southern Europe[2]. However, useful information for the prediction and prevention of adult overweight can be obtained from the BMI at age 12 years[35]. Long-term follow-up studies of adolescents suggest that approximately 30% of all obese adult women were obese early in adolescence, whereas only 10% of obese adult males had onset of their obesity as teenagers[36]. Since this study was performed in Trogir, a town in southern Croatia, where »Mediterranean« way of diet was dominant, the obtained frequency of overweight /obesity among children and their parents was unexpectedly high. According to the International Obesity Task Force (IOTF) standard which includes overweight and obesity, we found 25.6% boys and 20.5% total overweight girls. The prevalence of total overweight in 10-year-old boys ranged from 4.5% in the Netherlands to 29.6% in Italy[14]. Higher rates of total overweight were observed in 10-year-old girls from 6.7% in the Netherlands to 31.4% in Italy[14]. Celi et al. showed that a large prevalence of overweight and obesity was observed in school subjects from three provinces of central Italy, and they suggested that from the comparisons of the prevalence rate, the new internationally agreed criteria seem more appropriate for epidemiological studies in their population[37]. In our population of 11-years old, the mean BMI of overweight children was 21.6±1.0 kg/m^2, and in obesity group was 26.3±2.49 kg/m^2. Our previous study showed that female adolescents with higher BMI demonstrated body mass dissatisfaction, meal skipping and dieting behavior[38]. Whereas, overweight / obesity is almost equal »problem« for boys as well as for girls in the observed population, therefore, preventative activities should include both sexes.

Our results may assist to identify those children who are more prone to becoming overweight and obese. Strategies for prevention of overweight and targeted interventions for prevention of the progression of overweight to obesity are urgently required in school-aged children in order to stem the epidemic of overweight in the adult population[39]. Parental overweight or obesity may identify children at risk for a range of unhealthy behaviors, and promotion of a healthy lifestyle should be primarily targeted to overweight families[40]. Health professionals may be more effective in preventing childhood obesity by focusing on these goals that they share with mothers, because she has a central role in shaping early diet and activity patterns for her child[41].

In conclusion, according to our study, the association between parental and child overweight/obesity was significant. Furthermore, fathers' weight status and children overweight / obesity was stronger than with mothers' and their children. Family-based behavioral weight control treatment involves the parent in the modification of child and parent eating and activity change[42]. In our population the development of family based preventive programs for early adolescence overweight and obesity will be necessary. Furthermore, children whose parents are overweight or obese look for greater attention in this program.

REFERENCES

1. CHINN, S., R. J. RONA, B. M. J., 322 (2001) 24. — 2. LOBSTEIN, T., M. L. FRELUT, Obes. Rev., 4 (2003) 195. — 3. HANLEY, A. J. G., S. B. HARRIS, J. GITTELSOHN, T. M. S. WOLEVER, B. SAKSVIG, B. ZINMAN, Am. J. Clin. Nutr., 71 (2000) 693.— 4. JAMES, P. T., N. RIGBY, R. LEACH, Eur. J. Cardiovasc. Prev. Rehabil., 11 (2004) 3 — 5. WIEGAND, S., U. MAIKOWSKI, O. BLANKESTEIN, H. BIEBERMANN, P. TARNOW, A. GRUTERS, Eur. J. Endocrinol., 151 (2004) 199 — 6. YENSEL, C. S., D. PREUD'HOMME, D. M. CURRY, J. Pediatr. Nurs., 19 (2004) 238 — 7. INVITTI, C., G. GUZZALONI, L. GILARDINI, F. MORABITO, G. VIBERTI, Diabetes. Care., 26 (2003) 118 — 8. ANTONIĆ- DEGAČ, K., A. KAIĆ-RAK, E. MESAROŠ-KANJSKI, Z. PETROVIĆ, K. CAPAK, Paediatr. Croat., 48 (2004) 9. — 9. ČUBRILO-TUREK, M., Epidemiološki podaci debljine u Hrvatskoj. In: Proceedings. (Prvi Hrvatski kongres o debljini, Rabac, 2003). — 10. SERDULA, M. K., D. IVERY, R. J. COATES, D. S. FREEDMAN, D. F. WILLIAMSON, T. BYERS, Prev. Med., 22 (1993) 167. — 11. STETTLER, N., A. M. TERSHAKOVEC, B. S. ZEMEL, M. B. LEONARD, R. BOSTON, S. H. KATZ, V. A. STALLINGS, Am. J. Clin. Nutr., 72 (2000) 378. — 12. CARRIERE, G., Health. Rep., 14 (2003) 29. — 13. CUTTING, T. M., J. O. FISHER, K. GRIMM-THOMAS, L. L. BIRCH, Am. J. Clin. Nutr., 69 (1999) 608. — 14. BIRCH, L. L., Childhood Overweight: Family Environmental Factors. In: DIETZ, W., H., C. CHEN (Eds.): Obesity in childhood and adolescence (Nestlé Nutrition Workshop Series Pediatric Program, Vevey, Switzerland, 2001) 28. — 15. MORENO, L. A., M. JOYANES, M. I. MESANA, M. GONZALEZ-GROSS, C. M. GILL, A. SARRIA, Nutrition, 19 (2003) 481. — 16. EPSTEIN, L. H., J. MCCURLEY, R. C. MURDOCK, Addictive Behaviours, 16 (1991) 369. — 17. GARROW, J. S., J. WEBSTER, Int. J. Obes., 9 (1985) 147. — 18. COLE, T. J., M. C. BELLIZZI, K. M. FLEGAL, W. H. DIETZ, B. M. J., 320 (2000) 1240. — 19. MOSSBERG, H. O., Lancet., 2 (1989) 491.— 20. WANG, Z., C. M. PATTERSON, A. P. HILLS, Asia. Pac. J. Clin. Nutr., 11 (2002) 200. — 21. DANIELZIK, S., K. LANGNASE, M. MAST, C. SPETHMANN, M. J. MULLER, Eur. J. Nutr., 41 (2002) 132. — 22. FUENTES, R. M., I. L. NOTKOLA, S. SHEMEIKKA, J. TUOMILEHTO, A. NISSINEN, Horm. Metab. Res., 34 (2002) 406. — 23. SAFER, D. L., W. S. AGRAS, S. BRYSON, L. D. HAMMER, Int. J. Obes. Relat. Metab. Disord., 25 (2001) 1532. — 24. FOGELHOLM, M., O. NUUTINEN, M. PASANEN, E. MYOHYNEN, T. SAATELA, Int. J. Obes. Relat. Metab. Disord., 23 (1999) 1262. — 25. MOUSSA, M. A., M. B. SKAIK, S. B. SELWANES, O. Y. YAGHY, S. A. BIN-OTHMAN, Int. J. Obes. Relat. Metab. Disord., 18 (1994) 513. — 26. DAVISON, K. K., L. L. BIRCH, Int. J. Obes. Relat. Metab. Disord., 25 (2001) 1834. — 27. FUENTES, R. M., I. L. NOTKOLA, S. SHEMEIKKA, J. TUOMILEHTO, A. NISSINEN, Int. J. Obes. Relat. Metab. Disord., 27 (2003) 716. — 28. LAKE, K. L., C. POWER, T. J. COLEB, Arch. Dis. Child., 77 (1997) 376. — 29. MAGAREY, A. M., L. A. DANIELS, T. J. BOULTON, R. A. COCKINGTON, Int. J. Obes. Relat. Metab. Disord., 27 (2003) 505. — 30. LIEBERMAN, L. S., C. K. PROBART, Coll. Antropol., 16 (1992) 279. — 31. LIN, B.,H., C. L. HUANG, S. A. FRENCH, Int. J. Obes. Relat. Metab. Disord., 28 (2004) 536. — 32. VIGNEROVA, J., P. BLAHA, K. OSANCOVA, Z. ROTH, Econ. Hum. Biol., 2 (2004) 107. — 33. DANIELZIK, S., M. CZERWINSKI-MAST, K. LANGNASE, B. DILBA, M. J. MULLER, Int. J. Obes. Relat. Metab. Disord., 24 (2004) — 34. RASMUSSEN, F., M. JOHANSSON, Europ. J. Epidemiol., 14 (1998) 373. — 35. TRUDEAU, F., R. J. SHEPHARD, S. BOUCHARD, L. LAURENCELLE, Am. J. Human. Biol., 15 (2003) 187. — 36. BRADDON, F. E. M., B. RODGERS, M. E. J. WADSWORTH, J. M. C. DAVIES, B. M. J., 293 (1986) 299. — 37. CELI, F., V. BINI, G. DE GIORGI, D. MOLINARI, F. FARAONI, G. DI STEFANO, M. L. BACOSI, M. G. BERIOLI, G. CONTESSA, A. FALORNI, Eur. J. Clin. Nutr., 57 (2003) 1045. — 38. BRALIĆ, I., V. KOVAČIĆ, Public. Health., 119 (2005) 189. — 39. MAGAREY, A. M., L. A. DANIELS, T. J. BOULTON, R. A. COCKINGTON, Int. J. Obes. Relat. Metab. Disord., 27 (2003) 505. — 40. BURKE, V., L. J. BEILIN, D. DUNBAR, Int. J. Obes. Relat. Metab. Disord., 25 (2001) 147. — 41. JAIN, A., S. N. SHERMAN, D. L. CHAMBERLIN, Y. CARTER, S. W. POWERS, R. C. WHITAKER, Pediatrics, 107 (2001) 1138. — 42. WROTNIAK, B. H., L. H. EPSTEIN, R. A. PALUCH, J. N. ROEMMICH, Arch. Pediatr. Adolesc. Med., 158 (2004) 342.

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RIZIK ZA RAZVOJ PRETILOSTI TE PRETILOST KOD RODITELJA I DJECE

SAŽETAK

Rezultati analize pokazali su značajnu povezanost između rizika za razvoj pretilosti/pretilosti (OW/OB) kod roditelja i djece. Ispitanici su 318 parova majki i djece, te 336 parova očeva i djece dobi 11.3±0.4 godine iz Trogira, Hrvatska. OW/OB djece je definiran ekvivalentom indeksa tjelesne mase (ITM) 25 odnosno 30(kg/m^2). Prevalencija OW/OB kod djevojčica je 25.6%, a kod dječaka 20.5%. Tjelesna masa (p=0.003) i ITM majke(p=0.006) je veći u skupini pretile djece. Majke (p=0.009) i očevi (p=0.039) koji su u skupini OW/OB češće imaju OW/OB djecu. Korelacija između OW/OB djece i OW/OB očeva(odds ratio 3.2, 95% CI 1.5–6.8) je izrazitija nego između OW/OB djece i OW/OB majki (odds ratio 2.2, 95% CI 1.2–3.9). Povezanost OW/OB majki i OW/OB kćeri (p=0.017) je izrazitija nego između OW/OB majki i OW/OB sinova(p=0.12). Značajna je korelacija ITM djece i ITM očeva (r=0.265, p<0.0001), kao i ITM majke (r=0.173, p=0.002). Djeca roditelja koji su OW/OB trebaju pojačani nadzor u budućim programima za prevenciju OW/OB.